OFP
Osteopathic Family Physician

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

January/February, 2018
Volume 10 | Number 1
ofpjournal.com

acofp American College of Osteopathic Family Physicians
Advocacy • Education • Leadership
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 Cognitive Exam	**Electronic Testing** Regional Sites September 22, 2018	**April 1, 2018** *Late fee through June 1*
Family Medicine / OMT Certification / OCC Cognitive Exam	**AOA OMED Conference** **San Diego, CA** October 6 - 10, 2018 October 5 - 7, 2018	**April 1, 2018** *Late fee through June 1*

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

JAN/FEB, 2018
VOLUME 10 | NUMBER 1

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

Janis Coffin, DO
Practice Management

Philip Collins, DO
Patient Education

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

Rose Hall, DO
Family Medicine

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

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

Sarah Mitchell, DO
Family Medicine

Wadsworth Murad, DO
Psychiatry

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

Mischa Pollard, MD
Women's Health, Child Sexual Abuse, Pediatrics, Obstetrics

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

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, Emerency Medicine

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

Osteopathic Family Physician is looking for...

SPECIALTY PEER REVIEWERS

OFP PEER REVIEWER QUALIFICATIONS & EXPECTATIONS:

- Familiarity with the *Osteopathic Family Physician* editorial standards and compliance with those standards.
- Dependability – Be responsible, prompt, and maintain fine attention to detail.
- Objectivity –Evaluate the submission based on established criteria.
- Communicate – Interact in a professional manner. Be direct, kind and concise.
- Computer literacy- Microsoft Word, Adobe PDFs and working with electronic submission process of Scholar One is required.
- Respect the confidentiality inherent in the review process.
- A good article takes 1-3 hours to review and a flawed article may take up to 10 hours.

SPECIALTY TOPICS

CALL FOR SPECIALTY REVIEWERS IN THE FOLLOWING TOPICS:

- Allergy
- Dermatology
- Nutrition
- Neurology
- OMM
- Pediatrics
- Psychology
- Rheumatology

CONTACT INFORMATION

Please email belindab@acofp.org your CV and what type of articles you are qualified to peer review based on your specialty area(s). We recognize the time and effort and will be respectful to send articles that are worthy of reviewing and respect your time and limitations.

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
- Acute & Chronic Urticaria: Evaluation & Treatment
- Aseptic & Bacterial Meningitis: Evaluation, Treatment, & Prevention
- Chronic Kidney Disease: Detection & Evaluation
- Disorders of Puberty: An Approach to Diagnosis & Management
- Epilepsy: Treatment Options
- Lupus: Review article with osteopathic component
- OMT Treatments for Pediatric Conditions: Systematic Review
- Premenstrual Syndrome & Premenstrual Dysphoric Disorder
- Primary Care Approach to Eye Conditions
- Probiotics for Gastrointestinal Conditions: A Summary of the Evidence
- Review of Rheumatology Arthritis: with osteopathic component
- Strategies to reduce physician stress and burnout
- Treating psychosis, delirium, and dementia in the elderly
- Update on Office-Based Strategies for the Management of Obesity

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

Amy Keenum, DO, PharmD
Editor-in-Chief

Ronald Januchowski, DO, FACOFP
Associate Editor

acofp American College of Osteopathic Family Physicians

EDITOR'S MESSAGE

Winter is a Great Time for CME

Amy J. Keenum, DO, PharmD, Editor, *Osteopathic Family Physician*

We recently placed a call for patient education handout submission, and the response was strong both in number and quality. Osteopathic physicians know what it means to write information at the literacy level of the patient, and we are all getting practice with this in our respective electronic health records. I wonder how osteopathic family physicians incorporate information into the busy day-to-day practice if the data is not in the electronic health record? The colleague who sits next to me reaches into a file drawer a few times a day to offer printed patient information. I tend to copy and paste website locations into the patient instruction section and send patients to reliable websites. As an osteopathic family physician, I rely on the patient information incorporated into my electronic health record the most. It is fast, easy and hopefully, it is peer-reviewed.

We continue to offer clinical images and are getting common or essential pictures which patients kindly let us share for education. This edition has a typical case, *Paronychia*, as well as a critical case, *Traumatic Eye Injury in a 14-Year-Old Male*. One we treat ourselves and the other we should not miss because it requires a prompt specialty referral. That's how it goes in family medicine.

The article, *Aortic Aneurysm: Clinical Guidelines for Primary Care Physicians*, reviews how to prevent, screen, diagnosis and follow aortic aneurysms. In some regions patients are referred to vascular surgery and may be followed by the specialist. In rural and underserved areas family physicians can follow and educate the patient when a referral is required. This article nicely presents the information with helpful tables for easy reference.

In *Giant Cell Arteritis*, the authors review its pathophysiology, patient symptomatology, differential diagnosis, and treatment. Be sure to check out the video that shows a temporal artery biopsy; the video link is at the end of the article.

It's starting to get cold outside, and winter is a great time to do continuing medical education. Check out all the CME opportunities in our OFP calendar of event listings, as there are several opportunities to earn CME under the sunny skies of Florida, Texas, and California or stay close to home if you live in the Midwest and avoid driving on icy roads and airport delays.

 Print ISSN: 1877-573X

FROM THE PRESIDENT'S DESK

Leaning Into the Winds of Change Swirling Around Osteopathic Medicine

Rodney M. Wiseman, DO, FACOFP *dist*.
2017 - 2018 ACOFP President

> *When the world changes around you and when it changes against you – what used to be a tail wind is now a head wind – you have to lean into that and figure out what to do because complaining isn't a strategy.*
>
> *- Jeff Bezos, CEO, Amazon*

I've been practicing osteopathic medicine for 39 years, and have held appointed and elected offices for state and national membership associations during most of those same years. I've been swept along by tail winds and been buffeted by head winds – not just figuratively, but also literally when Hurricane Harvey hung over Houston for several days.

Knowing the forecast in advance helped us prepare for and survive a serious storm. And just a few weeks later we experienced the celebration for the Houston Astros' first World Series championship – the ultimate "bad news/good news" story for 2017.

Like any other year, 2018 will have its share of tail winds and head winds. Knowing the forecast in advance will help us prepare and survive, hopefully experiencing the joys of practicing osteopathic family medicine, beyond the travails. Here's my 2018 forecast.

Single Accreditation System

At deadline for this column, approximately 35 of the 150 osteopathic family medicine residency programs that had only been AOA accredited had not yet submitted the application for ACGME accreditation under the Single Accreditation System, and we are hopeful that the majority of the remaining programs will have submitted applications by the December 31, 2017 deadline.

From this point forward, ACOFP's efforts will be to have these programs also achieve Osteopathic Recognition status with ACGME, thereby maintaining osteopathic training in postdoctoral education. We know that 95 family medicine programs already are on the path to Osteopathic Recognition, which is two-thirds of programs covering all specialties.

MDs as ACOFP Members

The seismic shift to the Single Accreditation System also opens doors for allopathic physicians to be trained in family medicine residency programs with Osteopathic Recognition, thereby raising the prospect that MDs may also desire membership in the ACOFP.

At its March 21-22, 2018 meeting, ACOFP Congress of Delegates will debate the extent to which MDs should become ACOFP Active Members. The ACOFP Board surveyed the general membership and key constituencies in 2017, and will be presenting its recommendations to the Congress for its ultimate decision, followed by implementation after Constitution & Bylaws amendments are passed at the 2019 ACOFP Congress.

The membership survey revealed a wide range of opinions – everything from full membership parity for MD and DO members, to continuing the current criteria for DOs only, and several hybrid options in between. What would A. T. Still do?

Osteopathic Continuous Certification

The winds of change continue to swirl around Osteopathic Continuous Certification (OCC), as reflected in actions at the past two AOA House of Delegates meetings.

Physician certification should always prioritize quality and patient safety, but the profession also is calling on the AOA and its specialty certifying boards to develop a more meaningful, cost-effective method of certifying physicians throughout their careers. We are eager to see how the AOA will advance OCC reforms in the years ahead.

Health System Reform

Based on the shifting winds of health care reform that we experienced in 2017, the prospects for 2018 are just as uncertain, including the direction to be charted by a new Health and Human Services (HHS) Secretary, Alex Azar.

Reform to date has been focused on the Centers for Medicare and Medicaid Services' (CMS) value-based payment program. As physicians, we can count on the Merit-Based Incentive Payment System (MIPS) continuing, with increasing incentives for those who demonstrate increased quality outcomes, reduced costs, and improved patient satisfaction. For those who do not report, or do not demonstrate improved quality, there are year-over-year increasing penalties.

The ACOFP Board believes that more overarching health system reform is necessary, with greater incentives for patient access to primary care physicians. Primary care physicians deserve increased payment parity with other specialists so that they can provide the most innovative and cost-effective care. CMS has made recent changes to expand payment for Chronic Care Management (CCM),[1] care coordination and telehealth services, for which ACOFP has advocated. These changes also positively impact rural physicians, clinics, and hospitals.

Continuing Medical Education

I hope that your 2018 calendar includes participation in ACOFP's CME events. The ACOFP will offer almost 100 Category 1-A CME credits at these venues:

- March 22-25 ACOFP Annual Convention
  Austin, Texas
- August 24-26 Intensive Update & Board Review
  Rosemont, Illinois
- October 6-9 ACOFP at AOA OMED
  San Diego, California

In addition to live CME events, we are expanding the content available to you on-demand through the ACOFP E-Learning Center.

Leadership Stability

While the person holding the ACOFP President's gavel changes yearly, we do have consistency in pursuit of ACOFP's strategic plan and policy initiatives.

Dr. Duane Koehler will succeed me as ACOFP President, and I know that his experience in practice and as an educator will well serve the specialty throughout the year ahead.

Be assured that the ACOFP Board will be leaning into the winds of change headed our direction in 2018. We welcome your continued support, your innovative ideas, your constructive criticism, and your active participation!

Osteopathically yours,

Rodney M. Wiseman, DO, FACOFP *dist.*
2017-2018 ACOFP President

REFERENCES:

1. Department of Health and Human Services Centers for Medicare and Medicaid Services. Chronic Care Management Services Changes for 2017.https://www.cms.gov/Outreach-and-Education/Medicare-Learning-Network-MLN/MLNProducts/Downloads/ChronicCareManagementServicesChanges2017.pdf. Accessed December 20, 2017.

REVIEW ARTICLE

Aortic Aneurysms: Clinical Guidelines for Primary Care Physicians

Jordan E. Wong, BS, OMS I & Peter Zajac, DO, FACOFP

University of Pikeville - Kentucky College of Osteopathic Medicine

KEYWORDS:

Abdominal Aortic Aneurysm

Cardiology

Endovascular Aneurysm Repair

Marfan Syndrome

Thoracic Aortic Aneurysm

Aortic aneurysms (AA) are permanent, localized abnormal dilation of the wall of the aorta, the largest artery in the body, occurring as a result of medial degeneration of the arterial wall, generally, as a result of increased aortic hypertension or genetic predisposition. Risk factors for AAs are similar to those of other cardiovascular diseases. Tobacco use is strongly associated with aneurysm formation and dilation, and patients diagnosed with AA should be advised to stop smoking. An abdominal aorta with a diameter greater than 3.00 cm is generally considered aneurysmal. By convention, a thoracic aorta with a diameter greater than 4.50 cm is generally considered aneurysmal. No specific laboratory tests exist to diagnose an AA, and testing should be ordered supplementary to imaging studies. Dedicated imaging studies offer definitive identification or exclusion of potential AAs, but the imaging modality used is largely dependent upon patient-related factors. Patients with small aneurysms may be candidates for medical management, however, any patient with an aortic diameter greater than 5.00 - 5.50 cm should be referred for immediate surgical consultation. With the majority of AAs asymptomatic prior to rupture, it is important that primary care physicians understand how to properly evaluate and diagnose patients at risk for developing an AA as well as the short and long-term management of patients diagnosed with an AA.

INTRODUCTION

An aortic aneurysm (AA) is a permanent, localized abnormal dilation of more than 50% of the normal diameter of the wall of the aorta, the largest artery in the body.[1,2] Aortic aneurysms develop as a result of medial degeneration of the arterial wall, and proteolytic degradation of the associated elastic tissues.[3] Degeneration of the arterial architecture coupled with hypertensive hemodynamic stress results in aneurysmal dilation.[3,4] While the etiology of an AA is not fully understood, risk factors for an AA are similar to those of other cardiovascular diseases.[2] Risk factors for the development of an AA include age greater than 65 years, male sex, trauma, hypertension, hypercholesterolemia, atherosclerosis, tobacco use, and a familial history of vascular aneurysm.[3,5,6,7] In the United States, AA, and dissection (I71, ICD-10, excluding aortic ectasia, syphilitic aortic aneurysm, traumatic aortic aneurysm) were the primary causes of 201,985 deaths in the United States between 1999 and 2014 (50,010 between 2010 and 2014); cases were predominantly male (59%), white (91.6%), and age older than 65 years (80%).[8] Patients with an AA are at increased risk of dissection, pulmonary/arterial embolism, stroke, and myocardial infarction.[9] With the majority of AAs asymptomatic prior to rupture, it is important that primary care physicians understand how to properly evaluate and diagnose patients at risk for developing an AA as well as the short and long-term management of patients diagnosed with an AA.

CORRESPONDENCE:
Peter Zajac, DO, FACOFP | PeterZajac@upike.edu

 Print ISSN: 1877-573X

PREVENTION

Primary prevention or risk factor reduction for AAs is aimed at counseling the patient on health-related behaviors, such as improved diet and smoking cessation. The Dietary Approaches to Stop Hypertension (DASH), a diet rich in fruits and vegetables with reduced saturated and total fat, is a recognized dietary recommendation effective in reducing blood pressure, and with higher adherence being associated with cardiovascular risk factor reduction.[10] Further, adherence to a Mediterranean diet, a diet rich in fruits and vegetables that is supplemented with olive oil or nuts, is also a well-established protective factor against cardiovascular disease.[11] Dietary sodium restriction is strongly advocated for the prevention and treatment of hypertension, however, despite the abundance of studies on its efficacy, current evidence suggests a J-shaped association between sodium intake and cardiovascular disease.[12] Tobacco use is a significant risk factor for AA with one trial demonstrating population attributable risk at or above 47%, and another estimating that smoking accounted for 75% of all abdominal aortic aneurysms (AAA) greater than 4.0 cm in diameter; therefore, effective strategies for smoking cessation should be advocated.[13,14]

SCREENING

Guidelines

The United States Preventive Services Task Force (USPSTF) recommends one-time screening for AAA with ultrasonography in men ages 65 to 75 years who have ever smoked, and that clinicians selectively offer to screen for AAA in men ages 65 to 75 years who

have never smoked. Currently, the USPSTF concludes that current evidence is insufficient to assess risks and benefits of screening women ages 65 to 75 who have ever smoked, and does not recommend screening women who have never smoked.[6] Recurrent screening in men age 75 or older does not appear advantageous.[15] Although the USPSTF does not provide recommendations for one-time screening for thoracic aortic aneurysm (TAA) the application of AAA guidelines may be beneficial. The American College of Cardiology/American Heart Association (ACC/AHA) guidelines issued in 2005 recommend that men who are 65 to 75 years of age who have ever smoked should undergo a physical examination and one-time ultrasound screening for detection of AAAs, further, men 60 years of age or older who are either the siblings or offspring of patients with AAAs should also undergo physical examination and ultrasound screening for detection of aortic aneurysms.[16] The European Society of Cardiology recommends screening in all men greater than 65 years and considered in women greater than 65 years and tobacco smoking.[17]

Reimbursement

Although coverages may vary by insurer, physicians submitting claims for reimbursement for an ultrasound of the abdominal aorta will use CPT code G0389 when ordered during an initial preventive physical examination (IPPE). Eligible beneficiaries are those who: have received a referral for an ultrasound screening as a result of an IPPE, have not been previously furnished a covered AAA screening ultrasound examination under the Medicare program; and/or has a familial history of AAA or is a man age 65 to 75 who has smoked at least 100 cigarettes in his lifetime. If ordered during a visit that is not an IPPE, physicians will use CPT code 76775. Physicians submitting claims for payment by insurers will use ICD-10 code Z13.6, Z82.49 and Z87.891. Specific coverage for the screening of TAAs has not been established, and will vary by the insurer (See Summary: Table 1).

TABLE 1: Summary of CPT and ICD-10 codes of reimbursement

CODE	DESCRIPTION OF CODE
76000	Ultrasound, abdominal, real time with image documentation; complete
76705	Ultrasound, abdominal, real time with image documentation; limited (eg, single organ, quadrant, follow-up)
76770	Ultrasound, retroperitoneal (eg, renal, aorta, notes), real time with image documentation; complete
76775	Ultrasound, retroperitoneal, (eg, renal, aorta, nodes), real time with image documentation; limited
G0389	Ultrasound B-scan and/or real time image documentation; for abdominal aortic aneurysm (AAA) screening
Z13.6	Encounter for screening for cardiovascular disorders
Z82.49	Family history of ischemic heart disease and other diseases of the circulatory system
Z87.891	Personal history of nicotine dependence

DIAGNOSIS

Physical Examination

Detection of an AAA is difficult given that the majority are asymptomatic, and diagnoses made are often an incidental finding on radiographic studies. Physical examination is limited and associated with a large population of false-negative and false-positive findings.[3,4,9] Abdominal palpation has only moderate sensitivity for detecting AAAs with one study demonstrating 68% sensitivity and 75% specificity.[3,18] Abdominal palpation sensitivity is positively associated with AAAs of increasing diameter.[18] The most common finding in asymptomatic AAAs large enough to warrant intervention is a palpable pulsatile mass extending from the xiphoid to the umbilicus.[3,9] Abdominal palpation sensitivity is often affected by obesity, abdominal distention, and small aneurysm size.[3] Somatic findings correlated with AAAs can be manifested as ecchymotic, erythemic, and edematous tissue abnormalities.

Clinical features of symptomatic AAA include the presence of a Grey-Turner's sign — the extravasation of blood into the subcutaneous tissues producing flank ecchymosis, as consequence of an extensive retroperitoneal hematoma from ruptured AAA.[19] Further, Grey-Turner's sign may be coupled with periumbilical ecchymosis (Cullen's sign), ecchymosis of the proximal thigh (Fox's sign), and discoloration of the scrotum (Bryant's sign).[20,21] Abdominal pain is also common, however, abdominal pain may be associated with acute gastritis, bowel obstruction, ischemic bowel, mild pyelonephritis, emergent pancreatitis, and generalized musculoskeletal pain, and should be considered during the process of forming a differential. In hemodynamically unstable patients, abdominal pain may be symptomatic of appendicitis, diverticulitis, cholelithiasis, perforated peptic ulcer, myocardial infarction, or pulmonary embolism rather than an AAA. Acute limb ischemia can also be present with AAAs as a consequence of a distal thromboembolism originating from atherothrombotic debris from the AAA.[22] Pain varies with diameter and location of the aneurysm, and whether or not the aneurysm has ruptured (free or contained).[21] Anterior intraperitoneal rupture presents as sudden severe abdominal or back pain and collapse. The subsequent hemorrhaging often results in exsanguination and death.[21] Posterior retroperitoneal rupture also often manifests as back pain with or without associated abdominal pain and hypotension, however, the rupture is often contained allowing time for treatment.[21] Vertebral erosion may be resultant of a chronic contained rupture of an AAA presenting as chronic back pain.23 Auscultation of the aortic and femoral arteries may reveal the presence of bruits although their absence does not exclude the presence of an AAA3[24] *(See summary: Table 2, page 12).*

Like AAAs, TAAs are typically asymptomatic with diagnoses made incidentally on radiographic or electrocardiographic studies and are not easily detectable until a catastrophic complication occurs (i.e., a dissection or rupture).[25] Other symptoms include vocal cord palsy presenting as hoarseness resultant of vagus, or recurrent laryngeal nerve compression.[26] TAA compression of the trachea may cause a significant deviation, wheezing, dyspnea, or tussis.[9] Hemoptysis may be a sign of aneurysmal erosion into the trachea.[27] Dysphagia or hematemesis may be caused by esophageal compression or aortoesophageal fistula.[28] Dilation of the aortic root can mimic symptoms of congestive heart failure (CHF) due to aortic insufficiency.[9] Chest pain may be associated with acute aortic dissection,

TABLE 2:

Table 2A: Summary of risk factors, symptoms, and differential diagnoses for abdominal aortic aneurysm	
Abdominal Aortic Aneurysm	
Risk Factors	Age greater than 65 years male sex, hypertension, hypercholesterolemia, atherosclerosis, tobacco use, and familial history of vascular anuerysm
Symptoms	Abdominal pain, palpable pulsatile mass, flank ecchymosis (Grey-Turner's sign), periumbilical ecchymosis (Cullen's sign), ecchymosis of the proximal thigh (Fox's sign), discoloration of the scrotum (Bryant's sign), and acute limb ischemia. Bruits in aortic of femoral vessels
Differential Diagnoses	Acute gastritis, bowel obstruction, ischemic bowel, mild pyelonephritis, emergent pancreatitis, and generalized musculo-skeletal pain. Appendicitis, diverticulitis, cholelithiasis, perforated peptic ulcer, myocardial infarction, and pulmonary embolism
Table 2B: Summary of risk factors, symptoms, and differential diagnoses for thoracic aortic aneurysm	
Thoracic Aortic Aneurysm	
Risk Factors	Age greater than 65 years male sex, hypertension, hypercholesterolemia, atherosclerosis, tobacco use, and familial history of vascular anuerysm
Symptoms	Chest pain, vocal cord palsy presenting as hoarseness, tracheal deviation, wheezing, dyspnea, tussis, hemoptysis, dysphagia or hematemesis. Aortic regurgitation or other pathological murmurs.
Differential Diagnoses	Congestive heart failure, acute dissection, acute pericarditis, infective endocarditis, myocardial infarction, pulmonary embolism, and superior vena cava syndrome

acute pericarditis, infective endocarditis, myocardial infarction, pulmonary embolism, and superior vena cava syndrome. Cardiac auscultation for aortic regurgitation and other pathological murmurs may be revealing. Arterial perfusion differentials in both upper and lower extremities, as well as cardiac tamponade, may be evident (See summary: Table 2A and 2B).

Laboratory Studies

No specific laboratory tests exist to diagnose an AA. Laboratory testing should be ordered supplementary to imaging studies and cardio-pulmonary status studies. Blood should be drawn for complete blood count (CBC), prothrombin time/partial thromboplastin time (PT/PTT) with the international normalized ratio (INR) to evaluate for infection or bleeding disorders. Arterial blood gases, liver and kidney function tests, lipid panel, and blood lactate levels may be used to assess respiratory and metabolic status. HbA1c, while not diagnostic for an AA, should be used to test for Type 2 Diabetes Mellitus. Interestingly, some studies have demonstrated an inverse relationship between HbA1c and aneurysm expansion.[29,30] While not conclusive, hematocrit may be lowered in patients with a ruptured aneurysm. Disseminated intravascular coagulopathy (DIC) is a rare complication of an AA. Therefore coagulation studies should be ordered.[31,32]

Imaging Studies

Normal values for the intra-luminal diameter of the aortic root and ascending aorta have been reported as between 3.00 - 3.36 cm for males and 2.90 - 3.11 cm for females at end-systole.[33,34] The suprarenal diameter of the aorta tapers as it descends, and has been reported to be between 2.45 - 2.40 cm and 2.39 - 2.43 cm mid-descending to the diaphragmatic aorta in females and males, respectively.[35] An intra-luminal diameter less than 3.00 cm is considered normal. It is important to note that these measurements are heavily confounded by some factors including age, sex, body size, the location of the measurement, method of measurement, and the robustness of the type of imaging used.[35] By convention, a thoracic aorta with an intra-luminal diameter greater than 4.50 cm is considered aneurysmal.[35,36] The USPSTF defines an abdominal aorta with a diameter greater than 3.00 cm to be aneurysmal.[6] Definitive identification or exclusion of AAs requires dedicated imaging studies. While multiple imaging modalities can be used to evaluate potential AAs, selection of the most appropriate modality may depend upon patient-related factors such as hemodynamic stability, renal insufficiency, and contrast allergies.[35] Transesophageal echocardiography (TEE) and ultrasonography (US) serve as imaging modalities used to screen AAAs and TAAs respectively. US is the primary technology used to screen patients for an AAA. It is a noninvasive and inexpensive modality and offers the benefit of not requiring the use of contrast agent over computed tomography (CT).[9,37] US is suboptimal in obese patients, patients with increased bowel gas, and has increased inter-observer variation.[38] TEE offers considerable advantages in the diagnosis of TAAs. TEE offers high sensitivity/specificity, short duration, and is readily available.[39] Given the proximity of the esophagus to the aorta, TEE permits high-quality images to be obtained without interference from the thoracic wall or lungs.[39] TEE, however, cannot reliably image the distal ascending aorta and the aortic arch and is highly dependent upon an experienced echocardiographer.[40] Although TEE is a relatively invasive procedure requiring esophageal intubation, it is considered to be a safe procedure.[40,41] Cardiovascular contraindications including induced vagal and sympathetic reflexes, non-sustained ventricular and supraventricular tachycardia, atrial fibrillation, 3rd-degree block, angina, and myocardial ischemia should be considered.[41] The ACC/AHA guidelines issued in 2010 recommend that low to intermediate risk patients receive a chest x-ray during screening, as it may either establish an alternative diagnosis or demonstrate findings that are suggestive of a TAA. Plain radiography in patients with AAA may suggest an aneurysm secondary to a paravertebral curvilinear calcification.[42] Plain radiographs in patients with a TAA may suggest an enlarged thoracic aorta secondary to a calcified aortic wall similar to that seen in the atherosclerotic disease. Indirect findings suggestive of TAA include a widened mediastinum, although mediastinal masses may mimic aortic aneurysms; tracheal deviation may also be evident.[43] Ultimately, plain radiography is inadequate to definitively conclude the presence of a TAA or an AAA due to magnification effects, and often poor visualization of the aorta. Magnetic resonance imaging (MRI), CT, and angiography should be used when more definitive imaging is required and should be considered only when weighed with the potential benefits and risks.

Surveillance

The natural history of AAs shows that as aneurysms increase in size, the rate of expansion increases, and the risk of rupture increases (See summary: Table 3A and 3B).[16,44,45,46] For patients found to have AAAs on initial screening, ACC/AHA guidelines issued in 2005 recommend regular surveillance every six months to three years, depending on aneurysm size (See summary: Table 3A and 3B).[16] While no specific recommendations exist for TAAs, one study recommended regular surveillance every one to two years, depending on aneurysm size (See summary: Table 4A and 4B).[47]

TABLE 3:

Table 3A: Summary of expansion rates and risk of rupture for abdominal aortic aneurysm	
Aneurysm Diameter	**Annual Expansion Rate**
3.0 to 3.9 cm	1 to 4 mm
4.0 to 6.0 cm	3 to 5 mm
> 6.0 cm	7 to 8 mm
Aneurysm Diameter	**Annual Risk of Rupture**
< 4.0 cm	< 0.5%
4.0 to 4.9 cm	0.5 to 15%
5.0 to 5.9 cm	3 to 15%
6.0 to 6.9 cm	10 to 20%
7.0 to 7.9 cm	20 to 40%
> 8.0 cm	30 to 50%
Table 3B: Summary of expansion rates and risk of rupture for thoracic aortic aneurysm	
Aneurysm Type	**Annual Expansion Rate**
Aortic arch	5.6 mm
Ascending aorta	0.2 to 2.8 mm
Descending aorta	1.9 to 3.4 mm
Aneurysm Diameter	**Annual Risk of Rupture**
< 4.0 cm	0%
4.0 to 4.9 cm	2%
5.0 to 5.9 cm	3%
> 6.0 cm	7%

MANAGEMENT

Medical

Medical management of small and preoperative AAs generally consists of strict blood pressure control. A goal systolic blood pressure between 100 and 120 mm Hg has been recommended.[48] The AHA strongly recommends "stringent control of hypertension, lipid profile optimization, smoking cessation, and other atherosclerosis risk-reduction measures should be instituted for patients with small aneurysms not requiring surgery, as well as for patients who are not considered surgical or stent graft candidates."[49] Prophylactic beta-adrenergic blocking agent (beta blocker) therapy, may be beneficial in reducing the rate of aortic dilation by reducing the force of myocardial contraction.[43] However, while several animal and clinical studies have indicated a significant effect of beta-blockers on aneurysm growth rate, recent studies have not clearly shown to reduce aneurysm expansion rates.[50] Angiotensin-converting enzyme inhibitors (ACE inhibitors) have been demonstrated to "stimulate and inhibit matrix metalloproteinases (MMPs), and the degradation of extracellular matrix in AAs,"[41] Patients receiving ACE inhibitor therapy were significantly less likely to present with a ruptured aneurysm compared with those who were not receiving therapy.[50,51,52] Statin therapy is likely to be effective in preventing the growth of an AA and reduce the likelihood of adverse events by reducing MMP expression.[50,51,52] Meta analysis of eleven observational comparative studies suggested a significant reduction in AAA growth in patients receiving statin therapy compared to no therapy.[50] Tobacco use is strongly associated with aneurysm formation and dilation, and patients diagnosed with AA should be advised to stop smoking, and be offered smoking cessation interventions.[3,5,6,7,50,51,52]

TABLE 4:

Table 4A: Summary of surveillance intervals for abdominal aortic aneurysm	
Aneurysm Diameter	**Surveillance Interval**
< 3.0 cm	No surveillance
3.0 to 3.9 cm	US every two to three years
4.0 cm to 5.4 cm	US to CT every six to twelve months
>5.4 cm	Referral for surgical consultation
Table 4B: Summary of surveillance intervals for thoracic aortic aneurysm	
Aneurysm Diameter	**Surveillance Interval**
< 3.0 cm	No surveillance
3.0 cm to 3.9 cm	Every two years
> 4.5 cm	Annually
> 5.0 cm	Referral for surgical consultation

Osteopathic Manipulative Medicine

The use of manipulative techniques is contraindicated in patients with an aortic aneurysm due to the increased risk of rupture. However, osteopathic principals can be applied during the physical examination to aid in the diagnosis of an AA. Viscerosomatic reflexes are somatic dysfunction that develops in response to visceral pathology. Irritation, such as with compression from an aneurysm, results in activation of general visceral afferent neurons in surrounding tissues that project to the spinal cord. Prolonged afferent stimulation can stimulate interneurons that synapse with anterior horn motor neurons producing palpable tissue texture changes. Given that an aortic aneurysm can develop anywhere along the length of the aorta, viscerosomatic reflex somatic dysfunction will vary widely.

Referral

Patients for which medical management is refractory, with AAAs that exceed 1 cm of expansion per year or TAAs that exceed 0.5 cm of expansion per year, with symptomatic AA, or with aortic diameters greater than 5.5 cm should be referred for immediate surgical consultation (See Summary: Table 5). If surgical intervention is warranted, there are two main approaches: open and endovascular aneurysm repair. Prophylactic surgical repair is the most effective management to prevent rupture/dissection with 30-day mortality risk at 11.7% in open TAA repair versus 2.1% in EVAR TAA repair.[53] Endovascular repair is less invasive, has decreased morbidity and mortality, and is preferred in patients who are at high risk of complications from open surgical repair.[16] The Immediate Management of the Patient with Ruptured Aneurysm: Open Versus Endovascular repair (IMPROVE) trial reported 30-day mortality risk at 37.4% in open AA repair versus 35.4% in EVAR AAA repair. A Dutch trial reported 30-day mortality risk at 25% and 21% for open AAA and EVAR AAA repair, respectively.[53] The UK EVAR trial suggested that EVAR was associated with a significantly lower operative mortality than open surgical repair, but that these differences were not seen long-term.[54] Conversely, the Open Versus Endovascular Repair (OVER) trial reported that EVAR led to increased long-term survival among younger patients, but not among older patients suggesting that EVAR might be the more appropriate treatment for younger patients.[55] However, despite the many benefits of EVAR, graft failure and the associated risk of persistent sac enlargement requires lifelong surveillance.[56,57] CT/CT-angiography is recommended at one month, six months, and one-year post-EVAR repair to identify an endoleak, or persistent blood blow in the aneurysm sac extrinsic to the endograft, or other aberrant pathologies.[56,57] Open repair, while more invasive, is advantageous given that it is suitable for aneurysm repair in all areas, while EVAR has been associated with increased complications from upper extremity ischemia with aneurysms of the aortic arch.[58] Postoperative surveillance is minimal following open repair, and follow up CT imaging is recommended at five-year intervals.[56,57]

Special Considerations

Genetic predisposition (e.g., Marfan syndrome, Loeys-Dietz, Ehlers-Danlos syndrome) may also contribute to the development of an AA.[9] Special consideration should be made for patients with genetic syndromes that predispose them to the development of TAAs. Annual imaging is recommended for patients with Marfan syndrome if the stability of the aortic diameter is documented.[49]

TABLE 5:

Summary of indications for referral

INDICATION
Medical management is refractory
AAA growth greater than 1 cm per year; TAA growth greater than 0.5 cm per year
Symptomatic AA
Aortic diameter greater than 5.5 cm

More frequent imaging should be considered in patients with an aortic diameter of 4.5 cm or greater or if the aortic diameter shows significant growth.[49] Patients with Loeys-Dietz should obtain complete aortic imaging at initial diagnosis, and at six months to establish if enlargement is occurring.[49] Annually, patients should obtain magnetic resonance imaging from the cerebrovascular circulation to the pelvis.[49] Administration of beta-blockers has been recommended in patients with Marfan's syndrome unless contraindicated.[48] One study examining patients with Marfan syndrome reported that propranolol-treated patients had a 73% lower rate of dilation and mortality than placebo, however, later randomized studies in patients with an AAA failed to report similar findings.[50] Losartan (Cozaar®, Hyzaar®) an angiotensin receptor blocker (ARB), appears to exert a protective effect in a mouse model of Marfan syndrome through modulation of TGF-beta activity. However, no protective effect has been demonstrated in patients with AAAs.[50,51,52]

SUMMARY

Aortic aneurysms occur as a result of medial degeneration of the arterial wall generally as a result of increased aortic hypertension or genetic predisposition. Dedicated imaging studies offer definitive identification or exclusion of potential AAs, but the imaging modality used is largely dependent upon patient-related factors. Medical management of small AAs includes prophylactic beta blocker therapy, ACE inhibitors, ARBs, and statin therapy. There is large variability between patients presenting with an AA, and, therefore, no definitive therapy has been recommended for all patients. Studies evaluating a mouse model of the Marfan syndrome have suggested that Losartan (Cozaar,® Hyzaar®) an angiotensin receptor blocker, is advantageous. Patients for which medical management is refractory, with AAAs that exceed 1 cm of expansion per year or TAAs that exceed 0.5 cm of expansion per year, with symptomatic AA, or with aortic diameters greater than 5.5 cm should be referred for immediate surgical consultation, and postoperative surveillance should be followed accordingly.

AUTHOR DISCLOSURES

No relevant financial affiliations.

REFERENCES

1. Piechota-polanczyk A, Jozkowicz A, Nowak W, et al. The Abdominal Aortic Aneurysm and Intraluminal Thrombus: Current Concepts of Development and Treatment. Front Cardiovasc Med. 2015;2:19.
2. Goldfinger JZ, Halperin JL, Marin ML, Stewart AS, Eagle KA, Fuster V. Thoracic Aortic Aneurysm and Dissection. J Am Coll Cardiol. 2014;64(16):1725-1739. doi:10.1016/j.jacc.2014.08.025
3. Keisler B, Carter C. Abdominal aortic aneurysm. Am Fam Physician. 2015;91(8):538-43.
4. Van der Vliet A. Abdominal aortic aneurysm, The Lancet , Volume 349, Issue 9055 , 863 - 866
5. Vardulaki KA, Walker NM, Day NE, et al. Quantifying the risks of hypertension, age, sex and smoking in patients with abdominal aortic aneurysm. Br J Surg. 2000;87(2):195-200. PMID: 10671927.
6. U.S. Preventive Services Task Force. Screening for abdominal aortic aneurysm: recommendation statement. Ann Intern Med. 2014;161(4):281-290.
7. Jahangir E, Lipworth L, Edwards TL, et al. Smoking, sex, risk factors and abdominal aortic aneurysms: a prospective study of 18 782 persons aged above 65 years in the Southern Community Cohort Study. J Epidemiol Community Health. 2015;69(5):481-8.
8. Centers for Disease Control and Prevention, National Center for Health Statistics. Multiple Cause of Death 1999-2014 on CDC WONDER Online Database, released 2015. Data are from the Multiple Cause of Death Files, 1999-2014, as compiled from data provided by the 57 vital statistics jurisdictions through the Vital Statistics Cooperative Program. Accessed at http://wonder.cdc.gov/mcd-icd10.html on Aug 12, 2016 11:40:38 AM
9. Isselbacher EM. Thoracic and Abdominal Aortic Aneurysms. Circulation. 2005;111:816-828, published online before print February 14, 2005. http://dx.doi.org/10.1161/01.CIR.0000154569.08857.7A
10. Struijk EA, May AM, Wezenbeek NLW, Fransen HP, Soedamah-Muthu SS, Geelen A, Boer JMA, van der Schouw YT, Bueno-de-Mesquita HB, Beulens JWJ (2014) Adherence to dietary guidelines and cardiovascular disease risk in the EPIC-NL cohort. Int J Cardiol 176:354–359. doi:10.1016/j.ijcard.2014.07.017
11. Georgousopoulou EN, Mellor DD, Naumovski N, et al. Mediterranean lifestyle and cardiovascular disease prevention. Cardiovasc Diagn Ther. 2017;7(Suppl 1):S39-S47.
12. Smyth, A., O'Donnell, M., Mente, A. et al. Curr Hypertens Rep (2015) 17: 47. doi:10.1007/s11906-015-0559-8.
13. Sode BF, Nordestgaard BG, Grønbæk M, Dahl M. Tobacco smoking and aortic aneurysm: two population-based studies. Int J Cardiol. 2013;167(5):2271-7.
14. Lederle FA, Johnson GR, Wilson SE, et al. The aneurysm detection and management study screening program: validation cohort and final results. Aneurysm Detection and Management Veterans Affairs Cooperative Study Investigators. Arch Intern Med. 2000;160(10):1425-30.
15. Stanley JC, Veith F, Wakefield TW. Current Therapy in Vascular and Endovascular Surgery. Elsevier Health Sciences; 2014.
16. Hirsch AT, Haskal ZJ, Hertzer NR, et al. ACC/AHA 2005 Practice Guidelines for the management of patients with peripheral arterial disease (lower extremity, renal, mesenteric, and abdominal aortic): A collaborative report from the American Association for Vascular Surgery/Society for Vascular Surgery, Society for Cardiovascular Angiography and Interventions, Society for Vascular Medicine and Biology, Society of Interventional Radiology, and the ACC/AHA Task Force on Practice Guidelines (Writing Committee to Develop Guidelines for the Management of Patients With Peripheral Arterial Disease): Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation; National Heart, Lung, and Blood Institute; Society for Vascular Nursing; Trans-Atlantic Inter-Society Consensus; and Vascular Disease Foundation. Circulation. 2006;113:e463–e654.
17. Erbel R, Aboyans V, Boileau C, et al. Corrigendum to: 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases. Eur Heart J. 2015;36(41):2779.
18. Fink HA, Lederle FA, Roth CS, Bowles CA, Nelson DB, Haas MA. The Accuracy of Physical Examination to Detect Abdominal Aortic Aneurysm. Arch Intern Med. 2000;160(6):833-836. doi:10.1001/archinte.160.6.833.
19. Grey TG. Local discoloration of the abdominal wall as a sign of acute pancreatitis. British Journal of Surgery. 7 (27): 394–395. doi:10.1002/bjs.1800072711
20. Jim J, Thompson RW. Clinical features and diagnosis of abdominal aortic aneurysm. Available at: http://www.uptodate.com/contents/clinical-features-and-diagnosis-of-abdominal-aortic-aneurysm. Accessed September 14, 2016.
21. Assar AN, Zarins CK. Ruptured abdominal aortic aneurysm: a surgical emergency with many clinical presentations. Postgrad Med J. 2009;85(1003):268-73.
22. Nigro G, Giovannacci L, Engelberger S, Van den berg JC, Rosso R. The challenge of posttraumatic thrombus embolization from abdominal aortic aneurysm causing acute limb ischemia. J Vasc Surg. 2011;54(3):840-3.
23. Singla V, Virmani V, Modi M, Kalra N, Khandelwal N. Chronic rupture of abdominal aortic aneurysm with vertebral erosion: an uncommon but important cause of back pain. Ann Vasc Surg. 2014;28(8):1931.e1-4.
24. De Boer NJ, Knaap SFC, de Zoete A. Clinical detection of abdominal aortic aneurysm in a 74-year-old man in chiropractic practice. Journal of Chiropractic Medicine. 2010;9(1):38-41. doi:10.1016/j.jcm.2009.12.002.
25. Greenland P, Alpert JS, Beller GA, et al. 2010 ACCF/AHA guideline for assessment of cardiovascular risk in asymptomatic adults: executive summary: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. Circulation. 2010;122(25):2748-64.
26. Rizvi MM, Singh RB, Jain A, Sarkar A. Asymptomatic aortic aneurysm causing right vocal cord palsy and hoarseness: A rare presentation. Anesth Essays Res. 2014;8(3):397-400.
27. Tsui P, Lee JH, Maclennan G, Capdeville M. Hemoptysis as an unusual presenting symptom of invasion of a descending thoracic aortic aneurysmal dissection by lung cancer. Tex Heart Inst J. 2002;29(2):136-9.
28. Prokakis C, Charoulis N, Tselikos D, Koletsis EN, Apostolakis E, Dougenis D. Primary aortoesophageal fistula due to thoracic aortic aneurysm: successful surgical treatment. Tex Heart Inst J. 2009;36(6):607-10.
29. Taimour S, Zarrouk M, Holst J, et al. Aortic diameter at age 65 in men with newly diagnosed type 2 diabetes. Scand Cardiovasc J. 2017;:1-5.
30. Kristensen KL, Dahl M, Rasmussen LM, Lindholt JS. Glycated Hemoglobin Is Associated With the Growth Rate of Abdominal Aortic Aneurysms: A Substudy From the VIVA (Viborg Vascular) Randomized Screening Trial. Arterioscler Thromb Vasc Biol. 2017;37(4):730-736.
31. Kordzadeh A, Parsa AD, Askari A, Maddison B, Panayiotopoulos YP. Presenting Baseline Coagulation of Infra Renal Ruptured Abdominal Aortic Aneurysm: A Systematic Review and Pooled Analysis. Eur J Vasc Endovasc Surg. 2016;51(5):682-9.
32. Fernandez-bustamante A, Jimeno A. Disseminated intravascular coagulopathy in aortic aneurysms. Eur J Intern Med. 2005;16(8):551-60.
33. Mao SS, Ahmadi N, Shah B, et al. Normal thoracic aorta diameter on cardiac computed tomography in healthy asymptomatic adults: impact of age and gender. Acad Radiol. 2008;15(7):827-34.
34. Lin FY, Devereux RB, Roman MJ, et al. Assessment of the thoracic aorta by multidetector computed tomography: age- and sex-specific reference values in adults without evident cardiovascular disease. J Cardiovasc Comput Tomogr. 2008;2(5):298-308.

35. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with Thoracic Aortic Disease: a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. Circulation. 2010;121(13):e266-369.

36. Makaroun MS, Dillavou ED, Kee ST, et al. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. J Vasc Surg. 2005;41(1):1-9.

37. Chiu KW, Ling L, Tripathi V, Ahmed M, Shrivastava V. Ultrasound measurement for abdominal aortic aneurysm screening: a direct comparison of the three leading methods. Eur J Vasc Endovasc Surg. 2014;47(4):367-73.

38. Sparks AR, Johnson PL, Meyer MC. Imaging of abdominal aortic aneurysms. Am Fam Physician. 2002;65(8):1565-70.

39. Evangelista A, Avegliano G, Elorz C, González-Alujas T, García del Castillo H. Role of Transoesophageal Echocardiography in the Diagnosis of Acute Aortic Syndrome.

40. Ramaiah V, Kpodonu J, Rodriguez-Lopez JA. Endovascular and Hybrid Management of the Thoracic Aorta, A Case-based Approach. Wiley-Blackwell; 2008.

41. Mathur SK, Singh P. Transoesophageal echocardiography related complications. Indian J Anaesth. 2009;53(5):567-74.

42. Popescu BA, Savu O, Beladan CC, Rosça M, Lupescu I, Ginghin C. A paravertebral pulsatile mass. Circulation. 2012;126(24):e344-7.

43. Cronenwett JL, Johnston KW. Rutherford's Vascular Surgery, 2-Volume Set. Elsevier Health Sciences; 2010.

44. Brewster DC, Cronenwett JL, Hallett JW, et al. Guidelines for the treatment of abdominal aortic aneurysms. Report of a subcommittee of the Joint Council of the American Association for Vascular Surgery and Society for Vascular Surgery. J Vasc Surg. 2003;37(5):1106-17.

45. Oladokun D, Patterson BO, Sobocinski J, et al. Systematic Review of the Growth Rates and Influencing Factors in Thoracic Aortic Aneurysms. Eur J Vasc Endovasc Surg. 2016;51(5):674-81.

46. Davies RR, Goldstein LJ, Coady MA, et al. Yearly rupture or dissection rates for thoracic aortic aneurysms: simple prediction based on size. Ann Thorac Surg. 2002;73(1):17-27.

47. Patterson BO, Sobocinski J, Karthikesalingam A, et al. Expansion rate of descending thoracic aortic aneurysms. Br J Surg. 2016;103(13):1823-1827.

48. Ramanath VS, Oh JK, Sundt TM, Eagle KA. Acute aortic syndromes and thoracic aortic aneurysm. Mayo Clin Proc. 2009;84(5):465-81.

49. Elefteriades JA, Farkas EA. Thoracic aortic aneurysm: clinically pertinent controversies and uncertainties. Journal of the American College of Cardiology, 55 (2010), pp. 841–857

50. Danyi P, Elefteriades JA, Jovin IS. Medical Therapy of Thoracic Aortic Aneurysms, Are We There Yet?Circulation. 2011;124:1469-1476, published online before print September 26, 2011.

51. Mohler ER, Woo YJ, Greene CL. Management of thoracic aortic aneurysm in adults. Available at: http://www.uptodate.com/contents/management-of-thoracic-aortic-aneurysm-in-adults. Accessed September 14, 2016.

52. Dalman RL, Mell M. Management of asymptomatic abdominal aortic aneurysm. Available at: http://www.uptodate.com/contents/management-of-asymptomatic-abdominal-aortic-aneurysm. Accessed September 14, 2016.

53. Reimerink JJ, Hoornweg LL, Vahl AC, et al. Endovascular repair versus open repair of ruptured abdominal aortic aneurysms: a multicenter randomized controlled trial. Ann Surg. 2013;258(2):248-56.

54. Greenhalgh RM, Brown LC, Powell JT, et al. Endovascular versus open repair of abdominal aortic aneurysm. N Engl J Med. 2010;362(20):1863-71.

55. Lederle FA, Freischlag JA, Kyriakides TC, et al.; OVER Veterans Affairs Cooperative Study Group. Long-term comparison of endovascular and open repair of abdominal aortic aneurysm. N Engl J Med. 2012;367(21):1988-1997.

56. Ricotta JJ. Endoleak management and postoperative surveillance following endovascular repair of thoracic aortic aneurysms. J Vasc Surg. 2010;52(4 Suppl):91S-9S.

57. Chaikof EL, Brewster DC, Dalman RL, et al. The care of patients with an abdominal aortic aneurysm: the Society for Vascular Surgery practice guidelines. J Vasc Surg. 2009;50(4 Suppl):S2-49.

58. Zamor KC, Eskandari MK, Rodriguez HE, Ho KJ, Morasch MD, Hoel AW. Outcomes of Thoracic Endovascular Aortic Repair and Subclavian Revascularization Techniques. J Am Coll Surg. 2015;221(1):93-100.

Giant Cell Arteritis

Leonid Skorin, Jr., DO, OD, MS, FAAO, FAOCO[1] & Rebecca Lange, OD[2]

[1] Mayo Clinic Health System, Albert Lea, MN
[2] Whidbey Vision Care, Oak Harbor, WA

KEYWORDS:

Diplopia

Giant Cell Arteritis

Headache

Ophthalmology

Temporal Arteritis

Temporal Artery Biopsy

Vision Loss

Giant cell arteritis, also known as temporal arteritis, is a condition that can present in patients with a headache, scalp tenderness, anemia, jaw claudication, diplopia or sudden severe vision loss. The main differential diagnosis is non-arteritic anterior ischemic optic neuropathy. Upon suspected diagnosis of giant cell arteritis, laboratory workup for erythrocyte sedimentation rate, C-reactive protein, and complete blood count are performed. A temporal artery biopsy serves as confirmatory evidence of the disease. The immediate treatment for suspected giant cell arteritis is systemic steroids. This article will review giant cell arteritis, its pathophysiology, patient symptomatology, differential diagnosis, and treatment. Included in this review will be a video of a temporal artery biopsy.

INTRODUCTION

Giant cell arteritis (GCA), also known as temporal arteritis, is a chronic, granulomatous, inflammatory disorder with a predilection for the elastic tissue-rich, medium-to-large arteries in the head and neck.[1] Giant cell arteritis is a true ocular emergency.[2] Although GCA will likely present to an eye care practitioner when its most severe symptom of sudden vision loss occurs, it will often present initially to the primary care physician as a patient with a new onset headache, scalp tenderness or jaw claudication.[2] The goal of GCA management is to identify the disease before arteritic ischemic optic neuropathy, or permanent vision loss develops. Thus, it is important that every primary care physician be familiar with the symptoms, laboratory testing, and treatment of GCA.

EPIDEMIOLOGY

The likelihood that a primary care physician will be involved in the care of someone with GCA is high since this disease is the most common systemic vasculitis in North America.[3] The disease mainly affects Caucasians, especially those of Scandinavian descent.[2] It is rare in African Americans, Native Americans or those of Asian descent.[2] Age is the greatest risk factor for the disease with an overall prevalence of about 1 in 750 of those over the age of 50.[4] This prevalence increases with age and peaks in the age group 70-79 years.[5] Specifically, the incidence rates per 100,000 in the population increases from two individuals in the 50-59 year age group to 52 individuals in the age group 80 years and older.[5] Women are more commonly affected than men, by two to three times.[2] Other risk factors include those who have had a recent stroke or myocardial infarction, congestive heart failure, aortic aneurysm, anemia, or polymyalgia rheumatica.[1]

SYMPTOMS & SIGNS

A new onset headache, often in the temporal region and usually occurring on one side, is the most common symptom presenting to a primary care physician. Palpation along the temple will often denote a palpable, tender, nonpulsatile temporal artery. Other common symptoms include scalp tenderness, pain when brushing or styling one's hair and jaw claudication, particularly when chewing.[1] Recent weight loss or diplopia may also be reported.[1] Less common ailments may include general malaise or fatigue, changes in mental status, ear, throat or neck pain or facial swelling[6] *(Table 1, page 18)*.

Ocular Signs

Significant visual acuity loss, often resulting in counting fingers or worse vision, is a hallmark sign that the disease is progressing. Amaurosis fugax, which is a painless transient vision loss or blackening of vision, may occur in up to 40% of patients before permanent vision loss.[7] Pupil abnormalities, including tonic pupil, light- near dissociation (pupil does not constrict to light but does constrict to convergence at near), miotic Horner's syndrome pupil, or an afferent pupillary defect may also be noted.[6] Anterior segment examination may infrequently show ischemia including corneal

CORRESPONDENCE:
Leonid Skorin Jr., DO, OD, MS, FAAO, FAOCO
Skorin.Leonid@mayo.edu

 Print ISSN: 1877-573X

edema or fine keratic precipitates.[8] A posterior segment examination may show a pale, swollen optic disc that may also have flame-shaped hemorrhages, known as arteritic ischemic optic neuropathy (AION)[6] (Figure 1 and Figure 2 show AION with flame-shaped hemorrhages at two different time points).

DIFFERENTIAL DIAGNOSES

Non-arteritic ischemic optic neuropathy (NAION) is the most common ocular differential diagnosis from GCA, when it presents as AION. Patients with NAION are usually slightly younger than the typical AION patient, lack the subsequent complaints of those with GCA and usually present with less severe vision loss. NAION may have sectorial or segmental optic nerve edema (Figure 3) while patients with AION will have a pallid swollen disc.

Other ocular differentials include inflammatory optic neuritis which typically has pain on eye movements, compressive optic nerve tumor which presents with slowly progressive vision loss and retinal vascular occlusions such as vein occlusions that present with multiple diffuse retinal hemorrhages or artery occlusions, which present with retinal edema and a red macula (cherry red spot).[6]

TABLE 1:

Signs and Symptoms of Giant Cell Arteritis

	SYSTEMIC	OCULAR
Symptoms	- New headache - Scalp tenderness - Pain with hair styling - Jaw claudication - Recent weight loss - General malaise - Ear, throat, neck pain - Facial swelling	- Vision loss - Diplopia - Amaurosis fugax - Eye pain
Signs	- Palpable, tender, nonpulsatile temporal artery - Anemia - Change in mental status - Recent myocardial infarction - Congestive heart failure - Aortic aneurysm	- Abnormal pupils - Afferent pupillary defect - Anterior segment ischemia - Pale, swollen optic disc - Visual field defect - Central retinal artery occlusion - Cranial nerve 3, 4 or 6 palsy

A systemic differential is polymyalgia rheumatica (PMR). PMR is a disease of adults over age 50, with a prevalence that increases progressively with advancing age.[9] The lifetime risk of PMR is second only to rheumatoid arthritis as a systemic rheumatic disease in adults.[10] The primary presenting symptom of PMR is new-onset bilateral shoulder pain and stiffness.[11] Other symptoms include fatigue, depression, weight loss or a low-grade fever.[11] Many of these symptoms overlap with GCA. The overlap is considerable as 16-21% of patients with PMR have GCA on temporal artery biopsy, and symptoms of PMR are present in 40-60% of patients with GCA.[12]

Since a headache is a common finding in GCA, migraine and cluster headache should be considered in the differential. Both of these headache types have a much earlier age of onset, usually before the age of 40.[13] Also, the character of the headache types is different from a GCA headache that is usually painful to the touch in the temple region. A migraine is a unilateral, throbbing head pain or pulsing sensation, which may be preceded or accompanied by visual alterations, difficulty speaking, numbness of the face, nausea, vomiting and extreme sensitivity to light and sound.[14] Cluster headaches often wake the patient from sleep with a very rapid onset and peak intensity. They exhibit a boring or penetrating pain, and patients often have accompanying nasal congestion, lacrimation, rhinorrhea and conjunctival edema and injection.[15]

Jaw claudication as seen in GCA results from ischemia of the masseter muscle during chewing. Temporomandibular disorder (TMD) can mimic jaw claudication, but patients have the greatest risk of onset between the ages of 18 and 44.[16] Instead of ischemia, TMD symptoms are characterized by acute or chronic musculoskeletal pain triggered by jaw motion, dysfunction of the masticatory system, temporomandibular joint (TMJ) tenderness and cervical spinal dysfunction.[17] Clicking or popping noises with jaw function is a common finding with TMJ dysfunction.

PATHOPHYSIOLOGY

Giant cell arteritis is a vasculitis with a preference for the extracranial branches of the carotid artery including the temporal arteries, the terminal branches of the ophthalmic artery and the posterior ciliary arteries.[1] An integral part of this disorder is the presence of small calcifications within the internal elastic membrane (Figures 4, 5, and 6). The body reacts to these small calcifications with a release of foreign-body giant cells that accumulate and break down the internal elastic membrane calcifications.[18] This accumulation causes lymphocytes to surround these calcifications resulting in a widespread inflammatory reaction cascade. Following this event, lymphocytes are joined by plasma cells, mononuclear cells, multinucleated giant cells, and eosinophils, all of which obstruct the laminae of the involved vessels.[2,18]

WORK-UP

When a physician suspects the diagnosis of GCA, laboratory tests including C-reactive protein (CRP), complete blood count (CBC) and erythrocyte sedimentation rate (ESR) should be ordered immediately. Those with temporal arteritis will likely have a markedly elevated ESR (over 40mm/hr in 90% of confirmed cases).[2] However, ESR is dependent on age and sex and can be affected by anemia, which also affects many of those with temporal arteritis. Ten

FIGURE 1:

Arteritic ischemic optic neuropathy showing pallid swollen optic disc. Day 1, initial presentation

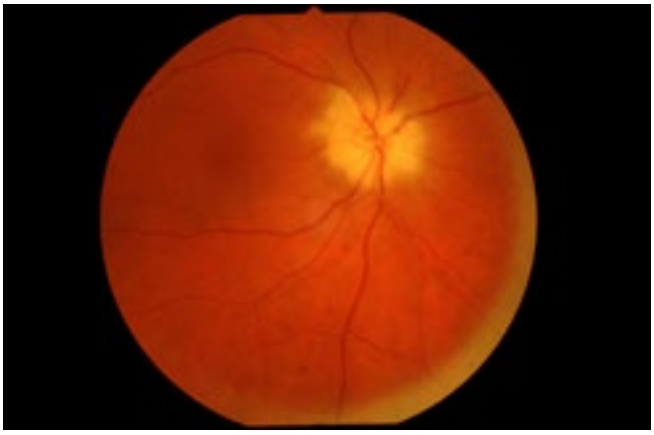


FIGURE 2:

Resolving disc edema after initiating steroid treatment. Day 19 presentation

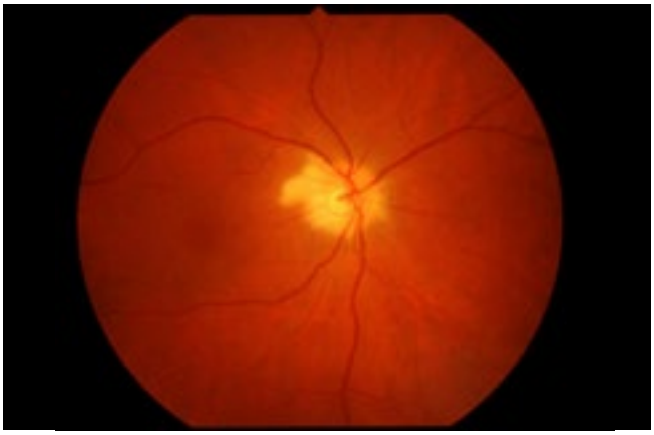


FIGURE 3:

Non-arteritic ischemic optic neuropathy showing segmental optic disc edema (arrow)

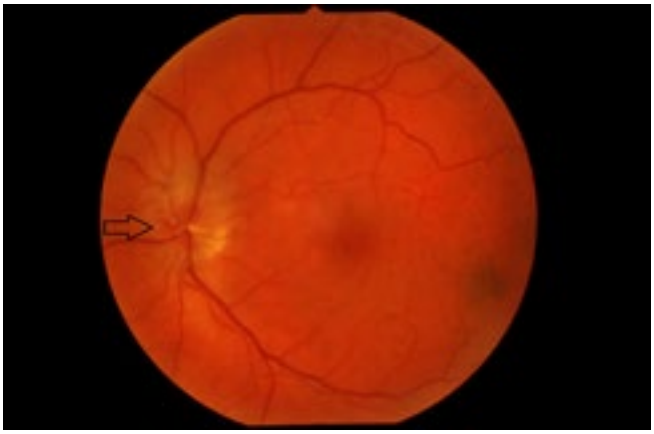


FIGURE 4:

Histology slide of normal temporal artery

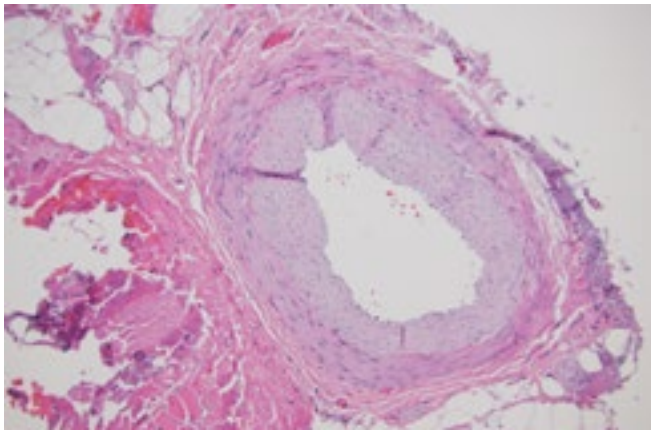


FIGURE 5:

Histology slide of artery with temporal arteritis

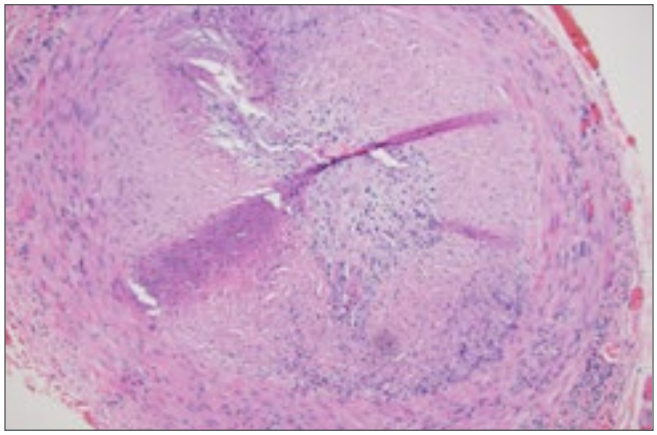
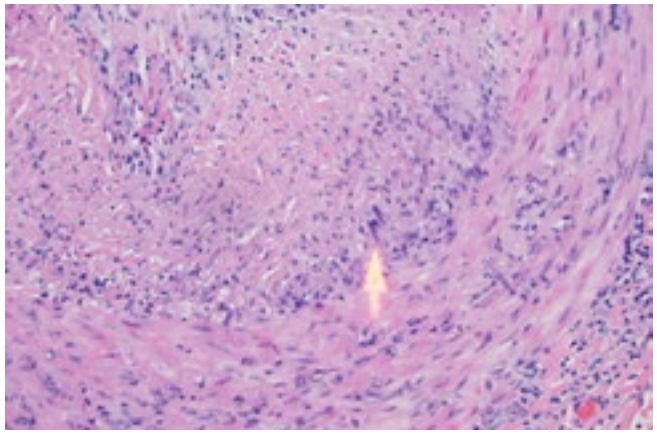


FIGURE 6:

Histology showing changes of lumen and increased vessel wall thickness with multinucleated giant cells (arrow)



to 15% of those with GCA may still have a normal ESR.[19] In patients who have the signs and symptoms of GCA yet a normal ESR value, a temporal artery biopsy should still be performed.[19] An elevated CRP value is also very likely in those with GCA. The CBC values may show mild normochromic normocytic anemia, normal leukocytes and differential count and an elevated platelet count.[9]

If the patient's laboratory findings are positive, oral prednisone should be initiated. If the patient also has ocular symptoms, they should be referred to an ophthalmologist or neuro-ophthalmologist. If there are no ocular complications referral to a rheumatologist or neurologist is indicated. Also, a temporal artery biopsy needs to be obtained within one week. A number of different surgical specialists can usually perform the biopsy. These would include surgeons specializing in ophthalmology, otorhinolaryngology, general or vascular surgery, plastics, oromaxillofacial or neurosurgery.

The definitive test for GCA is a temporal artery biopsy. The likelihood of a positive temporal artery biopsy is 1.5 times greater with an ESR of 47-107mm/hr, 5.3 times greater with a CRP greater than 2.45mg/dL and 4.2 times greater with a platelet count over 400,000µL.[20] If all three of these lab values are elevated, the likelihood of a positive temporal artery biopsy is eight times greater.[20] It is also suggested that there is a higher predictive power for a positive temporal artery biopsy if both CRP and platelets are elevated when compared to an elevated ESR alone.[20]

A color-duplex ultrasonography can be useful in assessing vascular inflammation. The accessible large arteries and the superficial temporal artery can be examined to look for the characteristic "halo sign" noted on ultrasonography.[11] This "halo sign" is a hypoechoic ring around the arterial lumen that shows the edematous thickening of the arterial wall due to inflammation.[11] While ultrasonography is noninvasive and may seem efficient, difficulties arise due to differences in criteria, technical equipment and the evaluation of different vessels.[11] Ultrasonography is thus used to supplement laboratory values and a temporal artery biopsy.

In addition, angiography of the aortic arch and its branches may serve to diagnose large-vessel involvement. Magnetic resonance angiography as well as magnetic resonance imaging of the cranium and chest can show the presence of increased aortic wall thickness and edema.[21] Increased thickness and edema are thought to suggest vascular inflammation, as is the hallmark of GCA. It has been suggested that imaging such as these may help to both diagnose and monitor the disease. Although the temporal artery biopsy is still the gold standard for diagnosis, these noninvasive modalities are helpful in monitoring GCA.[21]

TEMPORAL ARTERY BIOPSY

In suspected cases of GCA, a temporal artery biopsy should be performed on the side ipsilateral to any vision loss or ipsilateral to the most symptoms.[2] Due to potential vision loss, a physician should not wait for the biopsy results to initiate treatment. The biopsy is performed under monitored conscious sedation.[2] The portion of the frontal branch of the temporal artery to be excised is palpated, its course confirmed with ultrasonography, and then it is marked using a marking pen. A local anesthetic is injected along either side of the vessel markings. A #15 Bard-Parker blade is used to make the initial dermal incision, following the skin markings. Blunt dissection is performed to isolate the vessel. Cautery is applied as necessary to maintain hemostasis. When the artery has been localized, the patient is checked for ischemia by compressing the vessel. Titanium clips are used to clamp both the distal and proximal ends of the artery before it is excised (Figure 7). A specimen should be at least 5mm in length with the optimal length being 15mm, as skip lesions that contain no inflammation, may occur.[2,22] Also, there is a disagreement rate of approximately 23% between the right and left temporal arteries, so a negative biopsy result does not necessarily rule out the disease.[1] The subcutaneous tissue is closed with dissolvable 4-0 chromic suture. The incision is sealed with tissue adhesive and butterfly closures. A pressure dressing is then applied. The pressure dressing is removed in 24 hours.

TREATMENT

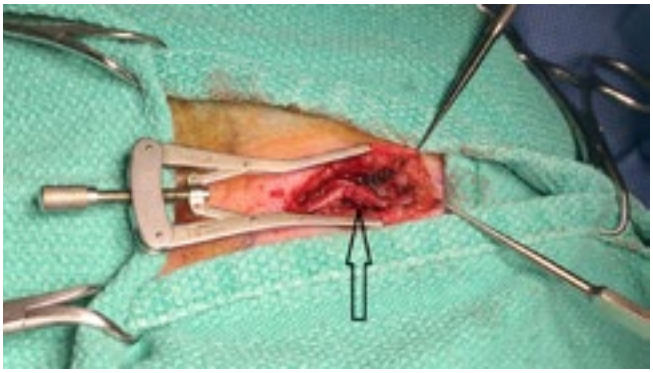
The traditional treatment for GCA is high dose oral steroids (100mg prednisone daily) and should be prescribed immediately after the diagnosis of GCA is suspected. In many cases, this will be able to provide complete symptomatic relief within 24-48 hours.[21] However, if vision loss is the presenting symptom, intravenous methylprednisolone 1-2g for three to five days is the recommended initiating treatment, followed by high dose oral steroids.[6] The physician should monitor both ESR and CRP levels every two to three weeks to assess treatment progression. If laboratory values suggest the disease is resolving, the steroids should be slowly tapered.[1] If the temporal artery biopsy is found to be negative, steroids should be stopped, and the patient should be investigated by the primary care physician for other causes of symptoms. If the temporal artery biopsy is found to be negative but the patient continues to present with persistent and significant signs and symptoms of GCA, the temporal artery on the opposite side should be biopsied. There are no known suggested osteopathic practices or principles for the treatment of GCA.

A new treatment, tocilizumab, was approved by the FDA in May 2017 for the treatment of GCA. Tocilizumab is an interleukin-6 receptor antagonist.[23] It was first approved for the treatment of adult rheumatoid arthritis in patients who have used at least one disease-modifying antirheumatic drug that was unable to provide necessary relief. When used in patients with GCA, those who received subcutaneous tocilizumab administered with oral prednisone had improved treatment outcomes over patients taking oral prednisone and placebo.[23] Sustained remission at week 52 occurred in 56% of the patients treated with tocilizumab weekly and in 53% of those treated with tocilizumab every other week, as compared with 14% of those in the placebo group that underwent a 26-week prednisone taper and 18% of those in the placebo group that underwent a 52-week prednisone taper.[23] Sustained remission was defined as an absence of symptoms of GCA, normal ESR and CRP laboratory tests, and tapering the use of oral prednisone.[23]

The key to a good prognosis in GCA is prompt proper therapy and management.[21] With treatment, a life expectancy similar to the general population is expected for most patients who have not had visual symptoms.[24] Those with vision loss from GCA usually will not recover their vision.[25] The goal of treatment is to stabilize and preserve vision in the unaffected eye.[2] Relapses of GCA are common with about one-third of patients having symptoms return.[26]

FIGURE 7:

Surgical image of temporal artery (arrow) before excision



Patients should be treated with the dose of oral prednisone given before relapse or a higher dose.[27] While relapses are most likely to occur within the first 18 months of treatment, they may also occur later.[26]

CONCLUSION

Giant cell arteritis is a chronic vasculitis that although often has visual symptoms, may present to the primary care physician first. It is important to be familiar with the signs and symptoms of this disease as initiating early treatment is essential for visual stability and to maintain a high quality of life. The most common symptoms are new onset headache, temporal pain, scalp tenderness, and jaw claudication.[1] When GCA is suspected, an inflammatory laboratory work-up should be immediately ordered including ESR, CRP and a full CBC. Also, a temporal artery biopsy should be ordered as it is still the gold standard for confirmation of the disease. As long as treatment is initiated right away, most patients experience remission of their symptoms.

View temporal artery biopsy video bit.ly/ofp_video

AUTHOR DISCLOSURES

No relevant financial affiliations.

REFERENCES:

1. Salvarani C, Cantini F, Boiardi L, et al. Medical progress: polymyalgia rheumatica and giant-cell arteritis. N Engl J Med. 2002; 347: 261-271.
2. Nesher G. The diagnosis and classification of giant cell arteritis. J Autoimmun. 2014; 48-49:73-75.
3. Gonzalez-Gay M, Vazquez-Rodriguez T, Lopez-Diaz M, et al. Epidemiology of giant cell arteritis and polymyalgia rheumatica. Arthritis & Rheumatism. 2009; 61: 1454-1461.
4. Boesen,P., Sorensen, S.F. Giant cell arteritis, temporal arteritis, and polymyalgia rheumatic in a Danish county. Arthritis Rheum. 1987; 30: 294-299.
5. Salvarani, C, Crowson, CS, O'Fallon, WM, et al. Reappraisal of the epidemiology of giant cell arteritis in Olmsted County, Minnesota, over a fifty-year period. Arthritis Rheum, 2004; 54: 264-268.
6. Gerstenblith AT, Rabinowitz, M P. Arteritic Ischemic Optic Neuropathy and Nonarteritic Ischemic Optic Neuropathy. In: The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. 6th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2012; 274-277.
7. Gonzalez-Gay MA, Blanco R, Rodriguez-Valverde V, et al. Permanent visual loss and cerebrovascular accidents in giant cell arteritis: predictors and response to treatment. Arthritis Rheum. 1998; 41:1497-1504.
8. McKillop E, Tejwani D, Weir C, et al. Anterior segment ischaemia with giant cell arteritis. Can J Ophthalmol. 2006; 41:201–203.
9. Salvarani C, Gabriel SE, O'Fallon WN, et al. Epidemiology of polymyalgia rheumatic in Olmsted County, Minnesota, 1970-1991. Arthritis Rheum. 1995; 38:369-373.
10. Crowson CS, Matteson EL, Myasaedova E, et al. The lifetime risk of adult-onset rheumatoid arthritis and other inflammatory autoimmune rheumatic disease. Arthritis Rheum. 2011; 63: 633-639.
11. Buttgereit F, Dejaco C, Matteson EL, et al. Polymyalgia rheumatica and giant cell arteritis: a systematic review. JAMA. 2016; 315: 2442-2458.
12. Salvarani C, Cantini F, Hunder GG. Polymyalgia rheumatic and giant cell arteritis. Lancet. 2008; 372: 234-245.
13. Lipton RB, Bigal ME, Diamond M, et al. Migraine prevalence, disease burden, and the need for preventive therapy. Neurology. 2007; 68:343-349.
14. Weatherall MW. The diagnosis and treatment of chronic migraine. Therapeutic Advances in Chronic Disease. 2015; 6: 115-123.
15. Weaver-Agostoni J. Cluster headache. Am Fam Physician. 2013; 88: 122-128.
16. Von Korff M, Le Resche L, Dworkin SF. First onset of common pain symptoms: a prospective study of depression as a risk factor. Pain. 1993; 55: 251-258.
17. Mehta NR, Forgione AG, Rosenbaum RS, et al. "TMJ" triad of dysfunctions: a biologic basis of diagnosis and treatment. J Mass Dent Soc. 1984; 33: 173-176, 212-213.
18. Nordborg C, Nordborg E, Petursdottir V. The pathogenesis of giant cell arteritis: Morphological aspects. Clin Exp Rheumatol. 2000; 18: S18-S21.
19. Keltner JL. Giant-cell arteritis: signs and symptoms. Ophthalmology. 1982; 89:1101-1110.
20. Walvick MD, Walvick MP. Giant cell arteritis: Laboratory predictors of a positive temporal artery biopsy. Ophthalmology. 2011; 118:1201–1204.
21. Ponte C, Rodrigues AF, O'Neill L, et al. Giant cell arteritis: Current treatment and management. World J Clin Cases. 2015; 3: 484-494.
22. Oh LJ, Wong E, Gill AJ, et al. Value of temporal artery biopsy length in diagnosing giant cell arteritis. ANZ J Surg. 2016; 1-5. DOI:10.1111/ans.13822.
23. Stone JH, Tuckwell K, Dimonaco M, et al. Trial of tocilizumab in giant-cell arteritis. N Engl J Med. 2017; 377: 317-328.
24. Matteson EL, Gold KN, Bloch DA, et al. Long-term survival of patients with giant cell arteritis in the American College of Rheumatology giant cell arteritis classification criteria cohort. Am J Med. 1996; 100: 193-196.
25. Rahman W, Rahman FZ. Giant cell (temporal) arteritis: an overview and update. Surv Ophthalmol. 2005; 50: 415-428.
26. Charlton R. Optimal management of giant cell arteritis and polymyalgia rheumatica. Ther Clin Risk Manag.2012; 8: 173-179.
27. Pountain G, Hazleman B. ABC of rheumatology: polymyalgia rheumatic and giant cell arteritis. BMJ. 1995; 310: 1057-1059.

IT'S MORE THAN A JOB

As an osteopathic family physician, every day is meaningful.

The ACOFP Career Center can help you find your perfect job. You can inventory your skills and accomplishments, proactively manage your career, and create a professional action plan tailored to your goals.

Jump start your career by adding or updating your professional profile today and gain access to valuable tools and resources.

To find a job or fill a position, visit www.acofp.org.

acofp American College of Osteopathic Family Physicians
Advocacy • Education • Leadership

TAKE ADVANTAGE OF YOUR MEMBER BENEFITS ON THE GO!

Download the ACOFP app to access the latest news and upcoming events. You can view CME resources, the OFP Journal, the ACOFP Career Center and more!

Download today!
www.acofp.org

Available on the iPhone App Store

Google play

acofp American College of Osteopathic Family Physicians

Paronychia

Korinn Vandervall OMS III,[1] Mary Ann Yehl DO,[2] & Lindsay Tjiattas-Saleski DO, MBA, FACOEP[3]

[1] VCOM Carolinas, Spartanburg, SC
[2] Family Medicine Red Bank, NJ
[3] Greenville Health System Emergency Department, Greenville, SC

A 73-year-old female with past a medical history of diabetes mellitus type II, hypertension and thyroid disease presented to the Urgent Care Center with right distal thumb pain and swelling (Figure 1). She had been seen two days prior, diagnosed with paronychia and prescribed cephalexin 500mg three times daily. She reported no improvement in the antibiotics and warm soaks. She did not report any associated fevers or chills. On her second visit, incision and drainage were performed. She discontinued cephalexin, and trimethoprim/sulfamethoxazole was started.

QUESTIONS

1. Acute paronychia treatment choices include:
   a. Warm water soaks 3-4 times daily
   b. Incision and drainage for abscesses
   c. Antibiotics for abscess or high-risk patients
   d. Only a and b
   e. All of the above

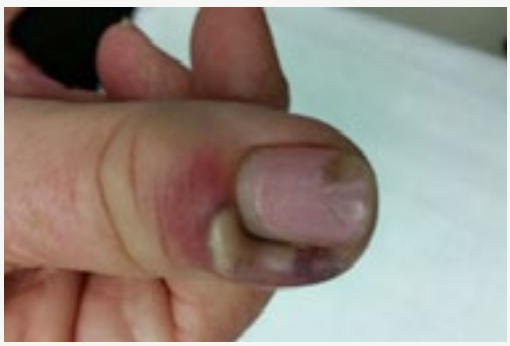
2. What are the risk factors for Acute Paronychia?
   a. Nail biting
   b. Diabetes Mellitus
   c. Artificial Nails
   d. HIV
   e. All of the Above

3. What are the recommended treatments for chronic paronychia?
   a. Discontinue inciting activity
   b. Antifungals Topical Steroids
   c. Amputation
   d. A and B
   e. A and C

4. The best antibiotic choices for acute paronychia induced from nail biting are:
   a. Vancomycin
   b. Clindamycin
   c. Clarithromycin
   d. Levofloxacin
   e. Azithromycin

5. Your resident explains he saw an acute paronychia and wishes to perform an incision and drainage. You note the patient has nail fold pain with a cluster of yellow lesions on an erythematous base. You stop him from performing because the diagnosis is:
   a. Chronic paronychia
   b. Herpetic whitlow
   c. Squamous cell carcinoma
   d. Felon
   e. Onychomycosis

FIGURE 1:
Thumb view



CORRESPONDENCE:
Lindsay Tjiattas-Saleski DO, MBA, FACOEP
LTjiattas-Saleski@ghs.org

ANSWERS

1. Acute paronychia treatment choices include:

   The correct answer is E) All of the above

   Patients with a mild paronychia infection without abscess may benefit from warm water soaks alone. Once an abscess develops, an incision and drainage are generally required to promote healing. Antibiotics are given to cover suspected organisms based on medical history.

2. What are the risk factors for Acute Paronychia?

   The correct answer is E) All of the Above

   Any condition that disrupts the integrity of the nail bed can predispose patients to acquiring acute paronychia. These risk factors include; nail biting, finger biting or sucking, manicures or artificial nail application. Medical conditions that compromise immunity such as diabetes, HIV, medications and chemotherapy can increase the risk of paronychia as well.

3. What are the recommended treatments for chronic paronychia?

   The correct answer is D) A and B

   Chronic paronychias are a result of repetitive trauma, inflammation, or exposure. This leads to an infection, most commonly, with candida. Treatment includes discontinuing the irritation or contact. Topical steroids to treat the dermatitis aid in the healing of nail folds, cuticle structure, and removal of habitat for candida. Antifungals are considered for resistant treatment, the antifungals can aid in reduced inflammation due to concomitant fungal infection if present.

4. The best antibiotic choices for acute paronychia induced from nail biting are:

   The correct answer is B) Clindamycin

   When choosing an antibiotic for paronychia, one must consider the patient's medical history, severity, and duration of infection. A patient who has had oral exposure from nail-biting or finger sucking needs coverage for anaerobic organisms. Vancomycin would be a good choice for an extensive infection that is suspected methicillin-resistant Staphylococcus aureus (MRSA) for a hospitalized patient. Clarithromycin, levofloxacin, and azithromycin would not provide coverage that is required.

5. Your resident explains he saw an acute paronychia and wishes to perform an incision and drainage. You note the patient has nail fold pain with a cluster of yellow lesions on an erythematous base. You stop him from performing because the diagnosis is:

   The correct answer is B) Herpetic whitlow

   All nail fold infections are not paronychias and its imperative to consider the differential diagnosis. Any vesicular lesion should be evaluated for herpes. Chronic paronychia appears as erythematous areas to nail folds. Squamous cell carcinoma presents typically as a tumor, ulceration, or fungating masses. A felon is a localized compartment syndrome at the palmar finger pad. Onychomycosis presents with irregular texture and color changes to the nail.

DISCUSSION

Paronychia is an infection of the nail fold, which can be an acute or chronic process.[1,2] This infection is associated with trauma or injury to the nail folds, which allows bacteria to invade.[1] Paronychia represents 35% of hand infections making it the most common hand infection in the United States.[3]

Acute paronychia is a painful pyogenic infection that presents with a rapid development of finger or toe pain, over the course of several hours to a few days.[1,2,4] Physical examination findings can vary from a red and tender swelling of the nail fold to obvious cellulitic changes or abscess formation.[4] Acute paronychia affects males and females equally, with a higher prevalence in children.[5] Risk factors include trauma to skin surrounding the nail, ingrown nails, manicured/sculptured nails, and thumb/finger sucking.[2] Repetitive biting of fingers and thumb sucking causes microtrauma allowing colonization of bacteria, most commonly Staphylococcus aureus but also Streptococcus pyogenes, Pseudomonas pyocyanea, and Proteus vulgaris.[2,5] If oral flora is part of the exposure the clinician should also consider aerobic Eikenella corrodens and anaerobic Fusobacterium and Peptostreptococcus.[2] The diagnosis of acute paronychia is clinical and should be considered when a patient presents with redness, pain, and warmth that developed acutely along the nail margin.[6]

Chronic paronychia is a progressive, inflammatory disorder lasting for more than six weeks and affects more than one nail fold.[1,8] It usually affects older individuals around the 5th and 6th decade, with a higher prevalence in females as compared to males.[5] Chronic paronychia affects patients that tend to have constant exposure to wet conditions including cooks, chefs, bartenders, housekeepers, and swimmers.[2,7] It is caused by many of the same mechanisms as acute paronychia but over an extended period.[1,7,8] The exposure to repetitive trauma and moist conditions leads to a multifactorial inflammation, usually dermatitis, that can become infected.[1,7,8] Chronic paronychia presents as a tender erythema and thickening of the nail folds, followed by loss of the cuticle.[1] These nail and tissue changes can lead to dystrophy of the nail plate.[1,2] Patients often have co-infection with Candida about 95% of the time, and less frequently may have co-infection with dermatophytes and molds such as Scytalidium Fusarium.[2] Unless preventative measures are initiated, the cycle of irritation and nail changes will continue.[1,7,8]

Paronychia can also be drug-induced, occurring with systemic retinoids (isotretinoin, etretinate), topical retinoids (tretinoin, tazarotene), antiretroviral drugs (indinavir), epidermal growth factor (EGF) receptor inhibitors (gefitinib and cetuximab), and anticancer mTOR inhibitors (everolimus).[1,9] Patients with drug-induced paronychia present with erythematous, swollen, and painful nail fold(s), shortly after beginning treatment with a new medication.[9] These lesions heal gradually after cessation of treatment and can be followed by onychomadesis, (proximal shedding of nail).[9]

DIFFERENTIAL DIAGNOSIS

Since paronychia is a clinical diagnosis, it is important to keep in mind other skin conditions that may present similarly including, but not limited to the following:

1. Felon: a localized compartment syndrome of the distal phalanx to the volar skin the finger pulp, generally from penetrating trauma.[2,10] Patients present with a tense, tender, swollen, erythematous finger pad, and severe throbbing pain.[8] Treatment involves incision and drainage.[10]
2. Herpetic whitlow: presents as a single vesicle or cluster of vesicles that arise on a digit after 3-4 days of skin irritation or minor trauma that have a clear or pale yellow appearance on an erythematous base.[11] The lesions are frequently located on the terminal phalanx of thumb, index, or long finger near the nail.[11]
3. Proximal onychomycosis: is a fungal infection of the nail that includes thickening, splitting, roughening, and discoloration of the nail.[6,12] Diagnosis is with KOH preparation for direct microscopy and isolation of organism in culture.[12]
4. Pyoderma gangrenosum: is a skin disorder that can occur on any location of the body, including the fingers and toes, and can develop as an erythematous pustule or nodule.[6,12] This lesion then rapidly progresses to form a necrotic ulcer.[12]

TREATMENT

Acute paronychia requires quick and effective treatment to prevent damage to the nail matrix.[1] If there is no evidence of abscess formation, treatment should start with warm compresses or antibacterial soap soaks of the affected digit three to four times a day for 10-15 minutes.[2,13] The patient can apply a topical anti-staphylococcal antibiotic, such as mupirocin, gentamicin, neomycin, or polymyxin B for 5-10 days.[2,13] Aluminum acetate soaks can also help reduce edema and provide a hostile environment to bacteria.[1,2]

If acute paronychia presents with abscess formation, incision and drainage are indicated.[2,13] Antibiotics are not routinely required after performance of incision and drainage.[4] However, if marked cellulitis or a possible MRSA infection is suspected, specific antibiotic coverage is determined by the exposure.[4] If exposure to oral flora is suspected, treatment includes clindamycin 300-450 mg three to four times daily for seven days, amoxicillin-clavulanate 875/125mg every 12 hours or 500/125mg three times a day for seven days.[2,5] Without oral flora exposure, treatment includes dicloxacillin 250mg three times a day for seven days or cephalexin 500mg two to three times a day for seven days.[2] If MRSA is suspected, treat with trimethoprim/sulfamethoxazole 160/800mg two times a day for seven days or doxycycline 100 mg two times a day for seven days.[2,5] If there is no clinical response to incision and drainage and antibiotic therapy after 48 hours, surgical intervention under local anesthesia may be required, commonly due to a deeper infection.[1]

Chronic paronychia tends to be much more challenging to resolve.[7] Treatment success is contingent on lifestyle changes as well as education on proper hand protection to prevent the recurrent microtrauma and irritant exposure.[1,2,8] If patients have exposure to unsanitary or dirty conditions, hands should be washed and dried thoroughly.[7] If hands tend to become too dry, patients can apply a hand cream or moisturizer after washing.[7] In the past, treatment has focused on antifungals, but chronic paronychia is currently understood to be a form of hand dermatitis from environmental exposure, not necessarily due to the Candida infection itself.[8] Typically, once the nail barriers are healed the Candida species are no longer able to colonize.[14] Topical steroid creams are found to be the most efficacious with first-line treatment including moderately potent to potent topical steroids such as betamethasone 0.05% applied two times a day for 7-14 days or methylprednisolone aceponate 0.1% cream at bedtime.[2,8,14] An alternative treatment choice for the topical steroids includes include tacrolimus 0.1% ointment twice a day for 3-6 weeks.[8,14] If a fungal co-infection is suspected, treatment can consist of topical econazole, clotrimazole, or nystatin up to three times a day.[1,2,8] Treatment can be continued until a decrease in inflammation and noted reattachment of cuticle, which can take up to 3 months.[1,2,8] Systemic antifungals are rarely needed, but for more severe cases that include the nail, longer courses of antifungals such as itraconazole, terbinafine or fluconazole may be indicated.[2] Surgical management for severe cases is considered after failure of conservative measures and medical therapy.[1,8] Surgical procedures include eponychial marsupialization, en bloc excision of proximal nail fold, and Swiss roll technique.[1,8]

PREVENTION & PROGNOSIS

Prevention is best accomplished with patient education.[3] It is essential for patients to refrain from causing trauma to the fingernails by avoidance of nail biting, finger sucking, excessive manicuring, cutting of cuticles, and adhering to proper nail care.[3,15] Avoidance of excessive exposure of hands to damp conditions and wearing rubber gloves with a cotton liner may also assist with prevention.[2] Nail trimming should involve use of clean clippers or sharp manicure scissors to form a rounded edge for fingernails and straight across for toenails.[15] Trimming after a bath or shower makes the nails easier to trim, and cuticles should not be manipulated as this can introduce bacteria to the region.[15]

With prompt treatment, paronychia has a good prognosis.[3] Acute paronychia usually clears entirely in a few days and is rarely recurrent.[7] If left untreated infection can worsen and potentially (but rarely) lead to complications such as felon formation, septic tenosynovitis and osteomyelitis.[3] Immunosuppressed patients and those that have neglected to obtain proper treatment may be more prone to complications. Chronic paronychia may require months of treatment for clearance, and it can take up to a year for nails to resume normal growth.[7]

AUTHOR DISCLOSURES
No relevant financial affiliations.

REFERENCES

1. Richard B. Mallett. Paronychia . In: Mark G. Lebwhol MD, Warren R. Heymann, MD, John Berth-Jones, FRCP, Ian Coulson BSc, MB, FRCP, 4th ed. *Treatment of Skin Disease: comprehensive therapeutic strategies*. 4th ed. Edinburgh: Sauders Elsevier Limited; 2014: 542-544. http://www.clinicalkey.com/dura/browse/bookChapter/3-s2.0-C20110052407
2. Robert a. Baldor MD. Paronychia. In: Frank J. Domino, Robert A. Baldor, 21st ed., Standard ed. The 5-minute clinical consult 2013. Philadelphia, Pa.: Wolters Kluwer Health/Lippincott Williams & Wilkins 2013.
3. Elizabeth M. Billingsley MD. Paronychia. Medscape. http://emedicine.medscape.com/article/1106062-overview#a4. June 2016. Accessed August 25 2016.
4. Philip Buttaravoli MD, Stephen M. Leffler MD. Paronychia. In: Philip Buttaravoli, 3rd ed. Minor Emergencies. Philadelphia, PA: Elsevier/Saunders; 2012: Chapter 149, 591-596.
5. Glenn G. Fort MD, MPH. Paronychia. In: Fred F. Ferri MD, FACP, ed. Ferri's Clinical Advisor 2017. Philadelphia, PA: Elsevier; 2017: 936-936.
6. Wingfield E. Rehmus MD. Acute Paronychia. Merck Manual. https://www.merckmanuals.com/professional/dermatologic-disorders/nail-disorders/acute-paronychia. Nov 2013. Accessed August 25, 2016.
7. American Osteopathic College of Dermatology. Paronychia Nail Infection. http://www.aocd.org/?page=ParonychiaNailInfe. Accessed date 10/22/16.
8. Relhan V, Goel K, Bansal S, Garg VK. Management of Chronic Paronychia. Indian Journal of Dermatology. 2014;59(1):15-20. doi:10.4103/0019-5154.123482.
9. Piraccini, B.M., & Alessandrini, A. Drug-related nail disease. Clinics in Dermatology. 2013: 31(5): 618-626. doi:10.1016/j.clindermatol.2013.06.013
10. Daniel J.A. Thornton & Tommy Lindau. (iii) Hand Infections. Orthopaedics and Trauma. 2010; 24 (3): 186-196. doi:10.1016/j.mporth.2010.03.016
11. James H. Rubright MD & Adam B. Shafritz MD. The Herpetic Whitlow. Journal of Hand Surgery. 2011; 36(2): 340-342. doi:10.1016/j.jhsa.2010.10.014
12. Patterson, James W and Gregory A. Hosler. The vasculopathic reaction pattern: Pyoderma gangrenosum. In: Patterson, James W and Gregory A. Hosler, 4th ed Weedon's Skin Pathology. Elsevier; 2016: Chapter 8.
13. Ritting AW, O'Malley MP, & Rodner CM. (2012). Acute paronychia. *The Journal Of Hand Surgery*, *37*(5), 1068-70; Quiz Page 1070. doi:10.1016/j.jhsa.2011.11.021
14. Iorizzo, M. Tips to Treat the 5 Most Common Nail Disorders: Brittle Nails, Onycholysis, Paronychia, Psoriasis, Onychomycosis. *Dermatologic Clinics*. 2015;*33*(2), 175-183. doi:10.1016/j.det.2014.12.001
15. Clevland Clinic. Paronychia (Nail Infection). http://my.clevelandclinic.org/health/diseases_conditions/hic-paronychia-nail-infection. Accessed date 10/22/16.

Traumatic Eye Injury in a 14-Year-Old Male

Lisa J. Hrushka, OMS-IV[1] & Eric S. Wernsman, DO, FACOEP[2]

[1] VCOM–Carolinas, Spartanburg, South Carolina
[2] Clinical Faculty, VCOM–Carolinas, Spartanburg, South Carolina

A 14-year-old caucasian male presents to the emergency department after being struck by a baseball to the left side of his face. The patient reports that while playing the outfield, he was hit by a line drive to the eye after losing the ball in the sun. He denies wearing glasses or contacts at the time of the injury. He complains of left eye pain and blurry vision but denies loss of consciousness or loss of vision after the accident. He also denies any nausea or vomiting. The patient describes his pain as throbbing which is made worse by eye-opening and bright lights. The patient has no other reported medical history.

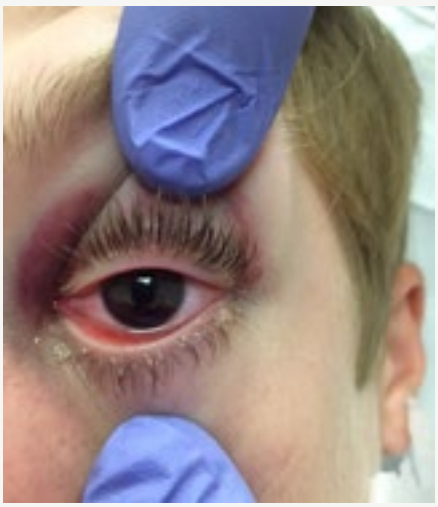
On physical examination, the patient is normocephalic with left periorbital ecchymosis and swelling to the left upper eyelid. Pupils are equal and reactive to light, and extraocular muscles are intact. A dark red fluid line is noted in the inferior anterior chamber of the left eye (Figure 1). The fluid line extends horizontally across the bottom third of the iris, almost to the level of the pupil. There is a distinct contrast in color to the iris; the natural green color is only visible above the level of the dark red fluid. There is no involvement of the sclera. The neck exam reveals no tenderness and the remainder of the physical exam is unremarkable. A CT scan of the head without contrast shows no acute intracranial hemorrhages or masses; however, a CT of the face and sinuses shows a nondisplaced fracture involving the superior orbital wall. After CT results are obtained, Tonopen testing shows the intraocular pressure to be normal on multiple readings and visual acuities are checked and are also normal. Prompt ophthalmological consultation is obtained, and the patient is initiated on Prednisolone drops and Cyclogyl. An eye shield is placed for protection until the patient's ophthalmology appointment the next day.

QUESTIONS:

1. What is the patient's most likely diagnosis?
   a. Hyphema
   b. Orbital compartment syndrome
   c. Retinoblastoma
   d. Ruptured globe
   e. Subconjunctival hemorrhage

2. What course of treatment is most important to protect the patient's vision?
   a. Bed rest
   b. Elevate the head of the bed
   c. Eye shield
   d. Limited eye movement
   e. Prompt ophthalmological consultation

FIGURE 1:
Left eye fluid collection



CORRESPONDENCE:
Eric S. Wernsman, DO FACOEP | golfdoc_94@yahoo.com

ANSWERS:

1. What is the patient's most likely diagnosis?

Correct Answer: A) Hyphema

When considering a traumatic injury to the eye, a hyphema is a collection of blood found inside the anterior chamber of the eye.[1] However, a ruptured globe and orbital compartment syndrome must be considered due to the substantial risk of vision loss or even enucleation. An irregularly shaped globe and drainage from the eye is suggestive of ruptured globe and proptosis, vision loss, and pupillary defect suggest orbital compartment syndrome. A subconjunctival hemorrhage is a collection of blood under the conjunctiva but above the sclera and outside the dome of the cornea.[1] Retinoblastoma is a rare cause of non-traumatic hyphema formation.[2]

2. What course of treatment is most important to protect the patient's vision?

Correct Answer: E) Prompt Ophthalmological consultation

Proper treatment of a hyphema is crucial to prevent vision loss as well as reduce the risk of re-bleeding. Bed rest allows the eye to heal and limiting eye movement reduces the chance of re-bleeding. Elevating the head of the bed while sleeping helps the body to absorb the blood in the eye and placing an eye patch helps protect the eye preventing further injury.[1] All options given are essentially correct; however, prompt ophthalmological follow up is still the most important treatment to maximize vision protection. Even a small crescent hyphema requires prompt evaluation and close follow up with Ophthalmology to prevent further vision loss or recurrent hemorrhage.[1]

DISCUSSION

A hyphema is a collection of blood in the anterior chamber of the eye that most commonly presents with painful, blurry vision.[3] The mechanism of injury is most often due to blunt or penetrating trauma and post intraocular surgery.[4] Blunt trauma to the eye can cause rupture of the vasculature supplying the iris and ciliary body.[1] The resultant bleeding can occlude the trabecular network in the angle of the eye which causes decreased drainage of the aqueous fluid. This allows the aqueous fluid to build up, increasing intraocular pressure.[1] Since vision can be affected by increasing intraocular pressure; every hyphema should be evaluated and followed daily by an ophthalmologist.[1] There is a higher incidence of hyphema in children under the age of 18 with a male predominance.[4] It may occur spontaneously in patients with iris melanoma, retinoblastoma, myotonic dystrophy, leukemia, sickle cell anemia and Von Willebrand disease and rarely can be a result of an infection.[1,4,5,6] Treatment goals involve reduction of increased intraocular pressure (IOP> 21 mmHg) and prevention of rebleeding to preserve vision.[5,7] One healthcare study involving children under the age of 18 reported that the majority of traumatic hyphemas spontaneously resolve within days to weeks.[4] In the study, out of the patients that had elevated IOP, 70% had normal IOP within one month of medical treatment while 12% had persistent elevated IOP requiring surgical intervention.[4]

Before evaluating a traumatic hyphema, it is important to examine and manage any life-threatening injuries, including any other blunt or penetrating trauma before assessing the ocular trauma.[7] Laboratory studies and CT scans of the head, neck, chest, and abdomen are widely utilized to ascertain any additional injuries in trauma patients. Indications for immediate ophthalmology consult and evaluation include orbital compartment syndrome and an open globe.[1] Orbital compartment syndrome results from rapidly elevated pressure within the limited space of the orbit.[1] Key findings on physical exam include proptosis, decreased visual acuity, subconjunctival hemorrhage, and an afferent pupillary defect.[1] If prompt treatment is not available, retinal ischemia can result, leading to possible permanent vision loss.[8] Open globe injury must be ruled out before a physical examination is performed as the exam can cause unwanted increased pressure on the eye.[1] Other indications that warrant an immediate consult is a traumatic hyphema associated with a grade III or IV injury (Figure 2), increased intraocular pressure, or those occurring in patients having a sickle hemoglobinopathy or other hematologic disorders as these have a higher risk for rebleeding and vison loss.[1,9]

Computed Tomography scans of the face/orbits are recommended to exclude a possible intraocular foreign body.[1] They are also recommended when severe swelling is present so that underlying structures can be evaluated for possible fractures or open globe.[1] Computed Tomography should be used with caution in adolescents due to higher radiation sensitivity in this population.[10] Estimates have shown that tumor rates can be as high or higher than one out of one thousand CT scans.[10] Therefore, every effort should be made to reduce unnecessary radiation in children. The benefits of scanning in children include visualizing bone, soft tissue, and blood vessels in a quick manner to exclude life-threatening injuries after a trauma.[10]

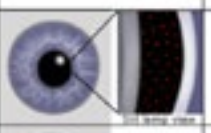
MEDICAL TREATMENT

After obtaining and documenting the visual acuity, management of a traumatic hyphema includes appropriate placement of an eye shield to the affected eye to protect the eye from further injury.[1,11,12] Dim lighting, bed rest, and elevation of the head of the bed should be employed to decrease stress on the blood vessels and promote clearance of the hyphema.[1,5,11,12] Once a ruptured globe is excluded, IOP should be determined. Patients experiencing nausea and vomiting should receive prompt treatment with an antiemetic to prevent a sudden increase in intraocular pressure that can be precipitated by emesis.[7] Pain control is important in these patients to perform a proper eye exam.[7] Topical analgesics such as tetracaine or proparacaine are safe options in patients without an open globe.[7] Topical eye drops should not, however, be sent home with the patient due to corneal toxicity.[7] Furthermore, the use of nonsteroidal anti-inflammatory medications is also discouraged due to platelet inhibition.[3]

For patients with increased intraocular pressure, beta blockers and carbonic acid anhydrase inhibitors (CAIs) should be used in conjunction with ophthalmology consultation.[1] However, in those patients with Sickle cell hemoglobinopathy, CAIs should be used with caution as it can cause further sickling of blood cells.[1,5] With all hyphemas, an eye shield is kept in place until evaluated by an ophthalmology .[1]

FIGURE 2:

Hyphema grading and prognosis

Grade	Anterior chamber filling	Diagram	Best prognosis for 20/50 vision or better
Microhyphema	Circulating red blood cells by slit lamp exam only		90 percent
I	<33 percent		90 percent
II	33-50 percent		70 percent
III	>50 percent		50 percent
IV	100 percent		50 percent

Adapted from Brandt MT, Haug RH. Traumatic hyphema: a comprehensive review. J Oral Maxillofacial Surg 2001; 59:1492.

When medically managing a hyphema, it's essential to hospitalize or maintain daily observation through outpatient ophthalmological evaluation to decrease associated complications.[5] Deciding to hospitalize is considered on an individual basis.[3] Outpatient care is appropriate for reliable patients having grade I or II hyphemas (see Fig. 2).[3] Patients having sickle cell anemia, increased intraocular pressure, hyphemas greater than 50%, or vision loss should be considered for admission.[3] Patients should be screened for history of bleeding disorders as this can increase chances of rebleeding.[1] Returning to normal activities is recommended only after complete resolution of the hyphema and the patient has been cleared by the ophthalmologist.

AUTHOR DISCLOSURES

No relevant financial affiliations.

REFERENCES:

1. Walker, R. A., Adhikari, S. (2016). Eye Emergencies. In Tintinalli, J. E., Stapczynski, J. S., Ma, O. J., Yealy, D. M., Meckler, G. D. & Cline, D. M. (Eds.). *Tintinalli's Emergency Medicine a comprehensive study guide*. (8th edition, pp. 1566-1569). New York, NY: McGraw-Hill Medical.
2. Shields, C L., Ghassemi, F., Tuncer, S., et al. Clinical spectrum of diffuse infiltrating renioblastoma in 34 consecutive eyes. Opthamology 2008; 115;2253.
3. Sharma, R., Brunette, D. D. (2014). Opthalmology in Marx, J. A., Hockberger, R. S., Walls, R. M. (Eds.). Rosen's Emergency Medicine Concepts and Clinical Practice. (8th Edition, pp. 910-914). Philadelphia,PA: Elsevier.
4. Soohoo, J. R., Davies, B. W., Braverman, R. S., Enzenauer, R. W., & Mccourt, E. A. (2013). Pediatric traumatic hyphema: A review of 138 consecutive cases. Journal of American Association for Pediatric Ophthalmology and Strabismus, 17(6), 565-567. doi:10.1016/j.jaapos.2013.07.007
5. Lenihan, P., & Hitchmoth OD, D. (2014). Traumatic Hyphema: A Teaching Case Report. The Journal of Optometric Education, 39(3). Retrieved May 1, 2016
6. Turbert, D. (2016, March 01). What Causes Hyphema? Retrieved May 02, 2016, from http://www.aao.org/eye-health/diseases/hyphema-cause
7. Andreoli, C. M., & Gardiner, M. F. (2015, January 20). Traumatic hyphema: Management. Retrieved May 1, 2016, from http://www.uptodate.com/contents/traumatic-hyphema-management?source=machineLearning
8. Rowh, A. D., Ufberg, J. W., Chan, T. C., Vilke, G. M., & Harrigan, R. A. (2015). Lateral Canthotomy and Cantholysis: Emergency Management of Orbital Compartment Syndrome. The Journal of Emergency Medicine, 48(3), 325-330. doi:10.1016/j.jemermed.2014.11.002
9. Pollock, T., Laliberte, I., Wu, J., & Lyons, C. (2008). Traumatic hyphema and immune thrombocytopenic purpura: Late rebleeds associated with low platelet count. Canadian Journal of Ophthalmology, 43(6), 717-717. Retrieved May 4, 2016
10. Riccabona, Michael. "CT in Children: Why and What to Consider for CT in Children." European Journal of Radiology 82.7 (2013): 1041-042. Web.
11. Walton, W., Hagen, S. V., Grigorian, R., & Zarbin, M. (2002). Management of Traumatic Hyphema. Survey of Ophthalmology, 47(4), 297-334. doi:10.1016/s0039-6257(02)00317-x
12. American Association for Pediatric Ophthalmology and Strabismus. (2016, January). Retrieved May 24, 2016, from http://www.aapos.org/terms/conditions/58

LETTER TO THE EDITOR

Patients & Doctors - Facebook Friends?

To the Editor of the Osteopathic Family Physician,

It has been over a month since I left the area I had practiced Family Medicine for over nine years. During that time I had the privilege to take care of numerous patients. When I was in the process of changing jobs, I consulted a lawyer and followed his advice regarding informing patients of my departure. Since I have left, I have been astonished by the number of Facebook friend requests and other ways former patients have tried to contact me. I do have a personal Facebook page, which is in the private setting, and most of the requests have come through this aspect of social media. With these requests, I have been confused on how to handle this situation. In my research, I am not alone. My question is regarding what to do in the case of patients contacting their former or current doctor on Facebook.

Many articles have been written about what a doctor is to do when contacted by patients to be "friends" on social media. An example of this was in the Curbside Consultation of the American Academy of Family Physicians in 2011 when an active social media family physician user was "friended" by her patient.[1] In this article the author, Dr. Chang Chretien stated that "regarding universally accepted standards for interacting with patients using social media, it is kind of like the Wild West."[1] This article was published in 2011, and although a lot of medical societies and specialty colleges have released guidelines on this subject since then, it can be confusing for the clinical practicing physician. A study showed that "13 of the 28 [US medical schools] have publically available student guidelines or policies giving explicit guidance on social media."[2,3] This is in contrast to the 95.45% of US medical schools website having a Facebook presence.[2]

Social media use is expanding, as is the internet in the practice of medicine. There are "dangers and opportunities for social media [use] in medicine."[3] An estimate from 2011 showed that approximately 90% of physicians use social media.[3] With these type of statistics, the numbers can only go up in 2018. Particularly among young physician, social media is getting more popular and evolving.[4] There is a lot of new found opportunity for social media use in medicine. There is a sense of community that can develop, and many positives can come about with this increase in use. These positives can especially be noteworthy in socially isolated areas, and many rural areas.[3]

Patients can be confused on certain aspects of social media and diversity exists about what is deemed appropriate in the internet world. Some hospitals have been using social media to promote their services and goals.[3] With this happening, the patient can question if the hospital uses Facebook why can't the physician? Problems arise when the patient and physician experience "blurred boundaries."[5] As Dr. Chang Chretien told the patient who questioned if she should be friends with her patients, she should be "cautious."[1] With social media, there is always a risk of patient confidentiality being violated.[6] If you are "friends" with a patient on social media, they can see everything you post. With all of these potential issues, it is recommended doctors know their "digital footprint."[6] A New Zealand study revealed that many doctors did not know the privacy setting of their Facebook account.[7]

Some patients do not realize the blurred boundaries and issues social media may cause. I think it is easy to "friend" someone and have gotten a variety of reactions when asking colleagues if this has ever happened to them. Some patients may feel a significant connection to their physician, even after their physician has left practice, and may want to stay in touch. But there are boundaries and the nature of our profession is such that there are risks to interactions outside of the doctors' office.

Since social media carries social, ethical, and professional risks[8] it is important to realize all the possible potential problems with online access to patients. The AMA Journal of Ethics released an article outlining "Professional Guidelines for Social Media Use - a Starting Point."[9] In this article, the guidelines released by the Federation of State Medical Board (FSMB) and the American College of Physicians (ACP) were presented.[9] The Federation of State Medical Board (FSMB) – "Model Guidelines for the Appropriate Use of Social Media and Social Networking in Practice" reiterated the importance of professionalism, privacy, and awareness of the possible pitfalls of social media. The summary described examples where social media and the physician can have issues.[10] These guidelines serve to remind the physician of the ethical considerations that occur when using social media.[10]

 Print ISSN: 1877-573X

As osteopathic family physicians, we have the privilege of taking care of patients. In a way, we already enter into a relationship with a patient the minute we walk through the exam door. Whether the osteopathic family physician chooses to communicate with their patients in a controlled, safe setting through social media avenues is their choice. There are guidelines available to help the physician see the dangers of social media, and also its potential positives. So the answer to the question outlined in the beginning of this article has not been answered. It is a personal choice, one that is not clear-cut, but neither is most of medicine. I am curious as to the number of osteopathic family physicians also experience this issue, and what they do in a similar situation.

B. Riley

Bernadette Riley, DO, FACOFP, FILM
Associate Professor
Department of Family Medicine
NYIT College of Osteopathic Medicine
Northern Blvd, Old Westbury, NY, 11568
Phone 516 686- 3989
Fax 516 686 3748
briley@nyit.edu

REFERENCES:

1. Chang Chretien, K., "Should I Be "Friends" with My Patients on Social Networking Web Sites?" AAFP July 1, 2011: 84 (1): 105-108.
2. Kind, T et al., "Social Media Policies at US medical schools" Medical Education Online 2010, 15: 5324; 1-8.
3. George, D et al., "Dangers and opportunities for social media in medicine" Clin Obstet Gynecol September 2013; 56 (3)
4. Guseh, J et al., "Medical Professionalism in the age of online social networking" Journal of Medical Ethics 2009; 35; 525-525.
5. GMC- UK Guidance "Doctors' Use of Social Media" www.gmc-uk.org/guidance assessed November 13, 2017.
6. Donahue, P et al., "Managing your digital footprint: How to use social media intelligently" Journal of the American Academy of Physician Assistants: May 2011, Volume 24 (5); p E1-E4.
7. MacDonald, J et al., "Privacy professionalism and Facebook: a dilemma for young doctors" Med Education August 2010; 44 (8) 805-813.
8. Pillow, M et al., "Social Media Guidelines and Best Practices: Recommendations from the Council of Residency Directors Social Media Task Force" Editorial Western Journal of Emergency Medicine, Volume XV, No 1: February 2014.
9. Kind, T., "Professional Guidelines for Social Media Use: A Starting Point" AMA Journal of Ethics May 2015, Vol 17 No 5; 441-447.
10. The Federation of State Medical Board (FSMB) – "Model Guidelines for the Appropriate Use of Social Media and Social Networking in Practice" April 2012- House of Delegates FSMB

EVERYBODY KNOWS YOUR NAME

WHERE HEALTH IS PRIMARY.

Health is Primary

BROUGHT TO YOU BY AMERICA'S FAMILY PHYSICIANS

Long-term relationships between doctors and patients build trust and lead to better outcomes.

Family doctors work with their patients throughout their lives. We want to give all patients access to this kind of continuing care.

Let's make health primary in America.
Learn more at **healthisprimary.org**.

HealthIsPrimary **#MakeHealthPrimary**

CALENDAR OF EVENTS

2018

JANUARY 12 - 14, 2018

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

JANUARY 24 - 27, 2018

29th Annual Osteopathic Winter Seminar
Clearwater Beach, Florida
www.pcomsociety.com

JANUARY 25 - 28, 2018

Missouri ACOFP - Winter Family Medicine Update
Independence, Missouri
www.msacofp.org

FEBRUARY 2 - 4, 2018

ACOFP Future Leaders Conference
New Orleans, Louisiana
www.acofp.org

MARCH 22 - 25, 2018

ACOFP Annual Convention & Scientific Seminars
Austin, Texas
www.acofp.org

AUGUST 2 - 5, 2018

acofpCA'42
California ACOFP Annual Scientific Medical Seminar
Anaheim, California
www.acofpca.org

OCTOBER 5 - 9, 2018

OMED®17
AOA/ACOFP Osteopathic Medical Conference & Exposition
San Diego, California
www.acofp.org

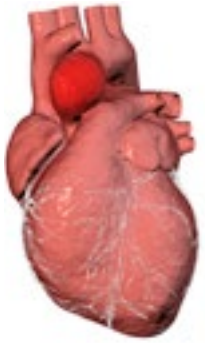
CME Resource: Osteopathic Family Physician Offers 2 Hours of 1-B CME

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

AORTIC ANEURYSMS

Malathi Amarnath, DO, Sandra Carnahan, DO, & Gabrielle Koczab, DO
Amy J. Keenum, DO, PharmD, Editor • Ronald Januchowski, DO, FACOFP, Health Literacy Editor



The aorta is the main blood vessel that carries blood from the heart to all the arteries in the body. It travels down the back of the chest and abdomen and then branches into two large arteries by the hips. An aneurysm is a stretch or bulge in the vessel that is caused by long-term vessel wall damage. There are multiple ways to damage vessels including smoking, elevated blood pressure, and elevated cholesterol. An aortic aneurysm usually does not have symptoms, but it can tear and cause severe chest and back pain, significant internal bleeding and even death. Fortunately, there are ways to prevent this from happening. Stopping smoking, controlling your blood pressure and cholesterol will help reduce risks. Your physician may get a screening ultrasound to check on the blood vessels.

PREVENTATIVE MEASURES INCLUDE:

- Quit smoking. The nicotine causes damage to the blood vessel walls, which makes it easier for the vessels to stretch and then tear.
- Control blood pressure with lifestyle changes and medication. High blood pressure creates a lot of stress on the vessels. This can lead to vessel wall damage and therefore stretching of the vessel wall.
- Control cholesterol and triglyceride levels with lifestyle modifications and medication. The buildup of fatty, cholesterol deposits on the inside of vessels narrow the area that blood can pass through and damages the vessel wall to which it is attached.
- Lifestyle changes can include exercise and a heart-healthy diet that is high in omega-three fatty acids. It is recommended that adults should have at least 30 minutes of cardio-intense workout three times per week like walking or running.
- USPSTF recommends all men have a screening ultrasound at the age of 65 to look for an aortic aneurysm. This is because white males are at a higher risk of developing an aneurysm, especially those that smoke. There has not been a definite recommendation for women, but if there is any history of aneurysms or any concerns, then you should speak to your doctor.

MEDICAL CARE & TREATMENT OPTIONS:

Once an aortic aneurysm is diagnosed, it requires close monitoring. Surgery may be required if the diameter of an aneurysm grows greater than 5.5cm. If you have a family history of aneurysms or are a male age 65 or greater, please call your Osteopathic Family Physician to discuss any necessary screenings. In case you develop any severe tearing chest pain that radiates to your back, you should call your doctor or 911 right away.

SOURCE(S): ***U.S. Preventive Services Task Force, Society for Vascular Surgery***

acofp American College of Osteopathic Family Physicians

SPORTS RELATED EYE INJURIES

Ante T. Pletikosic, DO & Louis Leone, DO, FAOASM
Amy J. Keenum, DO, PharmD, Editor • Ronald Januchowski, DO, FACOFP, Health Literacy Editor

Many people experience sports and recreation-related eye injuries each year. In fact, the number is estimated to be around 100,000 with approximately 42,000 going to the ER for treatment. Sports-related injuries cause over 13,000 people to go blind yearly and is the leading cause of blindness in children in the United States. Sports can have a low, high, and very high risk of eye injury. Low-risk sports do not use a ball, puck, bat, stick, and do not involve body contact. Some include swimming, gymnastics, and cycling. High-risk sports involve what low-risk sports do not and include sports such as baseball, basketball, hockey, football, lacrosse, tennis, and water polo. Very high risk does not use eye protectors and involve full body contact such as boxing, wrestling, and contact martial arts. Protective eyewear includes goggles and safety glasses, safety shields and eye guards designed for a particular sport. Most frequent injuries involve baseball, basketball and racquet sports. The good news is that about 90% of serious eye injuries are preventable by using appropriate protective eyewear.

PREVENTATIVE MEASURES INCLUDE:

- Your Osteopathic Family physician should examine you before you play sports and can help you choose eye protection for your specific sport
- Tell your doctor if you have any family history of retinal problems or if you have any eye problems. If any risk factors are present, an ophthalmologist should check you before doing any very high or high-risk sports
- Regular eyeglasses, sunglasses, and contacts do not protect your eyes from blunt injuries
- Protective eyewear is made of ultra-strong material. They are about 10x more impact resistant than normal plastic and do not reduce vision
- All participating athletes should wear protective eyewear, not only those who wear prescription glasses or contacts
- Most protective eyewear can match your prescription if needed
- Currently, most youth sports leagues do not require the use of eye protection. Parents and coaches must strongly encourage that children wear safety eyewear whenever they play and encourage sports leagues to adopt a policy on protective eyewear.
- Choose eye protectors that meet or exceed the standards of the American Society of Testing and Materials (ASTM)

MEDICAL CARE & TREATMENT OPTIONS:

If an eye injury occurs, please call your Osteopathic Family Physician immediately. If you are unable to reach your Family Physician call 911 or go to the emergency room, even if the eye injury appears to be minor. Any delay in medical treatment can potentially result in permanent vision impairment, loss, or even blindness.

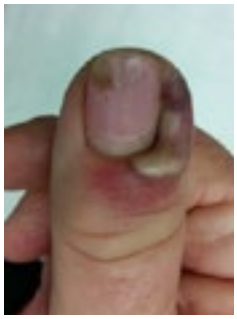
SOURCE(S): ***American Academy of Family Physicians, National Eye Institute, American Academy of Ophthalmology***

acofp American College of Osteopathic Family Physicians

TREATMENT OF PARONYCHIA (NAIL INFECTIONS)

Michael Majetich, DO & Louis D. Leone, DO, FAOASM

Amy J. Keenum, DO, PharmD, Editor • Ronald Januchowski, DO, FACOFP, Health Literacy Editor



Paronychia is a skin infection that occurs around a fingernail or toenail. It can be acute lasting less than six weeks or can be chronic and persist for more than six weeks. It is most likely to occur following a break in the skin, such as with trauma, nail-biting, and ingrown nails. It can also be more common in patients with diabetes, bad immune systems, poor circulation, or those who work with their hands in water a lot. Symptoms include pain, redness, swelling, and sometimes the development of a pus-filled blister. Your physician may get cultures to look for a specific bacterial infection. Treatments may include soaks in warm water, antibiotic use, cutting the pus pocket open, and sometimes even removal of the nail to help the pus drain. Depending on the extent of your infection, acute paronychia should clear within a few days to a few weeks.

PREVENTATIVE MEASURES INCLUDE:

- If a pus pocket is present, your physician may need to open this up to let the pus drain.
- Simple treatment may include soaking the affected area in warm water several times a day and applying a topical antiseptic or antibiotic ointment.
- If no pus pocket is present, your physician may have you do an antiseptic soak such as Chlorhexidine.
- Oral antibiotics may be necessary for severe or prolonged bacterial infections.
- If no response occurs with soaks and topical ointment use, then your physician may prescribe an oral antibiotic, especially if a pus pocket is present. Antibiotics commonly include doxycycline, clindamycin, or penicillin.
- In areas where there is a lot of methicillin-resistant Staphs. Aureus (MRSA) present, different antibiotics may have to be used. Sometimes these need to be used as a preventative measure.
- If a herpes simplex infection is suspected, you may be prescribed an antiviral medication.

MEDICAL CARE & TREATMENT OPTIONS:

Acute paronychia usually clears within a few days and will rarely recur in healthy people. Chronic paronychia may last for several months or longer and may recur in predisposed individuals. If you develop worsening of symptoms, please call your Osteopathic Family Physician. Paronychia is a treatable condition, and your physician can work with you to optimize your treatment. In case of an emergency, you should call your doctor or 911 right away.

SOURCE(S): ***American Academy of Family Physician, UpToDate.com and Dermnetnz.org***

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

Non-Profit Org.
U.S. Postage
PAID
Carol Stream, IL
PERMIT NO.
1746

FITNESS + NUTRITION
FOR PHYSICIANS & THEIR PATIENTS
HEALTH·EXPO

NEW THIS YEAR

ACOFP 55TH Annual Convention & Scientific Seminars

acofp '18
ANNUAL CONVENTION & SCIENTIFIC SEMINARS

www.acofp.org

MARCH 22 - 25, 2018

AUSTIN, TEXAS

32 Category 1-A CME Credits Anticipated
Including Pre-Con Credits Beginning on
Monday, March 19