OFP

Osteopathic Family Physician

THE OFFICIAL PEER-REVIEWED PUBLICATION OF THE AMERICAN COLLEGE OF OSTEOPATHIC FAMILY PHYSICIANS

SEPTEMBER / OCTOBER, 2018

Volume 10 | Number 5

ofpjournal.com

acofp | AMERICAN COLLEGE OF OSTEOPATHIC FAMILY PHYSICIANS

www.acofp.org

Guide for...

READERS

Osteopathic Family Physician (ISSN 1877-573X) is published bimonthly by the American College of Osteopathic Family Physicians. Periodicals postage paid at Arlington Heights, IL and additional mailing offices.

USA POSTMASTER

Send address changes to:

American College of Osteopathic Family Physicians
Membership Department:

330 East Algonquin Rd, Suite 1
Arlington Heights, IL, 60005
membership@acofpca.org

CUSTOMER SERVICE

(orders, claims, online, change of address)

American College of Osteopathic Family Physicians

330 East Algonquin Rd, Suite 1
Arlington Heights, IL 60005

800-323-0794 | membership@acofp.org

YEARLY SUBSCRIPTION RATES

United States & Possessions:
Individual $116 | Institution $208 | Student $57

All other countries: *(prices include airspeed delivery)*

Individual $146 | Institution $26 | Student $74
Single issues $42

To receive student/resident rate, orders must be accompanied by name of affiliated institution, date of orders must be accompanied by name of affiliated institution, date of term and the signature of program/residency coordinator on institution letterhead. Orders will be billed at the individual rate until proof of status is received. Current prices are in effect for back volumes and back issues.

ADVERTISING INFORMATION:

Advertising orders and inquiries can be sent to:

Matt Van Wie
804-550-2312 | matt@esvw.com

AUTHOR INQUIRIES

For inquiries relating to the submission of articles (including electronic submission) please visit www.ofpjournal.com.

Content details for questions arising after acceptance of an article, especially those relating to proofs will be provided by the publisher.

You can track accepted articles and view Author Guidelines through Scholar One at mc04.manuscriptcentral.com/ofp.

The paper used in this publication meets the requirements of ANSI/NISO Z39.48-1992 (Permanence of Paper).

AUTHORS

For a full and complete Guide for Authors, please go to:
mc04.manuscriptcentral.com/ofp.

REPRINTS:

For queries about author reprints, or to order 100 or more reprints for education, commercial or promotional use, contact ACOFP at 800.323.0794 or email ashleyd@acofp.org.

EXAM SCHEDULE
CERTIFICATION & OCC (RECERTIFICATION)

AOBFP EST. 1972

EXAMS	LOCATIONS	POSTMARK DATE
Family Medicine / OMT Certification / OCC Performance Evaluation Only	**ACOFP Annual Convention** **Chicago, IL** March 21 - 24, 2019 exam dates TBD	**October 1, 2018** *Late fee through December 1*
Family Medicine / OMT Certification / OCC Cognitive Exam	**Electronic Testing** Regional Sites May 4, 2019	**October 1, 2018** *Late fee through December 1*
Family Medicine / OMT Certification / OCC Cognitive Exam	**Electronic Testing** Regional Sites September 28, 2019	**April 1, 2019** *Late fee through June 1, 2019*
Family Medicine / OMT Certification / OCC Performance Evaluation Only	**AOA OMED Conference** Fall, 2019 exam dates TBD	**April 1, 2019** *Late fee through June 1, 2019*

AMERICAN OSTEOPATHIC BOARD OF FAMILY PHYSICIANS
If you have questions, please call 847.640.8477 or email aobfp@aobfp.org.

OFP
Osteopathic Family Physician
The Official Peer-Reviewed Publication of the
American College of Osteopathic Family Physicians

SEPT/OCT, 2018
VOLUME 10 | NUMBER 5

CONTENTS

OSTEOPATHIC FAMILY PHYSICIAN SPECIALTY PEER REVIEWERS

Nazem Abdelfatta, MD
Family Medicine, Obstetrics, Women's Health

Richard L. Averitte, Jr, MD
Dermatology

Dana Baigrie, DO
Clinical Images

Jeffrey Benseler, DO
Radiology

Shagun Bindlish, MD
Diabetes and Endocrinology

John Bissett, DO
Clinical Images

Warren Bodine, DO
Sports Medicine & Family Medicine

Grace Brannan, PhD
Statistics/Design

Natasha Bray, DO
Ethics

Omar Bukhari, MD
Research

Janis Coffin, DO
Practice Management

Philip Collins, DO
Patient Education

Danielle Cooley, DO
OMM

Rob Danoff, DO
Emergency Medicine, Preventive

Robin Devine, DO
Statistics/Design

Brian Downs, DO
HIV, Wound Care

Dennis Eckles, DO
Diabetes, Rural Medicine

Gail Feinberg, DO, FACOFP
Academic

Matthew Goldfinger
Neurology, OMM

Patricia Happel, DO
Nutrition and Obesity

Nadia Hasan, DO
Clinical Images

Robert Hunter, DO
Health Policy, Hospice/Palliative Care, ER, Diabetes, Wound Care

Ronald P. Januchowski, DO
Military & Rural/Underserved

Holly Kanavy, DO
Dermatology

Amy Keenum, DO, PharmD
Healthy Literacy, International & Patient Education

Uzma Khan, DO
Family Medicine

Frank Komara, DO
Geriatrics

Paul Lazar, DO
Adult Family Medicine, Geriatrics, Academic

Mana Lazzaroto, DO
Clinical Images

Ehab Mady, DO
Vascular

Mohammad Mansour, MD
Inpatient Medicine, Cardiology, Pulmonary, Geriatrics, Obstetrics

Sarah Mitchell, DO
Family Medicine

Merideth Norris, DO, FACOFP
Addiction

Michael O'Connell, DO
Pain, Rehabilitation, Musculoskeletal, Neurology, & Sports Medicine

Jon Parham, DO
Preventive Medicine, Pulmonary, Public Health, Geriatrics, Medical Errors

Raena Pettitt, DO
Disease Prevention & Wellness

Kim Pfotenhauer, DO
Diabetes

Prabhat Pokhrel, MD, MS, PhD, FAAFP
Pharmacology, Cardiology, Nephrology, Pulmonology

M. Jay Porcelli, DO, FACOFP *dist*.
Pain Management

Joseph Reyes, DO
Pain Management

Bernadette Riley, DO
Medical Education, Academic, Simulation Medicine, Physician Leadership, Health Policy

Mark Rogers, DO, MA, CAQSM, FAAFP
Family Medicine, Sports Medicine, OMM, Medical Ethics

Lawrence Sawicki, DO
Clinical Images

Kary Schroyer, DO
Direct Primary Care

Christopher Scuderi, DO
Family Practice, Practice Management

Jay Shubrook, Jr., DO, FACOFP
Endocrinology

Leslie Sleuwen, MD
Community Medicine

Frederick Stine, DO
Pediatrics, Nephrology, Emergency & Critical Care

Lindsay Tjiattas-Saleski, DO
Clincial Images, Emergency Medicine

Johnathon Torres, DO
OMM

Chad Uptigrove, DO
Obstetrics, Residency Training

Michael Watkins, DO
OB/GYN & Women's Health

Stuart Williams, DO
OMM

Barbara Wolf, DO
Psychology

William Woolery, DO, PhD, FACOFP
Geriatrics

Julian Vega, DO
Clinical Images

Sheldon Yao, DO
Cardiology

Peter Zajac, DO, FACOFP
Patient Education

2018 STUDENT ACADEMIC & PEER REVIEW WRITING INTERNS

Sanjhavi Agarwal
Western University of Health Sciences College of Osteopathic Medicine of the Pacific

Vaidehi Ambai
Philadelphia College of Osteopathic Medicine

Benjamin Berthet
A.T. Still University - School of Osteopathic Medicine in Arizona

Ethan Charles Blocher-Smith
Marian University College of Osteopathic Medicine

Lange Clancy
A.T. Still University - School of Osteopathic Medicine in Arizona

Ashton Dixon
University of Pikeville – Kentucky College of Osteopathic Medicine

Alice Doong, DO
Michigan State University College of Osteopathic Medicine

Kelcey Dunaway
A.T. Still University - School of Osteopathic Medicine in Arizona

Thomas Espisito
A.T. Still University - School of Osteopathic Medicine in Arizona

Joanna Ghobrial
A.T. Still University - School of Osteopathic Medicine in Arizona

Jessica Gonzalez Haugen
Western University of Health Sciences College of Osteopathic Medicine of the Pacific

Harmandeep Grewal
A.T. Still University - School of Osteopathic Medicine in Arizona

Ryne Jenkins
Western University of Health Sciences College of Osteopathic Medicine of the Pacific

Liana Kobayashi
A.T. Still University - School of Osteopathic Medicine in Arizona

Chris Koerber
A.T. Still University - School of Osteopathic Medicine in Arizona

Anna Marzvanyan
A.T. Still University - School of Osteopathic Medicine in Arizona

Amelia Ni
A.T. Still University - School of Osteopathic Medicine in Arizona

Wen Ze Niu, PhD
Western University of Health Sciences College of Osteopathic Medicine of the Pacific

Jay Patel
A.T. Still University - School of Osteopathic Medicine in Arizona

Matthew Petersen
Western University of Health Sciences College of Osteopathic Medicine of the Pacific

Elenie Philippas
Western University of Health Sciences College of Osteopathic Medicine of the Pacific

Meghan Piccinin
Michigan State University College of Osteopathic Medicine

Aisha Shamsi
Western University of Health Sciences College of Osteopathic Medicine of the Pacific

Sarah Shu
Western University of Health Sciences College of Osteopathic Medicine of the Pacific

Jonathan Swan
A.T. Still University - School of Osteopathic Medicine in Arizona

Elysia Tjong
A.T. Still University - School of Osteopathic Medicine in Arizona

Fangfei Xiao
Western University of Health Sciences College of Osteopathic Medicine of the Pacific

Jessica Yi
A.T. Still University - School of Osteopathic Medicine in Arizona

OFP
Osteopathic Family Physician
JOURNAL

2018 CALL FOR PAPERS

Osteopathic Family Physician is the ACOFP's official peer-reviewed journal
The bi-monthly publication features original research, clinical images and articles about preventive medicine, managed care, osteopathic principles and practices, pain management, public health, medical education and practice management.

RESERVE A TOPIC

Reserve a review article topic today by emailing ACOFP Managing Editor, Belinda Bombei at belindab@acofp.org. Please provide your name and the review title you would like to reserve. Once you reserve a review article topic, you will receive an email confirmation from ACOFP. This will initiate a three-month deadline for submission. If the paper is not received within three months, the system will release the review article topic for other authors to reserve. Articles submitted for publication must be original in nature and may not be published in any other periodical. Materials for publication should be of clinical or didactic interest to osteopathic family physicians. Any reference to statistics and/or studies must be footnoted. Material by another author must be in quotations and receive appropriate attribution. ACOFP reserves the right to edit all submissions. Visit ofpjournal.com to view author guidelines, policies, and manuscript checklist.

CLINICAL IMAGES

We are seeking clinical images from the wards that covers essential concepts or subject matter to the primary care physician.
Please provide a brief synopsis of how the case presented along with 1-4 questions and approximately 1 page of education with reference to the image and questions.

REVIEW ARTICLE TOPICS

- ADHD Management in Primary Care with Osteopathic Component
- Detecting, Managing & Treating Patients with Personality Pathology in Primary Care Settings
- Disorders of Puberty: An Approach to Diagnosis & Management
- Lupus: Review Article with Osteopathic Component
- OMT Treatments for Pediatric Conditions: Systematic Review

RESEARCH TOPICS

We are seeking original clinical or applied research papers. Original contributions include controlled trials, observational studies, diagnostic test studies, cost-effectiveness studies, and survey-based studies. The OFP will accept basic scientific research only if the work has clear clinical applications. For randomized controlled trials, study flow diagrams must be submitted. For all other types of original contributions, flow diagrams are encouraged. Original contributions should be 3000 words with no more than 50 references and 5 tables or figures. OFP requires you to submit a 250-word abstract, along with four to six keywords.

The content should include the following:

Abstract
Introduction
Methods
Results
Discussion
Conclusions
Acknowledgments

Ronald Januchowski, DO, FACOFP
Editor-in-Chief

Paula Gregroy, DO, MBA, CHCQM, FAIHQ
Associate Editor

acofp AMERICAN COLLEGE OF OSTEOPATHIC FAMILY PHYSICIANS

EDITOR'S MESSAGE

What Do Offensive Lineman, Police Officers, Pregnant Women & Soldiers Have in Common?

Ronald Januchowski, DO, FACOFP, Editor, *Osteopathic Family Physician*

Welcome to autumn!

As the cooler season comes upon us, this issue of OFP has some timely articles of interest. With the changing of seasons comes a return to the gridiron, and for those physicians participating, as sideline physicians for football, the article on concussions will be of value. Closed head injuries are in the medical literature as well as in the lay press and this article provides a good summary of this topic. It will be a good read as you ponder over the tackling rules changes while watching on Sundays. What do offensive linemen, police officers, pregnant women, and soldiers have in common? Read the article on meralgia paresthetica to answer that question. You may appreciate the osteopathic approach to this diagnosis and perhaps gain a better insight into additional diagnostic or treatment considerations for your patient with this condition. Staying on the musculoskeletal theme, there are two excellent review articles on joint pain, covering the usual to the obscure. I would be happy to hear feedback on whether this partial theming is worthwhile to our readers. As you enjoy the cooler temperatures of fall, be sure to watch out for mosquito bites, which leads to the final article in the issue. Zika virus, while not as prevalent in the mainstream media recently, is representative of an infectious disease with an insect vector.

Happy reading – and good luck trying to balance an egg on its end during the autumnal equinox on September 22nd! (It's claimed that a special property of the equinox allows eggs to be balanced on their ends that day.)

 Print ISSN: 1877-573X

FROM THE PRESIDENT'S DESK

A Different Side to the Opioid Crisis

Duane G. Koehler, DO, FACOFP *dist*.
2018 - 2019 ACOFP President

As family physicians, we care for extended families: mothers, fathers, brothers, sisters, etc. That is how many family physicians view their patients. The family link defines who they are to each other, and to the family physician who treats them.

But not every patient is seen this way. Those patients who are taking opioids are often immediately branded as "addicts," even if they are on opioids for good reason. They need opioids to function when they have documented chronic pain unresponsive to other medications and treatments.

Family physicians will see chronic pain patients first. It is up to them to determine if the pain is legitimate or not. Sometimes this is easy; there is radiographic evidence telling the history of the pain. Sometimes it is not that easy and you only have the patient's words, your hands, and a pain scale of numbers or faces. Then there is the final element of trust – do you trust the patient?

Some of our colleagues are afraid they will lose their license if they write one more opioid prescription for any patient because of the volumes of press on the opioid crisis. But the government is not looking for the family doctor who has a few patients suffering from chronic pain and may be on opioids long-term. They are looking for those who write excessive numbers of prescriptions, consistently, and have a volume that is out of the ordinary for their area. They are looking for diversion of opioids; they are looking for patterns of behavior that signal a problem that needs to be monitored.

A recent article told the story of a man who had a musculoskeletal condition that caused him debilitating pain. He had exhausted all procedures and other prescription medications. Several years back, his primary care doctor gave him an opioid prescription for his chronic pain. It worked so well that he could get up and do things with his wife. He was almost leading a normal life thanks to opioid therapy. He was no longer depressed and his wife was no longer his caretaker. This prescription changed two lives for the better.

Then the news of the opioid crisis hit. When the man went to his PCP, the physician would not prescribe opioids to him anymore. The physician said, "I am not going to lose my license over prescribing opioids." The man went home, and within hours the pain returned. The next day it was unbearable. He could not move around or go on walks with his wife, who became his caretaker again. Things continued to spiral downward. One day, the man did not see any reason to go on and ended his life. The cause of death was a self-inflicted gunshot wound; not a drug overdose.

We don't hear stories of how opioids improve and sometimes, save lives. We hear the stories of people who overdose, by accident or on purpose, by combining opioids with other non-opioid drugs such as cocaine. We hear about the illicit purchase and use of street drugs like heroin to replace opioids. We are all victims of the news we absorb. It has caused many physicians to see patients on opioids through a lens that is only negative.

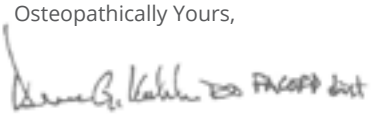
The CDC recently recognized that the rapid increase in deaths by drug overdose is driven in large part by synthetic opioids produced and sold illegally, including heroin, fentanyl and fentanyl analogs like carfentanil, which is 100 times more potent than fentanyl. This contributes to the damaging press on opioids, even though it is not the fault of a physician.

Though doctors cannot always control how patients use the medication they prescribe, the CDC does outline steps physicians can take in attempt to improve care and reduce risks in the *CDC Guideline for Prescribing Opioids for Chronic Pain*.[1] As osteopathic physicians we have OMT, which could be tried first or in addition to first-line, non-opioid analgesic therapy.

I am not advocating that every patient who is on opioids needs them, nor am I suggesting you start prescribing opioids if you are not comfortable. But, each patient who comes through your door needs something from you that they can't find anywhere else: compassion, health advice, support, direction. These are just part of what osteopathic physicians offer; a wholistic approach to treating patients.

ACOFP members will see patients on opioids – it is a guarantee. But, will we see them all as addicts, or as someone's father, brother, mother, sister? If needed, will we help them find a pain clinic or a behavioral health resource? Will we follow up with them to be sure they are getting what they need? Will we treat them in an osteopathic way?

Osteopathically Yours,



Duane G. Koehler, DO, FACOFP *dist*.
2018 - 2019 ACOFP President

REFERENCE:

1. Dowell D, Haegerich TM, Chou R. CDC Guideline for Prescribing Opioids for Chronic Pain — United States, 2016. MMWR Recomm Rep 2016;65 (No. RR-1): 1–49. DOI: http://dx.doi.org/10.15585/mmwr.rr6501e1

acofp

PREPARE FOR THE BOARD EXAM *with* ACOFP

ONLINE PRACTICE QUESTIONS

Boost your confidence with practice questions as you prepare for the Board exam. This tool features over 150 multiple choice questions with a detailed explanation of each answer.

Intensive Update & Board Review Online

Earn up to 15 1-B CME credits with fast-paced, 30-minute didactic video presentations on key topics in contemporary family medicine.

OMT ESSENTIALS

OMT Video Procedures Online

View step-by-step demonstrations of 150 OMT procedures. Sort and select the videos according to anatomic area to be treated, type of OMT procedure or chapter in the ACOFP textbook *Somatic Dysfunction in Osteopathic Family Medicine, Second Edition.*

OMT Textbook

Somatic Dysfunction in Osteopathic Family Medicine, Second Edition covers diagnostic work-up, the clinical rationale for OMT for specific diseases and conditions, manipulative techniques for a variety of patient types, and procedures for common conditions.

GET STARTED AT
acofp.org

RESEARCH ARTICLE

Meralgia Paresthetica: A 5 Model Osteopathic Approach

Martin Torrents, DO, MPH, MBA & Rajat Malik, DO

Touro College - OMM Department, Middletown, New York

KEYWORDS:

Meralgia Paresthetic

Osteopathic Medicine

Osteopathic Manipulative Treatment

Meralgia Paresthetica is the diagnostic term used to describe a neuropathy of the lateral femoral cutaneous nerve and typically presents with numbness, tingling, paresthesias or any other sign of nerve impingement along the anterior and lateral thigh. This condition is quite debilitating and bothersome for patients and typically is underdiagnosed in the outpatient setting, partially due to symptoms being attributed to other hip or lumbar spine causes. This article will provide an osteopathic perspective on this condition with all of its probable causes as well as a reference for a structured approach to managing the patient with osteopathic manipulative medicine. In addition, home exercises, stretches, and behavioral adaptations will be mentioned in order to maintain the results of the osteopathic manipulative treatment. A case report will be presented and specific findings related to this condition will be explained. Osteopathic manipulative treatment, along with behavioral retraining, should be considered as treatment options and offered to patients prior to pursuing more invasive therapeutic measures.

INTRODUCTION

Meralgia paresthetica (MP) is the diagnostic term given to a patient presenting with a mononeuropathy of the lateral femoral cutaneous nerve (LFCN) and typically presents with numbness, tingling, paresthesias, or any other sign of nerve impingement along the anterior and lateral thigh. It is specifically due to compression of this nerve as it passes over the anterior superior iliac spine and then under the inguinal canal at the lateral end as it progresses. It may be more commonly seen in individuals who are obese, have diabetes, or wear their pants too tight at the waist. Presenting clinical signs and symptoms may be sufficient for obtaining the diagnosis, however, electrodiagnostic studies may be useful in confirming the diagnosis and quantifying the degree of nerve involvement.[1] Treatment can vary from activity modification and a holistic approach to TENS unit applications, analgesics and local and/or systemic anti-inflammatories. Surgical interventions with neurolysis or neurectomies for those with significant weakness and atrophy or focal conduction blocks on electrodiagnostic examinations have also been used as treatment modalities.[2] Given that this condition can be considered an inflammatory mononeuropathy based on its clinical presentation, relieving compression of the nerve along its course with osteopathic manipulation and behavioral modifications can provide symptomatic relief and restore normal functioning of the lateral femoral cutaneous nerve.

CORRESPONDENCE:
Martin Torrents, DO, MPH, MBA | martin.torrents@touro.edu

 Print ISSN: 1877-573X

MP typically occurs in patients ranging from 30 to 60 years of age and has an approximate incidence of 4-10/10,000 people.[3] The age group of 55-64 years old had the highest incidence of MP in both men and women.[1] It is estimated that the actual incidence may be a little higher due to under-reporting of symptoms and physicians attributing MP to other causes of hip and/or thigh pain. The incidence rates are similar between men and women, ruling out gender as a determinant.

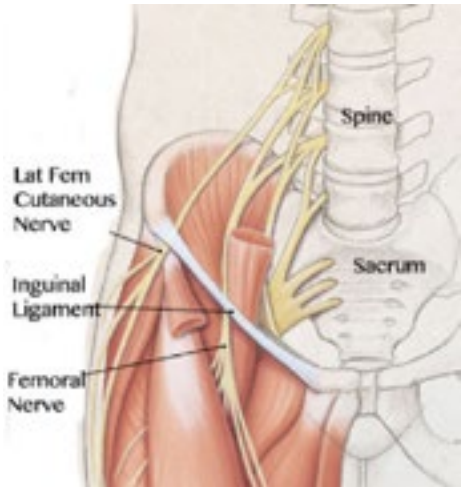
In addition to these factors, there are several behavioral and genetic causes that can contribute to the symptoms of MP. Consistent use of tight belts, tight sweat pants, corsets, or body armor have been associated with the development of symptoms of MP.[4,5] It is suspected that any extra added weight applied to the waist belt can also contribute (i.e. keys, cell phone/pager holders, gun holsters, etc.). Chronic repetitive positioning of the lower extremity in an extended or externally rotated position may also impinge on the nerve. MP as a post-op complication after orthopedic hip surgery, hernia repairs, or being in a prone position during surgery has also been noted.[6,7] In addition, pelvic crush fractures and pelvic osteotomies have also been linked with the prevalence of MP.[7] Among common genetic causes, patients with diabetes mellitus type 2 (DMT2) are seven times more likely to develop MP and there is a strong association of MP as a precursor to DMT2.[1] Obesity, pregnancy, and increased BMI have been associated with MP due to increased abdominal girth.[1,8] Other causes of MP include benign pelvic masses, tumors along the iliac crests, and osteoid sarcomas.[7]

ETIOLOGY

A mononeuropathy of the lateral femoral cutaneous nerve (LCFN) is what brings about the symptoms of MP. Originating from the L2 and L3 spinal nerves, the lateral femoral cutaneous nerve enters the pelvic cavity lateral to the psoas muscle. It continues towards the ASIS over the iliacus muscle. Many variants can be found for its path as it makes a right angle to exit the pelvic cavity above, below, or through the inguinal canal. Whichever course it takes, it is usually within 5cm of the ASIS. Therefore, it passes through the lacuna muscularis, a passageway inferior to the inguinal canal that takes the nerve to the sartorius muscle. In this passageway it may travel along the iliopsoas muscle, which, if in a contracted state, may compress some nerve fibers. After passing this location, the nerve passes superficially over the sartorius muscle and branches into the anterior and posterior branches (Figure 1). There is also a common variant of the nerve piercing though the sartorius muscle. The anterior branch continues deep to the tensor fascia lata for about 5-10 cm down the lateral thigh and then pierces the fascia to become subcutaneous, where it may be subjected to mechanical stress. The lateral femoral cutaneous nerve takes a long and complicated pathway, which presents many opportunities for the nerve to become entrapped. As with any nerve, prolonged chronic repetitive trauma may lead to trophic changes of the skin, neuropathic pain, and underlying tissue changes.

FIGURE 1:

Anatomy of LCFN and Cutaneous Sensory Distribution



There are several pathological changes that may affect the LFCN under prolonged compression or entrapment. In addition to the disordered orientation of nerve fiber bundles intermixed with increased connective tissue components that can be present in compression neuropathies,[9,10] an autopsy study of 12 nerves revealed that the LFCN demonstrated local demyelination and Wallerian degeneration. Greater relevance for this case were the additional findings of endoneurial vascular changes and polarized internodal swellings suggesting that mechanical factors are responsible for the symptoms of MP.[10,11,12]

These findings suggest that an osteopathic structural exam on the patient with MP should be considered an essential component of the physical examination in order to identify areas of mechanical compression on the LFCN by means of somatic dysfunctions of the lumbar spine, pelvis, sacrum, and lower extremities.

CASE PRESENTATION

A 68 year old overweight female with a history of bilateral mastectomy secondary to breast cancer presented with numbness and tingling of the anterior and lateral aspect of the right lower extremity for the past several weeks. Given the presentation and excluding other causes of her symptoms, she had been diagnosed with MP by her PCP. She was then provided with NSAIDs to use as needed and referred to a neurologist for treatment. The neurologist, recognizing the neuropathic component of her symptoms, prescribed Gabapentin three times daily for symptoms. This medication did not provide the sufficient relief that the patient was expecting and produced significant side effects (i.e. weight gain). She was also referred to a general surgeon but had no interest in pursuing surgical intervention at this point. The patient had heard of osteopathic manipulation through a family member and decided to see if it would be particularly helpful. She had stopped taking Gabapentin by the time of this initial visit, and denied any recent trauma, surgery or prior injury to the region.

Upon eliciting a detailed history and performing an osteopathic structural exam, patient stated that she forgot to mention that she spends hours a day knitting on the couch in which she tucks her right foot under her buttocks and keeps her lower extremity flexed and externally rotated. Physical exam findings demonstrated normal stance, gait, muscle strength testing and reflexes of lower extremities. She did have diminished sensation to touch on the anterior and lateral aspect of the right thigh. Her BMI was found to be 29, otherwise vital signs and other physical exam findings were normal.

Osteopathic exam findings were consistent with the following: bilateral hypertonicity to the psoas muscles right sided more severe than left, L2 flexed, rotated, and sidebent right, positive ASIS compression test on right with innominate findings consistent with a posteriorly rotated and out-flared pelvis. A restriction in excursion of the right hemidiaphragm was noted during the inspiratory phase of thoracic respiration. In addition, a chronically hypertrophied tensor fascia lata muscle with tenderness to palpation and a taut iliotibial band were also noted on her right.

MANAGEMENT WITH OSTEOPATHIC MANIPULATIVE TREATMENT

Due to the several factors that may contribute to the symptoms of MP, management of the patient must encompass a multifactorial approach. Reports of successful treatment modalities range from surgery to electroacupuncture to off-label use of medications to behavioral modifications.[2,4,8] Given this variety, principles of patient-centered care with emphasis on the five models of osteopathic diagnosis and treatment would be beneficial for MP. The five models are the biomechanical model, the respiratory-circulatory model, the neurological model, the metabolic-energy model, and the behavioral model.[13,14] The patient above was treated with different modalities in order to fully address all components contributing to her symptoms, while maintaining specific emphasis on these models.

MUSCLE ENERGY TECHNIQUE TO INNOMINATE SOMATIC DYSFUNCTION

A few muscle energy techniques were applied to the patient to address structural abnormalities that may have been compromising the LFCN in order to address the biomechanical model. Particular attention was directed at the innominate somatic dysfunctions and to the lumbar spine.

For the innominate posterior rotation dysfunction, the patient laid supine slightly diagonal so that the right sacroiliac joint was off the edge of the table. The physician was standing on the right side of the table while his cephalad hand was placed over the patient's left ASIS to prevent the patient from rolling off the table. The caudad hand was placed on the distal femur, just proximal to the knee. The physician's caudad hand passively extended the patient's right hip, bringing the innominate into anterior rotation, until the edge of the restrictive barrier was reached (Figure 2). Be careful not to pass the barrier or over extend the hip as this may stretch and further irritate the LCFN. The physician instructed the patient to lift the right leg toward the ceiling while the physician applied an equal counterforce. This isometric contraction was maintained for 3-5 seconds, after which the patient was instructed to stop and relax. Once the patient has completely relaxed, the physician extended the patient's right hip to the edge of the new restrictive barrier. Three to five repetitions of these directions were performed after which reassessment of the area was performed.[15]

For the right out-flared innominate dysfunction: The patient laid supine while the physician stood on the left side of the table. The patient's right hip and knee were flexed to approximately 75 degrees and the right foot was positioned just lateral to the left knee. The physician's cephalad hand was placed under the patient's right innominate, grasping the medial aspect of the right PSIS. The physician's caudad hand was placed on the patient's right knee and adducted it until the edge of the restrictive barrier was reached (Figure 3). The physician instructed the patient to abduct the right hip while the physician applied an equal counterforce. This isometric contraction was maintained for 3-5 seconds, then the patient was instructed to stop and relax. Once the patient completely relaxed, the physician further adducted the patient's right knee to the edge of the new restrictive barrier and drew traction laterally on the right PSIS. Three to five repetitions of these directions were performed after which reassessment of the area was indicated.[15]

MUSCLE ENERGY TECHNIQUE TO LUMBAR SD (L2FRSR)

The patient was placed in the right lateral recumbent position with the knees and feet together and shoulders and pelvis perpendicular to the table. The physician stood in front of the patient and with his cephalad hand he contacted the L2-L3 level. With his caudad hand, the physician flexed the patient's hips and knees until motion was localized at the L3 vertebral segment. The patient was instructed to straighten her lower leg and with his caudad hand the physician passively extended it to achieve motion at the L3 vertebral segment. The patient's left top leg was flexed and the foot placed in the popliteal space of the extended, bottom leg. The physician then switched hands so that the caudad hand was monitoring the segment. The now cephalad hand grasped the patient's lower arm and gently pulled it in an anterior and caudad direction until a rotational motion was felt at the L2-L3 level. The lower arm was then moved posterior and superior to induce left side bending. The patient should be grabbing the head of the table now with the upper body in a supine position. The patient was then instructed to take a deep breathe, exhale completely and while doing so reach further inferiorly by grasping the edge of the table with the superior arm and to continue this for a few respiratory cycles. The physician now grasped distal to the knee of the patient's top leg and elevated it until side bending of the lumbar spine was achieved to the motion barrier while monitoring the segment with the cephalad hand (Figure 4). The patient was asked to push her elevated foot down toward the table for 3 to 5 seconds, while the physician provided an isometric resistance. The patient was instructed to relax for 3-5 seconds after which the physician further elevated the foot until motion was noticed at the new barrier. The procedure was repeated at least three times after which the somatic dysfunction improved. Finally, a passive stretch was performed after the last repetition.[16]

FIGURE 2:

Posterior Innominate Muscle Energy

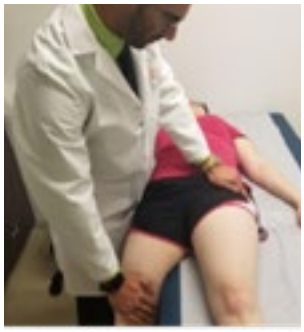


FIGURE 3:

Outflare Innominate Muscle Energy

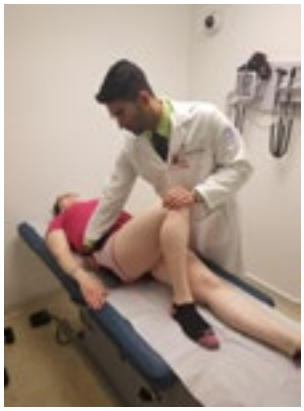


FIGURE 4:

Lumbar Spine Muscle Energy

FIGURE 5:

Diaphragmatic Lift Technique

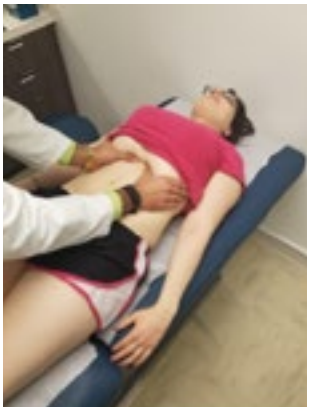
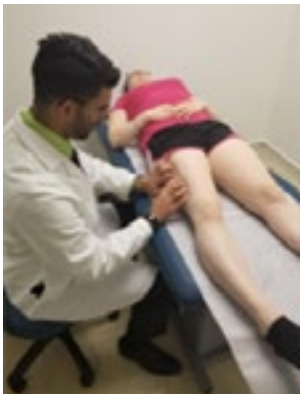


FIGURE 6:

Peripheral Nerve OMT of LFCN

FIGURE 7:

Peripheral Nerve OMT of LCFN Perforating Branches



DIAPHRAGMATIC LIFT TECHNIQUE

Due to the connections of the psoas muscle with the crural muscles at the posterior abdominal wall, it would be common to find diaphragmatic restrictions associated with psoas muscle spasms. Relieving dysfunctions in the respiratory-circulatory realm by treating the diaphragm was considered to be significant to bring about changes in the patient's' condition. Dr. William G. Sutherland, an original student of the founder of osteopathic medicine, Dr. Andrew T. Still, described a diaphragmatic lift technique in which the ultimate goal would be to "draw the diaphragm cranially, elevating the floor of the thorax, drawing upward on the abdominal contents, promoting venous and lymphatic drainage from the lower half of the body."[17] Based on his description of this technique, the physician can stand on the side that is easiest for them facing the patient. Then the physician's thumbs were placed right inferior to the sternum at the costosternal junctions while the rest of the fingers were spread around the lateral edges of the ribs towards the patient's head. A cephalad and lateral lift is then exerted by the physician, lifting the diaphragm. The patient was instructed to exhale while the physician continued to lift the diaphragm in a cephalad and lateral direction. Tension was maintained and held while the patient inspired and was slightly increased during exhalation (Figure 5). Concomitantly, the patient was instructed to exhale immediately after inhaling and to not hold the breath in, while the physician continued to engage new barriers with cephalad and lateral tractions. Finally, when no more advancement of fingers were noted, the patient was told to rapidly exhale, while simultaneously doing the valsalva maneuver (closing throat and increasing intrathoracic pressure) and expanding her chest.

PERIPHERAL NERVE OSTEOPATHIC TREATMENT OF LFCN

A direct treatment to the lateral femoral cutaneous nerve would specifically address the neurological model of osteopathic diagnosis and treatment. The LFCN is usually treated in the region of its anterior branch which runs two fingerbreadths subcutaneously on a horizontal line over the pubic bone. In this technique, and as with perforating skin branches of other nerves, palpation of the nerve was done from distal to proximal, always at the skin surface. The LFCN specifically runs from the middle to the upper third of the leg. In this nerve lesion, at the anterior aspect of the tensor fascia lata, there was a small, hardened, and pressure-sensitive deepening in the skin. For the actual treatment, the patient was in a supine position with the legs extended and the physician's thumb glided from the middle of the leg to the anterior lateral surface while maintaining pressure (Figure 6). The treatment released the tension in the tissue and at the same time released the perforating branch from impingements. The perforating points can also be felt and identified. Applying pressure while gliding from the middle of the leg to the anterior lateral surface, as in the previous technique, can help release tension and further release perforating branches from impingement (Figure 7). Multiple pain pressure points were discovered and even disappeared completely after a few manipulations.[19]

BEHAVIORAL INTERVENTION

The metabolic-energy model attempts to optimize the patient's internal homeostasis by establishing a healthy relationship between energy production and expenditure. In this case, due to body habitus and body mass index, the patient was screened for the presence of DMT2 by obtaining a Hemoglobin A1c measurement, which was 5.4 (within normal limits). The importance of exercise and a well balanced diet were enforced during this encounter and the patient stated interest in following up appropriately. A referral to a nutritionist was then provided.

To address aspects of the behavioral model, the patient was instructed to remove aggravating factors that may compress the waist line in any manner: use of tight belts, tight pants, body armor, etc. She was also instructed to be cautious as to how she normally sits on the couch and to avoid prolonged sitting with the right leg tucked under her buttock. To maintain the biomechanical changes achieved with OMT, the patient was also advised to perform a stretching exercise at least twice a day which particularly encourages free motion of the diaphragm and posterior abdominal wall attachments.

EXERCISES – STANDING PSOAS & ACHILLES STRETCH

One's gait is truly important when diagnosing lower extremity somatic dysfunctions. Hypertonic muscles can alter gait and create new and chronic somatic dysfunctions. In this case, it was important to ensure that the psoas, hamstrings, quadriceps, gastrocnemius, and soleus muscles were appropriately stretched for an effective gait with proper swing phase and heel strike. The patient was encouraged to thoroughly stretch the psoas muscle by increasing lumbar lordosis and extending slightly at hip; then to transition into a stretch for the Achilles tendon, which particularly in females, tends to contract due to frequent use of high heels. Effective stretching of this tendon will assist in normal gait mechanics. Standing two feet from the wall and, facing it with feet shoulder-width apart, place palms on the wall at shoulder height. From this position it is important to keep elbows fully extended and attempt to extend the lower back to stretch muscles that support the lumbar spine by gently arching the pelvis towards the wall. The patient was instructed to maintain this position and breathe slowly and deeply for at least ten respiratory cycles. To transition into the Achilles tendon stretch, have the patient return the pelvis to a relatively neutral position and instruct to simply bend the knees as much as possible while keeping the heels on the floor. While maintaining this position breathe slowly and deeply for at least ten respiratory cycles (Figure 8). Repeat both stretches at least twice a day but more if tolerated.[18]

FIGURE 8:

Standing Psoas and Achilles Stretch

FOLLOW-UP

The patient returned for a follow-up encounter one week after the first visit with a renowned sense of empowerment. Her symptoms had not returned with the same severity at all, on the contrary, she stated that if she felt any tingling or numbing of her thigh she would immediately stand up and perform the stretching exercises indicated for her. This would temporarily ease and eventually stop the progression of symptoms altogether. Continuously performing the stretches and being very aware of the resting position of her right hip during her knitting on the couch allowed her to be free of symptoms of MP. She did not feel the need to take an NSAID for her MP but was content on the fact that if she had to resort to it she would without issues.

Osteopathic structural evaluation at follow-up was only significant for a restricted right pelvic diaphragm which was not evident at the initial visit. This could be a new somatic dysfunction or a compensatory mechanism after other somatic dysfunctions were treated. A simple pelvic diaphragm release was performed in a supine position, which the patient tolerated very well. The patient was instructed to continue with the exercises and stretches and to return to the office if symptoms returned, persisted, or worsened.

CONCLUSION

The etiology of MP can be one that is multifactorial, ranging from musculoskeletal dysfunctions to genetic causes. Given this wide array of causes, the treatment of MP should encompass a multifactorial approach. Utilizing the tenets of osteopathic medicine which state that "structure and function are intimately related" as well as "the body is capable of self-healing, self-regulating mechanisms" can provide the physician with an in-depth manipulative treatment rationale for MP and other mononeuropathies. In addition, once this approach is combined with a motivated patient's willingness to partake in a home exercise program and suggested behavioral modifications for relieving compression of the nerve, a successful outcome can be obtained. Empowering the patient with responsibility for their health by use of exercises and stretches also allows application of another tenet which is "the human is a combination of mind, body and spirit" at a deeper level of self-consciousness and self-realization. An osteopathic approach can be key in treating musculoskeletal causes and modifying behavioral contributions that has been refractory to other treatment options. Although both surgical and non-surgical approaches have been shown to be effective in treating the symptoms of MP, osteopathic manipulation should also be considered before more invasive treatment modalities.

AUTHOR DISCLOSURES:
No relevant financial affiliations.

REFERENCES

1. Parisi, T. J., Mandrekar, J., Dyck, P., Klein, C. J. "Meralgia Paresthetica: Relation to Obesity, Advanced Age, and Diabetes Mellitus." Neurology Vol 77.16 (2011): 1538-1542.
2. Ruiter, GC, De Johannes, W., Wurzer, AL, and Kloet, A. "Decision Making in the Surgical Treatment of Meralgia Paresthetica: Neurolysis versus Neurectomy." Acta Neurochirurgica Vol 154.10 (2012): 1765-772.
3. Slobbe, AM., Van, RMD., Bernsen, AM., Bohnen, S.M., Bierma-Zeinstra, A., and Koes, BW. "Incidence Rates and Determinants in Meralgia Paresthetica in General Practice." Journal of Neurology Vol 251.3 (2004): 294-97.
4. Fargo, MV., and Konitzer, LN. "Meralgia Paresthetica Due to Body Armor Wear in U.S. Soldiers Serving in Iraq: A Case Report and Review of the Literature." Military Medicine Vol 172.6 (2007): 663-65.
5. Scott, K., and Kothari, MJ. "Evaluating the Patient with Peripheral Nervous System Complaints." The Journal of the American Osteopathic Association Vol 105.2 (2005): 71.
6. Peker, S., Ay, B., Sun, I., Ozgen, S., and Pamir, M. "Meralgia Paresthetica: Complication Of Prone Position During Lumbar Disc Surgery." The Internet Journal of Anesthesiology Vol 8.1 (2004).
7. Edelson R., Stevens, P. "Meralgia paresthetica in children". J Bone Joint Surg Am (1994): 76:993–9.
8. Skaggs, C.D., Winchester, B.A.,Vianin, M., and Prather, H. "A Manual Therapy and Exercise Approach to Meralgia Paresthetica in Pregnancy: A Case Report." Journal of Chiropractic Medicine Vol 5.3 (2006): 92-96.
9. Rakel, R. Textbook of Family Practice, 6th Ed. Philadelphia, PA: Saunders Elsevier 2002. Pages 1393-1454.
10. Robbins, S., Cotran, R., Kumar, V., Abbas, A., Aster, J. Pathologic Basis of Disease. Philadelphia, PA: Saunders Elsevier, 2015.
11. Herskovitz, S., Scelsa, S., Schaumburg, H. Peripheral Neuropathies in Clinical Practice. New York: Oxford UP, 2010.
12. Harney, D., Patijn, P. "Meralgia Paresthetica: Diagnosis and Management Strategies." Pain Medicine. Vol 8.8 (2007): 669–677.
13. Chila, A. Foundations of Osteopathic Medicine, 3rd Ed. Baltimore, MD: Lippincott Williams & Wilkins, 2011.
14. DiGiovanna, E., Schiowitz, S., Dowling, D. An Osteopathic Approach to Diagnosis and Treatment, 3rd Ed. Baltimore, MD: Lippincott Williams & Wilkins, 2005. Pages 246-247.
15. Nicholas, A., Nicholas, E. Atlas of Osteopathic Techniques, 2nd Ed. Baltimore, MD: Lippincott Williams & Wilkins, 2012. Pages 280, 287.
16. DeStefano, L. Greenman's Principles of Manual Medicine, 4th Ed. Baltimore, MD: Lippincott Williams & Wilkins, 2001. Pages 312-314.
17. Sutherland, W. Teachings in the Science of Osteopathy. Sutherland Cranial Teaching Foundation, Inc, 1990. Page 276.
18. Fulford, R. Dr. Fulford's Touch of Life. New York, NY: Pocket Books, 1996. Page 183.
19. Barral, JP., Croibier, A. Manual Therapy for the Peripheral Nerves. London, UK: Churchill Livingstone, 2007. Pages 196-197.

REVIEW ARTICLE

Zika Virus - A Review for Family Physicans

Marie Florent-Carre, MPH, DO,[1] Gina Foster-Moumoutjis, MD,[1]
Cyril Blavo, MS, DO, MPH & TM, FACOP,[1] Luzan Phillpotts, DO, MPH,[1] Traci-lyn Eisenberg, DO,[1] &
Bindu S. Mayi, MSc, PhD[2]

[1] Dr. Kiran C. Patel College of Osteopathic Medicine, Nova Southeastern University
[2] College of Medical Sciences, Nova Southeastern University

KEYWORDS:

Zika Virus

Nucleic Amplication Testing

Guillain-Barre Syndrome

Microcephaly

Prevention and Wellness

Mosquito-borne viruses have been on the rise in recent years. This becomes especially critical when you consider a virus with a particularly harrowing consequence, like the Zika virus (ZIKV) and infant microcephaly. From its obscure history in Africa, ZIKV diverged into an African and Asian strain, the latter strain traveling from Asia to the islands in the Pacific Ocean and ultimately to the Americas, including the United States. ZIKV spread through this immunologically naïve population through mosquito bites, sexual transmission, intrauterine transmission and blood transfusion. Zika symptoms may include fever, joint pain, and maculopapular rash, all of which are common to other mosquito-borne illnesses such as dengue and chikungunya, also seen in similar locations. Awareness of the symptoms, differential diagnosis and the testing algorithm for Zika may improve detection and facilitate early management. Tests for ZIKV include nucleic amplification assays, IgM serology, and the plaque reduction neutralization test, with decision to test determined by symptom status, pregnancy status, and degree of exposure. While the majority of patients with ZIKV are asymptomatic, the most severe complications are Guillain-Barré Syndrome for adults and Congenital Zika Syndrome for infants born to infected mothers. Since the mainstay of management is supportive care and surveillance, preventive strategies are critical in prevention of these complications. Prevention is multimodal including mosquito population control, mosquito bite prevention, and actions to prevent sexual transmission. Through counseling and early detection, the family medicine practitioner has the unique opportunity to prevent the spread of ZIKV and its potential complications.

INTRODUCTION

In 2016, the World Health Organization (WHO) declared Zika virus (ZIKV) infection a public health emergency of international concern.[1] The virus had spread to 26 countries and territories in the Americas, infecting thousands of individuals and causing complications such as Guillain-Barré Syndrome (GBS) in adults and severe microcephaly in infants born to infected mothers. The increased cases of microcephaly caught the attention of public health authorities and health professionals, compounded by the fact that microcephaly had not been associated with any arbovirus infection to date. Since ZIKV is a reemerging virus with the potential to cause complications, it is important for health professionals to be informed about ZIKV so they can recognize this threat. This paper provides a comprehensive description of the history, transmission, clinical manifestations, diagnosis and prevention of ZIKV infection.

CORRESPONDENCE:
Marie Florent-Carre, MPH, DO | mflorent@nova.edu

 Print ISSN: 1877-573X

ZIKV belongs to the Flaviviridae family of viruses, which also consists of other important human pathogens such as West Nile encephalitis virus (WNEV), Dengue virus (DENV), Yellow fever virus and Japanese encephalitis virus (JEV).[2] Before the current outbreak, ZIKV had an unremarkable history. In 1947, Yellow Fever researchers first isolated the virus from a caged Rhesus monkey in the Zika forest of Uganda.[3] From Africa, ZIKV made its way to Asia, where in 1954, it was isolated from mosquitoes in Malaysia and by 1977, it had been isolated from patients in Indonesia.[4,5] Phylogeny reveals two distinct lineages of ZIKV, African and Asian. In 2007, the Asian lineage of ZIKV affected about 70 percent of the population on Yap island in the Federated States of Micronesia, presumably after having arrived from Southeast Asia by air travel.[6] Data implicate this Asian lineage in the ZIKV outbreak in the Americas.[7] After Yap Island, ZIKV infected French Polynesia, other islands across the Pacific Ocean, and subsequently Brazil and the Americas, where it gained notoriety for its complications of microcephaly and GBS.[8] Based on phylogeny, researchers postulate that ZIKV was introduced to Brazil sometime between August 2013 and July 2014.[9] ZIKV infection of this immunologically naïve population may have facilitated its rapid spread in Brazil and neighboring countries. It is thought that the virus circulated undetected for about a year, either because of subclinical

infections or because of misdiagnosis of Zika (ZIKV disease) as one of the other endemic arboviral illnesses, such as Dengue and Chikungunya.

On July 29, 2016, the continental United States (U.S.) recorded its first case of a local, mosquito-borne ZIKV infection in Miami-Dade County, Florida.[10,11] Following this initial report, ZIKV infection was reported in Hidalgo County, Texas.[11,12] ZIKV is not the first arbovirus to cause an outbreak in the United States. The CDC website accounts for provisional Zika cases in the U.S. and its territories, according to which the total number of symptomatic Zika cases in the U.S. from 2015 to 2018 have been 5,700. The number of presumed locally transmitted cases was 231, with Florida showing the highest number at 220 and Texas showing 11 cases. There have been short-lived outbreaks with other arboviruses, such as DENV and Chikungunya virus (CHIKV), in regions of Texas and Florida, when the Aedes aegypti mosquito populations were abundant.[13] In 2016, mosquito surveillance data revealed that approximately one out of 1,600 Aedes aegypti mosquitoes were infected with ZIKV, an infection rate that is consistent with outbreaks of DENV and CHIKV.[14,15] Furthermore, research has shown that human ZIKV infection correlates with abundant Aedes aegypti populations.[16] Using phylogenic analyses of ZIKV from clinical and mosquito samples without passage through cell culture, researchers estimate that ZIKV was likely introduced into Florida at least two months prior to its first detected human case.[15] Because of international travel, outbreaks in the U.S. will likely correspond with ZIKV outbreaks in other parts of the world where Aedes aegypti mosquitoes are in abundance.

MODES OF TRANSMISSION

Current evidence supports the following modes of transmission of ZIKV to humans: mosquito-borne transmission, sexual transmission, intrauterine transmission and transmission through blood transfusion.[17,18] While the Aedes aegypti mosquito is most commonly responsible for transmission of ZIKV to humans, Aedes albopictus can also transmit ZIKV.[19,20] Transmission of ZIKV through sexual intercourse has been reported in male-to-female, male-to-male, and female-to-male sexual contact.[17, 21-27] In addition to the four modes of transmission, rare cases of intrapartum transmission and transmission from laboratory exposure have also been reported.[17,18]

CLINICAL MANIFESTATIONS & MANAGEMENT

The most common symptoms associated with human ZIKV infection include fever, a maculopapular rash, arthralgias, and non-purulent conjunctivitis. Other symptoms that have been reported from the outbreak in Yap Island include headache, myalgias, retro-orbital pain, edema and vomiting,[6,28] as shown in Table 1. Approximately 80 percent of individuals infected with ZIKV are asymptomatic.[6] The incubation period of ZIKV infection varies between three and 14 days.[29] The course of the infection is typically self-limiting, with symptoms lasting for several days to a week.[30] Severe disease requiring hospitalization is uncommon and mortality remains low, with one death reported in 153 hospitalized patients.[11,31] Supportive care of symptomatic patients and fetal surveillance of pregnant women is the mainstay of management for people infected with ZIKV.[31,32]

TABLE 1:

Symptoms of Zika

MOST COMMON SYMPTOMS OF ZIKA	ADDITIONAL SYMPTOMS
Fever	Retro-orbital pain
Maculopapular rash	Edema
Headache	Vomiting
Arthralgia	
Non-purulent conjunctivitis	
Myalgias	

COMPLICATIONS OF ZIKA VIRUS INFECTION IN ADULTS

The complication of greatest concern in adults infected with ZIKV is Guillain-Barré syndrome (GBS), an immune mediated progressive neurologic disorder characterized by tingling, progressive weakness, autonomic dysfunction and pain.[33] GBS has been associated with many infections, most notably Campylobacter jejuni, but also other flaviviruses such as JEV, WNEV and DENV.[33, 34, 35] During the French Polynesia ZIKV outbreak of 2013-2014, 32,000 people sought medical attention for possible ZIKV infection and 42 patients were identified with GBS.[35] This represents a 20-fold increase over baseline incidence of GBS in this population.[34] Associations between the incidence of ZIKV and GBS have been observed during outbreaks in Brazil, Colombia, Dominican Republic, El Salvador, Honduras, Panama, Suriname, and Venezuela.[36, 37] A WHO expert panel reviewed the data and concluded that ZIKV is a trigger for GBS.[35]

COMPLICATIONS IN INFANTS BORN TO MOTHERS INFECTED WITH ZIKV DURING PREGNANCY

A comprehensive review of the literature relative to ZIKV infection in French Polynesia, Brazil, U.S. and Spain identified a constellation of symptoms, now recognized as Congenital Zika Syndrome (CZS).[38] The main features of this syndrome are abnormal cranial morphology, brain anomalies, neurologic sequelae, ocular anomalies, and congenital contractures, with specific examples including fetal brain disruption sequence, cerebral cortex thinning, epilepsy, cataracts, and arthrogyropsis.[38] Continued surveillance and reporting should help better define this syndrome in the future.

The breadth of impact of maternal intrauterine ZIKV infection in the U.S. is still being monitored. The CDC, in collaboration with state, territorial, tribal, and local health departments, has created a registry known as the U.S. Zika Pregnancy Registry (USZPR),

which monitors birth defects in infants born in the U.S. to women potentially infected with ZIKV during pregnancy.[39] A detailed list of possible birth defects and instructions on how to report them is available through the CDC.[40] As of January 2018, the USZPR has reported 17 pregnancy losses with evidence of ZIKV-associated birth defects and 6,106 completed pregnancies with laboratory evidence of possible ZIKV infection among women in the U.S., District of Columbia, U.S. Territories and Freely Associated States.[41] Of the 6,106 completed pregnancies, there were 247 live-born infants with evidence of ZIKV-associated birth defects (4%). A 2017 study evaluated 442 pregnant women and pregnancy outcomes from the USZPR and concluded that 6% of infants had evidence of birth defects, with the rate of birth defects increasing to 11% when the mother was infected in the first trimester.[41, 42] Microcephaly was the most common defect, reported at a rate of 4%, which falls between the rates of birth defects seen in French Polynesia (0.95%) and estimated in Bahia, Brazil (13.2%).[43, 44]

DIFFERENTIAL DIAGNOSIS

Considering current epidemiologic trends, the most relevant differential diagnoses to consider are CHIKV and DENV. High fever (≥104˚F) is an important distinguishing characteristic of Dengue while fever and severe joint pain may be more indicative of Chikungunya.[45, 46] Table 2 provides an inclusive list of differential diagnoses, including other mosquito-borne illnesses and infections that present with fever, rash and arthralgia or myalgia. Currently, the CDC recommends testing for DENV and CHKV for patients at risk of clinically compatible illness.[47] If other diagnoses are suspected at the time of presentation, specific testing should be pursued as well.[47] While awaiting laboratory confirmation of ZIKV, it is important to avoid the use of nonsteroidal anti-inflammatory medications in all patients with suspected DENV due to the increased risk of hemorrhagic complications.[48]

DIAGNOSTIC APPROACH TO PATIENTS WITH POSSIBLE ZIKV EXPOSURE

Currently, there are three main arms of testing for ZIKV infection: Nucleic amplification testing assays (NAT), Anti-Zika IgM serology assays (Zika IgM), and the plaque reduction neutralization test (PRNT). As of January 2018, the FDA has approved 14 NAT and 5 Zika IgM assays for emergency use.[49] A positive NAT result represents the presence of ZIKV RNA in the host specimen sample. NAT is routinely performed on serum and urine samples; however, other fluid and tissue samples, such as cerebrospinal fluid, placental tissue, and amniotic fluid are also under investigation.[50, 51] Since ZIKV RNA is only transiently present in any body fluid, a positive NAT suggests an acute infection.[50, 52, 53] ZIKV IgM assays detect anti-ZIKV IgM antibodies that normally become detectable within 1 week of symptom onset.[32] Though data is limited, one recent study showed that ZIKV IgM may persist for up to 43 days in serum and that the duration of detection varies based on the assay being used.[54] Thus, positive ZIKV IgM is suggestive of ZIKV infection, but does not identify the timing of infection.[47] Given a large cross reactivity between flavivirus antibodies and a false positive rate up to 27% in the U.S., PRNT is used as a confirmatory test to differentiate ZIKV IgM antibodies from other flavivirus antibodies present in the host.[50, 55] However, in areas of high DENV circulation, PRNT does not differentiate between ZIKV and DENV IgM.[56] Thus, PRNT should not be used in areas such as Puerto Rico, which have high rates of DENV infection.[50]

TABLE 2:

Differential Diagnoses

PRESENTATION	DIAGNOSES
Fever, Rash and Arthralgia/Myalgia	Zika Lyme Disease Dengue Rickettsiosis Chikungunya
Fever and Rash	Rubella Measles Parvovirus Adenovirus Scarlet Fever Ehrlichiosis
Fever and Arthralgia/Myalgia	Yellow Fever Leptospirosis West Nile Encephalitis* Malaria

**Most cases are asymptomatic*

Given the changing epidemiology of ZIKV infection and the limitation of available testing, the CDC continuously updates its algorithm for laboratory testing and confirmation of diagnosis.[32] Table 3 summarizes the most recent testing recommendations for pregnant and non-pregnant individuals, released in January 2018 and July 2017 respectively. The most recent updates recommend initial testing with NAT of both serum and urine and/or ZIKV IgM testing.[32, 50, 51] The choice of test is based upon type of exposure, symptom, pregnancy status, and time since symptom onset.[32, 50, 51] Exposure is further categorized as ongoing or limited risk of ZIKV transmission. The term ongoing exposure refers to living in or frequent travel to an area with a risk of Zika or sexual intercourse with a partner who lives in or frequently travels to an area with a risk of Zika. The term limited exposure refers to limited travel to an area with a risk of Zika or sexual intercourse with a partner who has traveled to an area with a risk of Zika. The CDC travel pages provide updated information regarding areas worldwide and in the U.S. at risk of ZIKV infection.[57, 58] The term symptomatic refers to patients presenting with any of the following symptoms of ZIKV infection: maculopapular rash, conjunctivitis, fever or arthralgia.[47]

PREVENTION OF MOSQUITO-BORNE TRANSMISSION OF ZIKV

For people living in or traveling to areas at risk of Zika, preventing contact between mosquitoes and humans is the primary method of reducing infection. Bite prevention should be multimodal, including use of insect repellant, protective clothing, and environmental precautions. Insect repellants containing the following active ingredients have been approved and are endorsed by the CDC and Environmental Protection Agency (EPA): N,N-Diethyl-meta-toluamide (DEET), picaridin, IR35, Oil of Lemon Eucalyptus, para-menthane-diol, and 2-undecanone.[59] The most effective repellents against the Aedes aegypti mosquito contain

TABLE 3:

CDC recommendation for laboratory testing and result interpretation for patients with possible exposure to ZIKV

TIMEFRAME	INITIAL TEST INITIAL TEST	INTERPRETATION OF TEST RESULTS
Symptomatic Nonpregnant		
< 2 weeks from symptom onset	NAT serum and urine for ZIKV	Positive: diagnostic for acute ZIKV Infection Negative: ZIKV unlikely
> 2 and < 12 weeks from symptom onset	IgM serology for ZIKV	Positive or equivocal: PRNT testing to confirm diagnosis, if ZIKV ≥ 10 and DENV <10: confirms ZIKV infection, though timing of infection cannot be determined Negative: ZIKV unlikely
Symptomatic Pregnant		
Up to 12 weeks from symptom onset	NAT serum and urine for ZIKV and IgM for ZIKV*	Either urine or serum NAT Positive and IgM positive: diagnostic for acute ZIKV infection NAT not concordant** and IgM negative: retest original positive sample (urine or serum) for NAT, if negative then test serology 2 weeks from date of original test NAT and IgM negative: no evidence of ZIKV infection
Asymptomatic Pregnant: Ongoing Exposure		
Upon initiation of prenatal care	NAT urine and serum for ZIKV***	Positive: diagnostic for ZIKV infection, no further Zika testing needed Negative: ZIKV infection in pregnancy cannot be ruled out. Retest with NAT two more times in pregnancy.ŧ
Asymptomatic Pregnant: Limited Exposure		
Upon initiation of prenatal care if case-by-case decision makingł suggests testing	NAT urine and serum for ZIKV	Positive: diagnostic for ZIKV infection, no further Zika testing needed Negative: ZIKV infection in pregnancy cannot be ruled out. Retest with NAT two more times in pregnancy.ŧ

Abbreviations: *NAT = Nucleic amplification testing; ZIKV = Zika virus; PRNT = Plaque-reduction neutralization test Information from references (32, 47, 50)*

** Given the extended presence of viral particles in body fluids during pregnancy, all symptomatic pregnant women undergo both NAT-serum/urine and ZIKV IgM testing at initial presentation of symptoms*

*** Positive urine NAT and negative serum NAT or negative urine NAT and positive serum NAT*

**** CDC does not currently recommend IgM serology because IgM can persist for months after ZIKV infection, thus a positive serology result cannot reliably determine if the infection took place before or during pregnancy.*

ŧ At non-coinciding prenatal visits, additional testing may be warranted depending on local trends in ZIKV transmission

ł Given high rate of false positives of NAT and IgM in areas of low-risk, case-by-case decision making between provider and patient should inform the decision to test.

DEET (>5%) and picaridin (16%). However, products containing 25% DEET provide protection for the longest duration of time.[60, 61] The American Academy of Pediatrics cautions against the use of DEET containing products in infants less than 2 months of age.[62]

Aedes aegypti bite during the day and night and dwell both indoors and outdoors. Long sleeved shirts, long pants, socks, and hats are recommended to cover bare skin.[63] In areas where there is no permethrin resistance, permethrin treated clothes are also effective at repelling mosquitoes.[64, 65] Environmental precautions can also decrease exposure. Keeping windows and doors closed or screened and using air conditioning, can reduce the number of mosquitoes present in daytime and nighttime environments. Covering any holes or gaps between doors, walls, and screens can further secure the indoor environment.[65] If sleeping outside, insecticide treated bed nets are recommended.[64, 65]

To prevent continued dissemination of ZIKV, it is also important to prevent transmission from infected humans to mosquitoes. Because humans infected with ZIKV are viremic during the first week of illness and can transmit the virus to a biting mosquito, it is recommended that patients with known ZIKV infection take precautions to prevent mosquito bites during this time.[66, 67] Travelers returning from areas of possible ZIKV infection should prevent mosquito bites for three weeks, which safely covers the range of the ZIKV incubation period and the length of viremia.[66]

According to the CDC and the U.S. EPA, one of the most effective means of decreasing a mosquito population is to remove the habitat and thus interrupt the life cycle at the larval and adult stages. Monitoring areas with standing water is an important aspect of mosquito population control since mosquitoes rely on water for two stages of their life cycle. Areas with standing water, such as old tires, gutters, or toys containing water should be drained, scrubbed and covered. Birdbaths, fountains, or potted plant trays should be emptied and scrubbed once a week. The addition of larvicides to breeding habitats is very effective in reducing populations in nearby locations. Aerial spraying of adulticides, either by aircraft, truck-mounted or backpack sprayers, is effective in reducing the total number of adult mosquitoes over larger areas.[65]

PREVENTION OF SEXUAL TRANSMISSION OF ZIKV & PRECONCEPTION COUNSELING TO PREVENT INTRAUTERINE TRANSMISSION

Though not as common as transmission from mosquitoes, sexual transmission poses an important modifiable risk that, if addressed, can help prevent birth defects associated with ZIKV. To reduce the risk of sexual transmission of ZIKV, all men and women who live in or travel to areas at risk of ZIKV infection should use barrier methods or abstain from vaginal, anal or oral sex.[22] Furthermore, to help prevent possible adverse outcomes in pregnancy, ZIKV prevention counseling should be a routine part of care for women of reproductive age. To promote early detection of ZIKV before and during pregnancy, the American College of Obstetricians and Gynecologists (ACOG) recommends that all women should be informed of the common symptoms of ZIKV infection (Table 1).[68] If pregnancy is not desired, strategies to prevent pregnancy, including family planning and contraceptive options, should be discussed.[22]

For women with ongoing ZIKV exposure who desire pregnancy, the health professional should discuss the possible adverse outcomes of ZIKV infection during pregnancy. Women and their partners should be informed of mosquito bite prevention strategies and reassured that if used according to product labels, EPA registered mosquito repellents can be used safely before and during pregnancy.[69] If a woman with ongoing ZIKV exposure does become pregnant, she should initiate prenatal care immediately and undergo ZIKV testing.[32]

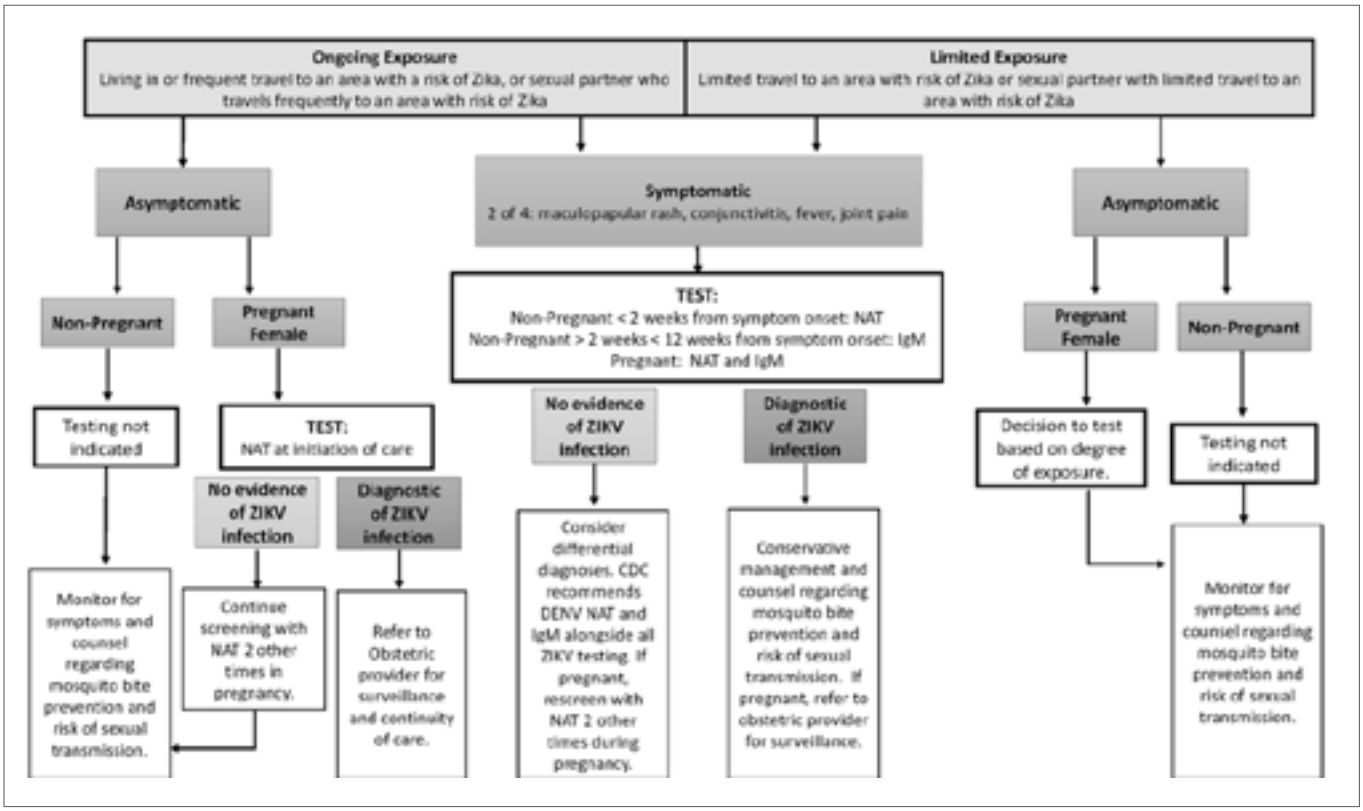
For women with limited ZIKV exposure who desire pregnancy, the health professional should recommend avoiding travel to areas at risk of ZIKV infection.[32] If she or her sexual partner must travel to these areas, they should follow the recommendations listed above to help prevent mosquito bites while traveling. If the woman is possibly exposed to ZIKV through travel or sexual contact with a partner who has traveled, she should prevent conception for the next eight weeks either by abstaining from or using condoms during sexual intercourse.[22, 32] ZIKV may be passed through sexual transmission from an asymptomatic male.[22, 70, 71] and ZIKV RNA has been found in semen up to 188 days after symptom onset.[22, 72] Because of this evidence, the CDC recommends that males should abstain from sex or use condoms for six months following possible exposure.[22] If females become symptomatic, they should avoid unprotected sex for eight weeks from the date of symptom onset or Zika diagnosis. If males become symptomatic, they and their female partners should avoid unprotected sex for six months from the date of symptom onset or Zika diagnosis in the male.[22] Should the woman become pregnant, she should initiate prenatal care immediately. The need for ZIKV testing would be determined based on the degree of possible exposure and shared decision making.[32]

CONCLUSION

An approach to patients with possible ZIKV exposure is illustrated in Figure 1. Armed with this knowledge, through counseling and early detection, the medical community can help prevent the spread of ZIKV and its potential complications. In the absence of FDA approved antivirals and vaccines, the onus for preventing ZIKV infection falls on mosquito eradication efforts, as well as early detection and management. The rapid emergence of ZIKV in the Americas highlights the importance of a gene-based, global surveillance network that can identify the increasing presence of new or reemerging viruses and implement prevention initiatives such as mosquito eradication and immunization programs when available.

FIGURE 1:

Primary Care Approach to Patient with Possible ZIKV Exposure.



AUTHOR DISCLOSURES:
No relevant financial affiliations

REFERENCES

1. WHO statement on the first meeting of the International Health Regulations (2005) (IHR 2005) Emergency Committee on Zika virus and observed increase in neurological disorders and neonatal malformations. 1 February 2016. http://www.who.int/mediacentre/news/statements/2016/1st-emergency-committee-zika/en/
2. Kuno G, Chang GJ, Tsuchiya KR, Karabatsos N, Cropp CB. Phylogeny of the genus Flavivirus. J Virol. 1998;72(1):73-83.
3. Dick, GWA. Zika virus (II). Pathogenicity and physical properties. Transactions of The Royal Society of Tropical Medicine and Hygiene.1952;46(5):521–534. https://doi.org/10.1016/0035-9203(52)90043-6
4. Marchette NJ, Garcia R, Rudnick A. Isolation of Zika virus from Aedes aegypti mosquitoes in Malaysia. Am J Trop Med Hyg. 1969;18(3):411-5.
5. Olson JG, Ksiazek TG, Suhandiman, Triwibowo. Zika virus, a cause of fever in Central Java, Indonesia. Trans R Soc Trop Med Hyg. 1981;75(3):389-93.
6. Duffy MR, Chen TH, Hancock WT, Powers AM, Kool JL, et al. Zika virus outbreak on Yap Island, Federated States of Micronesia. N Engl J Med. 2009;360(24):2536-43. doi: 10.1056/NEJMoa0805715.
7. Haddow AD, Schuh AJ, Yasuda CY, Kasper MR, Heang V, et al. Genetic Characterization of Zika Virus Strains: Geographic Expansion of the Asian Lineage. PLoS Negl Trop Dis 2012; 6(2): e1477. doi:10.1371/journal.pntd.0001477
8. Musso D, Gubler DJ. Zika virus. Clin Microbiol Rev 2016; 29:487–524. doi:10.1128/CMR.00072-15.
9. Metsky HC, Matranga CH, Wohl S, Schaffner SF, Freije CA et al. Zika virus evolution and spread in the Americas. Nature 2017;546(7658):411-415. doi: 10.1038/nature22402.
10. Florida Department of Health Daily Zika Update: the Department Responds to Local Zika Cases. July 29, 2016. http://www.floridahealth.gov/newsroom/2016/07/072916-local-zika.html
11. Hall V, Walker WL, Lindsey NP, et al. Update: Noncongenital Zika Virus Disease Cases — 50 U.S. States and the District of Columbia, 2016. MMWR Morb Mortal Wkly Rep 2018;67:265–269. DOI: http://dx.doi.org/10.15585/mmwr.mm6709a1
12. McCarthy M. First US case of Zika virus infection is identified in Texas. BMJ 2016;352:i212. doi: 10.1136/bmj.i212.
13. Adalja AA, Sell T, Bouri N, Franco C. Lessons Learned during Dengue Outbreaks in the United States, 2001–2011. Emerg Infect Dis. 2012;18(4):608-614. https://dx.doi.org/10.3201/eid1804.110968
14. CDC. Chikungunya virus in the United States. August, 2015. Augusta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/chikungunya/geo/united-states.html
15. Grubaugh ND, Ladner JT, Kraemer MUG, Dudas G, Tan AL, et al. Genomic epidemiology reveals multiple introductions of Zika virus into the United States. Nature 2017;546(7658):401-405. doi: 10.1038/nature22400.

17. Dzul-Manzanilla F, Martínez NE, Cruz-Nolasco M, Gutiérrez-Castro C, López-Damián L, et al. Evidence of vertical transmission and co-circulation of chikungunya and dengue viruses in field populations of Aedes aegypti (L.) from Guerrero, Mexico. Trans R Soc Trop Med Hyg. 2016;110(2):141-4. doi: 10.1093/trstmh/trv106.
18. Gregory CJ, Oduyebo T, Brault AC, et al. Modes of transmission of zika virus. J Infect Dis. 2017;216:S875-S883.
19. Sharma A, Lal SK. Zika virus: Transmission, detection, control, and prevention. Frontiers in microbiology. 2017;8:110.
20. Wong PJ, Li MI, Chong C, Ng L, Tan C. Aedes (stegomyia) albopictus (skuse): A potential vector of zika virus in singapore. PLoS Neglected Tropical Diseases, 2013;7(8): 1.
21. Grard G, Caron M, Mombo IM, et al. Zika virus in Gabon (Central Africa) – 2007: A new threat from aedes albopictus? PLoS neglected tropical diseases. 2014;8(2):1.
22. Sherley M, Ong C. Sexual transmission of zika virus: A literature review. Sexual health. 2017.
23. Petersen EE, Meaney-Delman D, Neblett-Fanfair R, et al. Update: Interim Guidance for Preconception Counseling and Prevention of Sexual Transmission of Zika Virus for Persons with Possible Zika Virus Exposure — United States, September 2016. MMWR Morb Mortal Wkly Rep 2016;65:1077-1081. DOI: http://dx.doi.org/10.15585/mmwr.mm6539e1.
24. Foy BD, Kobylinski KC, Chilson Foy J,L., et al. Probable non-vector-borne transmission of zika virus, Colorado, USA. Emerging infectious diseases. 2011;17(5):880-882.
25. Hills SL, Russell K, Hennessey M, et al. Transmission of zika virus through sexual contact with travelers to areas of ongoing transmission - continental United States, 2016. MMWR Morb Mortal Wkly Rep 2016;65(8):215-216.
26. Venturi G, Zammarchi L, Fortuna C, et al. An autochthonous case of Zika due to possible sexual transmission, Florence, Italy, 2014. Euro Surveill 2016;21:30148.
27. Deckard DT, Chung WM, Brooks JT, et al. Male-to-male sexual transmission of Zika virus – Texas, January 2016. MMWR Morb Mortal Wkly Rep 2016;65:372–4.
28. Davidson A, Slavinski S, Komoto K, Rakeman J, Weiss D. Suspected female-to-male sexual transmission of Zika virus – New York City, 2016. MMWR Morb Mortal Wkly Rep 2016;65:716–7.
29. PAHO. Epidemiological Alert: neurological syndrome, congenital malformations, and Zika virus infection. Implications for public health in the Americas. 1 December 2015. Washington, DC: World Health Organization, Pan American Health Organization.
30. Krow-Lucal ER, Biggerstaff BJ, Staples J. Estimated Incubation Period for Zika Virus Disease. Emerg Infect Dis. 2017;23(5):841-845. https://dx.doi.org/10.3201/eid2305.161715
31. CDC. Symptoms. May 2017. https://www.cdc.gov/zika/symptoms/symptoms.html
32. CDC. Clinical Evaluation and Disease. December, 2017. https://www.cdc.gov/zika/hc-providers/preparing-for-zika/clinicalevaluationdisease.html
33. Oduyebo T, Polen KD, Walke HT, et al. Update: Interim Guidance for Health Care Providers Caring for Pregnant Women with Possible Zika Virus Exposure – United States (Including U.S. Territories), July 2017. MMWR Morb Mortal Wkly Rep 2017;66:781-793. DOI: http://dx.doi.org/10.15585/mmwr.mm6629e1.
34. Rodríguez Y, Rojas M, Pacheco Y, Acosta-Ampudia Y, Ramírez-Santana, C, Monsalve, DM, et al. Guillain–Barré syndrome, transverse myelitis and infectious diseases. Cell Mol Immunol 2018; doi: 10.1038/cmi.2017.142. [Epub ahead of print]
35. Oehler E, Watrin L, Larre P, Leparc-Goffart I, Lastère S, Valour F, et al. Zika virus infection complicated by Guillain-Barré syndrome – case report, French Polynesia, December 2013. Euro Surveill. 2014;19(9):pii=20720. https://doi.org/10.2807/1560-7917.ES2014.19.9.20720
36. Krauer F, Riesen M, Reveiz L, Oladapo OT, Martínez-Vega R, Porgo TV, et al. Zika virus infection as a cause of congenital brain abnormalities and Guillain–Barré syndrome: systematic review. PLoS Med 2017;14(1):e1002203. doi:10.1371/journal.pmed.1002203
37. Smith DW, Mackenzie, J. Zika virus and Guillain-Barré syndrome: another viral cause to add to the list. The Lancet, 2016;387(10027):1486-1488.
38. Dos Santos T, Rodriguez A, Almiron M, Sanhueza A, Ramon P, de Oliveira WK, et al. Zika virus and the Guillain–Barré syndrome – case series from seven countries. N Engl J Med 2016;375(16):1598-1601.
39. Moore CA, Staples JE, Dobyns WB, et al. Characterizing the pattern of anomalies in congenital zika syndrome for pediatric clinicians. JAMA pediatrics. 2017;171(3):288-295.
40. CDC. US Zika and pregnancy infancy registry. January, 2018. GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/pregnancy/zika/research/registry.html
41. CDC. Zika and Pregnancy, Technical and Clinical Information. January, 2018. GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/pregnancy/zika/research/technical-clinical.html
42. CDC. Outcomes of Pregnancies with Laboratory Evidence of Possible Zika Virus Infection, 2015-2018. January, 2017. Atlanta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/pregnancy/zika/data/pregnancy-outcomes.html
43. Honein MA, Dawson AL, Petersen EE, et al. Birth defects among fetuses and infants of US women with evidence of possible zika virus infection during pregnancy. Jama. 2017;317(1):59-68.
44. Cauchemez S, Besnard M, Bompard P, et al. Association between zika virus and microcephaly in french polynesia, 2013-15: A retrospective study. Lancet (London, England). 2016;387(10033):2125-2132.
45. Johansson MA, Mier-y-Teran-Romero L, Reefhuis J, Gilboa SM, Hills SL. Zika and the risk of microcephaly. N Engl J Med. 2016;375(1):1-4.
46. WHO. Dengue and Severe Dengue. April, 2017. Geneva: World Health Organization. Retrieved from: http://www.who.int/mediacentre/factsheets/fs117/en/
47. CDC. Chikungunya Virus: What You Need to Know. September, 2015. Atlanta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/chikungunya/pdfs/factsheet_chikungunya-what-you-need-to-know.pdf
48. CDC. Zika virus testing guidance: symptomatic non-pregnant individuals with possible zika virus exposure. August, 2017. Atlanta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/zika/pdfs/testing-algorithm-symptomatic-nonpregnant.pdf
49. PAHO. Tool for diagnosis and care for patients with suspected arboviral disease. March, 2017. Washington, DC: World Health Organization, Pan American Health Organization. http://iris.paho.org/xmlui/handle/123456789/33895
50. FDA. Zika Virus Emergency Use Authorization. January, 2018. Silver Spring, MD. US Food and Drug Administration. Retrieved from: https://www.fda.gov/medicaldevices/safety/emergencysituations/ucm161496.htm#zika
51. CDC. Guidance for US Laboratories Testing for Zika Virus Infection. July 24, 2017. Atlanta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/zika/laboratories/lab-guidance.html

52. CDC. Zika and Pregnancy, Testing and Diagnosis. January, 2018. Atlanta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/pregnancy/zika/testing-follow-up/testing-and-diagnosis.html -

53. Lanciotti RS, Kosoy OL, Laven JJ, et al. Genetic and serologic properties of zika virus associated with an epidemic, yap state, micronesia, 2007. Emerging infectious diseases. 2008;14(8):1232-1239.

54. Paz-Bailey G, Rosenberg ES, Doyle K, et al. Persistence of zika virus in body fluids - preliminary report. N Engl J Med. 2017.

55. Pasquier C, Joguet G, Mengelle C, et al. Kinetics of anti-ZIKV antibodies after zika infection using two commercial enzyme-linked immunoassays. Diagn Microbiol Infect Dis. 2018;90(1):26-30.

56. Woods CR. False-positive results for immunoglobulin M serologic results: Explanations and examples. Journal of the Pediatric Infectious Diseases Society. 2013;2(1):87-90.

57. Lindsey NP, Staples JE, Powell K, et al. Ability to serologically confirm recent zika virus infection in areas with varying past incidence of dengue virus infection in the united states and U.S. territories in 2016. J Clin Microbiol. 2018;56(1).

58. CDC. World maps of areas with risk of Zika transmission. February, 2018. Atlanta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://wwwnc.cdc.gov/travel/page/world-map-areas-with-zika

59. CDC. Guidance for areas with local Zika transmission in the continental United States and Hawaii. August, 2017. Atlanta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/zika/geo/domestic-guidance.html

60. Centers for Disease Control and Prevention. CDC Yellow Book 2018: Health Information for International Travel. Chapter 2 (19). New York: Oxford University Press; 2017.

61. Uc-Puc V, Herrera-Bojórquez J, Carmona-Carballo C, et al. Effectiveness of commercial repellents against Aedes aegypti (L.) in yucatan, méxico]. Salud publica de Mexico. 2016;58(4):472-475.

62. Fradin MS, Day JF. Comparative efficacy of insect repellents against mosquito bites. N Engl J Med. 2002;347(1):13-18.

63. American Academy of Pediatrics Committee on Environmental Health. Pesticides. In: Etzel RA, ed. Pediatric Environmental Health. 2nd ed. Elk Grove Village, IL: American Academy of Pediatrics; 2003:348-350.

64. EPA. Success in Mosquito Control: An Integrated Approach. October, 2016. The United States Environmental Protection Agency. Retrieved from: https://www.epa.gov/mosquitocontrol/success-mosquito-control-integrated-approach

65. Goodyer LI, Croft AM, Frances SP, et al. Expert review of the evidence base for arthropod bite avoidance. Journal of travel medicine. 2010;17(3):182-192.

66. CDC. Response to Zika, Counseling Travelers. September, 2017. Atlanta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/zika/pdfs/travelcounceling-fs.pdf

67. CDC. Zika Key Messages. October, 2017. Atlanta, GA: US Department of Health and Human Services, CDC. Retrieved from: https://www.cdc.gov/zika/pdfs/Zika-Key-Messages.pdf

68. Krow-Lucal ER, Biggerstaff BJ, Staples J. Estimated Incubation Period for Zika Virus Disease. Emerging Infectious Diseases. 2017;23(5):841-845. doi:10.3201/eid2305.161715.

69. ACOG. Practice Advisory, Interim Guidance Care for Obstetric Patients During a Zika Virus Outbreak, Reproductive Counseling. September, 2017. The American College of Obstetricians and Gynecologists. https://www.acog.org/Clinical-Guidance-and-Publications/Practice-Advisories/Practice-Advisory-Interim-Guidance-for-Care-of-Obstetric-Patients-During-a-Zika-Virus-Outbreak

70. ACOG. Practice Advisory, Interim Guidance Care for Obstetric Patients During a Zika Virus Outbreak, Prevention. October, 2016. The American College of Obstetricians and Gynecologists. https://www.acog.org/Clinical-Guidance-and-Publications/Practice-Advisories/Practice-Advisory-Interim-Guidance-for-Care-of-Obstetric-Patients-During-a-Zika-Virus-Outbreak

71. Fréour T, Mirallié S, Hubert B, et al. Sexual transmission of Zika virus in an entirely asymptomatic couple returning from a Zika epidemic area, France, April 2016. Euro Surveill 2016;21:30254.

72. Brooks RB, Carlos MP, Myers RA, et al. Likely sexual transmission of Zika virus from a man with no symptoms of infection—Maryland, 2016. MMWR Morb Mortal Wkly Rep 2016;65:915–6.

73. Nicastri E, Castilletti C, Liuzzi G, Iannetta M, Capobianchi MR, Ippolito G. Persistent detection of zika virus RNA in semen for six months after symptom onset in a traveller returning from Haiti to Italy, February 2016. Euro surveillance : bulletin Europeen sur les maladies transmissibles = European communicable disease bulletin. 2016;21(32).

REVIEW ARTICLE

Approach to Polyarthritis for the Primary Care Physician

Arielle Freilich, DO, PGY2 & Helaine Larsen, DO

Good Samaritan Hospital Medical Center, West Islip, New York

KEYWORDS:

Polyarthritis

Synovitis

Joint Pain

Rheumatic Disease

Rheumatology

Complaints of joint pain are commonly seen in clinical practice. Primary care physicians are frequently the first practitioners to work up these complaints. Polyarthritis can be seen in a multitude of diseases. It can be a challenging diagnostic process. In this article, we review the approach to diagnosing polyarthritis joint pain in the primary care setting. Starting with history and physical, we outline the defining characteristics of various causes of arthralgia. We discuss the use of certain laboratory studies including sedimentation rate, antinuclear antibody, and rheumatoid factor. Aspiration of synovial fluid is often required for diagnosis, and we discuss the interpretation of possible results. Primary care physicians can initiate the evaluation of polyarthralgia, and this article outlines a diagnostic approach.

INTRODUCTION

Polyarticular joint pain is a common complaint seen in primary care practices. The differential diagnosis is extensive, thus making the diagnostic process difficult. A comprehensive history and physical exam can help point towards the more likely etiology of the complaint. The physician must first ensure that there are no symptoms pointing towards a more serious diagnosis, which may require urgent management or referral. It is then important to differentiate between true articular pain versus pain which arises from outside the joint. Distinguishing whether the arthritis has an inflammatory component can further narrow down the differential diagnosis. Inflammatory conditions will often cause synovitis with warmth and swelling, as well as prolonged morning stiffness. These conditions also cause constitutional symptoms, and patients may report other associated complaints such as rash or ocular involvement. Physical exam of a patient with osteoarthritis, a non-inflammatory condition, will often reveal osteophytes and crepitus, as opposed to an inflammatory arthritis, which will demonstrate findings of synovitis. Laboratory and imaging studies are often nonspecific, but may help rule in or rule out certain diagnoses. Much of the initial workup for polyarthritis can be done in the primary care setting using proper clinical investigation and appropriate diagnostic studies (See Figure 1).

CORRESPONDENCE:

Arielle Freilich, DO PGY2 | Afreilich1@gmail.com

 Print ISSN: 1877-573X

PATIENT HISTORY

Although laboratory studies can shed much light on a possible diagnosis, a detailed history and physical examination remain crucial in the evaluation of polyarticular symptoms. The vast differential for polyarticular pain can be greatly narrowed using a thorough history.

Emergencies

During the initial evaluation, the physician must first exclude any life-threatening conditions, which may present with arthritic-type symptoms. Urgent orthopedic evaluation should be considered in the setting of significant injury to a joint. A warm, swollen joint should raise concern for infection before a diagnosis of gout or pseudogout is given. While constitutional symptoms such as fever, weight change, or fatigue may indicate a systemic rheumatic disease, sepsis from infection must be ruled out. Weakness may be a sign of muscle dysfunction, such as myositis or a degenerative neuromuscular disorder. Radiculopathy must be considered in the presence of neurogenic pain. Once we have ruled out these serious diagnoses, we may further focus on the patient's symptoms.[1]

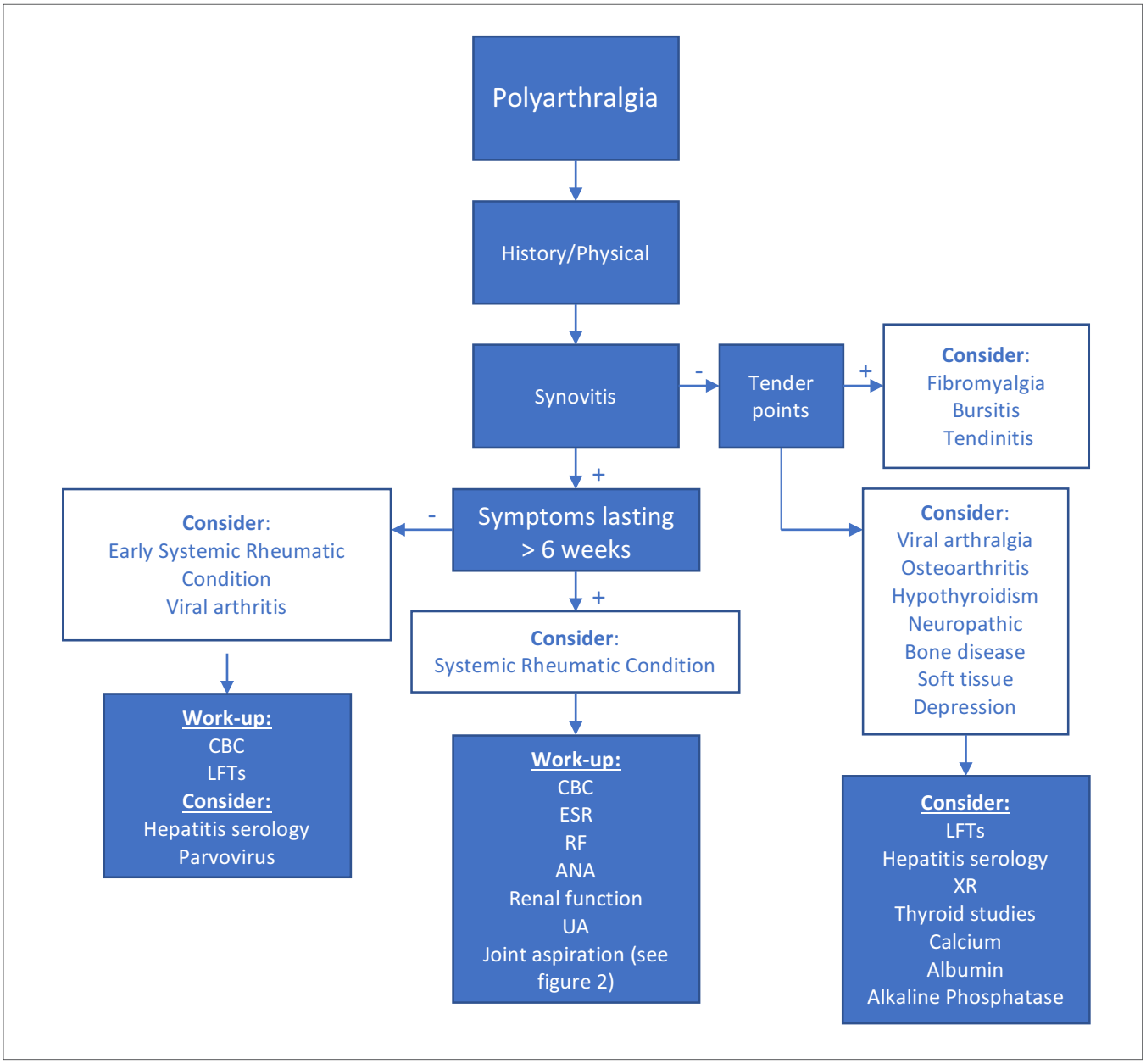
Joint Involvement

Characterizing the joint symptoms a patient is experiencing is an important aspect of the diagnostic approach. The physician must determine whether the patient is experiencing true joint inflammation as opposed to periarticular pain as seen with tendonitis or bursitis. Usually, joint swelling will not be seen in non-arthritic conditions.[2] Arthritis is defined as joint inflammation alongside joint pain. This is in contrast to arthralgia, which lacks the inflammatory component. It is important to make this distinction. Markers of inflammation include surrounding erythema, warmth, tenderness, and swelling of the joint.[3]

The quality of pain a patient reports can help guide the physician. True articular pain would be expected to improve with rest of the joint and worsen with movement or stress. Neurogenic pain would cause feelings of

FIGURE 1:

General approach for evaluation of polyarthritis symptoms[1]



numbness, burning, or a "pins and needles" sensation. This type of pain will not worsen with joint movement and is often more severe at night. Claudication, or pain from arterial insufficiency, will promptly improve with rest. Inflammatory arthritis would cause pain which persists even with rest.[1,4]

The timeframe of symptoms must be established. Acute symptoms persist anywhere from hours to two weeks. Symptoms lasting more than two weeks are considered chronic. Chronic symptoms may be constant or intermittent. Investigating these patterns may help identify certain triggers or associated conditions.[4]

Rheumatoid arthritis (RA) classically affects multiple joints in a bilateral and symmetrical pattern. Patients will present with pain, stiffness, and swelling of joints. Morning stiffness is a common complaint and often lasts more than one hour after awakening.[5] Other systemic rheumatic diseases, such as systemic lupus erythematosus (SLE) and polymyalgia rheumatica, may present with similar patterns. Spondyloarthropathies, including ankylosing spondylitis, reactive arthritis, psoriatic arthritis, and arthritis associated with inflammatory bowel disease are characterized by the presence of the HLA-B27 gene. Joint involvement with these conditions is typically asymmetric and commonly involves inflammation of the sacroiliac joints and spine. Enthesitis, inflammation of the insertion site of tendons or ligaments, is the hallmark of the spondyloarthropathies.[6]

The joints involved can provide important clues. Rheumatoid arthritis will usually affect the proximal interphalangeal joints (PIP), as well as the metacarpophalangeal joints (MCP), but will not involve the distal interphalangeal joints (DIP). This differentiates between osteoarthritis which will involve the DIP and PIP, but spares the MCP.[7] SLE affects similar joints as RA, however there is rarely bone destruction.[2] Any of these joints can be affected with psoriatic arthritis, gout, pseudogout, and sarcoid. Lyme disease differs as inflammation of the hand is not commonly seen.[3] Early Lyme often causes a migratory arthralgia, later progressing to arthritis of mainly large joints.[8] In one study, 51 percent of patients developed arthritis weeks to months after being diagnosed with Lyme disease. The majority of patients experienced knee effusions, but other large and small joints, such as temporomandibular joint, were also involved. Many patients experienced recurrent attacks, with some progressing to chronic arthritis.[9]

Bacterial arthritis from a nongonococcal source classically occurs in one joint, with only 10 to 19 percent of cases having polyarticular involvement. Gonococcal arthritis is typically polyarticular and may have a migratory pattern. Systemic signs such as fevers, chills, and a vesicular rash are common.[8]

Various viral infections may cause arthritic symptoms. An episode of polyarthritis may be the early signs of a fulminant hepatitis B infection, causing joint pain and fevers during a prodromal phase.[8] HIV must be considered, as early disease may cause various forms of arthralgia. Patients may experience episodes of oligoarthritis as well as symmetrical polyarthritis.[2,8] Parvovirus B19 may not cause the frequently classic "slapped-cheek" rash in adults. While most adults with parvovirus infection are asymptomatic, approximately 60 percent of women who are symptomatic will develop arthropathy. These patients will often complain of polyarthritis which is symmetric and affects small joints such as hands, knees, wrists, and ankles. While most see resolution of symptoms in one to three weeks, 20 percent of women will continue to have pain which could last years. In one study on parvovirus B19, 48 percent of adults studied described a symmetrical polyarthropathy. Most complained of pain and stiffness. Several women experienced recurrent episodes of joint symptoms after resolution of the infection.[10] Children are less likely than adults to experience parvovirus-related arthropathy. As opposed to adults, children usually have involvement of large joints in an asymmetric pattern.[11]

If a patient experiences joint tenderness and swelling which fully resolves, followed by recurrent attacks, crystal-induced arthritis, such as gout, must be considered. Generally, one joint is affected with each attack. The initial presentation may involve multiple joints along with fever. Episodes may last one to two weeks, while symptom-free periods could last years early on in the course[2] (See Table 1).

Fibromyalgia is a condition associated with arthralgia that may be confused for polyarthritis. Patients will complain of pain of the back, elbow, trochanter, medial knee, and anterior chest. This pain can be localized to specific tender points. Symptoms are often symmetric. It is often associated with stiffness which is worse in the morning.[12]

TABLE 1:

Differential Diagnosis of Polyarthritis [2,3]

SYSTEMIC RHEUMATIC CONDITIONS	Rheumatoid arthritis Systemic lupus erythematosus Scleroderma Polymyalgia rheumatica Polymyositis Still's Disease Sjögren's syndrome Sarcoidosis
INFECTIOUS	Viral Bacterial (gonococcal vs nongonococcal) Other (Lyme, tuberculosis)
CRYSTAL-INDUCED	Gout Pseudogout
SPONDYLOARTHROPATHIES	Ankylosing spondylitis Psoriatic arthritis Inflammatory bowel disease Reactive arthritis Juvenile idiopathic arthritis
SYSTEMIC VASCULITIS	Henoch-Schönlein purpura Polyarteritis nodosa Granulomatosis with polyangiitis
ENDOCRINE DISORDERS	Hyperthyroidism Hypothyroidism Hyperparathyroidism
OTHER	Osteoarthritis Fibromyalgia Amyloidosis Malignancy

TABLE 2:

Patterns of Joint Symptoms [2]

SYMPTOM PATTERN	DEFINITION	EXAMPLES
INTERMITTENT	Episodes of polyarthritis symptoms with complete resolution between attacks	Gout Pseudogout Reactive arthritis
MIGRATORY	Joint symptoms resolve and then reappear in different joints	Lyme disease Gonococcal arthritis
ADDITIVE	Symptoms in joints persist, with addition of further joint involvement over time	Systemic lupus erythematosus Rheumatoid arthritis Osteoarthritis

A possible sequelae to certain urogenital and enteric infections is reactive arthritis. The most common causes are *Chlamydia trachomatis*, *Salmonella*, *Shigella*, *Campylobacter*, and *Yersinia*. Patients with this condition will experience an additive polyarthritis usually affecting large joints in an asymmetric pattern. Joint symptoms typically present one or two weeks following the infection[6] (See Table 2).

Extra-articular Manifestations

Many rheumatologic causes of polyarthritis will also present with constitutional and systemic signs. Fever, weight loss, and fatigue may be seen. Multisystem involvement of diseases like RA and SLE may present with rash, adenopathy, mucosal ulcers, Raynaud's, xerostomia, and keratoconjunctivitis sicca.[1] Psoriatic arthritis may be suspected in the setting of a history of psoriasis and the classic nail findings such as hyperkeratosis and pitting.[13] The spondyloarthropathies may feature ocular involvement and mucosal lesions.[6]

The inflammation associated with RA stretches way beyond the boundaries of the joints. Subcutaneous and pulmonary nodules may develop. Vasculitis and peripheral neuropathy are also associated with the disease. Patients may develop pericarditis with associated effusions. Some potential ocular findings include episcleritis or scleritis.[7]

Similarly to RA, gout can result in cutaneous nodules referred to as tophi. These crystal deposits can distort the joint space as well as cause pain. Differentiating between RA nodules and tophaceous nodules involves aspiration and analyses of the fluid.[2]

In addition to the synovitis seen with rheumatoid arthritis, the spondyloarthropathies also feature enthesitis. These conditions also present with spinal inflammation and dactylitis, or "sausage digits."[14]

Fever may be seen in conditions other than rheumatic causes. Infection must always be entertained in the setting of fever. Gout and pseudogout are also known to cause fevers during attacks. As previously mentioned, SLE may involve fever. Spiking fevers preceding joint symptoms may be a sign of Still's disease.[15]

Identifying certain rashes can aid in diagnosis. As discussed earlier, history of a "slapped-cheek" rash can indicate parvovirus B19. This classic rash is described as lacy, erythematous, and maculopapular in appearance.[16] SLE often involves a light-sensitive rash which classically involves the face but can also be seen between joints. History of a target-shaped rash can point towards a diagnosis of Lyme disease, although the rash is usually resolved prior to the onset of joint symptoms.[15]

PHYSICAL EXAM

During the exam, it is important to assess joint motion, joint integrity, and exact location of pain to help determine whether a patient is experiencing a mechanical abnormality, soft tissue disease, or true joint disease. Synovitis will present with effusion, warmth, and joint pain with movement. Further analysis will be required, as noninflammatory conditions may also present with joint swelling and effusion.[1,2]

Soft tissue abnormalities, such as bursitis, tendinitis, or injury to a muscle will usually present in a predictable manner. Patients will usually have intact passive range of motion but will experience decreased active range of motion secondary to pain. Point tenderness

to the affected area will often be seen. Decreased active as well as passive range of motion should raise concern for synovitis or structural joint damage. Stability of the joint must be assessed, as laxity may indicate ligament damage.[1] Osteoarthritis should be considered if crepitation is observed without erythema or warmth.[4] Septic arthritis can present with a painful, warm, erythematous joint. Based on patient history, a diagnosis of crystal-induced arthritis may be more likely.[8]

LABORATORY STUDIES

After a thorough history and physical, laboratory investigations should be ordered when appropriate. It is unnecessary to do further testing once a mechanical or extraarticular problem is identified. There are a variety of tests that may help further guide diagnosis.

Erythrocyte Sedimentation Rate & C-Reactive Protein

Both erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are nonspecific markers of inflammation. They are useful in differentiating between inflammatory and noninflammatory processes. ESR can be elevated in many diseases, and is therefore not diagnostic on its own. For example, levels will be increased in the setting of rheumatic conditions, malignancy, and infection. In the acute phase of inflammation, CRP is more reliable than ESR. Non-inflammatory conditions such as diabetes, renal disease, and dysproteinemia can increase ESR, as ESR can be affected in the setting of abnormal red blood cells. While ESR is not diagnostic, an elevated ESR level alongside a classic history and physical can further support a diagnosis of rheumatic disease or other inflammatory process.[1,4]

Antinuclear Antibody

Antinuclear antibody (ANA) may be useful when considering a diagnosis of SLE. It is highly unlikely a patient with negative ANA results will have a diagnosis of SLE.[1] Five to ten percent of the population will have positive ANA testing without a rheumatologic diagnosis, so it is important to consider clinical presentation alongside the laboratory testing. Positive ANA without the clinical features of a rheumatic condition should not drive a diagnosis.[3] Although it cannot provide a definite diagnosis as discussed, the higher the ANA titer is, the more likely a patient is to have SLE.[1]

Rheumatoid Factor & Anti-Citrullinated Peptide Antibodies

Rheumatoid factor (RF) is a nonspecific marker. It may be identified in not only rheumatoid arthritis, but also Sjögren's syndrome, SLE, vasculitis, and chronic infections such as hepatitis C and tuberculosis.[1] RF should only be ordered in the setting of moderate suspicion for rheumatoid arthritis.[1] The test has poor sensitivity and specificity. Approximately 20 percent of rheumatoid arthritis patients lack RF, while five to ten percent of patients without the disease will have positive rheumatoid factor.[17]

Serum Uric Acid

While uric acid in excess can predispose individuals for developing gout, hyperuricemia alone is not diagnostic for the condition.[18] Measuring serum uric acid during a suspected attack does not aid with diagnosis, as uric acid levels may be normal or even low during this time. Uric acid levels become useful for monitoring chronic gout.[1]

Synovial Fluid

Analysis of synovial fluid becomes a very important diagnostic modality when a crystal-induced arthritis or infection is suspected. In order to confirm these diagnoses, the joint must be aspirated. It is important to send the fluid for Gram stain and culture, cell count, and microscopy to identify crystals.[4] Gout is diagnosed with the presence of monosodium urate crystals which are negatively birefringent on microscopy.[4,18] Synovial fluid containing at least 2,000 per mm^3 of white blood cells is considered inflammatory in nature. If the sample contains over 50,000 per mm^3, infection is likely (*Table 3*).[19] Fluid culture will confirm the presence of bacteria and guide the proper antibiotic treatment. While cell counts and culture are pending, any patient with fever and joint effusion should be treated with concern for septic arthritis[4] (See Table 3, Figure 2).

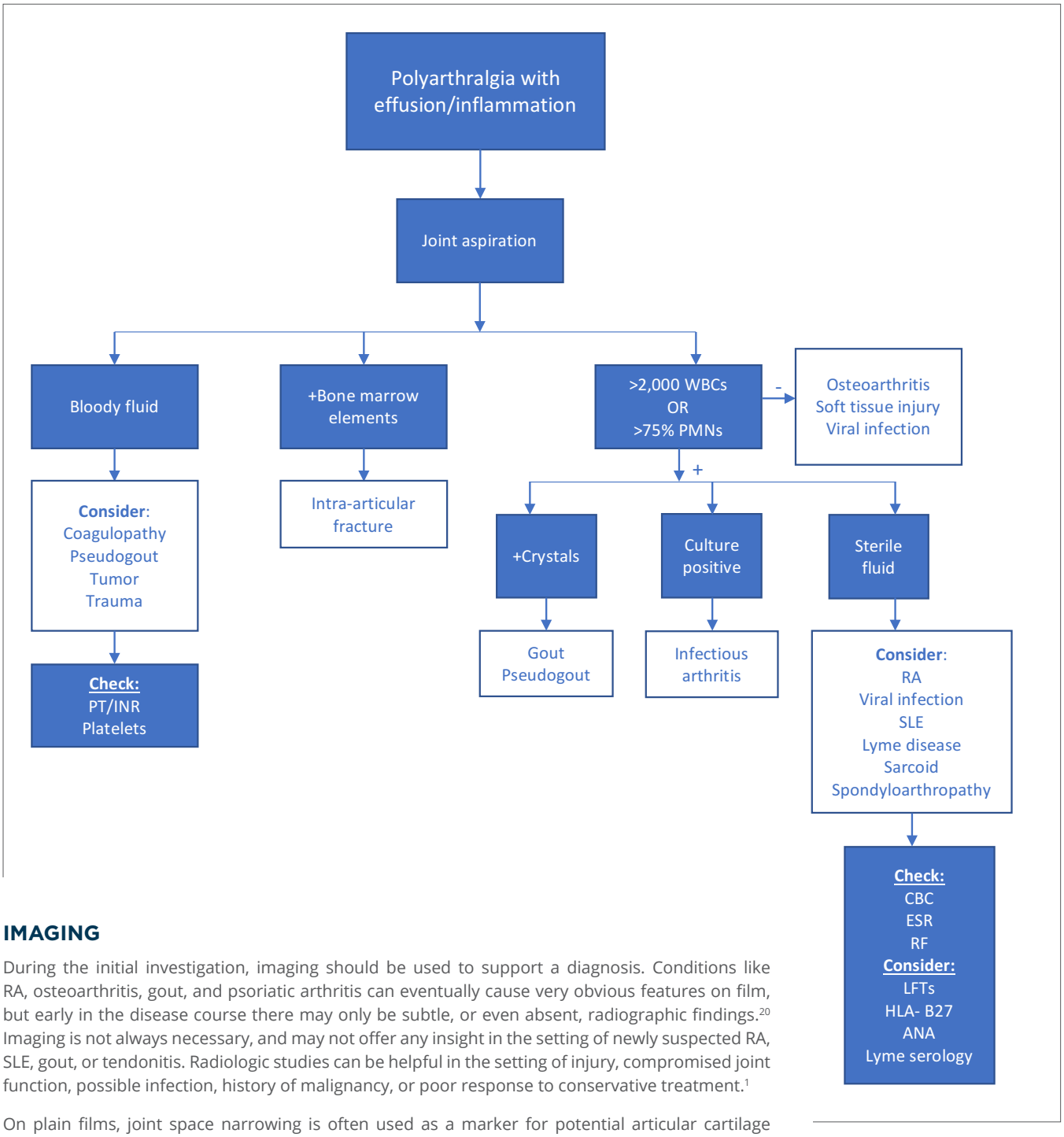
TABLE 3:

Synovial Fluid Analysis[3]

CLASSIFICATION	WBC count	Polymorphonuclear leukocyte count	Examples
NORMAL FLUID	0-200 per mm^3	<25%	
NONINFLAMMATORY	<2,000 per mm^3	<25%	Osteoarthritis
INFLAMMATORY	2,000-50,000 per mm^3	>75%	Rheumatoid arthritis Psoriatic arthritis Gout
SEPTIC JOINT	>50,000 per mm^3	>90%	Septic arthritis Gout Reactive arthritis

FIGURE 2:

Interpretation of synovial fluid aspirate[1]



IMAGING

During the initial investigation, imaging should be used to support a diagnosis. Conditions like RA, osteoarthritis, gout, and psoriatic arthritis can eventually cause very obvious features on film, but early in the disease course there may only be subtle, or even absent, radiographic findings.[20] Imaging is not always necessary, and may not offer any insight in the setting of newly suspected RA, SLE, gout, or tendonitis. Radiologic studies can be helpful in the setting of injury, compromised joint function, possible infection, history of malignancy, or poor response to conservative treatment.[1]

On plain films, joint space narrowing is often used as a marker for potential articular cartilage damage in early osteoarthritis (OA), but studies show there is not a clear correlation, and this finding cannot accurately diagnose or rule out OA.[21] In early RA, conventional radiography may show nonspecific signs such as soft tissue swelling.[1] Identification of erosions early on has been shown to indicate likely progression of structural joint damage. As prompt treatment is required for good prognosis in RA, it is crucial to identify these early erosions. Ultrasound and MRI are found to better detect bone erosions than plain films, and can be very important in monitoring RA progression.[22] Conventional radiography is the first line in detecting structural damage from seronegative spondyloarthropathies such as ankylosing spondylitis, but MRI can be useful in detecting early inflammatory changes.[23]

OSTEOPATHIC PRINCIPLES

Although there is no definitive evidence regarding the benefit of osteopathic manipulative treatment (OMT) in treating arthritic pain, some studies have shown that including OMT in the treatment for certain chronic pain syndromes is efficacious.[24] It is important to discuss the concept of facilitation while speaking of polyarthritis. Facilitation occurs when a segment of neurons remain in a state of partial or sub-threshold excitement. These groups of neurons enter a facilitated state when they receive input from a certain stimulus, which may be somatic in nature. Inflammation and tension at a joint can cause sensitization of a neuronal segment, resulting in facilitation of the segment. Over time, the facilitated segment can deliver hyperstimulated output to the region of the joint, resulting in tissue texture changes, restrictions, and tenderness, all of which will worsen the patient's condition. The sympathetic innervation for the upper extremities stems from T2-T8, while the lower extremities are innervated by T11-L2. These regions should be evaluated for tissue texture changes by the osteopathic physician. Physicians may choose to use myofascial release, articulation, and counterstrain techniques to normalize sympathetics and reverse facilitation to improve healing.[25]

SUMMARY

In order to accurately diagnose a complaint of polyarthritis, the physician must employ an extensive history and physical to help narrow the broad differential. If there is a history of trauma or the patient presents with localized bone pain, appropriate imaging should be done to rule out fracture or a tumor. If synovitis is present for greater than 6 weeks, a systemic rheumatic disease must be ruled out through further testing (ESR, RF, ANA). If the synovitis is present for less than 6 weeks, the patient will require close follow up, as this may be a sign of early rheumatic disease or perhaps a viral arthritis. Patients found to have effusion often require joint aspiration for further evaluation. The contents of the aspiration can point to a likely diagnosis, and further testing may be required for confirmation. If there is no effusion or signs of inflammation at the joint site, the physician should evaluate whether there are trigger points or point tenderness. The presence of these focal findings can point to bursitis, tendinitis, or fibromyalgia. Lack of point tenderness may indicate osteoarthritis or a soft tissue injury.[1]

Primary care physicians are often the first clinicians to evaluate patients suffering from polyarthritis, and they are a crucial part in identifying patients who require prompt treatment to ensure the best possible outcomes.

AUTHOR DISCLOSURES:
No relevant financial affiliations.

REFERENCES:

1. Guidelines for the Initial Evaluation of the Adult Patient with Acute Musculoskeletal Symptoms. Am College of Rheumatology Ad Hoc Committee on Clinical Guidelines. Arthritis Rheumatol. 1996;39(1):1-8. http://onlinelibrary.wiley.com/doi/10.1002/art.1780390102/pdf
2. Meador R, Schumacher HR. Evaluating and Treating Patients with Polyarthritis of Recent Onset. Hosp Physician. 2003;39(3):37-45. http://turner-white.com/pdf/hp_mar03_recent.pdf
3. Mies Richie A, Francis ML. Diagnostic Approach to Polyarticular Joint Pain. Am Fam Physician. 2003;68(6):1151-1160. https://pdfs.semanticscholar.org/44dc/31ab2f0cffa1ffd372d5faefb476006fe8d8.pdf
4. Pujalte GA, Albano-Aluquin SA. Differential Diagnosis of Polyarticular Arthritis. Am Fam Physician. 2015;92(1):35-41. https://www.aafp.org/afp/2015/0701/p35.html
5. Majithia V, Geraci SA. Rheumatoid Arthritis: Diagnosis and Management. Am J Med. 2007;120:936-939.
6. El-Gabalawy HS, Duray P, Goldbach-Mansky R. Evaluating Patients with Arthritis of Recent Onset: Studies in Pathogenesis and Prognosis. JAMA. 2000;284(18):2368-73.
7. Sangha O. Epidemiology of Rheumatic Diseases. http://ow.ly/soBG30lCxtd
8. Pinals RS. Polyarthritis and Fever. N Engl J Med. 1994;330(11):769-74.
9. Steere AC. Musculoskeletal Manifestations of Lyme Disease. Am J Med. 1995;98(4A):44S-51S.
10. Woolf AD, Campion GV, Chishick A, Wise S, Cohen BJ, Klouda PT, Caul O, Dieppe PA. Clinical Manifestations of Human Parvovirus B19 in Adults. Arch Intern Med. 1989;149(5):1153-56
11. Oğuz F, Akdeniz C, Ünüvar E, et al. Parvovirus B19 in the acute arthropathies and juvenile rheumatoid arthritis. J. Paediatr. Child Health. 2002;38:358-362.
12. Barth WF. Office Evaluation of the Patient with Musculoskeletal Complaints. Am J Med. 1997;102(1A):3S-10S.
13. Salomon J, Szepietowski JC, Proniewicz A. Psoriatic Nails: A Prospective Clinical Study. J Cutan Med Surg. 2003;7(4):317-21. http://journals.sagepub.com/doi/abs/10.1007/s10227-002-0143-0?url_ver=Z39.88-2003&rfr_id=ori%3Arid%3Acrossref.org&rfr_dat=cr_pub%3Dpubmed&
14. McGonagle D, Gibbon W, Emery P. Classification of Inflammatory Arthritis by Enthesitis. Lancet. 1998;352(9134):1137-1140.
15. Schumacher HR. Arthritis of Recent Onset. Postgrad Med. 1995;97(4):52-54, 57-59, 63.
16. Sabella C, Goldfarb J. Parvovirus B19 Infections. Am Fam Physician. 1999;60(5):1455-1460. https://www.aafp.org/afp/1999/1001/p1455.html
17. Shmerling RH, Delbanco TL. How Useful is the Rheumatoid Factor? Arch Intern Med. 1992;152(12):2417-20.
18. Agudelo CA, Wise CM. Gout: Diagnosis, Pathogenesis, and Clinical Manifestations. Curr Opin Rheumatol. 2001;13(3):234-239.
19. Shmerling RH, Delbanco TL, Tosteson AN, et al. Synovial fluid tests. What should be ordered? JAMA. 1990;264(8):1009-14.
20. Renfrew, DL. Polyarthritis, Musculoskeletal Masses, and Osteoporosis. Symptom Based Radiology. Neenah, WI: Radiology Associates of the Fox Valley; 2014. http://www.symptombasedradiology.com/TableContents/PDFs/SBR%20Chapters/SBR%2013.pdf. Accessed November 12th, 2017.

21. Fife RS, Brandt KD, Braunstein EM, et al. Relationship Between Arthroscopic Evidence of Cartilage Damage and Radiographic Evidence of Joint Space Narrowing in Early Osteoarthritis of the Knee. Arthritis Rheum. 1991;34(4):377-382. http://onlinelibrary.wiley.com/doi/10.1002/art.1780340402/pdf

22. Baillet A, Gaujoux-Viala C, Mouterde G, et al. Comparison of the Efficacy of Sonography, Magnetic Resonance Imaging and Conventional Radiography for the Detection of Bone Erosions in Rheumatoid Arthritis Patients: a Systematic Review and Meta-analysis. Rheumatology (Oxford). 2011;50(6):1137-1147. https://academic.oup.com/rheumatology/article/50/6/1137/1786432

23. Mandl P, Navarro-Compán V, Terslev L, et al. EULAR Recommendations for the Use of Imaging in the Diagnosis and Management of Spondyloarthritis in Clinical Practice. Ann Rheum Dis. 2015;74(7):1327-1339. http://ard.bmj.com/content/74/7/1327

24. Gamber RG, Shores JH, Russo DP, et al. Osteopathic manipulative treatment in conjunction with medication relieves pain associated with fibromyalgia syndrome: Results of a randomized clinical pilot project. J Am Osteopath Assoc. 2002;102(6):321-5. http://jaoa.org/article.aspx?articleid=2092685

25. Savarese RG, Capobianco JD, Cox JJ. Facilitation. OMT Review: A Comprehensive Review in Osteopathic Medicine. 3rd edition. United States. 2009.

REVIEW ARTICLE

Current Concepts in the Office-Based Treatment of the Concussed Athlete

Robert Franks DO, FAOASM, Danielle Chase, PhD & Ronald Torrance II, DO

KEYWORDS:

Sport-Related Concussion

Head Injuries

Neuropsychological Testing

Vestibular Therapy

Neuro-Optometry

Context: Concussion has emerged as one of the most challenging conditions in medicine. With the currently changing and evolving diagnosis and treatment paradigms in concussion management, a comprehensive review of the current literature was necessary. This article focuses on the in-office evaluation, diagnosis and treatment of the concussed athlete as opposed to on-field assessment. This article will guide the primary care physician through a logical evaluation and treatment process for this challenging injury.

Evidence Acquisition: A thorough and comprehensive review of the current literature was performed via PubMed, Medline, and Google Scholar.

Study Design: Clinical Review.

Level of Evidence: Level 5

Results: Upon review of the current medical and allied health literature, a systematic and step-wise approach to obtaining key historical points, physical examination, medical imaging, additional diagnostic testing, and treatment both medically and by allied health professionals was developed to give primary care physicians a structured protocol to follow in the diagnosis and treatment of concussion in the office-based setting.

Conclusion: This article gives primary care physicians a current and progressive approach to the diagnosis, evaluation, work-up, and treatment of the concussed athlete in the in-office setting concerning concussion management.

INTRODUCTION

Sports Related Concussion (SRC) has emerged as one of the most challenging conditions in medicine. Continued media coverage both in the news and entertainment has created much confusion amongst athletes about treatment options and future consequences regarding this injury. Thus, SRC has continued to be a challenging condition to diagnose and treat, and athletes often present with several of the following questions when seeking treatment:

- Is this really SRC?
- Are the symptoms that I am having normal for SRC?
- Is rest still best?
- Are there treatments now for SRC?
- How long will it take me to recover?
- Can I go to school and/or work?
- When can I return to sports?

The goals of the treating physician are to determine the presence or absence of SRC, reassure the patient that his/her symptoms are consistent with SRC if diagnosed, and provide the patient with an individualized treatment plan to provide for optimal recovery.

Further, the physician should aim to prevent Second Impact and Post Concussion Syndrome, mitigate symptoms, decrease symptom duration, determine if the athlete is safe to return to learn, and if appropriate, play, and reduce the possibility of cumulative effects of SRC including its controversial association with Chronic Traumatic Encephalopathy.

As SRC is continually evolving in terms of nomenclature, diagnosis, and treatment, ongoing review of the current concepts of SRC is essential not only for sports medicine physicians, but for all practitioners. Only by continually reviewing the literature and individualizing treatment to the symptoms of the athlete, will the physician be successful in determining if and when the athlete can be safely returned to sport.

CORRESPONDENCE:

Robert Franks DO, FAOASM | r.robert.franks@rothmaninstitute.com

 Print ISSN: 1877-573X

CLINICAL RECOMMENDATION	EVIDENCE RATING	REFERENCES
Medical imaging yields no information in concussion management unless to determine if structural brain injury has occurred	A	3
There is no currently endorsed laboratory test or biomarker by the CISG	A	3
VOMS is significantly helpful in discerning vestibulo-ocular symptomatology in association with concussion	A	6
Vision therapy may be a helpful modality to use in concussion management.	B	20,21
Rest should be the mainstay of continued care in the concussed athlete	C	3
There is limited data to support the use of medications in concussion management except for symptom control.	B	1,3,4,8
Neuropsychological testing and treatment secondary to is a definitive assistant in concussion management	B	3
Cognitive behavioral therapy is emerging as an adjunct to treatment in the concussed patient with associated mood symptomatology	B	22

BACKGROUND:

SRC is the most common head injury in athletics, with less that 10 percent resulting in loss of consciousness.[1] An estimated 1.1 to 1.9 million sport and recreationally related concussions occur in the United States every year in those under age 18.[2] Concussion occurs most frequently in collision and contact sports, and can be caused by a head to head collision between athletes, or by a blow to the head from the ground or other objects.[1] Impact forces to the chest, back, neck, or face that radiate to the head may also cause a concussion.

The basic definition of SRC, developed by the Concussion in Sport Group (CISG) and released in the Spring 2017 Consensus Statement on Concussion in Sport – the 5th International Conference on Concussion in Sport Berlin 2016, is as follows:

"SRC is a traumatic brain injury induced by biomechanical forces. Several common features that may be utilized in clinically defining the nature of a concussive head injury include:

- SRC may be caused either by a direct blow to the head, face, neck or elsewhere on the body with an impulsive force transmitted to the head.
- SRC typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously. However, in some cases, signs and symptoms evolve over a number of minutes to hours.
- SRC may result in neuropathological changes, but the acute clinical signs and symptoms largely reflect a functional disturbance rather than a structural injury and, as such, no abnormality is seen on standard structural neuroimaging studies.
- SRC results in a range of clinical signs and symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive features typically follows a sequential course. However, in some cases symptoms may be prolonged.

The clinical signs and symptoms cannot be explained by drug, alcohol, or medication use, other injuries, or other comorbidities."[3]

The gold standard, which has remained in place since the first CISG in 2004, is that if there is any doubt SRC has occurred, the athlete should be removed from the competition immediately and seen by a practitioner with experience in SRC evaluation and management within the following 48-72 hours for a more thorough examination.[4]

MEDICAL MANAGEMENT:

Management of SRC, per the 2016 CISG Consensus Statement, consists of the 11 Rs of SRC – Recognize, Remove, Re-evaluate, Rest, Rehabilitation, Refer, Recover, Return to sport, Reconsider, Residual effects and sequelae, and Risk reduction.[3]

In the office setting, assessment often begins before the athlete enters the examination room. As the athlete enters a new environment, the reaction of his/her vestibular-ocular system to this new environment often yields clinical information that is observable prior to obtaining a history or physical examination.

Athletes are encouraged to bring with them a family member or second historian as well as any information that may help in the evaluation of the current concussive episode, including:

- Any radiological studies such as CT Scan or MRI
- Any computerized neurocognitive testing, including both baseline and post-testing (i.e. IMPACT, etc.)
- The Sport Concussion Assessment Tool (SCAT5) and Balance Error Scoring System (BESS) scores
- The Pre-participation Physical Examination Exam (PPE)

These materials and historians are helpful as the athlete often cannot recall his/her history due to the injury and parents/guardians are often challenged in recalling an accurate history due to the complexity of SRC and the concussion office visit.

HISTORY:

A detailed history is the most important component of the athlete encounter. Questions that should be asked during the encounter:

1. How many head injuries has the athlete had in the past?
   - When did they occur?
   - How did they occur?
   - What type of symptoms did the athlete have?
   - How long did the symptoms last?
   - Did they lose consciousness?
   - Did they have amnesia (anterograde or retrograde)?
2. Are the current symptoms and injury associated with loss of consciousness or amnesia, and what type?
3. Did the athlete report on-field dizziness?
4. Does the athlete have a history of motion sickness?
5. What kind of headache does the athlete have and does it worsen with exertion or schoolwork?
6. Does the athlete get dizzy with movement?
7. Does the athlete get fatigued at the end or a certain point in the day?
8. Is the athlete more sensitive to light or noise?
9. Is the athlete more distracted?
10. Does the athlete have trouble falling asleep or staying asleep?
11. Is the athlete moody or irritable?
12. Does the athlete feel “foggy” or “removed”?
13. How many practices or games has the athlete missed?
14. How many school days or tests has the athlete missed?
15. Has the athlete’s symptoms affected his/her classes or grades?
16. How long did it take the athlete to recover from past concussions?
17. Did the athlete have incidents/symptoms from any hits to chest, neck or face that radiated to the head (e.g., whiplash) that were not reported as a concussion?

The answers to the above history questions can mimic other sports medicine conditions seen in competition, such as heat illness or dehydration. Categorization of symptoms often makes it easier to correlate certain aspects of physical examination findings to establish treatment. The strongest and most consistent predictor of slower recovery from SRC is severity of the patient’s initial symptoms on the first day, or initial few days, after injury.[3] Patients with co-morbid migraine headaches and mood disorders are often at risk for protracted recovery. While AD/HD and other learning disabilities require more careful evaluation and follow-up, they do not seem to be solely responsible for protracted recovery.[3]

While several classification systems have been used in the past, more recently the University of Pittsburgh Medical Center (UPMC) Classification System offered categorization of concussion symptoms into six different categories, which can occur in isolation or in any combination, and include cervicogenic, vestibular, ocular, post-traumatic migraine, cognitive/fatigue, and anxiety/mood.[5]

For the purposes of this paper, a modified version of the UPMC Classification System containing the following categories is reviewed:

1. Cervicogenic
   - Cervical Strain
   - Tension Headache
2. Cognitive
   - Attention
   - Fogginess
   - Fatigue
   - Cognitive slowing
3. Emotionality
   - Depression
   - Anxiety
   - Sadness
   - Nervous
   - Irritable

4. Sleep Disturbance
    - Difficulty falling asleep
    - Inability to remain asleep
    - Sleeping more than usual
    - Sleeping less than usual

5. Vestibular
    - Dysfunction in balance and coordination

6. Ocular
    - Inability of the ocular system to work appropriately
        - » Difficulty with convergence
        - » Difficulty with divergence
        - » Difficulty with smooth pursuits
        - » Difficulty with saccadic eye movement
        - » Difficulty with accommodation
        - » Difficulty with VOR (Vestibulo-ocular reflex) or VMS (Visual Motion Sensitivity)

PHYSICAL EXAM:

A complete physical examination is an essential component of the in-office evaluation. This exam begins with a detailed neurological examination assessing vital signs, analyses of speech and gait, cranial nerve testing, visual field testing, upper extremity and lower extremity sensation, range of motion and strength, deep tendon reflexes, Romberg, Pronator Drift, Tandem Walk, Heel to Shin, Finger to Nose Testing, and the Vestibular-Ocular Motor Screen.

The Vestibular/Ocular Motor Screen (VOMS) is one of the more valuable resources in evaluation, diagnosis, and treatment of concussion. Given the importance of the vestibular-ocular system to the recovery of the concussed athlete, it is highly recommended to include VOMS testing as part of the standard physical examination.[6]

VOMS begins with examination of smooth pursuits, performed similarly to traditional extra ocular muscle testing. Smooth pursuits are examined horizontally and vertically with the examiner looking for non-physiological nystagmus, inability to track, or dizziness/nausea *(Image 1, page 36).*

Saccadic eye movement is then measured as the examiner stands away from the athlete's eyes, with his/her fingers 12 inches apart horizontally and then vertically on either side of the athlete's head. The athlete is instructed to look back and forth between the examiner's fingers for 15 seconds. The examiner is again looking for non-physiological nystagmus, latency of onset, speed, accuracy, and conjugate movement. Failure of the test occurs with delayed or inaccurate saccades, or disconjugate eye movement *(Images 2 and 3, page 36).*

Evaluation of the Vestibular-ocular reflex (VOR) is an examination of gaze stability. Testing is performed by observing the athlete while s/he stares at his/her own thumb while moving the head horizontally, and then vertically, without blurriness or dizziness. Similar to saccadic eye movements, VOR testing is performed for approximately 15 seconds. Test failure is characterized by the inability of the athlete to maintain focus/stabilize gaze on his/her thumb in either the vertical or the horizontal positions *(Image 4, page 36).*

The Visual Motion Sensitivity Test, (VMS) measures response to optokinetic activity. On this test, the athlete focuses on his/her thumb while moving the head and thumb in tandem, both horizontally and then vertically, while maintaining focus on the thumb for 15 seconds. Failure of this test is characterized by the athlete's inability to follow the "fixed" object, i.e., his/her thumb *(Images 5 and 6, page 36).*

Near point of convergence (NPC) is the point nearest to the bridge of the athlete's nose at which s/he is able to maintain binocular fusion while focusing on a target moving inward at approximately 1 to 2 seconds/centimeter. While the research is mixed, distances of NPC in children greater than 6 cm, and NPC in adults greater than 8 cm, are considered a failed examination[7] *(Image 7, page 36).*

If at any time during these tests the athlete has a return of concussive symptoms, this is also a failed test.

In addition to ocular testing, balance testing is recommended as part of the overall physical examination. Balance testing can be performed in a variety of different ways. Commercially, there are applications available that offer clinicians a modality from which to measure balance from their mobile devices. There are also several commercially available force-plate technology apparatuses that can measure an athlete's balance. The Balance Error Scoring System (BESS) is a simple test that can be performed without technology, is part of the Sport Concussion Assessment Tool 5th Edition (SCAT5), and measures the athlete's balance in three positions - double leg, single leg, and tandem stances - on first a firm and then foam surface with the eyes closed. Athletes lose points on the test for each error.[3,4,6,8,9] Due to subjective differences between test administrators, BESS testing results should be approached with caution.

IMAGING

The CISG has recognized that neuroimaging is usually normal and contributes little to evaluating concussion. Neuroimaging is used primarily when there has been prolonged disturbed consciousness, when the athlete presents with focal neurological deficits or persistent cognitive symptoms, or when there is suspicion of a structural abnormality, cerebral bleed, or seizure activity. Computed Tomography (CT) is used to rule out hemorrhages and fractures. Magnetic Resonance Imaging (MRI) is used to assess structural and functional abnormalities, and is being used more often as there is no radiation exposure. The weighted images produced by diffusion-tensor imaging (DTI) often can elucidate structural brain abnormalities in protracted cases or post-concussion syndrome.[10] Functional MRI (fMRI), which is typically performed while the athlete simultaneously completes a cognitive task, has been used more extensively in concussion research but is not yet standard of clinical care.[1,3,4,8,9]

IMAGE 1:

IMAGE 2:

IMAGE 3:

IMAGE 4:

IMAGE 5:

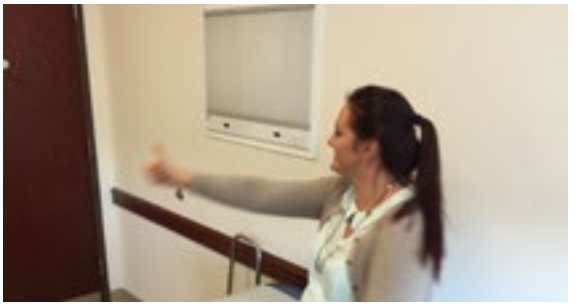


IMAGE 6:

IMAGE 7:

Image interpretation:
Image One – Smooth Pursuits
Image Two, Three – Saccades
Image Four – Vestibular-Ocular Reflex Test
Image Five, Six – Visual Motion Sensitivity Test
Image Seven - Near Point of Convergence Test

LABORATORY TESTING:

There currently are no definitive fluid biomarkers or genetic testing used in the evaluation of SRC.[3] Though the CISG has endorsed no fluid biomarkers or genetic testing in the evaluation of SRC, the FDA has just endorsed the first laboratory blood test used to evaluate mTBI/concussion.[11]

POST CONCUSSION TREATMENT:

Patient Education

Symptomatic athletes should be immediately removed from play.[3] In the immediately concussed athlete, first-line treatment in concussion management should be complete mental and physical rest for no more than the first 24 - 48 hours, including no physical education or sports. Use of electronics should be eliminated or significantly decreased until the patient is asymptomatic with use, and then the patient should be encouraged to engage in progressive cognitive and physical activity below the threshold of symptom exacerbation.[3] Should symptoms reemerge with use, the patient should be encouraged to back off of electronics until symptom free, and then begin to reengage until symptomatic, and then continue in this cycle in an attempt to elongate his/her ability to engage in the use of electronics while symptom free, i.e., "push and recover, push and recover."

Immediately, and through the course of recovery, diet should consist of three meals a day, including encouragement of a protein diet, with adequate and increased hydration of at least 80 ounces per day. Mild exercise such as walking, stretching, or moderate yoga should be encouraged. Sleep should be 7 to 9 hours, without napping, and no electronic devices should be used in the bedroom at bedtime.[5] The athlete should also be counseled at this time on the necessity of an individualized treatment paradigm to lessen symptoms, decrease duration of concussion, prevent possible long term complications of concussion based on the individual patient's case, and to explain to the athlete the requirements of when s/he can return to learn, and if appropriate, return to play.

Treatment is often executed by a multi-disciplined team

Vestibular Therapy

Vestibular therapy is a cornerstone of concussion treatment targeting dizziness, vertigo, balance, vision, and visual discrimination associated with concussion. Vestibular therapy uses current vestibular, physical, and occupational therapy maneuvers. There are five main categories of the exercises done in vestibular therapy through which the athlete progresses given sequential successes.[10]

1. Coordination of the eyes and head.
    - Exercises target rehabilitation of deficits found on examination of VOR, VMS, smooth pursuits, anticipatory gaze shifts, image targeting, vergence, accommodation, and/or saccadic eye movements.
2. Sitting balance exercises.
    - These can include maintaining balance while sitting upright, weight shifting side-to-side, or bouncing.
3. Standing static balance exercises.
    - These can include the athlete standing in place while upright or weight shifting. Athletes can also be asked to stand on one leg, stand on the rocker board, or stand with one foot on a step. This category includes sit to stand exercises.
4. Standing dynamic balance exercises.
    - These can include the athlete standing and moving without walking. The athlete may walk in place, step forward, step backward, step side to side, step up and down, and/or turn around.
5. Ambulation exercises.
    - In these exercises the athlete moves forward, backward, on stairs, skips, jogs, or runs. The modifiers of these exercises include posture, surface, size of base support, position of arms, position of trunk, direction of head movements, direction of whole body movements, visual input, presence or absence of a cognitive task, and special circumstances.

Vestibular exercises are recorded in frequency and duration

Vestibular therapists often use true physical therapy modalities for comorbid cervical or peri-scapular strain and/or tension headache. Treatment may include manual therapy, range of motion exercises, and strength training.

Osteopathic physicians are specifically trained in osteopathic manipulative treatment and injection therapy which can assist in the treatment of concussed athletes. After a thorough structural examination, osteopathic manipulative techniques that can be considered include gentle range of motion (ROM) exercises, stretching exercises, myofascial release, and muscle energy.

Exertion therapy is being used more frequently in concussion management. In addition to decreasing deconditioning, it can be helpful in distinguishing anxiety and/or depression symptoms from those truly associated with concussion. Symptoms that peak with exertion, yet diminish with cessation of activity and return to rest are often related to SRC, while those that remain once exertion has ceased and rest has been restored, often can be related to associated anxiety. Exertion typically begins with static exercises, followed by exercises challenging the vestibulo-ocular system. Judgment of the athlete's exertional tolerance allows the therapist to progress the athlete through a continuum, typically from the exercise bicycle, to the elliptical, the treadmill, resistance training, and finally to sport specific activity.

NEUROPSYCHOLOGICAL ASSESSMENT/CONSULTATION:

Baseline and post-injury neuropsychological (NP) assessment was not considered as a requirement by the CISG; however, it often yields useful information.[3] NP is multifaceted, and typically consists of either short, often computerized, screeners, or more formal, comprehensive, pencil and paper neuropsychological batteries. Computerized NP tests, e.g. ImPACT (Immediate Post-Concussion Assessment and Cognitive Testing), can be administered by a wide variety of clinicians and offers snapshots of an athlete's cognition that are easily replicable in short periods of time given acceptable practice of effects.[4,12] Furthermore, it has been demonstrated that the addition of NP testing to symptom self-report forms "adds value" to the clinician's ability to diagnose SRC correctly, in those with true SRC.[13]

While computerized measures are more easily applicable for baseline testing, when an athlete's recovery becomes protracted, the use of a comprehensive NP battery, administered and interpreted by a neuropsychologist trained to evaluate subtle neurocognitive deficits, is often recommended by the treating physician. 4 When NP assessment is deemed medically necessary, the medical necessity for the inclusion of measures assessing effort and performance validity is implied.[14] Measurement of an athlete's effort on NP testing is critical to determining validity of test results and subsequently offering information to the physician in making RTP decision.[15]

It has been commonly accepted that in order to return to play more quickly, athletes always will provide maximum effort on NP testing. However, as effort relates to the sports medicine population, it was demonstrated that over three comprehensive NP testing sessions, approximately 28% of this athletic population was sub-optimally engaged for at least one session, and there is a subgroup of young athletes who were disengaged in testing over the course of the entire three-session NP evaluation, regardless of gender, history of previous concussion, psychiatric or learning diagnoses, or use of prescription medication.[16] While these data refer to comprehensive neuropsychological testing, administered by a doctoral level neuropsychologist, research has demonstrated that athletes may exert suboptimal effort on baseline computerized neuropsychological testing, in the presence of sports medicine physicians and related clinicians, as well. It has been demonstrated that within ImPACT, review of failure to identify distractors increases the clinician's ability to detect malingering when used with the already embedded validity scales.[17]

COGNITIVE REHABILITATION THERAPY:

Following NP assessment, Cognitive Rehabilitation Therapy (CRT) may be recommended to support the athlete in his/her area(s) of deficit, which may include NP domains such as memory, attention, executive functioning, and/or processing speed, etc. Therapy may be with a cognitive rehabilitation specialist working on specific skill set training and strategies for learning, and/or home-based, skill specific computer programs. Strategies targeted in CRT may be incorporated into the athlete's 504 Accommodations Plan or Individualized Education Program (IEP). Research demonstrates that while there has been efficacy of CRT following concussive injuries, successful rehabilitation may be mediated by type of injury, age, and timing of intervention.[18,19]

NEURO OPTOMETRIC CONSULTATION:

Disturbances to the neuro-optometric system are one of the most common and challenging sequelae of SRC. These changes can include problems with convergence, divergence, smooth pursuits, saccades, accommodation, VOR, and VMS. Collectively, disturbances to the visual system secondary to concussion are known as Post Trauma Vision Syndrome (PTVS). Symptoms resulting from PTVS include: convergence insufficiency, high exophoria or exotropia, accommodative dysfunction, low blink rate, difficulties in attention/concentration, ocular motor difficulties, and visuospatial distortions with associated neuromotor affects.[20] Physicians may need to consider curtailing driving in patients with PTVS *(Table 1).*

While vestibular therapy often incorporates treatment of the neuro-optometric system, continued symptomatology within the neuro-optometric system often necessitates referral to a neuro-optometrist.

OPTOMETRIC VISUAL DEVICES/VISION THERAPY:

The three primary optometric treatments include in-office therapy, in-home therapy, and lenses, with in-office and in-home therapies often used in tandem. Therapies target vergence, accommodation, tracking, visual memory, and/or visual attention, and must be balanced with the academic, homework, and therapy load of the athlete as they often are a trigger for headache in the symptomatic athlete.

Glasses are often used temporarily with concussed athletes, and may include corrective lenses, Irlen lenses, lenses with an anti-refractive coating, or prisms.[21] Intra-office vision therapy and home-based therapies typically include active treatments such as "pencil-push ups," therapy with base-in prisms, computer programs, stereoscopes, and free-space fusion cards. Though research is mixed, a combination of in-home and office-based therapies has yielded the most efficacy.[7]

PSYCHIATRIC/PSYCHOLOGICAL CONSULTATION:

Psychiatric and/or psychological consult has definitive application in SRC management. Early detection of athletes with either premorbid, emerging, or comorbid anxiety/depressive symptoms is critical to successful SRC management. There is increasing evidence that athletes with post concussive symptoms respond well to Cognitive Behavioral Therapy;[22] however, the training and type of clinician is critical to the possible success of the intervention. Depression is considered the paramount psychiatric condition for serious complications related to the treatment and recovery, as many symptoms of major depression mirror those of SRC (e.g. difficulties with attention, concentration, cognition, fatigue, irritability, headaches, relational, and occupational and academic problems, etc....)[23]

Athletes with significant psychiatric symptomatology may require consultation with a psychiatrist regarding pharmacological intervention. Given the potential side effects of psychiatric medications, particularly the risks of suicidal ideation in the adolescent population with the use of SSRIs, psychiatrists are an integral part of the SRC team. Oftentimes, pharmacological and non-pharmacological approaches are utilized simultaneously.[24]

COGNITIVE BEHAVIORAL THERAPY

Cognitive behavior therapy (CBT) is often useful for those athletes who demonstrate true psychopathology and related symptoms. When considering CBT for an athlete, referral to a doctoral level psychiatrist or psychologist should be considered, as the specific recommendation for CBT is typically provided by a trained psychiatric practitioner, following formal neuropsychological testing or psychiatric examination. Current evidence exists that

TABLE 1:

Common Neuro-Optometric Conditions and Their Effects on Concussion

CONDITION	PHYSIOLOGY	MANIFESTATION
Convergence insufficiency	Insufficient ability to use eyes as a "team" to converge/lock on a target	Difficulties with reading
Ocular motor dysfunction	Inability of the eyes to work together to track a moving target and switch fixation points	Accurate visual scanning and exploration; reading or copying from a smartboard
Accommodation Infacility	Inability to maintain clarity while shifting attention/focus from one distance to another rapidly and accurately	Maintenance of focus at reading distance; academic efficiency; comfort focusing on an object
Visual intake-visual memory	The inability to obtain maximum visual information in the shortest possible time and retain this information over an adequate period of time	Reading comprehension and spelling
Visual-motor integration	The inability to integrate visual input with motor output.	Copying notes from a whiteboard or projector screen, or transferring answers to test papers similar to Scantron formats
Fusional Dysfunction	Binocular dysfunction affecting near and distant visual tasks	Blur and/or double vision

CBT may be an effective treatment for post-concussion syndrome, and there is limited evidence that rehabilitation programs that include a psychotherapeutic/mindfulness and/or relaxation element are beneficial for persisting symptoms.[22] In contrast with previously held beliefs, dispersion of information, education, and reassurance alone may not be as convincingly efficacious as active treatments.[22] Furthermore, research does not support the use of educational materials alone to affect behavior change.[25] Therapy can be done in an outpatient or school setting, and should be coordinated with the athlete's 504 Plan or IEP.

MEDICATIONS

There is limited data to support the use of medications in SRC.[3] Medications are not recommended in the first 10 to 14 days of SRC management as athletes typically recover on their own,[1,3,4,8] and if considered, they should be prescribed by experienced SRC clinicians, and are used to control specific symptoms, modify the underlying pathophysiology of SRC, or to shorten symptom duration. Non-steroidal anti-inflammatory medications (NSAIDs) may cause rebound headache; however, they may be considered in concurrent cases of cervical or peri-scapular strain, or tension headache. Use of acetaminophen is considered a better choice than NSAIDs; however, it also can cause rebound headache. Tramadol or tramadol/acetaminophen can manage headaches in those athletes over 18 with no comorbid seizure history. Muscle relaxants are helpful for cervicogenic headache. Data is mixed on the efficacy of "vitamin therapy" for headache control. Vitamins B2, Omega 3 Fatty Acids, magnesium, and coenzyme Q often help with migraine and have been successful in some concussed athletes. True post-traumatic migraine (headache associated with nausea and photophobia and/or phonophobia) can be treated with propranolol, verapamil, amitriptyline, nortriptyline, or the triptans. The social/emotional symptoms of SRC are often treated with selective serotonin reuptake inhibitors (SSRIs) or lorazepam. Sertraline is often successful in the treatment of the psychological effects of SRC; however, escitalopram is commonly prescribed. Cognitive and attention symptoms are often treated with stimulant medications, and methylphenidate or atomoxetine are often used in the SRC population. Amantadine, an antiviral drug, has been shown to assist with cognition.[26] Clonazepam is often used in patients with significant vestibular symptoms often accompanied by anxiety. Insomnia is often treated with melatonin, diphenhydramine, hydroxyzine, trazodone, and more rarely, zolpidem.

NEUROLOGY CONSULTATION:

Consultation with Neurology is considered in certain situations. Neurology co-management is helpful in situations where the concussed athlete is dealing with true neurological co-morbidities. These situations would include:

1. Abnormal medical imaging
2. Pre/Co-morbid headache syndromes
3. Pre/Co-morbid Attention-Deficit/Hyperactivity Disorder (AD/HD)
4. Pre/Co-morbid neurological conditions
5. Any newly diagnosed or suspected neurological conditions

CURRENT CONCEPTS IN EDUCATION

As SRC is evolving as one of the most challenging conditions in medicine, effectively managing Return to Learn (RTL) is evolving as one of the most challenging aspects in education. While there are standardized Return to Play (RTP) protocol, current RTL protocol continue to evolve – though are consistently of a stepwise approach.[27]

Return to Learn:

Further complicating effective RTL, pre-existing AD/HD, learning disabilities, depression, and/or anxiety may worsen symptoms and protract recovery[28] *(Table 4, page 42).*

Practitioners may facilitate recovery by allowing a brief absence from school initially, as overtaxing cognition may worsen symptoms and possibly prolong recovery.[29] However, students typically return to school while symptomatic, as a primary goal of SRC management is to keep disruptions to the student's life to a minimum. School based accommodations target balancing the level of the athlete's academic workload with the progression of the athlete's cognitive recovery, and are monitored carefully as academic difficulties post-injury impact symptom report, and may also be associated with the emersion of anxiety and/or depression.[27,29]

Medical and education providers must work reciprocally to communicate the academic expectations and accommodations necessary to support a gradual RTL process.[30] Providing symptomatic students with subject-specific supports during the post-injury recovery period is necessary, particularly as students progress through more demanding phases of academia.[27] There is a general consensus that communication between the athlete and his/her caregivers and medical and educations teams is critical to maximizing recovery outcomes.[30]

Post concussion, athletes often experience academically based difficulties related to physicality, cognition, and emotionality, all of which may increase with schoolwork, computer work, and testing.[29] *(Tables 2 and 3)*

Return to Play:

Athletes should be encouraged to engage in tolerated exercise activity during the course of recovery. Activity should begin with walking, and is often dictated by performance in exertion therapy. The decision for RTP to actual sport for athletes continues to be determined by the experience and knowledge of the attending or team physician. There continues to be no timeline or formula with which to return an athlete to normal game play. All RTP decisions should be individualized, [31] and factors that warrant further consideration of return to contact/collision sports include the following:[3]

1. Smaller impacts causing worsening symptoms or longer recovery with repetitive SRCs.
2. Extended symptom numbers, severity, or duration in athletes with repetitive SRC.
3. Extended time for formal or computerized neuropsychological testing to return to baseline level.
4. Increased frequency of repetitive SRCs or shortened periods between repetitive SRCs.
5. Age of athlete and number of SRCs sustained.
6. Role of pre-existing conditions and co-morbidities, and their influence on concussion symptoms/duration.
7. Impact of athlete's daily/regular medications on SRC.
8. Role of type of sport, rules of sport, and athlete's behavior in sport, related to risk of further SRC.

At the discretion of the attending or team physician, athletes should be considered to begin the RTP program when they have met the following criteria:

1. Symptom Free for 24 to 48 hours without the assistance of medication.[4]
2. 24 to 48 hours of executed normal activity, which for a student athlete in a full contact sport, would be full activity in school (if school is in session) without accommodations, and for a student in a noncontact sport, such as swimming, cross country, etc., would be full activity in school (if school is in session) with minimal accommodations. The goal being to return the athlete to the highest level of sport safely allowed in the subset of student athletes who have unremitting residual symptoms.
3. Though controversial, if computerized neuropsychological testing is used in management, and baseline testing was performed, there should be return of computerized neurocognitive testing to baseline; if no baseline was obtained, computerized neurocognitive testing should be at least average for age and sex matched peers.

Progression through the RTP protocol should proceed stepwise. If post concussive symptoms recur, the athlete should return to the previous asymptomatic level, and then begin progression again within 24 hours. The steps should proceed as follows in Table 5, (*page 42*): [3,8,9,10]

If the athlete is unable to complete any step of the RTP program, or cannot compete the program without symptoms, s/he should be referred back to the physician for reassessment.

While SRC has emerged as one of the most challenging conditions in medicine, today there are objective measures that have made in-office evaluation and management of SRC more manageable for the clinician. Immediate recognition of SRC and early intervention by the physician can decrease symptomatology, lessen duration of SRC, and prevent long term complications. While considering the modifying factors associated with SRC, the decision to return an athlete to sport continues to rely on the individual experience and practices of each attending or team physician. As every State, and the District of Columbia, now has a concussion law, knowledge of not only the diagnosis and treatment of SRC, but also the State and/or scholastic guidelines dictating return to learn and play, is imperative to every physician treating SRC. As these continue to be updated and revised, it is critical that any physician caring for concussed athletes remain updated on current changes to the literature in order to provide immediate, comprehensive, and individualized care to the athlete, as well as to allow for a more rapid return to learn, and if appropriate, return to play.

TABLE 2:

Common Academic Based Difficulties and Their Expressed Symptoms with Concussion and Suggested Accommodations

SYMPTOM	COMPLAINTS	DIFFICULTIES	ACCOMMODATIONS
Physical	Headaches, photo/phonophobia, Dizziness, imbalance, dissomnolence, exertional sensitivity	Irritability, gait, arousal, sleep, and concentration, which may worsen in loud and bright environments, or with over-activity	Rest breaks, sunglasses, preferential seating, environmental alternatives to lunchroom, assemblies, hallways, preferential class transition, later start time, shortened day, reduce cognitive or physical demands; complete work in small increments
Cognitive	Attention/Concentration, i.e. Short focus on lecture, classwork, homework Memory working / consolidation / retrieval Processing speed Cognitive fatigue	Holding instructions in mind, reading comprehension, math calculation and applications, writing, Retaining new information, accessing learned information Keep pace with work demand, process verbal information effectively Decreased arousal/activation, poor basic attention, working memory, and concentration Student may push through symptoms to prevent falling behind	Repetition, written instructions, note-taking, calculator, short reading passages, chunking, recognition cues Extended time, slow down verbal info, comprehension checking Rest breaks during classes, homework, and exams, workload reduction, alternate forms of testing
Emotional	Anxiety Depression/Withdrawal Irritability	Withdrawal from school/friends (stigma/activity restrictions) Poor stress tolerance, alienate peers/teachers	Engage student with friends at lunch/recess Reduce stimulation, stressors, and workload, rest breaks, shorter assignments, break down tasks[13]

TABLE 3:

Role of Members of the Academic Support Team in Concussion and Suggested Plan of Action

SCHOOL PERSONNEL	STUDENT NEEDS	PLAN
Certified Athletic Trainer and/or school nurse	The person of first contact who disseminates information and coordinates the treatment of the athlete with the educational and medical team	Often Liaison between family, coaches, academic and medical team.
Guidance Counselor	Short-term academic accommodations	Works with the student and medical team to develop a Section 504 Accommodations Plan
General Education Teacher	Translate plan to classroom	Working knowledge of Section 504 Accommodations related to concussion in the classroom
Child Study Team	Long-term educational intervention	Works with Guidance Counselor to transition 504 to an Individualized Education Program (IEP) – An IEP signifies that the student now requires a specialized education curriculum administered in an inclusion classroom by a special education teacher as opposed accommodations in a general education classroom with a regular education teacher
Special Education Teacher	Translate plan to classroom	Working knowledge of how to educate students with brain injuries in the classroom who require specialized education using a specialized curriculum

TABLE 4:

Return to Learn[3]

AIM	ACTIVITY	GOAL
Daily activities without symptoms	Reading, texting, screen time beginning with 5 to 15 minute intervals	Gradual return to typical activities
School activities	Homework, reading, or other cognitive activities	Increase tolerance to cognitive work
Return to school part time	Gradual reintroduction of schoolwork. May begin with half day with breaks if needed	Increased academic activities
Return to school full time	Gradually work to full school day	Return to full academic activities and perform makeup work

TABLE 5:

Return to Play[3]

STEP:	LEVEL:
1.	No activity with athlete at complete rest
2.	Light aerobic exercises (i.e. walking, swimming, stationary cycling). Goal is to increase heart rate to 70% of maximal effort. No resistance training.
3.	Sport specific training (i.e. skating drills for hockey, or running drills for soccer) Goal to add movement No head impact activities
4.	Noncontact drills progressing to more complex drills Goal is to add exercise, coordination, and cognitive load Progressive resistance training may begin at this step
5.	Full contact training after medical clearance Goal is to restore confidence and assess functional skills by coaching staff for return to gameplay

ACKNOWLEDGMENTS:
The authors would like to thank Jessica Kempa, ATC, and Kimberly Merlino for their contributions to this manuscript, which are greatly appreciated by the authors.

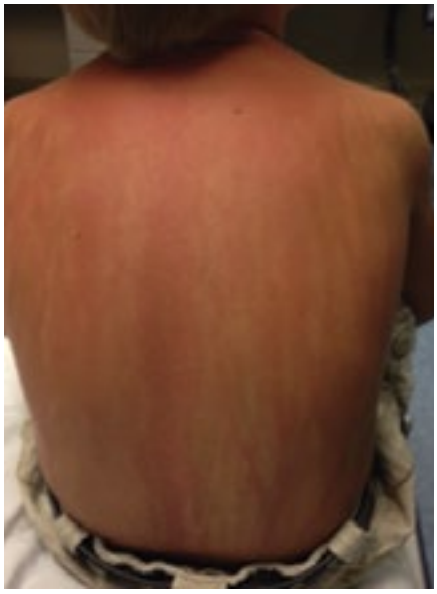
AUTHOR DISCLOSURES:
No relevant financial affiliations.

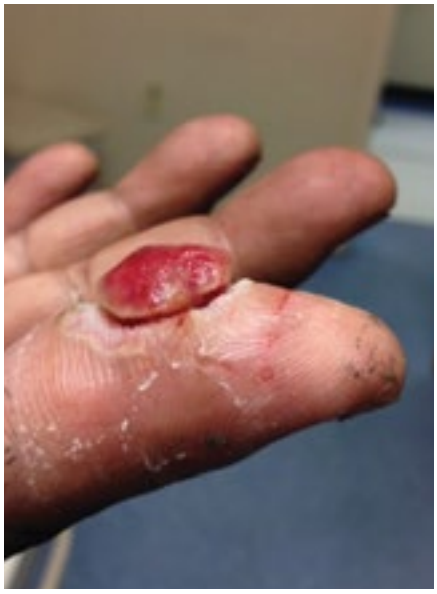
REFERENCES:

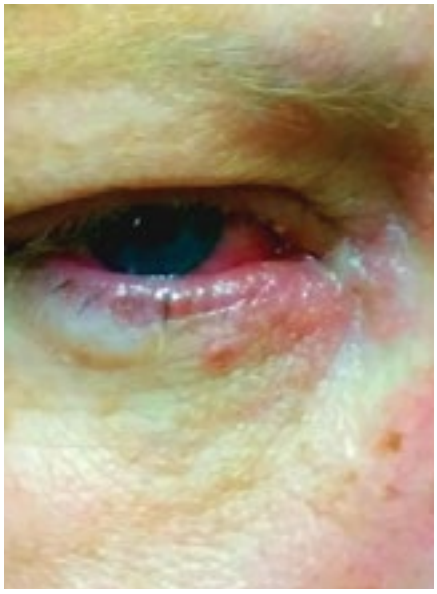
1. Johnson KM, McCrory P, Mohtadi NG, Meeuwisse W. Evidence Based Review of Sport-Related Concussion. Clinical Journal of Sport Medicine. 2001;11:150-9.
2. Bryan MA, Rowhani-Rahbar A, Comstock RD, Rivera F. Sports- and Recreation-Related Concussions in US Youth. Pediatrics. 2016 Jul;138(1) .
3. McCrory P, Meeuwisse W, Dvorak J, Bailes, J, Broglio S, Cantu Ret al. Consensus Statement on Concussion in Sport – the 5th International Conference on Concussion in Sport held Berlin, October 2016. Br J Sports Med 2017; 0-10.
4. McCrory P, Meeuwisse W, Aubry M, Cantu B, Dvorak J, Echemendia RJ, et al. Summary and Agreement Statement of the 4th International Conference on Concussion in Sport, Zurich 2012. Br J Sports Med. 2013 Feb;47:250-258.
5. Collins M, Kontos A, Maroon J, Fu F. Emerging Frontiers in Concussion: Advancements in Assessment, Management, and Rehabilitation; 2013 June 7-9; Pittsburg PA.
6. Mucha A, Collins MW, Elbin RJ, Furman JM, Troutman-Enseki C, DeWolf RM, et al. A Brief Vestibular/Ocular Motor Screening (VOMS) Assessment to Evaluate Concussions: Preliminary Findings. Am J Sports Med. 2014 Oct 26;42(10): 2479-86.
7. Cooper J, Jamal N. Convergence Insufficiency- a major review. Optometry Review. American Optometric Association. 2012.
8. McCrory P, Meeuwisse W, Johnston K, Dvorak J, Aubry M, Molloy M, et al. Summary and Agreement Statement of the 3rd International Conference on Concussion in Sport, Zurich 2008. Clinical Journal of Sports Medicine. 2009 May 19(3); 185-200.
9. Virji-Babul N, Borich M R, Makan N, Moore T, Frew K, Emery C A, et al. Diffusion tensor imaging of sports-related concussion in adolescents. Pediatric Neurology. 2013 June;48(1), 24-29.
10. Alsalaheen BA, Whitney SL, Mucha A, Morris LO, Furman JM, Sparto PJ. Exercise prescription patterns in patients treated with vestibular rehabilitation after concussion. Physiother Res Int. 2013 Jun; 18(2): 100-8.
11. Rabin, T. (2018). FDA authorizes marketing of first blood test to aid in the evaluation of concussion in adults: New quick testing option to help reduce need for CT scans, radiation exposure for patients. Available from the Federal Drug Administration at https://www.fda.gov/newsevents/newsroom/pressannouncements/ucm596531.htm.
12. Schatz P, Pardini JE, Lovell MR, Collins MW, Podell K. Sensitivity and specificity of the ImPACT test battery for concussion in athletes. Arch Clin Neuropsychol. 2006;21(1):91-99.
13. Van Kampen DA, Lovell MR, Pardini JE, Collins MW, Fu FH. The "value added" of neurocognitive testing after sports-related concussion. Am J Sports Med. 2006;34(10):1630-1635.
14. Bush SS, Ruff RM, Troster A, Barth J, Koffler SP, Pliskin NH, et al. NAN position paper: Symptom validity assessment: Practice issues and medical necessity. Archives of Clinical Neuropsychology. 2005.
15. Heilbronner RL, Sweet JJ, Morgan JE, Larrabee G J, Millis SR. American Academy of Clinical Neuropsychology Consensus Conference Statement on the Neuropsychological Assessment of Effort, Response Bias, and Malingering. Clin Neuropsychol. 2009 Sep;23(7):1093-129.
16. Chase D, Schatz P, Symk N, Franks RR (2018) The Stability of Engagement Over Comprehensive Neuropsychological Assessment in Student Athletes Diagnosed with Sports Related Concussion, Developmental Neuropsychology, 43:4, 345-355.
17. Schatz, P., Glatts, C. (2013). "Sandbagging" Baseline Test Performance on ImPACT, Without Detection, Is More Difficult than It Appears. Arch Clin Neuropsychol., May;28(3):236-44.
18. Rohling ML, Faust ME, Beverly B, Demakis G. Effectiveness of cognitive rehabilitation following acquired brain injury: a meta-analytic re-examination of Cicerone et al.'s (2000, 2005) systematic reviews. Neuropsychology. 2009 Jan; (1):20-39.
19. Dawson DR, Anderson ND, Binns MA, Bottari C, Damianakis T, Hunt A, Polatajko HJ, Zwarenstein M. Managing executive dysfunction following acquired brain injury and stroke using an ecologically valid rehabilitation approach: a study protocol for a randomized, controlled trial. Trials. 2013, 14:306.
20. Suter PS, Harvey LH. Vision Rehabilitation: Multidisciplinary Care of the Patient Following Brain Injury. 2011. P.9.
21. Doble JE, Feinberg DL, Rosner MS, Rosner AJ. Identification of Binocular Vision Dysfunction (Vertical Heterophoria) in Traumatic Brain Injury Patients and Effects of Individualized Prismatic Spectacle Lenses in the Treatment of Post concussive Symptoms: A Retrospective Analysis. PM&R 2010; 2 (4):244.
22. Al Sayegh A, Sandford D, Carson AJ. Psychological approaches to treatment of post concussion syndrome: a systematic review. Journal of Neurology, Neurosurgery and Psychiatry. 2010; 81 (10): 1128-34.
23. Iverson, G. Misdiagnosis of the persistent post concussion syndrome in patients with depression. Archives of Clinical Neuropsychology 2006;21: 303-310.
24. Vaishnavi S, Rao V, Fann JR. Neuropsychiatric problems after traumatic brain injury: unraveling the silent epidemic. Psychosomatics. 2009; 50(3): 198-205.
25. Mrazik, M., Dennison, C.R., Brooks, B.L., Yeates, K.O., Babul, S., Naidu, D. A qualitative review of sports concussion education: prime time for evidence-based knowledge translation. Br J Sports Med. 2015 August 25.
26. Reddy CC, Collins M, Lovell M, Kontos AP, Efficacy of amantadine treatment on symptoms and neurocognitive performance among adolescents following sports-related concussion. J Head Trauma Regabil. 2013 Jul-Aug;28(4):260-5.
27. Ransom DM, Vaughan CG, Pratson L, Sady MD, McGill CA, Gioia GA. Academic Effects of Concussion in Children and Adolescents. Pediatrics. 2015 May 11:135 (6). 1043-50.
28. Iverson GL, Atkins J, Zafonte R, Berkner P. Factors Influencing Post-Concussion-Like Symptom Reporting in Adolescent Athletes. 10th World Congress on Brain Injury, 2014 Mar 19-22. San Francisco, California.
29. Halsted ME, McAvoy, K, Devore, CD, Carl R, Lee, M, Logan, K, et. Al. Clinical Report: Returning to Learning Following a Concussion. Council on Sports Medicine and Fitness, And Council on School Health. Pediatrics. 2013 Oct 27; 132:5 948-957.
30. Gioa GA. Medical-School Partnership in Guiding Return to School Following Mild Traumatic Brain Injury in Youth. Journal of Child Neurology 1-16. Dec 2014.
31. Herring SA, Cantu RC, Guskiewicz KM, Putukian M, Kibler WB, et al. "Concussion (Mild Traumatic Brain Injury) and the Team Physician: A Consensus Statement -2011 Update. Medicine and Science in Sports and Exercise, 2011: 2412-2422.

NOW SEEKING

CLINICAL IMAGES







OSTEOPATHIC FAMILY PHYSICIAN

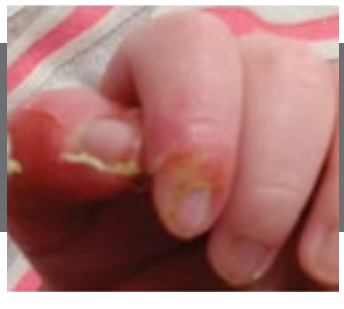
ACCEPTING SUBMISSIONS FOR THE SECTION TITLED "CLINICAL IMAGES."

This section showcases clinical images from the wards that cover essential concepts or subject matter to the primary care physician.

Each installment of "Clinical Images" comprises 1 or 2 medical images along with a brief synopsis of how the case presented along with 1- 4 questions and approximately 1 page of education with reference to the image and questions.

Submissions should be submitted online at ofpjournal.com via our Scholar One publication process.

OFP
OSTEOPATHIC FAMILY PHYSICIAN
The Official Peer-Reviewed Publication of the
American college of Osteopathic Family Physicians



Migratory Arthritis & Fever

Elizabeth Dockery, DO[1] & Rabab Elmezayen, MD, PhD[2]

[1] Family Medicine Resident, PGY-3 - Johnston Memorial Hospital, Abingdon, Virginia
[2] Clinical Faculty, Infectious Disease - Johnston Memorial Hospital, Abingdon, Virginia

A 55-year-old Caucasian female presented to the emergency department with fever and flu-like symptoms for the past three days. Review of symptoms was positive for shortness of breath, non-productive cough, sneezing, sore throat, body aches, nausea, vomiting, and a temperature of 103° F at home. She called her primary care office a few days prior to her ER visit and was empirically prescribed oseltamivir for influenza. After treatment with oseltamivir, symptoms did not resolve, and she developed new onset symptoms of red, swollen, painful joints and a new subcutaneous nodule on the lateral left foot. Her pain originated in her left MCP joint and then spread to her right elbow and right knee.

On physical examination, vital signs were T 102.8° F, HR 117, RR 20, BP 119/59, and SpO2 93% RA. The patient was in mild distress and frail appearing. On HEENT exam, the patient was normocephalic with a slightly erythematous oropharynx. She had a midline uvula and no other lesions. Eyes showed reddened sclera without any exudate or drainage. Lung sounds were diminished bilaterally, but otherwise clear to auscultation. Her heart rate was regular rate and rhythm, without any murmurs, rubs or gallops. Extremities revealed decreased range of motion, redness, edema, and tenderness to palpation of the right elbow *(Figure 1)* and right knee *(Figure 2)*. She also had an erythematous and tender 3 cm circular nodule on the left metatarsal/tarsal joint *(Figure 3).*

Urinalysis was suspicious for UTI. Remaining lab work was unremarkable. The patient was admitted to the hospital and started on empiric ceftriaxone IV daily for UTI.

QUESTIONS

1. What was the most likely diagnosis?
   a) Acute rheumatic fever
   b) Reactive arthritis
   c) Lyme disease
   d) Rheumatoid arthritis
   e) Septic arthritis

2. Based on the diagnosis criteria, how is infection with Group A Streptococcus confirmed in a patient with the suspected disease?
   a) Positive blood cultures
   b) Elevated ESR
   c) Rising anti-strepolysin O titers
   d) Elevated CRP
   e) All of the above

3. Which of the following is considered a major criterion for the presumed diagnosis?
   a) Fever > 38.5 C
   b) Arthritis
   c) ESR > 60mm in the first hour
   d) Prolonged PR interval
   e) Carditis

FIGURE 1:
Right elbow

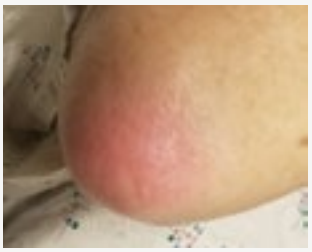


FIGURE 2:
Right knee

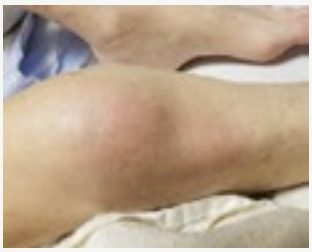
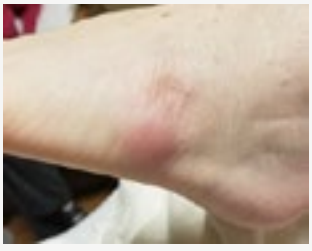


FIGURE 3:
Left lateral foot



CORRESPONDENCE:
Elizabeth Dockery, DO
elizabeth.dockery@balladhealth.org

ANSWERS

1. What is the most likely diagnosis?

Correct answer: a) Acute rheumatic fever

Acute rheumatic fever is characterized by a systemic inflammatory response secondary to group A streptococcus.[1] Presenting symptoms may include high fever, polyarthritis, rash, chorea, and subcutaneous nodules.

2. Based on the diagnosis criteria, how is infection with Group A Streptococcus confirmed in a patient with the suspected disease?

Correct answer: c) Rising anti-streptolysin O titers

The American Heart Association states that evidence of a preceding streptococcal infection for acute rheumatic fever can be diagnosed by either of the following: 1) an increased or rising anti-streptolysin O titer or other streptococcal antibodies, 2) a positive throat culture for group A beta-hemolytic streptococci, or 3) a positive rapid group A streptococcal carbohydrate antigen test.[2] A four-fold rise in anti-streptolysin O titers confirms the diagnosis of ARF after a recent streptococcal infection.[1]

3. Which of the following is considered a major criterion for the presumed diagnosis?

Correct answer: e) Carditis

Diagnosis of acute rheumatic fever is based on the Jones criteria, which was recently updated in 2015. Diagnosis is established by evidence of a preceding streptococcal infection in addition to 2 major criteria or 1 major criteria with 2 minor criteria. Major criteria include carditis, polyarthritis, chorea, erythema marginatum, and subcutaneous nodules. Minor criteria include polyarthralgia, fever (> 38.5 C), ESR > 60mm in the first hour, CRP >3.0 mg/dL, and prolonged PR interval on ECG. With established rheumatic heart disease or a reliable past history for acute rheumatic fever, three minor criteria may be sufficient to diagnose recurrent rheumatic fever.[1,2]

DISCUSSION

Acute rheumatic fever (ARF) is characterized by a systemic inflammatory response secondary to group A streptococcus.[1] This typically develops two to three weeks after a throat infection. The most common manifestations are carditis (50% – 70%) and arthritis (35% - 66%). Other common clinical manifestations are chorea (10% - 30%), subcutaneous nodules (< 10%), and erythema marginatum (< 6%).[1,2]

ARF is typically a syndrome of childhood, with most cases occurring in pediatrics aged 5 to 15 years.[1] However, attacks may occur at any age.[3] The etiology is not fully understood, but it is believed to involve cross reactivity of streptococcal antibodies with cardiac, synovial, and brain tissue.[1] Thus resulting in the clinical features of carditis, arthritis and chorea respectively.

Classically, carditis has been a clinical judgment based on auscultation of murmurs consistent with mitral or aortic valve regurgitation. However, Doppler echocardiogram has been found to be a reliable source of diagnosing carditis in the absence of auscultory findings and is recommended in all cases or confirmed or suspected acute rheumatic fever.[2] Pericarditis and myocarditis may also occur in acute rheumatic fever, and if severe enough, can result in congestive heart failure. The sequela of carditis can take weeks to months, so patients with an initial normal echocardiogram should have a repeat echo in 2 – 4 weeks.[2]

Polyarthritis associated with rheumatic fever is usually asymmetric, migratory, and involving the large joints such as the elbows, wrists, knees, and ankles.[2] The arthritis is typically self-limited, resolving in several weeks, but NSAIDs and salicylates have proven to be effective for rapid recovery.[1] Small joint involvement is less common.[2]

Chorea is characterized by involuntary, rhythmic movements of the trunk and extremities that is often associated with muscle weakness and emotional liability.[2] It is important to determine that the cause of chorea is not an underlying neurological disorder such as Huntington disease, Wilson disease or systemic lupus erythematous.[2] Chorea may also only be unilateral, so tics, hyperkinesis and conversion disorder should also be ruled out.2 Since chorea is a latent manifestation of ARF, it may not be possible to prove infection with GAS.[2]

Skin manifestations include erythema marginatum and subcutaneous nodules. Erythema marginatum is rare and occurs in less than 6% of cases.[1] It is described as a pink rash with central clearing that blanches with pressure and may worsen with heat.[2] Raised boarders with outward-spreading macules and papules are also characteristic.[1] It is usually located on the trunk and proximal extremities, sparing the face.[2] Subcutaneous nodules are also rare findings and usually appear during the first few weeks of the inflammatory phase.[1] They are firm protuberances typically found on the extensor surfaces of joints close to boney protuberances.[2] They are approximately 1 cm in size and can be singular or clustered.[2]

Fever accompanying acute rheumatic fever commonly exceeds 38.5 C orally and can be treated with antipyretics.[2] Elevated C-reactive protein and/or erythrocyte sedimentation rates are lab values consistent with ARF. Normal CRP and ESR should cause a clinician to seriously rethink the diagnosis of ARF.[2] First degree heart block can be used as a minor criteria for diagnosis in the absence of carditis. PR interval of 0.16 seconds in children younger than age 12 and 0.18 seconds in children 12 years of age and older are considered prolonged.[1]

Antibiotic treatment of acute rheumatic fever is usually penicillin. Symptoms such as fever and arthritis can be treated with salicylates or NSAIDS as mentioned above. Oral Penicillin VK 250 mg twice daily in children or 500 mg twice daily in adolescents for 10 days is recommended.[1] Another option is a one-time dose of intramuscular benzathine penicillin. Erythromycin is the recommended for patients with penicillin allergy.[1] Prophylaxis is recommended to help prevent recurrent episodes of acute rheumatic fever. The World Health Organization recommends intramuscular benzathine penicillin injections every three to four weeks for prophylaxis. The timeframe is debatable on the duration of prophylaxis treatment, but the consensus is a minimum of 10 years.[1]

HOSPITAL COURSE:

The patient's signs and symptoms of migrating polyarthritis, fatigue, sore throat, fever, and subcutaneous nodules were consistent with ARF. Her blood cultures were positive for streptococcus pyogenes (group A B-hemolytic streptococcus). CRP, ESR and anti-streptolysin-O titers were all markedly elevated. A throat culture was obtained 3 days after starting antibiotics, but was negative for Group A Streptococci. Echocardiogram obtained showed mild mitral and tricuspid regurgitation, but no valvular vegetations were noted. The patient had an allergy to penicillin and was continued on IV ceftriaxone for bacteremia. She improved and was discharged home on hospital day 7 with 1 more week of IV ceftriaxone and oral azithromycin 250 mg daily for 8 weeks after completing her IV antibiotic course.

CONCLUSION:

Acute rheumatic fever is usually not initially considered in the differential diagnosis for adults with fever and arthralgia. Though uncommon, adult-onset ARF is not rare in developing countries as sporadic incidences of small endemic areas have been reported in the United States since 1980.[4] From these incidences in the United States, carditis and arthritis were the main complaints, whereas throat pain was rare.[4] Therefore, ARF should be considered in older patients with new onset arthritis and fevers.

AUTHOR DISCLOSURES:
No relevant financial affiliations

REFERENCES:

1. Gewitz MH, Baltimore RS, Tani LY, et al. Revision of the Jones Criteria for the diagnosis of acute rheumatic fever in the era of Doppler echocardiography: a scientific statement from the American Heart Association. Circulation. 2015;131(20):1806-1818. DOI: 10.1161/CIR.0000000000000205. Epub 2015 Apr 23.
2. Webb RH, Grant C and Harnden A. Acute rheumatic fever. BMJ. 2015;351:h3443. DOI: 10.1136/bmj.h3443. http://www.bmj.com/content/351/bmj.h3443. Accessed February 18, 2018.
3. Ben-Dov I, Berry E. Acute rheumatic fever in adults over the age of 45 years: an analysis of 23 patients together with a review of the literature. Seminars in Arthritis & Rheumatism. 1980;10(2):100-110. PMID: 7292018. https://doi.org/10.1016/0049-0172(80)90003-7. Accessed February 16, 2018.
4. Nakashima D, Ueda K, Tsukuda K, et al. Adult-onset Acute Rheumatic Fever. Internal medicine. 2012;51(19):2805-2808. Epub 2012 Oct 1. PMID: 23037480. https://www.jstage.jst.go.jp/article/internalmedicine/51/19/51_51.7661/_article. Accessed February 16, 2018.

CALENDAR OF EVENTS

2018

OCTOBER 5 - 9, 2018

OMED®18
AOA/ACOFP Osteopathic Medical Conference & Exposition
San Diego, California
www.acofp.org

2019

JANUARY 18 - 20, 2019

Midwinter Osteopathic Family Practice Conference
Iowa Chapter the ACOFP
Des Moines, Iowa
www.ioma.org

JANUARY 24 - 27, 2019

Missouri Winter Family Medicine Update
Missouri Society of the ACOFP
Independence, Missouri
www.msacofp.org

JANUARY 24 - 27, 2019

MAOFP's Midwinter Family Medicine Update
Bellaire, Michigan
www.maofp.org

MARCH 21 - 24, 2019

ACOFP 56th Annual Convention & Scientific Seminars
Chicago, Illinois
www.acofp.org

OCTOBER 25 - 28, 2019

OMED®19
AOA/ACOFP Osteopathic Medical Conference & Exposition
Baltimore, MD
www.acofp.org

CME Resource: Osteopathic Family Physician Offers 2 Hours of 1-B CME

ACOFP members who read Osteopathic Family Physician can receive two hours of Category 1-B continuing medical education credit for completing quizzes in the journal. Visit the eLearning Center at www.acofp.org to access the quizzes.

acofp AMERICAN COLLEGE OF OSTEOPATHIC FAMILY PHYSICIANS

Zika: Ideas for Talking to Your Child About Zika

Jennalee Gaiser, DO

Ronald Januchowski, DO, FACOFP, Editor | Paula Gregory, DO, FACOFP, Health Literacy Editor

Zika virus disease (Zika) has been in the media and a hot topic for travelers. Children may have many questions about Zika and if they better understand the facts, they can better cope with disease outbreak. Speaking to your child with facts, and tailoring the conversation to their age and concerns can help them cope with Zika and spread understanding.

SHARE WITH THEM FACTS & WHAT YOU KNOW ABOUT ZIKA:

- It is extremely rare, fewer than 1,000 cases in the US per year.
- It is spread mostly from the bite of mosquitoes that are infected with Zika virus.
- You can only get Zika if you live or travel to an area where the mosquitos are known to be infected and you get bitten.
- Most people bit by a Zika infected mosquito will not actually get sick! Those that do, may only feel sick for a few days, and may not even know they have Zika virus.
- People that do get sick from Zika will display some flu-like symptoms that include some fever, rash, headache, joint pain, or red eyes.
- There is no specific treatment, vaccine, or medicine for Zika virus, therefore the best thing we can do is prevent the bite! Plenty of rest, rehydration and medicine for pain can help for those sick with Zika virus.
- Children can help adults be diligent in protecting everyone with insect repellant and appropriate clothing.
- Health care providers can do blood tests for Zika virus if they suspect infection.

ASK WHAT THEY KNOW & UNDERSTAND ABOUT ZIKA VIRUS

- Correct misinformation gently.
- Understand their fears and concerns about Zika.

EXPLAIN HOW THEY CAN HELP PREVENT THE SPREAD OF ZIKA VIRUS:

Best way is to prevent getting any mosquito bites. Things to prevent bites include:

- Long-sleeved shirts and pants
- Screens on windows and doors and/or air-conditioning
- Sleeping under a bed-net if traveling to area where Zika is prominent
- Using EPA-registered insect repellant, let an adult do this if child is younger
- Infants <2 months old should not use insect repellant
- Apply repellant to any skin not covered by clothing
- If using sunscreen in addition to repellant, apply sunscreen first, then repellant on-top
- Remove standing water from around your home or lodging. Places like buckets or tires are mosquito breeding grounds

If you have questions or concerns about talking to your child about Zika, or you want to understand more about your risk, please talk with your Osteopathic Family Physician.

SOURCE(S): *American Academy of Pediatrics, Centers for Disease Control and Prevention, and World Health Organization*

acofp AMERICAN COLLEGE OF OSTEOPATHIC FAMILY PHYSICIANS

Polyarthritis

Purwaai Chand Matriya, DO

Ronald Januchowski, DO, FACOFP, Editor | Paula Gregory, DO, FACOFP, Health Literacy Editor

Polyarthritis is joint pain that affects five or more joints. Joints that are inflamed are often red, warm, and swollen. Risk factors include old age, sex (more common in females), and genetics. Risk factors you can change include being overweight, infections, and trauma. Many different conditions can cause polyarthritis. These conditions are grouped into acute (symptoms lasting <6 weeks) or chronic (symptoms lasting >6 weeks). To make a diagnosis, your doctor will want to know when your symptoms started, what joints are involved, how many joints are involved, whether one or both sides of body are affected, and whether inflammation is present.

FEW COMMON CONDITIONS CAUSING POLYARTHRITIS:

- Osteoarthritis (OA): most common type of arthritis. Involves the breakdown of cartilage, which is located between joints. Weight-bearing joints, such as hip and knee, are most commonly affected. OA is a chronic form of arthritis. Both sides may be affected, but symptoms may be worse on one side more than the other.
- Infectious arthritis: caused by a virus (such as Parvovirus or HIV) or bacteria (such as Gonorrhea). Presents acutely with inflamed joints. Joints involved and symmetry of involvement vary with the type of infection. May also present with a fever.
- Rheumatoid Arthritis (RA): a type of arthritis where your own body is attacking the joints. It is a chronic condition and involves symmetrical inflammation of small and large joints. Typically will affect joints in hands, wrists and knees.
- Systemic Lupus Erythematous (SLE): chronic disorder that causes inflammation throughout the entire body, affecting joints and organs. Involves inflammation of small joints of hands, wrists, and knees in a symmetrical fashion.
- Ankylosing Arthritis: chronic condition that involves inflammation of spine and large joints, such as the hip, in a symmetrical fashion. More common in men.
- Psoriatic Arthritis: seen in individuals with psoriasis, a chronic disorder that attacks the skin and joints. Causes symmetrical inflammation of both small and large joints.

MEDICAL TREATMENT & OPTIONS:

You should see your Osteopathic Family Physician if you experience joint pain or swelling. Treatment for arthritis is targeted to decrease pain and damage to joints. Treatment choices include:

Medications

- **Non-steroidal anti-inflammatory (NSAIDS):** These drugs decrease pain and inflammation
- **Corticosteroids**: These suppress the immune system to help decrease inflammation. These can either be given orally or by injection directly to affected joint.
- **Disease-modifying anti-rheumatic drugs (DMARDS):** These also suppress the immune system. Often used to treat rheumatoid arthritis.
- **Analgesics**: These medications, such as acetaminophen (Tylenol®), are available over-the-counter and will help to reduce pain.
- **Antibiotics**: Used for infectious arthritis caused by bacteria. If the joint is infected by a virus, antibiotics will not be helpful. Arthritis caused by a virus typically will resolve on its own.

Non-pharmacological intervention

- Physical therapy can help strengthen joints and increase range of motion.
- Low impact exercise can help, such as walking, biking, or swimming.
- Hot or cold packs can provide pain relief. See which one works best for you, or alternate.

Should conservative management fail, your doctor may discuss surgical options with you.

SOURCE(S): *American College of Rheumatology*

"Arthritis". (2017, November 21). Retrieved from https://www.cdc.gov/arthritis/basics/index.html.

Pujalte, G.G., & Albano-Aluquin, S.A. (2015, July 1). Differential diagnosis of polyarticular disease. Retrieved from https://www.aafp.org/afp/2015/0701/p35.html.

Richie, A.M. (2003, September 15). Diagnostic approach to polyarticular pain. Retrieved from https://www.aafp.org/afp/2003/0915/p1151.html.

American College of Osteopathic Family Physicians
330 East Algonquin Road, Suite 1
Arlington Heights, IL 60005

acofp '19

MARCH 21 - 24, 2019

Sheraton Grand Chicago
Chicago, Illinois

Over 30 Category 1-A CME credits anticipated!

www.acofp.org