OFP

Osteopathic Family Physician

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

November / December, 2016

Volume 8 | Number 6

ofpjournal.com

acofp American College of Osteopathic Family Physicians

Advocacy • Education • Leadership

www.acofp.org

OFP
Osteopathic Family Physician
JOURNAL
www.ofpjournal.com

2017 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.

INSTRUCTIONS FOR AUTHORS

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Articles submitted for publication must be original in nature and may not be published in any other periodical.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.

RESEARCH PAPERS

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 | Acknowledgements

REVIEW ARTICLE TOPICS:

- Advances in Skin Care Diagnosis & Treatment
- Newborn Disorders & Nutritional Guidance
- Direct Primary Care: Emerging Practice Alternative
- Skin and Soft Tissue Infections: It's More than Just MSRA
- Patient Engagement *(Help define the science of engaged research, provide tangible examples of the impact of engaged research, or answer a question or controversy related to patient engagement.)*

Amy Keenum, DO, PharmD
Editor-in-Chief

Ronald Januchowski, DO, FACOFP
Associate Editor

acofp American College of Osteopathic Family Physicians

EXAM SCHEDULE

CERTIFICATION & OCC (RECERTIFICATION)

AOBFP EST. 1972

EXAMS	LOCATIONS	POSTMARK DATE
	ACOFP Annual Convention	
Family Medicine / OMT Certification / OCC Performance Evaluation Only	**Kissimmee, FL** March 16 - 19, 2017 March 14 - 17, 2017	**October 1, 2016** *Late fee through December 1*
Geriatric Medicine CAQ Certification / OCC Cognitive Exam	**Electronic Testing** Regional Sites April 1, 2017	**October 1, 2016** *Late fee through December 1*
Family Medicine / OMT Certification Cognitive Exam	**Electronic Testing** Regional Sites April 1, 2017	**October 1, 2016** *Late fee through December 1*
Family Medicine / OMT OCC Cognitive Exam	**Electronic Testing** Regional Sites May 20, 2017	**November 1, 2016** *Late fee through December 1*
	AOA OMED Conference	
Family Medicine / OMT Certification / OCC Performance Evaluation Only	**Philadelphia, PA** October 7 - 11, 2017 October 6 - 8, 2017	**April 1, 2017** *Late fee through June 1*
Family Medicine / OMT Certification / OCC Cognitive Exam	**Electronic Testing** Regional Sites October 21, 2017	**April 1, 2017** *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.

QM:7.0 | acofp QUALITY MARKERS™
access • analyze • report

www.acofpqualitymarkers.org | qualitymarkers@acofp.org

Extract

Extract patient outcomes data, tests, well-care visits, vaccines, etc. from your EMR

Actionable Reporting

Actionable reporting on 20 categories of care; over 200 total measures

Avoid Penalties

Avoid penalties by reporting data to CMS to meet quality reporting requirements (PQRS)

Improve Quality of Care

Improve quality of care by viewing your patients' data vs. CMS benchmarks

Identify Patients

Identify patients who have missed appointments, are due for annual wellness visits, or need to have tests done

Segment Patients

Segment patients by age, disease, testing, etc. to view and act on those at highest risk

Enhance Workflow

Use Quality Markers 7.0 to enhance workflow and pre-plan for patient visits

QUALITY MARKERS™ 7.0

- ✓ CMS Qualified Vendor for Reporting
- ✓ PQRS Approved Registry[1,2]
- ✓ Approved Qualified Clinical Data Registry (QCDR)[1]
- ✓ HIPAA Compliant
- ✓ Compatible with most EMRs

acofp American College of Osteopathic Family Physicians

[1]*A subset of Quality Markers measures qualify for PQRS and QCDR reporting.*

[2]*Provider is responsible to register with CMS as necessary and to have available the necessary data points for reporting requirements.*

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.

This journal and the individual contributions contained in it are protected under copyright by ACOFP. The following terms and conditions apply:

PHOTOCOPYING

Single photocopies of single articles may be made for personal use as allowed by national copyright laws. Permission of the Publisher and payment of a fee is required for all other photocopying, including multiple or systematic copying, copying for advertising or promotional purposes, resale, and all forms of document delivery. Special rates are available for educational institutions that wish to make photocopies for non-profit educational classroom use.

Permission may be sought directly from ACOFP:
800-509-9204 | membership@acofp.org.

DERIVATIVE WORKS

Subscribers may reproduce tables of contents or prepare lists of articles including abstracts for internal circulation within their institutions. Permission of the publisher is required for all other derivative works, including compilations and translations.

ELECTRONIC STORAGE OR USAGE

Permission of the Publisher is required to store or use electronically any material contained in this journal, including an article or part of an article.

Except as outlined above, no part of this publication may be reproduced, stored in a retrieval system or transmitted in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, without written permission of the Publisher.

Address permission requests to ACOFP at membership@acofp.org.

NOTICE

No responsibility is assumed by ACOFP for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions or ideas contained in the material herein. Because of rapid advances in the medical ciences, in particular, independent verification of diagnoses and drug doses should be made.

Although all advertising materials is expected to conform to ethical (medical) standards, inclusion in the publication does not constitute a guarantee or endorsement of the quality of value of such product or of the claims made of it by its manufacturer.

OFP

Osteopathic Family Physician

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

BOARD OF GOVERNORS

PRESIDENT
Larry W. Anderson, DO, FACOFP *dist.*

PRESIDENT-ELECT
Rodney M. Wiseman, DO, FACOFP *dist.*

VICE PRESIDENT
Robert C. DeLuca, DO, FACOFP *dist.*

SECRETARY/TREASURER
Duane G. Koehler, DO, FACOFP

IMMEDIATE PAST PRESIDENT
Kevin V. de Regnier, DO, FACOFP *dist.*

PAST PRESIDENT
Carol L. Henwood, DO, FACOFP *dist.*

GOVERNORS
Nicole H. Bixler, DO, MBA, FACOFP

Gautam J. Desai, DO, FACOFP

Brian A. Kessler, DO, FACOFP

David J. Park, DO, FACOFP

Gregory D. Smith, DO, FACOFP *dist.*

Bruce R. Williams, DO, FACOFP

SPEAKER
Elizabeth Palmarozzi, DO, FACOFP

RESIDENT GOVERNOR
Garrett L. Kirkpatrick, DO

STUDENT GOVERNOR
Andrew Paul Crow, OMS III

EXECUTIVE DIRECTOR
Peter L. Schmelzer, CAE

RESIDENT LIAISON
Dustin Mullens, DO
VCOM - Carolinas, Blacksburg, VA

WRITING INTERNS
Frederick Stine
University of Pikeville

STAFF LIAISONS
Belinda Bombei & Samantha Abramczyk
ACOFP, Arlington Heights, IL

EDITORIAL COMMITTEE

CHAIR
Peter Zajac, DO, FACOFP
Associate Professor of Family Medicine/Director of Clinical Skills/Research
University of Pikeville-Kentucky College of Osteopathic Medicine (UP-KYCOM)
Pikeville, KY

EDITOR
Amy J. Keenum, DO, PharmD
Chair Family & Community Medicine, Michigan State University, East Lansing, MI

ASSOCIATE EDITOR
Ronald Januchowski, DO, FACOFP
Associate Dean for Curriculum, VCOM Carolinas Campus, Spartanburg, SC

MEMBERS
David Buford, PhD, OMS III
William Carey University College of Osteopathic Medicine, Hattiesburg, MS

Ryan Christensen, DO
Chief Resident, McLaren-Oakland, Clarkston, MI

Tyler C. Cymet, DO, FACOFP
Chief of Clinical Education, American Association of Colleges of
Osteopathic Medicine, Chevy Chase, MD

Robin C. Devine, DO
Assistant Program Director, Grant Family Practice Residency, Columbus, OH

Paula Gregory, DO, MBA
Assistant Dean of Clinical Eduaction, Philadelphia College School of
Osteopathic Medicine, Suwanee, GA

Douglas W. Harley, DO, FACOFP
Family Medicine, Akron General Medical Center – Center for Family Medicine, Akron, OH

Patricia H. Kroth, DO
Associate Program Director FM Residency, Hunterdon Medical Center, Milford, NJ

Justin D. Puckett, DO
Medical Director, Complete Family Medicine, LLC, Kirkville, MP

Wayne J. Reynolds, DO
Family Medicine, Sentara Medical Group, Gloucester, VA

Jon Roberts, DO
Family Medicine, Winona, MO

Maurice S. Robinson, DO
Family Medicine, Robinson Family Practice, Vienna, IL

Richard M. Watson, DO
Program Director FM Residency Lankenau Medical Center, Wynnewood, PA

Abraham Wheeler
Librarian, Michigan State Libraries, East Lansing, MI

EMERITUS MEMBER
Merideth Norris, DO, FACOFP
Grateful Recovery, Kennebunk, ME

WRITING MENTOR
Jay H. Shubrook, Jr., DO, FACOFP
Professor, Touro University College of Osteopathic Medicine, Vallejo, CA

DEPARTMENT CHAIR
Brian A. Kessler, DO, FACOFP
Associate Dean for Clinical Affairs
Campbell University's Jerry M. Wallace School of Osteopathic Medicine, Lillington, NC

NOV/DEC, 2016
VOLUME 8 | NUMBER 6

CONTENTS

OSTEOPATHIC FAMILY PHYSICIAN SPECIALTY PEER REVIEWERS

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

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

Robert Grubb, DO
Sports Medicine

Rose Hall, DO
Family Medicine

Nadia Hasan, DO
Clinical Images

Edward Hosbach, DO
Injections

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

Sarah Mitchell, DO
Family Medicine

Wadsworth Murad, DO
Psychiatry

Merideth Norris, DO, FACOFP
Addiction

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

Michael O'Connell, DO
Pain, Rehabilitation, & Neurology

John Pham, DO
Family Medicine

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

Jay Shubrook, Jr., DO, FACOFP
Endocrinology

Daryn Straley, DO
Pulmonary

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

Peter Zajac, DO, FACOFP
Patient Education

2016 STUDENT PEER REVIEW INTERNS

Christopher Ackerman
Edward Via College of Osteopathic Medicine Carolinas Campus

Samuel Berry
University of Pikeville - Kentucky College of Osteopathic Medicine

Omar Bukhari
University of Pikeville - Kentucky College of Osteopathic Medicine

Nazaneen Farahani
University of Pikeville - Kentucky College of Osteopathic Medicine

Cassie Henninger
Edward Via College of Osteopathic Medicine – Carolinas Campus

John T. Herrion
Edward Via College of Osteopathic Medicine – Carolinas Campus

Loura Khallouf
Edward Via College of Osteopathic Medicine – Carolinas Campus

Benjamin Oldach
Ohio University, College of Osteopathic Medicine

Aaron Prado
Touro - Nevada Campus

Shandilya Ramdas
University of Pikeville - Kentucky College of Osteopathic Medicine

Frederick Stine
University of Pikeville - Kentucky College of Osteopathic Medicine

Chip Wiginton
Edward Via College of Osteopathic Medicine – Carolinas Campus

INSTRUCTIONS FOR AUTHORS:

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 apporpriate attribution. ACOFP reserves the right to edit all submissions. To submit a manuscript or to access additional submission guidelines visit mc04.manuscriptcentral.com/ofp.

All opinions expresssed in Osteopathic Family Physician are those of the authors and not necessarily those of the editors, ACOFP, or the insitution with which the authors are affiliated, unless expressley stated. Instructions for authors can be viewed online at mc04.manuscriptcentral.com/ofp.

Osteopathic Family Physician is looking for...

STUDENT WRITING INTERN

STUDENT INTERN RESPONSIBILITIES:

- Familiarity with OFP editorial standards and compliance with those standards.
- Dependability – Be responsible, prompt, and maintain attention to details.
- Communicate – Interact in a professional manner. Be direct, kind and concise.
- Respect the confidentiality inherent in the review process.
- Available for a 8am EST conference call with the OFP editors on a predetermined Friday to discuss, rank and decide which articles will be slated to a bimonthly issue for OFP; choose an image for the cover of a bimonthly issue; choose a topic for patient education handout.

STUDENT PEER REVIEWER INTERN

STUDENT PEER REVIEWER RESPONSIBILITIES:

- Familiarity with OFP editorial standards and compliance with those standards.
- Dependability – Be responsible, prompt, and maintain attention to details.
- Communicate – Interact in a professional manner. Be direct, kind and concise.
- 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.
- You will be asked to peer review at least 1 article during the 2017 journal year.
- You will be provided with peer reviewing how to's and an evaluation by an OFP editor of your submitted peer review.

Please email belindab@acofp.org with your CV by **December 1, 2016**, noting which internship you are interested in - either Writing or Peer Review, or both. You will be asked to sign a letter of commitment that outlines your duties and professional expectations. Limited spots available.

EDITOR'S MESSAGE

Why Do We Diagnose in Millimeters But Treat in Inches?

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

Sleep disorders and treatment is the lead article for this issue paired with an article on FDA approved medications for the treatment of obesity. The editors decided on the pairing due to the common nature of obstructive sleep apnea and obesity as the most common cause of the most frequently diagnosed hypersomnolent sleep disorder. This article makes a point that there are many other causes of sleepiness. As history is a key to any diagnosis consider a referral to a sleep medicine physician rather that directly to a sleep laboratory. Many times a diagnosis may be made by history alone or in other scenarios knowledge of the patient history may aid in the evaluation of the polysomnogram if ordered. Often the polysomnogram without significant sleep apnea will still yield a diagnosis if one knows the patient's habits, work schedule, medicines, etc. and can interpret the study with that additional light. A brief review and treatment of insomnia, commonly treated in family medicine is a helpful section of this article.

Family physicians have the option of completing further education and sleep boards without pediatric or internal medicine boards. It is one of the few areas of specialty medicine that is open to family physicians.

Otitis media is a common diagnosis in family medicine. Antibiotic choice and pain relief are discussed but the idea of no antibiotic was only briefly mentioned. High dose amoxicillin remains the treatment for mainstay of antibiotic treatment.

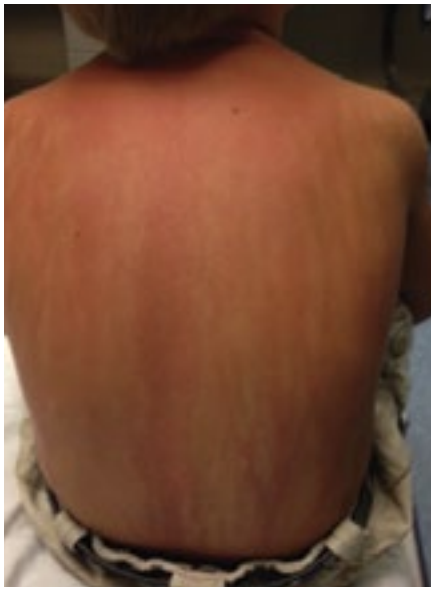
A nicely written review of the assessment and treatment of leg length discrepancy is included in this edition. It left me with one question. Why do we diagnose in millimeters but treat in inches? Did researchers determine the length data and the clinicians the treatment units?

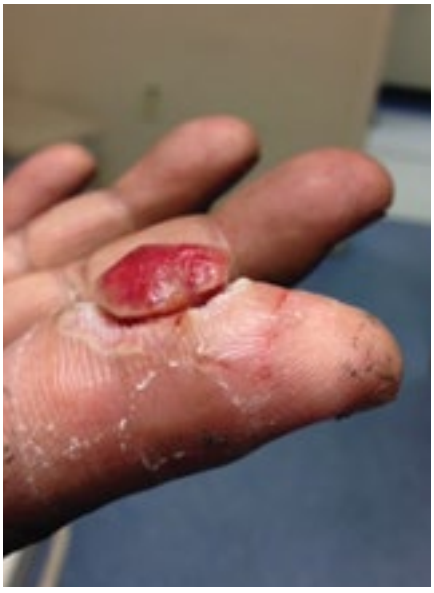
We have included two clinical images with discussion. Both depict painful skin lesions, their diagnosis and treatment.

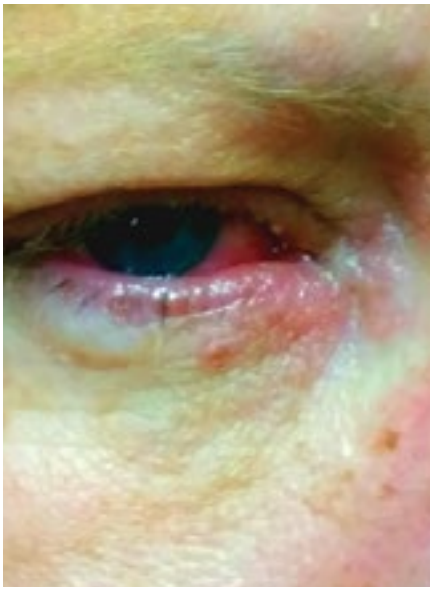
1877-5773X/$ - see front matter. © 2016 ACOFP. All rights reserved.

NOW SEEKING

CLINICAL IMAGES







Osteopathic Family Physician

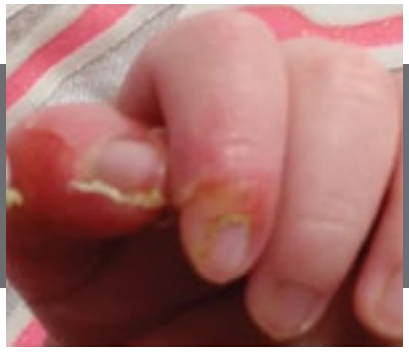
ACCEPTING SUBMISSIONS FOR THE SECTION TITLED "CLINICAL IMAGES."

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

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

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

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



FROM THE PRESIDENT'S DESK

Payment Readine$$, Part II: Advancing Care Information

Larry W. Anderson, DO, FACOFP *dist*.
2016 - 2017 ACOFP President

In the previous Issue of Osteopathic Family Physician, we looked at the new CMS category of Clinical Practice Improvement Activities (CPIA's). Since that issue, CMS has released the final payment Rule for 2017 and beyond. This article contains information from the Final Rule (MACRA), released October 14, 2016.

Advancing Care Information, in combination with Quality Reporting, Resource Use, and CPIA's , comprises the EP's Composite Performance Score (CPS). After further analysis by CMS, they arrive at the Payment Modifier for each EP based on the annual performance measures. The modifier will either be an incentive payment on top of the Physician Fee Schedule (PFS) amount, or a penalty against the PFS amount for your Medicare patients (this does not include Medicare Advantage or Medicare Shared Savings Patients).[1]

"Advancing Care Information" was previously called "Meaningful Use." In short, it requires an EMR and compliance with the switch to Electronic Medical Records for your practice. The new "Advancing Care Information" category provides a list of measures for physicians to attest to. The attestation of the measures is done via a CMS website. To have a link for the site for 2016 attestation sent to you, contact Debbie Sarason.*

ACI will be worth 25% of your Performance Score. Please see below for the other categories and their percentages.

To qualify for meeting ACI, you need to complete activities for a base score of 50 points. You do this by completing and attesting to 11 measures.

Base points – 50: Physicians/EP's must conduct a "security risk analysis" under the privacy requirement to receive these.[3] If you would like a summary of what is required by CMS, please contact Debbie Sarason.*

In addition, 80 points can be obtained by attesting to some or all of the "High-Priority" measures below:

- Protect patient health information
- Electronic prescribing
- Provide patient access to view, download, and transmit information
- Patient access to health information
- Send electronic summary of care record in transitions of care
- Incorporate electronic summary of care record in transitions of care
- Conduct clinical information reconciliation
- Submit data to an immunization registry (this would be a registry which is specific to the state you practice in)
- Submit data to a public health registry

To fully meet the ACI category for CMS, you need a total of 100 points. If you score less than 100 points, the 25% of the CPS score goes down proportionately.[1] The good news is that it is not an "all-or-nothing" category, as it was in previous years.

TABLE 1:

The Four CMS Categories Used to Determine an EP's Composite Performance Score[2]

Measurements	2017 - Percentage of CMS Composite Performance Score	Possible Point Score
Quality Reporting	60%	100 points
Resource Use	N/A for 2017 - Benchmark Year	No points for 2017
Advancing Care Information *(Previously Meaningful Use)*	25%	100 points
Clinical Practice Improvement Activities	15%	60 points

From the American College of Osteopathic Family Physicians.

The CMS EHR Registration and Attestation System is available for you to report ACI measures now. You only need to report for a contiguous 90-day period for 2016. To start the process go to: https://www.cms.gov/Regulations-and-Guidance/Legislation/EHRIncentivePrograms/index.html

In the next issue we will complete the review of the "Four Pillars of Payment" with an overview of "Quality Reporting," 60% of the CPS, and "Resource Use," which will not be counted towards the 2017 Performance Score. It will be a Resource Use "benchmark" for 2017. This benchmark will be the point of comparison for EP's performance in 2018 and beyond.

Look for updates on the "Payment and CMS Policy" webpage under "Practice Enhancement" on www.acofp.org.

With all the changes to Medicare reporting starting January 1, 2017, it is the right time to make ACOFP's Quality Markers 7.0™ part of your practice stabilization plan. ACOFP offers this population health and quality reporting platform at a significant Member Service discount. ACOFP's Quality Markers can also help Family Physicians qualify for points towards CMS' "Clinical Practice Improvement Activities." View your patient outcomes data like CMS does, improve quality, insure timely reporting, and gain incentives, rather than non-reporting penalties.

Sincerely,

Larry W. Anderson, DO, FACOFP *dist.*
ACOFP President

REFERENCES

1. www.cms.gov Accessed on September 29, 2016
2. Mullins, Amy. "Medicare Payment Reform: Making Sense of MACRA." Family Practice Management, March-April 2016; 23(2); 12-15
3. CMS eHealth University. Medicare and Medicaid HER Incentive Programs: Security Risk Analysis Tipsheet. Accessed September 29, 2016.

*Debbie Sarason
ACOFP Manager of Practice Enhancement & Quality Reporting
debbies@acofp.org | 847-952-5523

ACOFP QUALITY MARKERS

With all the changes to Medicare/CMS reporting starting January 1, 2017, it is the right time to make ACOFP's Quality Markers 7.0™ part of your practice stabilization plan. ACOFP offers this population health and quality reporting platform at a significant Member Service discount. ACOFP's Quality Markers can also help Family Physicians qualify for points towards CMS' "Clinical Practice Improvement Activities." Streamline CMS requirements and potentially gain incentives with ACOFP's Quality Markers.

For any questions or for more information, please contact Debbie Sarason, ACOFP Manager of Practice Enhancement & Quality Reporting at debbies@acofp.org or call 847-952-5523.

REVIEW ARTICLE

Obesity Pharmacotherapy: Present & Future

Luis Liu Perez, DO

Medical Director, Weight Management and Nutrition Clinic at Firelands Regional Medical Center

Keywords:

Obesity

Pharmacotherapy

Disease Prevention & Wellness

Obesity is a common, serious problem affecting many patients in every osteopathic family physician's practice and is also quickly becoming a pressing public health concern. The proper treatment of the obese patient rests on three pillars: proper nutrition, increased physical activity, and behavioral modification. Pharmacotherapy is an option to be considered in patients that don't respond adequately to a sensible treatment regimen targeting the above three elements. This article will review current approved short and long-term medications for the treatment of the obese patient, and briefly discuss some options that might be available in the future.

INTRODUCTION

Obesity is a common, serious problem affecting many patients in every family physician's practice. It is also quickly becoming a pressing public health concern, and imposes a tremendous economic burden on society. The Centers for Disease Control and Prevention reports that more than one-third of U.S. adults are obese (defined as a body mass index ≥30), and the estimated annual medical cost of obesity in 2008 was $147 billion dollars.[1]

As a complex, multifactorial disease, obesity is very challenging to treat, particularly in the setting of a busy primary care practice. A multidisciplinary approach is often needed to tackle the challenge of the obese patient, with the help of behavioral counselors, dietitians, and physical therapists. One cannot effectively treat obesity unless the condition is considered in the context of the whole patient (an "obese patient" instead of "obesity"). All aspects of the patient need to be addressed: mind, body, and spirit, as well as the patient's socioeconomic environment. By adopting a holistic approach to the care of the obese patient, family physicians are well-positioned to be at the forefront of medical bariatrics.

The proper treatment of the obese patient rests on three pillars: proper nutrition, increased physical activity, and behavioral modification.[2,3] Pharmacotherapy is an option to be considered in patients that don't respond adequately to a sensible treatment regimen targeting the above three elements. This article will review current approved medications for the treatment of the obese patient, and briefly discuss some options that might be available in the future.

PRESENT THERAPIES

Currently there are several approved medications for short and long-term management of the obese patient. As mentioned previously, these medications are not to be used as monotherapy, but should only be considered as adjunctive treatment in patients that don't respond adequately to a proper diet and exercise regimen. The obese patient should be required to continue with diet, exercise, and behavior modification efforts while under treatment with one of these medications. Table 1 *(pages 14 & 15)* provides a summary of all the medications discussed in this review.

SHORT-TERM MEDICATIONS

Sympathomimetic medications available for short-term treatment of the obese patient include phentermine, diethylpropion, and benzphetamine.[4,5,6] It's important for the physician to ensure he's complying with state laws when prescribing any of these medications, as many states have specific laws regulating the use of these agents. Phentermine and diethylpropion are schedule IV controlled substances, and benzphetamine is a schedule III controlled substance.

These medications act as appetite suppressants by stimulating the hypothalamus to release norepinephrine in the central nervous system. Phentermine has been shown to promote an average weight loss of about 8%.[7] They are available as once-daily doses, preferably to be taken in the morning to avoid insomnia. They are approved for short-term use only, typically no longer than 12 weeks, and only on patients with a BMI ≥ 30, or with BMI ≥ 27 with medical comorbidities (such as hypertension or diabetes). Once the medication is started, the physician should reassess the patient in 4 weeks and decide if response is adequate to justify continuing the medication.

Sympathomimetics should be avoided in patients with heart disease or uncontrolled hypertension. In patients with well-controlled hypertension, blood pressure should be monitored frequently. These agents should also be avoided in patients with severe psychiatric disease or a history of drug/substance abuse, pulmonary hypertension, hyperthyroidism, or glaucoma. If the patient has started taking monoamine oxidase inhibitors, 14 days should elapse prior to starting a sympathomimetic.

CORRESPONDENCE:
Luis Liu Perez, DO | perezlu@firelands.com

1877-5773X/$ - see front matter. © 2016 ACOFP. All rights reserved.

These agents are absolutely contraindicated in pregnancy, and appropriate contraception needs to be in place prior to prescribing these medications to women of child-bearing age. The prescribing physician should consider documenting a negative pregnancy test prior to prescribing these agents.

Most common adverse reactions include palpitations, tachycardia, tremors, insomnia, and hypertension.

These agents are among the most affordable for patients. They also carry the advantage that patients are not allowed to take these medications long-term, making them ideal as adjunctive treatment, forcing the patient to eventually rely on lifestyle changes for long-term weight loss.

LONG-TERM MEDICATIONS

Long-term medications are available for those patients with a BMI ≥ 30 (or with BMI ≥ 27 with medical comorbidities) who might need a longer course of treatment to achieve their weight loss goals. Although these agents are approved for treatment courses that exceed those of sympathomimetics, prudent use of these agents is still recommended. Patients should not rely solely on long-term weight loss medications and need to eventually modify their lifestyle and habits in order to continue to lose weight or to maintain their current weight. Physicians need to engage in a holistic treatment plan with their patients in order to avoid reliance on long-term pharmacotherapeutic agents for weight loss. No pill can take the place of proper nutrition, increased physical activity, and behavioral modification.

Orlistat, a pancreatic lipase inhibitor, stimulates weight loss by inhibiting the absorption of dietary fats,[8] and has been shown to promote a 10% weight loss.[9] Due to its minimal systemic absorption, this is the safest of all weight loss medications, and is also available over-the-counter without a prescription. At the recommended dose of 120mg three times daily prior to meals, this agent can inhibit absorption of dietary fats by approximately 30%. Orlistat is contraindicated in patients with chronic malabsorption syndromes or cholestasis. It's recommended that patients take a multivitamin due to the possibility of reduced absorption of fat-soluble vitamins while taking orlistat. Most common adverse reactions experienced are oily spotting, flatulence, and fecal urgency. Although this medication is the safest and is also available without a prescription, its use is limited due to the adverse effects patients experience.

Phentermine/topiramate, a combination agent approved in 2012, stimulates weight loss by the combined action of low-dose extended release phentermine, and the anorexiant effects of topiramate,[10] and has been shown to promote an average 10.5% weight loss,[11] making this combination agent the most effective weight loss medication currently available. This medication should be started at the lowest dose of 3.75mg/23mg for 14 days, then increased to the usual daily dose of 7.5mg/46mg. If after 12 weeks of treatment the patient has not lost at least 3% of body weight, the treating physician may choose to discontinue the medication or to increase the dose first to 11.25mg/69mg for 14 days and then to the maximum dose of 15mg/92mg. The medication should be tapered off and discontinued if the patient does not achieve at least 5% weight loss after 12 weeks of treatment at the maximum dose. This agent is contraindicated in pregnancy, or those patients with glaucoma, hyperthyroidism, or within 14 days of starting a monoamine oxidase inhibitor. Most common adverse reactions are paresthesias, dizziness, dysgeusia, insomnia, constipation, and dry mouth.

Naltrexone/bupropion is another combination agent available. It stimulates weight loss by affecting the appetite regulatory center of the hypothalamus, as well as the mesolimbic dopamine reward system. Naltrexone is an opioid antagonist, and bupropion is a weak inhibitor of dopamine and norepinephrine reuptake. The exact neurochemical effects of this drug combination that lead to weight loss are not fully understood.[12] Patients can expect an average of 7% weight loss.[13] Its typical daily dosage is 2 tablets of 8mg/90mg twice daily, but this dose should be arrived at after a slow titration period. This medication should not be used in pregnancy, uncontrolled hypertension, seizure disorder, or within 14 days of taking monoamine oxidase inhibitors. Because of the naltrexone component, this agent should also be avoided in patients taking opioids for chronic pain. Most common adverse reactions are nausea, constipation, headache, vomiting, dizziness, insomnia, and dry mouth. This combination agent carries a black box warning due to a risk of increased suicidal thoughts and behaviors, and increased risk of neuropsychiatric reactions.

Lorcaserin, a serotonin 2C receptor agonist, has an unknown mechanism of action, but is believed to stimulate weight loss by promoting satiety.[14] It has been shown to promote an average 6% weight loss.[15] It's dosed at 10mg twice daily. Response to therapy should be evaluated by week 12, and lorcaserin should be discontinued if the patient has not lost at least 5% body weight. This agent is contraindicated in pregnancy. Patients taking other serotonergic drugs, should be advised on the possibility of serotonin syndrome while on this agent. Previous medications in this class (i.e. fenfluramine) affected the 5-HT2B receptors in the heart, which caused valvular issues. Lorcaserin is selective for the 5-HT2C receptor, which is not present in the heart. However, patients should be monitored for the development of new heart murmurs while on this medication. Its use should be avoided in patients that have valvular heart disease or congestive heart failure. Most common adverse effects are headache, dizziness, fatigue, nausea, dry mouth, and constipation

Liraglutide, the most recent agent to be approved for weight loss, is an injectable glucagon-like peptide-1 (GLP-1) receptor agonist. It stimulates weight loss by affecting appetite regulation areas in the brain and slowing gastric emptying,[16] and has been shown to promote a 6-10% weight loss.[17] It's started at 0.6mg daily, and the dose is increased weekly until the maximum daily dose of 3mg is achieved. This medication should be discontinued if the patient has not lost at least 4% body weight after 16 weeks of treatment. Liraglutide is contraindicated in patients with a personal or family history of medullary thyroid carcinoma or Multiple Endocrine Neoplasia syndrome type 2, for which it carries a Black Box warning. There is also a risk of developing pancreatitis with this product. It should not be used in patients taking insulin or other GLP-1 receptor agonists. Most common adverse reactions are nausea, hypoglycemia, gastrointestinal upset, and headaches.

FUTURE THERAPIES

Two future therapies in development are worth mentioning: beloranib and fecal microbiota transplantation.

TABLE 1:

Drug Name (approximate monthly cost)	Mechanism of Action	Average Weight Loss	Contraindications
Phentermine ($36.99) Diethylpropion ($49.19) Benzphetamine ($140.89)	Appetite suppressant	8%	Heart disease Uncontrolled hypertension History of drug abuse Pulmonary hypertension Glaucoma Hyperthyroidism Pregnancy
Orlistat ($56 - $173)	Pancreatic Lipase Inhibitor	10%	Cholestasis Chronic Malabsorption Syndromes
Phentermine / Topiramate ($100.89 - $276.99)	Appetite suppressant	10.5%	Pregnancy Glaucoma Hyperthyroidism
Naltrexone / Bupropion ($230)	Affects appetite regulatory center of the hypothalamus & mesolimbic reward system	7%	Pregnancy Uncontrolled Hypertension Seizure Disorder
Lorcaserin ($170.75)	Promotes satiety	6%	Pregnancy Valvular heart disease Congestive heart failure
Liraglutide ($1231.64)	Affects appetite regulation areas in the brain & slows down gastric emptying	6 - 10%	Personal/family history of medullary thyroid carcinoma MEN type 2

Beloranib, an agent developed as a novel, first-in-class obesity therapy, is an inhibitor of methionine aminopeptidase 2 (MetAP2). It works by affecting the way the body metabolizes fat. This medication is currently being tested to treat Prader-Willi syndrome with plans to eventually have approval for severe and complicated obesity as well as obesity due to hypothalamic injury.[18] During Phase 1b studies, the drug demonstrated impressive weight loss, averaging about 2Lb per week. During its Phase 3 trials for treatment of Prader-Willi syndrome the drug was placed on complete clinical hold by the Food and Drug Administration (FDA) due to serious thromboembolic events leading to death in two study participants.[19] At the time of publication, it is unclear whether this agent will make it to routine clinical practice.

Fecal microbiota transplantation (FMT) is a fascinating, possible future adjunctive therapy in the treatment of obesity. A complete explanation of the role of the human intestinal microbiome in obesity is beyond the scope of this review article. The basic premise of FMT is that the human intestinal microbiome affects several of our body's processes, and an imbalance in the microbiome composition is associated with certain diseases (including obesity). With the introduction of donor feces into the patient's intestine, this balance can be restored or altered in order to effect weight loss.[20] This treatment is under investigation and is not FDA approved for weight loss. Currently the FDA only allows the use of FMT for treatment of recurrent C-difficile infections,[21] although not many institutions are capable of performing the procedure.

CONCLUSION

There are currently multiple medication options for the treatment of the obese patient, each with a unique mechanism of action and side-effect profile. The osteopathic physician is encouraged to consider these as adjunctive treatment in patients that are not successful in losing weight with a regimen of proper nutrition, increased physical activity, and behavioral modification. Consideration should be given to the cost of the medications and their insurance coverage, their contraindications and side effects, and the motivation of the patient to continue with their non-pharmacologic regimen, as these medications do not replace a holistic, multidisciplinary approach to the treatment of the obese patient.

REFERENCES

1. Adult Obesity Facts. Centers for Disease Control and Prevention. http://www.cdc.gov/obesity/data/adult.html. Accessed March 21, 2016.
2. Apovian CM et al. Pharmacological management of obesity: An Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2015;100(2):342-362.
3. Seger JC et al. Obesity Algorithm, presented by the Obesity Medicine Association. 2015-2016. www.obesityalgorithm.org. Accessed March 11, 2016.
4. Phentermine [package insert]. Horsham, PA: Teva Pharmaceuticals;2014.
5. Diethylpropion [package insert]. Cincinnati, OH: Merrell Pharmaceuticals;2003.

Adverse Events	Criteria to Continue Treatment	Comments
Palpitations Tachycardia Insomnia Tremors Hypertension	Assess for adequate response after 4 weeks of treatment	Most affordable medication (Phentermine) Dose in the morning to avoid insomnia
Oily spotting Flatulence Fecal urgency	5% weight loss by 12 weeks of treatment	Safest medication Adverse effects limit compliance
Paresthesias Dizziness Dysgeusia Insomnia Constipation Dry mouth	Must lose 5% body weight by 12 weeks of treatment at maximum dose	Most effective agent Cost/coverage limits its use
Nausea Vomiting Headaches Dizziness Insomnia Dry mouth	Must lose 5% body weight by 12 weeks of treatment	Black Box Warning due to increased risk of psychiatric adverse effects
Headache Dizziness Fatigue Nausea Dry mouth Constipation	Must lose 5% body weight by 12 weeks of treatment	Least incidence of adverse effects
Nausea Hypoglycemia GI upset Headaches	Must lose 4% body weight by 16 weeks of treatment	Black Box Warning on medullary thyroid carcinoma and MEN-2 Cost is a limiting factor

6. Benzphetamine [package insert]. New York, NY: Pfizer;2009.
7. Munro JF, Maccuish AC, Wilson EM, Duncan LJP. Comparison of continuous and intermittent anorectic therapy in obesity. BMJ. 1968;1:352-254.
8. Orlistat [package insert]. Nutley, NJ: Roche Laboratories;2009.
9. Sjostrom L et al. Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. Lancet. 1998 Jul 18;352(9123):167-72.
10. Phentermine/topiramate [package insert]. Mountain View, CA: Vivus;2014.
11. Garvey WT et al. Two-year sustained weight loss and metabolic benefits with controlled-release phentermine/topiramate in obese and overweight adults (SEQUEL): a randomized, placebo-controlled, phase 3 extension study. Am J Clin Nutr. 2012; 95: 297-308.
12. Naltrexone/bupropion [package insert]. Deerfield, IL: Takeda Pharmaceuticals; 2014
13. Apovian CM. A randomized, phase 3 trial of naltrexone SR/bupropion SR on weight and obesity-related risk factors (COR-II). Obesity. 2013;21(5):935-43.
14. Lorcaserin [package insert]. Zofingen, Switzerland: Arena Pharmaceuticals; 2012.
15. Smith SR et al. Multicenter, Placebo-Controlled Trial of Lorcaserin for Weight Management. N Engl J Med. 2010; 363:245-256.
16. Liraglutide [package insert]. Plainsboro, NJ: Novo Nordisk; 2014.
17. Pi-Sunyer X et al. A Randomized, Controlled Trial of 3.0 mg of Liraglutide in Weight Management. N Engl J Med. 2015; 373:11-22.
18. http://www.zafgen.com/zafgen/our-approach/beloranib. Accessed March 18, 2016.
19. Zafgen announces Beloranib IND placed on complete clinical hold. http://ir.zafgen.com/releasedetail.cfm?releaseid=945314. Accessed March 21, 2016.
20. Schneider A, Lashner B. Fecal Microbiota Transplantation. Lecture presentation at: The 10th Annual Obesity Summit; October 2, 2015; Cleveland Clinic Foundation, Cleveland, OH.
21. Guidance for Industry: Enforcement Policy Regarding Investigational New Drug Requirements for Use of Fecal Microbiota for Transplantation to Treat Clostridium difficile Infection Not Responsive to Standard Therapies. U.S. Food and Drug Administration. http://www.fda.gov/BiologicsBloodVaccines/GuidanceComplianceRegulatoryInformation/Guidances/Vaccines/ucm361379.htm. Accessed March 28, 2016.

REVIEW ARTICLE

An Update on Obstructive Sleep Apnea

Anthony Leazzo, DO, FAAP, FAOASM

Keywords:
Obstructive
Sleep
Apnea
CPAP
OSA
AHI
Disease Prevention & Wellness

Obstructive sleep apnea has become much more prevalent and is now considered the third most common respiratory condition affecting approximately 20 million Americans and an estimated 100 million people worldwide. It affects men, women and children alike. Obesity is thought to be one of the major contributors to the rise in cases. However, new technologies are starting to identify possible genetic links to the development of obstructive sleep apnea. Questionnaires and biometrics seem to be reliable methods to pre-screen people in an office setting, but the gold standard is still overnight, in-lab, polysomnography. Continuous positive airway pressure (CPAP) is the most effective treatment for all levels of severity of obstructive sleep apnea, but oral devices are also effective for mild-to-moderate cases. Novel treatments with oral suction devices, hypoglossal nerve stimulation, nasal expiratory positive airway pressure devices, and pharmaceuticals are all being investigated, but have shown mixed results in effectiveness. This article is an overview of Obstructive Sleep Apnea and a review of recent diagnostic and treatment options.

INTRODUCTION

Sleep is an opportunity for the body to conserve energy and restore its normal processes.[1]

The average American adult sleeps approximately 6.9 hours a night, which is less than the 7 - 9 hours recommended by many sleep experts.[2] While there are many reasons for lack of sleep, there is a growing concern over obstructive sleep apnea (OSA). OSA is defined as a recurrent cessation of breathing commonly from oropharyngeal collapse in adults[3] or enlarged tonsils and/or adenoids in children.[4]

OSA is increasing world wide and is now considered the third most common respiratory condition.[4] Concern for OSA is based on OSA's association with comorbid health conditions such as diabetes and hypertension, motor vehicle accidents among commercial drivers, and work related injuries and illnesses. These comorbidities have grave long term socioeconomic implications.

The insidious nature of OSA makes it sometimes difficult to recognize. The most commonly associated symptom is loud snoring,[5] but there are other symptoms also observed in people with OSA *(Table 1)*.

Prevalence

It is estimated that OSA affects over 20 million Americans, and over 100 million people world wide.[6,7] The overall prevalence is estimated at 9 - 24% and affects men 2:1 over women.[4] Recent data estimates that approximately 2 - 4% of children also suffer from OSA in an even distribution between boys and girls.[8] While OSA can effect adults and children at any age, children seem to peak between 2 and 8 years old, which corresponds with peak tonsillar growth.[6,8]

The growing occurrence of OSA also has much to do with the obesity epidemic plaguing society. The prevalence of OSA ranges from 11 - 46% in obese women and 33 - 77% in obese males between 30 and 69 years old.[9] Between 25 and 45% of obese children have OSA compared with 1 to 3% of their normal-weight counterparts.[5]

Commercial drivers (CD) with OSA pose a particularly challenging and dangerous problem. As a profession CDs are at higher risk for OSA given the sedentary nature of their occupation coupled with a high prevalence of obesity due to poor eating habits and lack of exercise. It is estimated that 45 - 50% of CDs in the United States are obese.[10] This is significant because CDs with OSA have a 2 - 11 fold increased risk of being involved in a motor vehicle accident, and OSA is estimated to be present in 15 - 30% of CDs who have crashed.[10] Additionally, 29% of CDs have experienced almost falling asleep at the wheel and 18.3% have experienced a near miss due to falling asleep while driving.[11]

Studies have shown that CDs generally do not report their symptoms and diagnoses accurately because of the economic and occupational implications of an OSA diagnosis.[10] An anonymous survey of US truck drivers showed that over 20% of drivers reported falling asleep at traffic lights, casting further doubt about the validity of screening questionnaires used during medical exams due to the potential for denial or deception on the part of the driver.[10]

CORRESPONDENCE:
Anthony Leazzo, DO, FAAP, FAOASM | leazzo_do@yahoo.com

1877-5773X/$ - see front matter. © 2016 ACOFP. All rights reserved.

OSA can cause a tremendous financial strain. There has been an increase in both direct and indirect medical costs due to OSA and its treatment in CDs, as well as nonfinancial costs such as loss of quality of life and premature death.[11] In general workers and commercial drivers with untreated OSA have more work disabilities, more work-related injuries, and higher rates of absenteeism and presenteeism compared to workers and drivers who do not have OSA.[11] It is estimated that individuals with sleep problems have a 1.62 % greater risk of injury and account for two thirds of the total financial costs on society once again compared to those workers who do not have OSA.[11]

Etiology

Multiple reasons exist as to why individuals suffer from OSA. Research conducted by Dongmei, MD and Jinmei, MD suggests that a first degree relative with OSA increases the risk for developing OSA by more than two-fold suggesting there may be a genetic component to the development of OSA.[4] Parish, MD's research further defines that there is no single sleep gene, and as such, sleep may be controlled or influenced by many genes.[12] Understanding the genetic basis of OSA is important to find new insights into its pathogenesis, the biologic basis for the disorder, and to develop new tests for the diagnosis of, and therapies for, patients with OSA.[12]

Changes in weight are inextricably liked to OSA. As little as a 10% weight gain can increase the apnea-hypopnea index (AHI) by 32%, conversely a weight loss of 10% will cause a reduction in AHI by 26%.[8] Current estimates suggest that about 58% of OSA cases can be attributed to excessive weight.[13]

The distribution of fat, particularly central/visceral obesity, is a more important risk factor versus fat size for OSA.[14,15] Studies have linked visceral obesity to the common OSA-related comorbidities *(Table 2)*.[8,14,16]

In addition to obesity, several phenotypes associated with OSA have been identified. In children they include: adenotonsilar hypertrophy, craniofacial malformation, and primary muscular disorders.[17,18] In adults they are: passive critical closing pressure of the upper airway and arousal threshold (how easily a person is awakened from sleep). The higher the arousal threshold, the more likely a person will become hypoxic while sleeping because it will take a larger stimulus to awaken them. Additional adult phenotypes include: oropharyngeal muscle responsiveness and loop gain. Loop gain is the measurement of how quickly the body responds to changing levels of carbon dioxide in the blood to signal a need to awaken. The higher the loop gain the more carbon dioxide needed to cause a person to awaken from sleep.[19]

While these are the two most common etiologies, there are ongoing studies investigating other sources for the development of OSA as well.

SCREENING & DIAGNOSIS

Biometrics

Biometric indices are helpful in the screening process. Table 3 lists the measurements that should be taken at the initial visit and then subsequently followed during the treatment process. Additionally, assessment for long face syndrome (infraorbital darkening, mouth

TABLE 1:

Additional symptoms encountered in adult and pediatric OSA.[5]

Symptom	Adult	Child
Witnessed pauses in breathing while asleep	√	√
Fitful sleep quality	√	√
Fatique	√	√
Dry mouth	√	√
Morning headaches	√	√
Excessive daytime sleepiness	√	
Irritability		√
Behavioral problems		√
Learning difficulties		√
Motor vehicle accidents	√	√
Poor academic performance		√
Restless leg syndrome	√	

TABLE 2:

Common OSA-related comorbidities.[8,14,16]

Cardiovascular disease
Chronic obstructive pulmonary disease
Depression
Diabetes
Hypertension
Increased cancer risk
Neurocognitive disease
Stroke

TABLE 3:

Areas to obtain biometrics data from in screening for OSA.[19]

Blood pressure
Body mass index (increased risk if > 30 kg/m2)
Heart rate
Height
Neck circumference (increased risk if > 17" in men and 16" in women)
Weight

breathing, elongated midface and nasal atrophy) and facial morphology need to be evaluated.[19]

People with mandibular retrognathia (anterior prominence of the chin ≥ 2 mm behind a virtual line drawn from the vermillion border of the lip to the chin), Mallampati classification 3 or 4, and Friedman palate position of 3 or 4 are at increased risk for OSA.[19] While both Mallampati and Friedman classifications correlated positively with predicting OSA severity based on AHI, Friedman correlated more strongly with OSA severity.[20]

In addition to the adult risk factors, children with enlarged tonsils (grades 2 - 4), small maxillary dimensions, greater posterior facial height, reduced maxillary protrusion or shorter and flattened dental arches have additional risks for OSA.[21]

Questionnaires

Currently there are numerous screening questionnaires used to help predict the presence of OSA. The most popular screening questionnaires are the Epworth Sleepiness Scale (ESS), the STOP-Bang questionnaire (SBQ), the STOP questionnaire (SQ) and the Berlin questionnaire (BQ). These various questionnaires have been compared to each other in terms of sensitivity, specificity and the ability to predict the severity of OSA. While each one has its strengths and weaknesses, the SBQ appears to be the best overall choice for screening questionnaires.

The SBQ had superior sensitivity and area under the curve than the ESS, BQ and SQ.[22] The SBQ correlated not only with polysomnography, but also with hypoxia and poor sleep quality.[23] Additionally the SBQ has an excellent sensitivity and a low specificity (AHI ≥ 5/hour 94.9% and 50% respectively; AHI ≥ 15/hour 96.5% and 28.6% respectively; AHI ≥ 30/hour 97.7% and 17.9% respectively) and could predict the severity of OSA.[23]

Although the SBQ seems superior, there are advantages to the other questionnaires as well. Even though the ESS had limited value screening for OSA, it was able to identify the severity of OSA, and is the most commonly used questionnaire to prescreen patients.[22,24] The Berlin questionnaire had good sensitivity and specificity for diagnosing OSA and assessing its severity, but is a very long questionnaire to complete and has a complicated scoring system.[22]

Genetic Markers

New research is looking at genetic biomarkers for OSA. Studies have indicated that increased expression of cysteinyl leukotrienes and changes in glucocorticoid receptor expression and activity has been reported in the tonsils and adenoids of children with OSA.[21] Other research is suggesting that increased prevalence of the rs1054135 polymorphism of the FABP4 genomic sequence is also seen in children with OSA.[12,21] Additional researchers are looking at Kallirein-1 uromodulin, urocortin-3 and orosomucoid-1 in children as these genes have had high correlations with OSA.[4,25]

Inflammatory markers may also be useful to consider. Many investigators accept CRP as a marker of inflammation and its regulation is thought to be interlukin (IL)-6, IL-8, and IL-10

dependent, and is increased in the presence of hypoxemia in adults with OSA.[16,26,27]

Other promising genetic associations are genes with different alleles for apolipoprotein E4 (ApoE4), tumor necrosis factor (TNF), specifically the TNF polymorphism TNFA rs1800629 and TNF-□, as well as angiotensin-converting enzyme (ACE).[14]

Polysomnography

Although questionnaires are useful for initial quick screenings, and genetic biomarkers are an upcoming tool, the gold standard for the diagnoses of OSA is polysomnography (PSG). For those patients that require PSG, there are a few options available. The gold standard is still an in-lab overnight PSG test that is continuously monitored and read by a sleep specialist.

However, not all patients can afford the cost of an in-lab test, and in some cases in-lab testing is not available due to geographical considerations. In those cases, in-home testing may be available. There are advantages and disadvantages to in-home testing. Some advantages are: improved patient access, lower cost, and greater acceptability by patients.[18] Some of the disadvantages include: decreased reliability, diagnostic limitations, and the portable monitoring devices can vary according to number and types of signals, sensors used, methods of scoring, and the criteria used to define respiratory apneas.[18]

Classification

The agreed upon classification for OSA is based on the number of apneas, or a complete arrest of breathing, plus hypopnea (where the airway is severely obstructed but breathing is not arrested resulting in a 30% reduction in thoracoabdominal movement compared to baseline and ≥ 4% oxygen desaturation that lasts ≥ 10 seconds), a night divided by sixty.[3,6,14] This is defined as the apnea-hypopnea index (AHI) or respiratory disturbance index (RDI).[6] An AHI of < 5 events/hour is considered normal, 5 - 15 events/hour is mild, 15 - 30 events/hour is moderate and > 30 events/hour is considered severe.[16,18]

TREATMENT

The main goals in the treatment of OSA are: relief of clinical manifestations, restoration of sleep quality, reduction of respiratory events, and correction of nocturnal oxygen desaturations. This can be accomplished through many different therapeutic interventions. The gold standard of which is nightly use of nasal continuous positive airway pressure (CPAP) in adults, and adenoidectomy and tonsillectomy in children.[5,10]

However, recent developments have shown that there may be options other than CPAP for adults with mild or moderate OSA and for children where tonsillectomy and adenoidectomy does not resolve their OSA. These alternatives include: oral devices, upper airway surgery, positional therapy, nasal expiratory positive airway pressure (nEPAP), myofunctional therapies and electrical stimulators.[7,19,24,25]

In addition to the modality chosen, lifestyle modifications have been of tremendous benefit. These modifications include weight loss, smoking cessation, and avoidance of alcohol or neurorelaxant drugs within 4 - 6 hours of retiring.[8,24,25]

Oral Devices

Oral devices work in different ways, but generally they apply pressure to the jaw to prevent retroglossal collapse. Studies have shown that this method may be preferable over CPAP in individuals with mild-to-moderate OSA, or for those with severe OSA who cannot tolerate CPAP or refuse surgery.[24,28] The devices are commonly mechanical in order to advance the mandible and lift the palate, or to retain the tongue in an anterior position.[8,29]

A new technology is emerging that instead of applying positive pressure to the upper airway, applies gentle oral suction sufficient enough to anteriorly and superiorly displace the soft palate and tongue.[29,30] There is a lack of universal response though, which may be due to insufficient enlargement of the retropalate space or collapse of the airway at different sites, requiring further work to better delineate its applicability.[29,30] Side effects of this therapy include oral and dental discomfort and dry mouth.[29,31] Currently only 30 - 40% of patients using this device have had successful treatment of their OSA.[29,31]

Surgical Interventions

Surgery was once the primary and only method of treating OSA. The advent of CPAP has caused surgery to become a secondary consideration. Surgical interventions are broadly divided into procedures aimed at attempting to cure OSA via upper airway reconstruction, or interventions to improve CPAP adherence, e.g. nasal septoplasty or polypectomy.[8] Currently evidence supporting surgery is lacking, and the consensus among experts is that the best candidates for surgery are patients with known craniofacial structural abnormalities.[25]

Bariatric surgery is becoming recommended for any adult with a BMI ≥ 40 kg/m^2 or a BMI ≥ 30 kg/m^2 with significant comorbidities such as type 2 diabetes, hypertension, chronic kidney disease, cardiovascular disease or chronic obstructive pulmonary disease.[25]

Sleeping Position

It is estimated that up to 60% of individuals have positional OSA, which is defined as supine AHI greater than two times that of non-supine AHI.[25,31] The treatment for this can be changing the sleeping position from supine to a side-sleeping position. This can be achieved through a simple "tennis ball" tee shirt or more sophisticated items worn by patients to prevent them from sleeping on their backs.

Nasal Expiratory Positive Airway Pressure (nEPAP)

nEPAP is a unidirectional flow resistance device. nEPAP regulates flow through the nostrils creating expiratory flow resistance without affecting inspiratory resistance.[30] While it appears that nEPAP may be an alternative to traditional CPAP for those with mild OSA, it is contraindicated for those patients whose oxygen saturation is less than 75% for more than 25% of the first four hours of the study, or for 10% of the entire study. Additionally, if the patient has persistent nasal blockage nEPAP is contraindicated.[30] Current data suggested that approximated 35 - 50% of patients would benefit from this type of therapy.[31]

Electrical Stimulation

Patients with moderate-to-severe OSA may not have consistent clinical benefit from CPAP owing to poor adherence to treatment or anatomically from excessive airway collapsibility.[32] While not yet approved for clinical use, human studies have shown that hypoglossal nerve stimulation (HGNS) shows promise in the treatment of OSA; especially if the BMI is ≤ 32 kg/m^2.[30] The nocturnal activation of lingual muscles is a way to overcome sleep-related decrease in pharyngeal dilator muscle tone in order to maintain a patent airway through stimulation of the genioglossus muscle.[33] HGNS has been shown to improve airflow in a dose-dependent manner, and decreases pharyngeal collapsibility during sleep.[33] Additionally, electrical stimulation of the hypoglossal nerve evokes a functional response of the tongue muscles.[32] This causes an anterior displacement of the tongue further augmenting the increase in the orpharyngeal space.[32] Unfortunately, isolated activation of the tongue through transcutaneous or direct intra-muscular means has been shown to have limitations for treatment of OSA without concomitant activation of the muscle to dilate the pharynx.[33]

Pharmaceutical Treatment

There is emerging evidence that suggests arousals from sleep may contribute to the severity of OSA in that if there is a decrease in one's ability to be aroused from sleep when apneic events occur then the apnea could be more severe and last longer.[30,33] Pharmaceutical research is being conducted to increase the ability of someone to be aroused from sleep when apneic events occur. Evidence suggests that arousals destabilize the ventilator response to stimuli and it has been hypothesized that arousal thresholds could be raised pharmacologically causing sleep-stage stability.[30] Additional research has suggested that increasing upper airway muscle tone may help treat OSA as well.[33] Eszopiclone is the drug being investigated, and while the results are encouraging, the studies have not shown a statistically significant improvement in AHI so further investigation is warrented.[30]

Myofunctional Therapy

Myofunctional therapy is a treatment program that suggests training the upper airway muscles, similar to athletes training other body muscles, would help stabilize the upper airway to prevent collapse and thus treat OSA. Thus isotonic and isometric exercising of the lips, tongue, soft palate and lateral pharyngeal wall muscles has been suggested to have a positive influence on reducing upper airway collapse during sleep.[33] Treatment times vary from as little as 5 minutes twice daily four days a week to 10 minutes 3 - 5 times a day for 2 - 3 months.[19] Current literature demonstrates that myofunctional therapy could serve as an adjunct to other OSA treatments because it has been shown to decrease AHI by approximately 50% in adults and 62% in children.[19] These exercises can be taught by any provider familiar with them, but generally are taught by a pulmonologist or sleep physician. While there are no formal osteopathic manipulative therapy techniques for OSA, these myofunctional therapies could be a potential area for further Osteopathic specific research.

ADHERENCE

Many studies have defined acceptable compliance as consisting of at least 4 hours of usage for more than 70% of nights.[8] Unfortunately about 23% of patients on average (ranging from 7 - 74%) abandon treatment, mostly in the first year of usage.[28,30] It seems that the first few weeks are crucial to further adherence and that any support at this stage will have a positive impact on future compliance.[28] Studies suggest that motivational telephone interviews that enable patients to discuss their experiences with CPAP and having medical staff present to review the patient's goals and give advice to the patients, demonstrated that these patient's CPAP compliance was 1 hour more per night at 6 months and 2 hours more per night at 1 year, than patients who did not receive this intervention.[28]

Overall long term efficacy of mandibular advancement oral devices is fairly good (76% after 1 year and 62% after 4 years) showing AHI stability from 1 - 2 years after implementation in treatment responders.[29] Previously there was no method of determining compliance except for patients reporting their usage, which was quite good at 90% using the device more than 4 hours a night more than half the nights a week at 5 years.[29] Recent advances in technology have helped with the development in intra-oral temperature sensors and microsensor thermometers with on-chip integrated readout electronics to allow objective determination of compliance similar to CPAP devices.[29]

CONCLUSIONS

Since the discovery of OSA, researchers have been trying to develop new ways to diagnose and treat OSA earlier and easier. Screening with questionnaires such as STOP-Bang are aiding in this process, while advancements at the genetic level to develop reliable biomarkers are also looking promising.

The definitive diagnostic tool is still lab-based overnight PSG. New advances in technology are making home-based technology better, and in the cases of suspected mild-to-moderate OSA, have been proven as effective as in-lab studies. In areas where there is limited access to in-lab studies, the newer home-based technology is proving to be very useful.

CPAP remains the gold standard treatment modality for OSA, especially in severe cases. New and novel therapies are being developed and some of these therapies have been shown to be very promising. Oral devices in cases of mild-to-moderate OSA have proven to be just as effective as CPAP. Training of the muscles of the oropharynx and HGNS are showing promise, but further studies are needed to prove the effectiveness of these strategies before they will be considered mainstream treatment options. Surgical interventions are quickly becoming passé except in the cases of craniofacial abnormalities or as adjunctive treatment for severe OSA. Unfortunately, pharmaceutical options have not proven to be of any benefit at this time.

Ultimately, compliance with treatment is the biggest struggle with OSA. The importance of compliance with treatment must be enforced and compliance must be monitored closely. It is essential that patients understand that the associated comorbid conditions such as diabetes and hypertension are made worse in the presence of OSA. Treatment must occur nightly in order to help treat these other diseases and mitigate any additional potential complications. Therefore, studies have shown that patients requiring OSA do much better with a multidisciplinary approach aimed at providing positive reinforcement as well as encouraging weight loss in the obese patient.

REFERENCES

1. Carter KA, Hathway NE, Lettiere, CF. Common sleep disorders in children. Am Fam Physician. 2014;89(5):368-377.
2. Ramar, K, Olson EJ. Management of common sleep disorders. Am Fam Physician. 2013;88(4):231-238.
3. Novakovic D, MacKay S. Adult obstructive sleep apnea and the larynx. Curr Opin Otolaryngol Head Neck Surg. 2015;23(6):464-9.
4. Dongmei Z, Yi X, Jinmei L. Genetics of obstructive sleep apnea/hypopnea syndrome. Chin Med J (Engl). 2014;127(17):3135-41.
5. Mofid M. Obstructive sleep apnea: The sleeping giant of the childhood obesity epidemic. JAAPA. 2014;27(10):27-30.
6. Barthlem G. Sleep-disordered breathing. Continuum (Minneap Minn). 2002:8(6)147-154.
7. Kezirian EJ, Goding GS Jr, Malhotra A, O'Donoghue FJ, Zammit G, Wheatley JR, et al. Hypoglossal nerve stimulation improves obstructive sleep apnea: 12 month outcomes. J Sleep Res. 2014;23(1):77-83.
8. Usmani ZA, Chai-Coetzer CL, Antic NA, McEvoy RD. Obstructive sleep apnea in adults. Postgrad Med J. 2013;89(1049)148-156.
9. Viswanath A, Ramamurthy J, Dinesh SP, Srinivas A. Obstructive sleep apnea: Awakening the hidden truth. Niger J Clin Pract. 2015:18(1)1-7.
10. Kales SN, Straubel MG. Obstructive sleep apnea in North American driver. Ind Health. 2014;52(1):13-24.
11. Allen AJMH, Bansback N, Ayas NT. The effect of OSA on work disability and work-related injuries. Chest. 2015;147(5):1422-1428.
12. Parish JM. Genetic and immunologic aspects of sleep and sleep disorders. Chest. 2013;143(5):1489-1499.
13. Quan SF, Budhiraja R, Clarke DP, Goodwin JL, Gottlieb DJ, Nichols DA, et al. Impact of treatment with continuous positive airway pressure (CPAP) on weight in obstructive sleep apnea. J Clin Sleep Med. 2013:9(10) 989-993.
14. Dewan NA, Nieto J, Somers VK. Intermittent Hypoxemia and OSA: Implications for comorbidities. Chest. 2015;147(1)266-274.
15. Liu KH, Chu WCW, To KW, Ko FWS, NG SSS, Ngai JCL, et al. Mesenteric fat thickness in associated with increased risk of obstructive sleep apnea. Respirology. 2014;19(1)92-97.
16. Ayas NT, Hirsch AAJ, Laher I, Bradley D, Malhotra A, Polotsky VY, Tasali E. New frontiers in obstructive sleep apnea. Clin Sci (Lond). 2014;127(4)209-216.
17. Arens R, Muzumdar H. Childhood obesity and obstructive sleep apnea syndrome. J Appl Physiol. 2010;108(2):436-444.
18. Corral-Peñafiel J, Pepin JL, Barbe F. Ambulatory monitoring in the diagnosis and management of obstructive sleep apnea syndrome. Eur Respir Rev. 2013;22(129)312-324.
19. Friedman M, Hamilton C, Samuelson CG, Ludgren M, Pott T. Diagnostic value of the Friedman tongue position and Mallampati classification for obstructive sleep apnea: A meta-analysis. Otolaryngol Head Neck Surg. 2013;148(4):540-547.

20. Shelgikar AV, Chervin R. Approach to and evaluation of sleep disorders. Continuum (Minneap Minn). 2013;19(1)32-49.

21. Kheirandish-Gozal L, Gozal D. Genotype-Phenotype interactions in pediatric obstructive sleep apnea. Respir Physiol Neurobiol. 2013;189(2)1-13.

22. Jinmei L, Rong H, Xu Z, Jiong Z. STOP-Bang questionnaire is superior to Epworth sleepiness scales, Berlin questionnaire, and STOP questionnaire in screening obstructive sleep apnea hypopnea syndrome patients. Chin Med J (Engl). 2014;127(17):3065-3070.

23. Jinmei L, Rong H, Xu Z, Jiong Z. STOP-Bang questionnaire in screening patients with obstructive sleep apnea hypopnea syndrome in sleep disordered breathing clinic. Chin Med J (Engl). 2014;127(17):1843-1848.

24. Dugan JA, Fagan MJ, Arand D. Obstructive sleep apnea. Primary Care Rep. 2015;9(7)1-18.

25. Canto GD, Pachêco-Pereira C, Aydinoz Secil, Major PW, Flores-Mir C, Gozal D. Diagnostic capability of biological markers in assessment of obstructive sleep apnea: A systemic review and meta-analysis. J Clin Sleep Med. 2015;11(1):27-36.

26. Archontogeorgis K, Nena E, Papanas N, Steiropoulos P. Biomarkers to improve diagnosis and monitoring of obstructive sleep apnea syndrome: Current status and future perspectives. Pulm Med. 2014;2014:930535.

27. Chiner E, Andreu AL, Sancho-Chust JN, Sánchez-de-la-Torre A, Barbé F. The use of ambulatory strategies for the diagnosis and treatment of obstructive sleep apnea in adults. Expert Rev Resp Med. 2013;7(3): 259-273.

28. Sutherland K, Vanderveken OM, Tsuda H, Marklund M, Gagnadoux F, Kushida CA, et al. J Clin Sleep Med. 2014;10(2):215-227.

29. Park JG, Morgenthaler TM, Gay PC. Novel and emerging nonpositive airway pressure therapies for sleep apnea. Chest. 2013;144(6): 1946-1952.

30. White DP. New therapies for obstructive sleep apnea. Semin Respir Crit Care Med. 2014;35(5):621-628.

31. Strollo PJ, Soose RJ, Maurer JT, deVries N, Cornelius J, Froymovich JT, et al. N Engl J Med. 2014;370(2):139-149.

32. Sutherland K, Cistulli PA. Recent advances in obstructive sleep apnea pathophysiology and treatment. Sleep and Biol Rhythms. 2015;13(1): 26-40.

33. Harada Y, Oga T, Chihara Y, Azuma M, Murase K, Toyama Y, et. Al. Differences n associations between visceral fat accumulation and obstructive sleep apnea by sex. Ann Am Thorac Soc. 2014;11(3):383-91.

Primary care physician opportunities with Geisinger

Renewed emphasis on caring

Geisinger is seeking BC/BE family medicine and internal medicine/pediatric physicians for primary care opportunities throughout our Pennsylvania service area. Medical school repayment up to $150,000. Resident/fellow stipend and relocation reimbursement also available.

If you want to make a difference in healthcare, we'd like to talk with you.

For more information, visit geisinger.org/careers or contact: Miranda Grace, Professional Staffing at mlgrace@geisinger.edu or 717-242-7109.

For positions with Holy Spirit–A Geisinger Affiliate, contact Lotoya Henry, Professional Staffing at lahenry@geisinger.edu or 717-972-4862.

Follow us: | LinkedIn

AA/EOE: disability/vet

Geisinger

CHECK OUT THE NEW

ACOFP APPAREL

NOW ONLINE!

acofp American College of Osteopathic Family Physicians

ACOFP polos, fleece jackets and windbreakers make the perfect holiday gift!

Buy it now at acofp.org.

REVIEW ARTICLE

Acute Otitis Media

Joe Kingery, DO,[1] Wanda Taylor, DO,[2] & Denielle Braun, DO[3]

[1]Associate Professor, East Kentucky Family Medicine Residency, University of Kentucky
[2]Assistant Professor, East Kentucky Family Medicine Residency, University of Kentucky
[3]PGY-2, East Kentucky Family Medicine Residency, University of Kentucky

Keywords:
Acute Otitis Media
Antibiotics
Upper Respiratory Illness
Ear Pain
Pediatrics
ENT

Acute otitis media (AOM) is one of the most common diagnoses made by physicians treating children. Approximately 80% of children will have at least one episode of AOM by one year of age, and up to 90% will have AOM by two to three years of age. AOM has been the most common condition for which antibiotics have been prescribed for children in the US. Numerous studies have looked at the diagnosis of AOM, how to differentiate it from Otitis Media with Effusion (OME), and its etiologies. New guidelines from the American Academy of Pediatrics requires otoscopic findings to diagnose AOM. Bacteria have been isolated in 50 to 90% of cases depending on diagnostic criteria used to diagnose AOM or OME. The most common bacteria isolated are Streptococcus pneumoniae, nontypeable Haemophilus influenza, and Moxarella catarrhalis. Thus, antibiotic therapy should be directed at those organisms. Currently, high-dose amoxicillin is recommended as a first-line antibiotic. However; new guidelines have significantly changed treatment with antibiotics in children. Those guidelines are reviewed in this article.

INTRODUCTION

Acute otitis media (AOM) is one of the most common diagnoses made by physicians treating children. Approximately 80% of children will have at least one episode of AOM by one year of age, and up to 90% will have AOM by two to three years of age.[1,2] AOM has also represented the most common condition for which antibiotics have been prescribed for children in the US. The etiology and appropriate treatment has been a long debate in medicine. One reason for this is likely the concern for complications if it were not treated with antibiotics. Also contributing to this is the wide variation in diagnosis and treatment that exists amongst different physicians.[3] In 2013, the American Academy of Pediatrics issued a significantly updated clinical practice guideline on the diagnosis and management of AOM. Among those recommendations were changes in treatment with antibiotics for children, clearer diagnostic criteria, and reiteration that complications are rare.

ETIOLOGY

AOM is usually caused by a cascade of events beginning with a viral upper respiratory illness (URI). The URI causes Eustachian tube (ET) dysfunction by causing inflammation and swelling of the ET and entrapment of fluid. That fluid then serves as a medium for growth of viruses or bacteria. Bacteria can be found in 50% to 90% of cases of AOM and OME.[2] In the case of bacterial infection, the most common bacteria isolated are Streptococcus pneumoniae, nontypeable Haemophilus influenza, and Moxarella catarrhalis.[3]

CORRESPONDENCE:
Joe Kingery, DO | joe.kingery@uky.edu

1877-5773X/$ - see front matter. © 2016 ACOFP. All rights reserved.

EPIDEMIOLOGY

Acute otitis media has a higher incidence rate in infancy and declines with age. Children less than 2 years of age have more visits with medical providers for AOM than those greater than 2 years old.[4] A peak occurrence has been observed between the ages of 6 to 12 months.[1] Several risk factors for acute otitis media in children have been identified. These include male gender, having parents who smoke, being cared for outside of the home, history of acute otitis media in other siblings and lack of breast feeding.[1,5] A decrease in the rate of visits to health care providers for AOM has been seen from 2004-2011.[4]

CLINICAL PRESENTATION

Younger children may present with a history of tugging or pulling at their ears, excessive crying, and fever. However; these symptoms are nonspecific and seen in many cases when AOM is not present.[6] Older children usually present with acute onset of fever and ear pain. Often times the onset in all age ranges are preceded by a viral upper respiratory illness. There has been different symptom scoring systems proposed to help diagnose AOM. Although history alone does not allow for diagnosis of AOM, the most validated scoring system is a 7-item parent-reported symptom score, the Acute Otitis Media Severity of Symptom Scale (AOM-SOS).[7] The AOM-SOS is presented in Table 1. The usefulness of the AOM-SOS is found with its day-to-day change with regards to AOM symptoms following diagnosis.

DIAGNOSIS

The diagnosis of AOM is more clearly defined in the latest American Academy of Pediatrics (AAP) guidelines that include otoscopic criteria *(See Table 2)*. In particular, AOM should not be diagnosed in

children who do not have middle ear effusion based on pneumatic otoscopy or tympanometry. AOM should be diagnosed in children presenting with moderate to severe bulging of the tympanic membrane or new onset of otorrhea not due to otitis externa. Additionally, AOM can be diagnosed in children who present with mild bulging of the tympanic membrane and recent (<48 hours) onset of ear pain (including holding, tugging, or rubbing of the ear in a nonverbal child).[7] The more strict criteria are aimed at decreasing antibiotic resistance and side effects from unnecessary antibiotic use. The differential diagnosis includes Otitis Media with Effusion, eczema, contact dermatitis, otitis externa, viral upper respiratory infection, and acute sinusitis. Red flag symptoms include fever unresponsive to therapy, meningeal signs including nuchal rigidity, significant tenderness over the mastoids, and significant lethargy or weakness.[8]

TREATMENT

Treatment is partly guided by age as well as symptoms. For all children without severe symptoms (moderate or severe otalgia, otalgia for at least 48 hours, or temperature 102.2 F or higher), no otorrhea and only unilateral AOM, observation can be considered without antibiotics. For those 2 years and older without any severe symptoms, bilateral AOM without otorrhea can also be observed *(see Figure 1, page 24)*. Pain can be quite severe and thus, should be treated appropriately. One of the challenges with stricter antibiotic use is educating patients, and particularly parents, about the evidence based medicine and complications that can occur when antibiotics are over utilized. The Centers for Disease Control and Prevention have published a lot of resources in their campaign "Get Smart". These include educational materials and information for patients and parents. As with most things that change in medicine, the challenge will be education and helping change a culture.[9]

ANALGESICS

Pain associated with AOM can be quite severe and last up to 7 days. Otalgia does not begin to improve for at least 24 hours after starting antibiotics. Therefore, analgesics should be prescribed or recommended during the duration of the ear pain. Common analgesics that show significant benefit are acetaminophen or ibuprofen. These will also help with the fever associated with AOM.

Although commonly prescribed, topical agents including benzocaine/antipyrine show little to no benefit. Any benefit that may exist from it does not last beyond 30 minutes.[10]

ANTIBIOTICS

Antibiotic therapy is aimed at targeting the most common causative organisms. When a decision has been made to treat with antibiotics and the child has not been treated with amoxicillin in the past 30 days, in the absence of an allergy, high-dose amoxicillin is recommended as the initial treatment. Amoxicillin is the first line choice due to multiple factors including efficacy against the most common causative organisms, acceptable taste profile, low cost, and narrow spectrum.[5] In the event the child has received amoxicillin within the last 30 days, has concurrent conjunctivitis, or in cases where coverage for beta lactamase positive organisms is desired, therapy should be high dose amoxicillin-clavulanate. Other alternative initial therapies that can be considered in cases of initial treatment failure or in patients with allergies include cefdinir, cefuroxime, cefpodoxime or ceftriaxone *(See Table 3, page 24)*.[5]

TABLE 1:

Acute Otitis Media Severity of Symptom Scale (AOM-SOS)

Symptoms	No	A Little	A Lot
Ear tugging / rubbing / holding			
Excessive crying			
Irritability			
Difficulty sleeping			
Decreased activity			
Decreased appetite			
Fever			

TABLE 2:

Diagnosis of Acute Otitis Meda

Findings	Quality of Evidence
Moderate to severe bulging of the tympanic membrane	Grade B
New onset of otorrhea not due to otitis extern	Grade B
Mild bulging of the tympanic membrane and recent onset of ear pain (holding, tugging, rubbing of ear if nonverbal)	Grade C

OSTEOPATHIC MANIPULATIVE THERAPY

There is a role for osteopathic manipulative therapy in AOM as well. One study showed that a standardized OMT protocol, which was administered along with standard care for AOM, resulted in faster resolution of middle ear effusion following AOM than standard treatment by itself.[11] One study suggests that there is a potential benefit of OMT as adjuvant therapy in those with recurrent AOM and may prevent or at least decrease surgical intervention and antibiotic use. Another study demonstrated the same findings.[12] OMT techniques that can be utilized include myofascial release, balanced membranous tension, balanced ligamentous tension, facilitated positional release, and counterstrain.[13]

COMPLICATIONS

While the overall incidence of complications from acute otitis media is low, clinicians should be able to recognize these should they occur. Infratemporal and intracranial complications occur in one in 100,000 children and in one in 300,000 adults per year.[14]

FIGURE 1:

Decision of antibiotic therapy

**Severe symptoms: moderate or severe otalgia, otalgia for at least 48 hours, or temperature 102.2° F or higher*

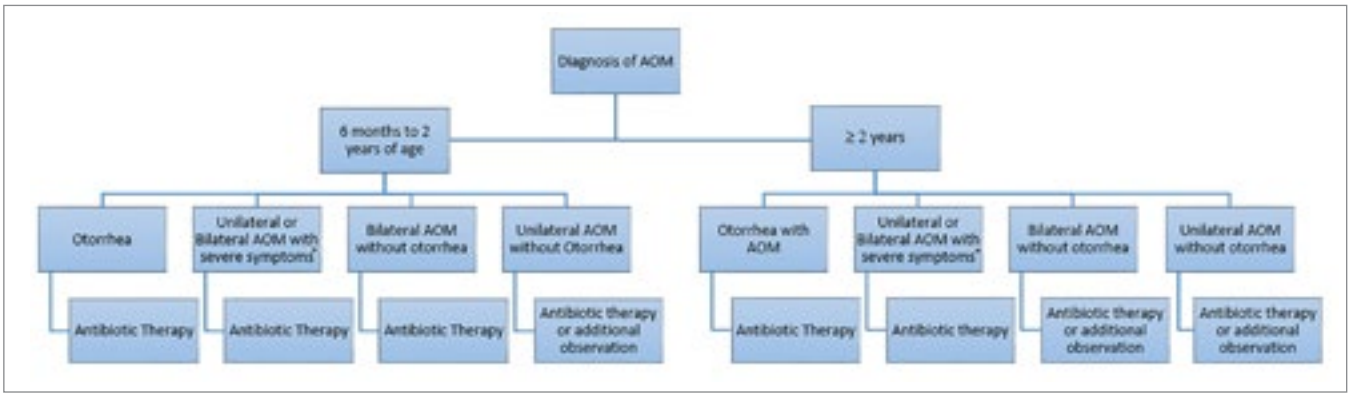


TABLE 3:

Initial immediate or delayed antibiotic treatment		**After 48 - 72 hrs of failure of initial antibiotic (or received amoxicillin w/in 30 days)**	
First-line treatment	Alternative treatment (if penicillin allergy)	First-line treatment	Alternative treatment
Amoxicillin *(80-90mg/kg per day in 2 div doses)*	*Cefdinir* *(14mg/kg per day in 1 or 2 doses)*	*Amoxicillin-clavulanate* *(90mg/kg per day of amox w/6.4 mg/kg per day of clavulanate in 2 div doses)*	*Ceftriaxone, 3 d clindamycin* *(30-40mg/kg per day in 3 div doses)* *+/- 3rd generation cephalosporin*
Amoxicillin-clavulanate *(90mg/kg per day of amox w/ 6.4mg/kg per day of clavulanate in 2 div doses)*	*Cefuroxime* *(30mg/kg per day in 2 div doses)*	*Ceftriaxone* *(50mg/kg IM or IV per day for 1 to 3 days, max 1g per day)*	Failure of 2nd antibiotic: *Clindamycin* *(30-40mg/kg per day in 3 div doses)* *+/- 3rd generation cephalosporin*
	Cefpodoxime *(10mg/kg per day in 2 div doses)*		
	Ceftriaxone *(50mg/kg IM or IV per day for 1 to 3 days, max 1g per day)*		

Chronic middle ear effusion can lead to conductive hearing loss which may be fluctuating or persistent. These children may score lower on tests of speech, language, and cognitive abilities. A perforation in the central portion of the tympanic membrane, may potentially lead to infection in the middle ear as well as the potential for mastoiditis, which could subsequently lead to abscess. In addition, infections of the temporal bone, meningitis, and intracranial infections, are a rare complication of otitis media more commonly seen in developing countries where access to medical care is limited.[14] Children with chronic suppurative otitis media or recurrent instances of acute otitis media are more at risk for mild to moderate conductive hearing loss, and may need referral to a specialist for tympanocentesis/tympanostomy tube placement. Children six months to 12 years of age with bilateral otitis media with effusion for 3 months or longer with documented hearing loss, or those children with recurrent acute otitis media with effusion at the time of assessment are candidates for tube placement.[5]

PREVENTION

There are several ways to help prevent AOM. One of the most common bacteria causing AOM has been significantly decreased by the routine use of the pneumococcal vaccine. A meta-analysis of five studies of AOM showed a 29% reduction in AOM caused by all pneumococcal serotypes among children receiving the vaccine before 24 months of age.[15]

Additionally, lifestyle changes can have significant impacts on rates of AOM. These include tobacco cessation, breast-feeding, avoiding supine bottle feeding, and reducing or eliminating the use of pacifiers after 6 months of age. Breast-feeding has been shown to be beneficial with most benefit seen if breastfeeding for at least 4 to 6 months. The largest impact has been seen in those with exclusive breastfeeding in the first 6 months of life.[16] An increase in OME and recurrent otitis media (ROM) is seen in children exposed to passive tobacco smoke.[17] Eliminating exposure to second-hand smoke is important for children to decrease chances of upper respiratory symptoms and OME as well as ROM. Supine bottle-feeding creates negative pressure in the oral cavity and in the bottle as fluid is removed from the bottle by sucking. To overcome the negative pressure in the bottle, the infant must suck excessively and causes the intraoral negative pressure to be transmitted to the middle ear via the ET which can then lead to OME and AOM.[18] Pacifiers have shown an increase in the incidence of AOM in children when used beyond the first 6 months of life.[19] Encouraging parents to reduce or eliminate the use of a pacifier beyond six months of age may help prevent AOM.

CONCLUSIONS

Acute otitis media remains one of the most common diagnosed conditions in children. New guidelines for the diagnosis of AOM have been developed in effort to reduce antibiotic overuse and subsequent resistance. According to new criteria AOM should be diagnosed when a patient presents with moderate to severe tympanic membrane bulging, mild bulging of the tympanic membrane with recent ear pain, or new onset of otorrhea. Analgesics are recommended for the duration of ear pain. Acetaminophen and ibuprofen have shown the most significant benefit in reducing pain. High dose amoxicillin is first line treatment and should be used whenever possible. Preventative measures including administration of the pneumococcal vaccine and lifestyle changes such as tobacco cessation, breast-feeding, inclined feeding, and eliminating pacifier use after 6 months of age have helped to reduce the incidence of AOM.

REFERENCES

1. Teele, D.W., J.O. Klein, and B. Rosner, Epidemiology of otitis media during the first seven years of life in children in greater Boston: a prospective, cohort study. J Infect Dis, 1989. 160(1): p. 83-94.
2. Harmes, K.M., et al., Otitis media: diagnosis and treatment. Am Fam Physician, 2013. 88(7): p. 435-40.
3. Coker, T.R., et al., Diagnosis, microbial epidemiology, and antibiotic treatment of acute otitis media in children: a systematic review. JAMA, 2010. 304(19): p. 2161-9.
4. Marom, T., et al., Trends in otitis media-related health care use in the United States, 2001-2011. JAMA Pediatr, 2014. 168(1): p. 68-75.
5. Uhari, M., K. Mantysaari, and M. Niemela, A meta-analytic review of the risk factors for acute otitis media. Clin Infect Dis, 1996. 22(6): p. 1079-83.
6. Rothman, R., T. Owens, and D.L. Simel, Does this child have acute otitis media? JAMA, 2003. 290(12): p. 1633-40.
7. Lieberthal, A.S., et al., The diagnosis and management of acute otitis media. Pediatrics, 2013. 131(3): p. e964-99.
8. Bethel, J., Otitis Media, in Paediatric Minor Emergencies. 2008. p. 164-165.
9. Prevention, C.f.D.C.a. Get Smart. 2016; Available from: http://www.cdc.gov/getsmart/.
10. Foxlee, R., et al., Topical analgesia for acute otitis media. Cochrane Database Syst Rev, 2006(3): p. CD005657.
11. Steele KM, C.J., Viola JH, Conte JA, Ridpath LC, Effect of Osteopathic Manipulative Treatment on Middle Ear Effusion Following Acute Otitis Media in Young Children: A Pilot Study. The Journal of the American Osteopathic Association, 2014. 114(6): p. 436-447.
12. Degenhardt BF, K.M., Osteopathic Evaluation and Manipulative Treatment in Reducing the Morbidity of Otitis Media: A Pilot Study. The Journal of the American Osteopathic Association, 2006. 106(6): p. 327-334.
13. Mills MV, H.C., Barnes LLB, Carreiro JE, Degenhardt BF, The Use of Osteopathic Manipulative Treatment as Adjuvant Therapy in Children with Recurrent Acute Otitis Media. Arch Pediatr Adolesc Med., 2003. 157(9): p. 861-866.
14. Ramakrishnan, K., R.A. Sparks, and W.E. Berryhill, Diagnosis and treatment of otitis media. Am Fam Physician, 2007. 76(11): p. 1650-8.
15. Pavia, M., et al., Efficacy of pneumococcal vaccination in children younger than 24 months: a meta-analysis. Pediatrics, 2009. 123(6): p. e1103-10.
16. McNiel, M.E., M.H. Labbok, and S.W. Abrahams, What are the risks associated with formula feeding? A re-analysis and review. Breastfeed Rev, 2010. 18(2): p. 25-32.
17. Ilicali, O.C., et al., Relationship of passive cigarette smoking to otitis media. Arch Otolaryngol Head Neck Surg, 1999. 125(7): p. 758-62.
18. Brown, C.E. and B. Magnuson, On the physics of the infant feeding bottle and middle ear sequela: ear disease in infants can be associated with bottle feeding. Int J Pediatr Otorhinolaryngol, 2000. 54(1): p. 13-20.
19. Niemela, M., et al., Pacifier as a risk factor for acute otitis media: A randomized, controlled trial of parental counseling. Pediatrics, 2000. 106(3): p. 483-8.

BRIEF REPORT

Short Leg Syndrome: A Common Cause of Low Back Pain

Kendi Hensel, DO, PhD[1] & Paul Crapo, OMS III[2]

[1]Associate Professor, Department of OMM, TCOM-UNTHSC
[2]TCOM-UNTHSC

Keywords:

Leg Length Discrepancy

Structural Exam

Low Back Pain

Osteopathic Manipulative Treatment (OMT)

Discrepancies in leg length are extremely common among the general population. Most people have few if any problems from a leg length inequality, but for some it can cause low back pain and other symptoms that are collectively termed Short Leg Syndrome. Low back pain stemming from Short Leg Syndrome is a common presentation that is missed too often in emergency departments and primary care clinics, because its prevalence and diagnostic findings are not well known by these providers. This paper reports a case of a 27 year-old Caucasian male with acute low back pain who first presented to an emergency department and was subsequently seen in a primary care clinic three times before being correctly diagnosed with Short Leg Syndrome. The prevalence and pathophysiology of Short Leg Syndrome are discussed. Obstacles hindering practitioners from making the correct diagnosis and solutions to those obstacles, such as encouraging the uniquely positioned Osteopathic community to assist in these efforts, are also discussed.

INTRODUCTION

A short leg, or limb length inequality (LLI), can occur because one leg is anatomically shorter than the other (an anatomic LLI), or occur because of somatic dysfunction (a functional LLI). Astoundingly, the estimated prevalence of anatomic LLIs nears 90%,[1] according to a 2005 review that compared research from 1970 to 2005 using Medline, CINAHL, and MANTIS databases. Not only did the author find that the prevalence of anatomic LLIs nears 90%, but that the average LLI is 5.22 millimeters (mm), that 14.8% of people have a LLI greater than 10 mm, and 2.6% have a LLI greater than 20 mm.[1] These figures are similar to statistics cited in a 1983 article comparing patients with chronic low back pain to symptom-free patients. In the symptom-free group, 15.6% had a LLI of 10 mm or more, and 2.2% had a LLI of 20 mm or more.[2] But the research on prevalence fails to answer the question: how large does a LLI have to be to cause symptoms? The data is conflicting on this point. Some articles say a LLI must reach approximately 5 mm before clinically apparent symptoms are produced, others 11 mm, and still others say as high as 30 mm.[2,3,4] In truth, it is unlikely that symptoms develop at a specific asymmetry threshold. Rather, symptoms can be absent or present in patients with a 5 mm LLI or 30 mm LLI, and the presence of symptoms is more likely related to how much time the patient spends standing or walking and the vigor of their physical activity.[1,2] Regardless, in a typical population where 90% of people have some degree of anatomic LLI, and when 50% of these LLIs are 5.22 mm or greater, it is prudent for practitioners to keep Short Leg Syndrome (SLS) high on their differential for patients presenting with low back pain (LBP).

CORRESPONDENCE:
Kendi Hensel, DO, PhD | kendi.hensel@unthsc.edu

1877-5773X/$ - see front matter. © 2016 ACOFP. All rights reserved.

SLS can be defined as an anatomic LLI that causes symptoms. Symptoms commonly include LBP, but may also include a shooting pain down the leg (sciatica), as well as pain over the sacroiliac joint, hip, outer thigh, knee, shin, ankle, and plantar fascia stemming from sacroiliac joint strain, greater trochanteric bursitis, iliotibial band strain, chondromalacia, shin splints, medial ankle synovitis, and medial plantar fasciitis respectively. Asymmetric landmarks involving the medial malleoli, anterior superior iliac spines (ASIS), posterior superior iliac spines (PSIS), iliac crests, and lumbar vertebrae almost invariably accompany SLS. The following predictable physical exam findings are found on the side with the shorter leg: 1.) superior medial malleolus, 2.) inferior ASIS, 3.) superior PSIS, 4.) inferior iliac crest, and 5.) contralateral side bending and ipsilateral rotation of the lumbar spine *(Table 1)*.[5,6,7,8,9] Opposite findings are found on the contralateral side of the LLI. With the exception of the medial malleoli, these changes occur in an effort to equalize the length of both legs. The innominate on the side of the LLI rotates anteriorly, thus lengthening the short leg, while the innominate on the contralateral side of the LLI rotates posteriorly, shortening the longer leg. The sacral base tilts toward the side with the LLI, dropping the iliac crest on the same side, causing the lumbar spine to side bend away and rotate toward the side with the short leg.

The diagnosis of SLS is clinical. Physicians should look for the above physical exam findings in patients with LBP, or other symptoms suspicious of SLS. However, caution is advised to avoid misdiagnosing SLS in a patient with a functional LLI. A functional LLI is an appreciable leg length discrepancy caused by somatic dysfunction that is usually the result of poor lower limb mechanics, such as excessive foot pronation. Not surprisingly, limb lengths equalize once the functional LLI is resolved, assuming leg lengths were equal in the first place. With the exception of the medial malleoli, exam findings on a functional LLI will be exactly opposite to that found in an anatomic LLI *(Figure 1)*.[10] Treatment for functional LLIs

varies depending on what caused the dysfunction. The treatment for a functional LLI stemming from excessive foot pronation is custom orthotics that corrects lower limb mechanics.

The best method to quantify anatomic LLIs is controversial. Commonly, practitioners measure from the medial malleoli of the short leg to its corresponding ASIS. However, due to disproportionate compensatory changes from patient to patient, this measurement has been criticized as inaccurate.[11,12] A second method is to measure the entirety of both limbs with plain radiographs, called a scanogram. Most current literature cites this as the most accurate method.[2] A third method used by the osteopathic and podiatric communities is to measure the declination of the base of the sacrum using radiography.[13]

Once the diagnosis is confirmed, various approaches exist to treat SLS, but all involve equalizing leg lengths through one method or another. The most common method is the use of a heel lift. Initial therapeutic heel lift size is also a matter of some debate. Some literature suggests starting with either a 1/16 or 1/8-inch lift depending on the health and age of the patient.[14] Other articles recommend starting out with a heel lift half the size of the LLI.[14] In either case, a patient typically begins wearing heel lifts smaller than the LLI itself to give the body time to decompensate. Patients then wear progressively larger heel lifts until the lift reaches the size of the LLI. Surgical approaches are reserved for severe cases, and include techniques such as the Ilizarov distraction method and subtrochanteric femoral shortening osteotomy.[15,16]

PATIENT PRESENTATION

A 27-year-old Caucasian male presented to the emergency department complaining of LBP. In the patient's history of present illness (HPI), he stated the pain had been present for four years, but that two months ago it worsened after he started working at a department store lifting and storing merchandise in the back of the store. The pain was sharp with no radiation, numbness, tingling, urinary retention, incontinence, bowel problems, or diaphoresis. He rated the pain a five out of ten. The symptoms were aggravated by movement and relieved by rest. Plain radiographs were taken of the patient's back which were negative for any acute bony abnormality. The patient was discharged home on cyclobenzaprine and meloxicam and told to follow up with a local resident clinic in one week.

At the resident clinic the next week, the patient's pain was still unrelenting. He was taught stretching exercises, given ibuprofen to replace meloxicam, and told to follow up in one week.

A week later, the patient's pain was still not better, despite being compliant with the exercises and medications. Ibuprofen was continued, cyclobenzaprine was discontinued, and nabumatone was added. Plain lumbar radiographs were re-ordered, a urinalysis was performed, and blood tests were taken. The patient was told to follow up in one week to discuss the results.

Another week later, the patient's symptoms were still present, matching the HPI recorded in the emergency department, except his pain was less severe. His past medical history consisted of chronic low back pain. His past surgical history was positive for a tumor removal from his right knee. His medications included ibuprofen 800 mg twice daily and nabumetone 500 mg twice daily. He denied knowledge of any drug allergies. The patient had recently quit smoking. His family history was negative. A review of systems was unremarkable except for that noted in the HPI. The physical exam showed normal sensation, motor function, gait, stance, and reflexes. A standing and supine osteopathic structural exam was performed which showed the following: tender to palpation lumbar paraspinal muscles, a superior left medial malleolus, an inferior left ASIS, a superior left PSIS, an inferior left iliac crest, and side-bent right rotated left lumbar vertebrae from L2 to L5. The patient's pubic symphysis was then gapped and landmarks were rechecked. His medial malleoli were almost symmetric, but the rest of the findings were nearly identical. Lumbar soft tissue and HVLA were performed and the patient felt slightly better. The lumbar radiographs, urinalysis, and blood tests were all negative, and they were discussed with the patient. At this point, heel lifts were not initiated. The patient was told to follow up in one month.

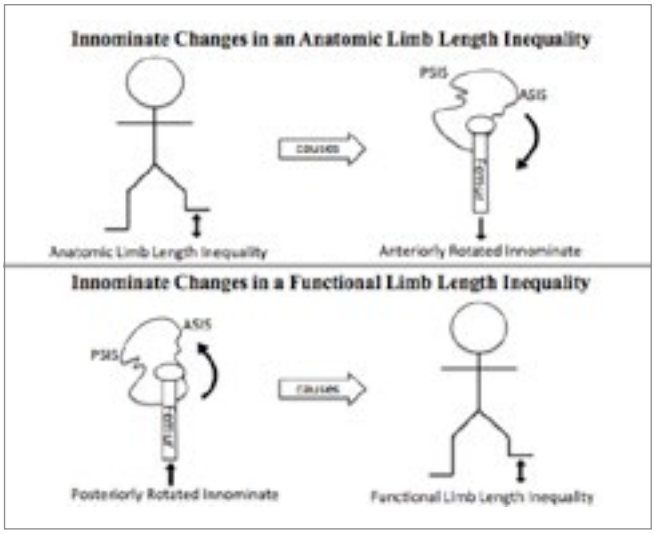
TABLE 1:

Physical Exam Findings in Short Leg Syndrome

Anatomic Landmarks (on the side of the short leg)	**Physical Exam Findings**
Medial Malleolus	Superior
Anterior Superior Iliac Spine (ASIS)	Inferior
Posterior Superior Iliac Spine (PSIS)	Superior
Iliac Crest	Inferior
Lumbar Spine	Contralateral side bending, ipsilateral rotation

FIGURE 1:

Innominate Changes in Anatomic & Functional Limb Length Inequalities



Innominate changes seen in limb length inequalities. The top part of the figure shows how the innominate rotates anteriorly to compensate for an anatomic limb length inequality. The bottom part of the figure shows how somatic dysfunction producing a posteriorly rotated innominate can cause a functional limb length inequality.

DISCUSSION

Several points regarding the treatment of this patient merit discussion. First, each of the doctors involved likely did not have SLS high on their differential diagnosis when they encountered this patient with low back pain. More than 85% of patients who present to primary care with LBP have a non-emergent and nonspecific cause,[17] the vast majority of which come from some type of strain on the structures that comprise the back itself (muscles, ligaments, tendons, disks, etc.).[18] Although it is unknown what percent of patients have low back pain due to SLS, knowing that the prevalence of LLIs in the general population is 90%, with a mean of 5.22 mm, and that clinical symptoms can be present in patients with LLIs as low as 5 mm,[1] should cause providers to place SLS high on their differential diagnosis for a patient with LBP. Had this been the case, the patient might have been diagnosed earlier. Second, it took almost one month before at least a sufficient structural exam was performed on the patient. Ideally, the patient would have been evaluated with a structural exam at his first presentation. The structural exam should have at least included a standing forward flexion test, and an evaluation of the medial malleoli lengths, ASISs, PSISs, iliac crests, and lumbar spine. For the reader's benefit, an additional section at the end of the article has been added on how to perform an adequate structural exam *(see Appendix, page 28)*.

Performing a good structural exam on a patient with LBP is analogous to performing a good cardiovascular exam on a patient with chest pain. Third, the practitioners involved were probably ill-equipped to correctly diagnose SLS. Most doctors have never been taught what physical exam findings to look for in SLS *(Table 1)*. Put together, these three aspects regarding this patient's several doctor-patient encounters make it easy to understand why a patient with SLS might be misdiagnosed. However, had the patient been diagnosed earlier, even in the emergency department, the correct treatment plan could have been initiated, and the interim pain, time, and money spent could have been avoided or minimized. The exact costs that SLS incurs on society are not known, but can be surmised when considered in the context of LBP. For LBP, 90 billion dollars of healthcare related expenses were spent nationally in 2010 (without factoring in lost opportunity cost such as days missed at work).[19] Additionally, of 291 conditions considered in the Global Burden of Disease 2010 Study, LBP ranked first in years lived with disability (YLDs) and sixth in the total burden of disease (Disability Adjusted Life Years or DALYs).[20] It seems reasonable to assume that if providers worldwide knew how to appropriately diagnose SLS, at least a small proportion of these costs would be reduced, given the prevalence and magnitude of anatomic LLIs in the general population.

CONCLUSION

SLS is an often missed diagnosis of LBP by providers. This can change if providers adequately educate themselves about the syndrome. On what specifically should they educate themselves? On two items: 1.) the prevalence of anatomic LLIs in the general population, and 2.) how to diagnose SLS from its characteristic physical exam findings. Regarding the prevalence, providers should remember that of the nonspecific 85% of LBP they will see, a substantial proportion will likely be due to SLS, given that 90% of the general population has an anatomic LLI, with a mean of 5.22 mm, and that clinical symptoms can be present in patients with LLIs as low as 5 mm. Regarding the diagnosis of SLS, providers should remember to perform adequate structural exams that at least include a standing forward flexion test, an evaluation of the medial malleoli lengths, the ASISs, PSISs, iliac crests, and the lumbar spine to determine if the following pattern of landmarks is appreciated on the side of the short leg: 1.) superior medial malleolus, 2.) inferior ASIS, 3.) superior PSIS, 4.) inferior iliac crest, and 5.) contralateral side bending and ipsilateral rotation of the lumbar spine. Additionally, disseminating this information to change SLS from being a commonly missed diagnosis of LBP to a common diagnosis of LBP will take considerable effort. The Osteopathic community is uniquely situated to help. Consider their focused education and diagnostic training in musculoskeletal complaints. Given these characteristics, they likely have the greatest potential to educate their colleagues across the nation on how to correctly diagnose SLS, thus, responsibility falls largely on their shoulders. Further research should be done to determine the proportion of patients with LBP that stems from SLS, the financial and other societal costs on the general population due specifically to SLS, and effective ways at disseminating knowledge on how to diagnose SLS effectively to the general medical community.

APPENDIX:

HOW TO PERFORM AN OSTEOPATHIC STRUCTURAL EXAM

Performing a structural exam on patients is critical to diagnose and treat them correctly, yet many providers do not remember how to perform one adequately. The purpose of this appendix is to teach clinicians how to perform a simple yet thorough osteopathic structural exam. Content contained herein has been summarized from the second edition of *The Atlas of Osteopathic Techniques*, by Alexander S. Nicholas and Evan A. Nicholas, 2012. Generally, a structural exam should include the following four main components, normally performed in the order provided:

I. Osteopathic Static Musculoskeletal Examination

II. Spinal Regional Range of Motion Testing

III. Osteopathic Layer-by-Layer Palpation

IV. Intersegmental Motion Testing

I. Osteopathic Static Musculoskeletal Examination

The goal of the static portion of the exam is to determine potential somatic dysfunction by identifying obvious structural asymmetries. To perform the static exam, visualize the patient from the anterior, posterior, and lateral views; then determine landmarks and compare symmetry.

Note or compare the following major landmarks from the ANTERIOR VIEW:

- Midgravitational line
- Head position in relation to shoulders and body
- Levelness of eyebrows
- Levelness of eyes
- Deviation of nasal bones and/or nose
- Angles of mouth
- Deviation of mentum
- Levelness of shoulders
- Depth of shoulders (anteroposterior relation)
- Thoracic symmetry
- Iliac crests
- Rotation of anterior superior iliac spine (ASIS)
- Levelness of patellae
- Pronation or supination of feet

Note or compare the following major landmarks from the POSTERIOR VIEW:

- Midgravitational line
- Head position in relation to shoulders and body
- Mastoid processes
- Neck to shoulder angles
- Levelness of shoulders
- Depth of shoulders (anteroposterior relation)
- Position of scapulae
- Erector spinae muscle prominence(s)
- Levelness of iliac crests
- Rotation of posterior superior iliac spine (PSIS)
- Levelness of greater trochanters
- Achilles tendons shape

Note or compare the following major landmarks from the LATERAL VIEW:

- Lateral midgravitational line connecting:
    - External auditory canal
    - Lateral head of humerus
    - Third lumbar vertebra
    - Anterior third of the sacrum
    - Greater trochanter of the hip
    - Lateral condyle of the knee
    - Lateral malleolus
- Head position in relation to shoulders and body
- Sternal angle
- Lordosis of cervical spine
- Kyphosis of thoracic spine
- Lordosis of lumbar spine
- Lumbosacral angle
- Flexion or extension of hips
- Flexion or extension of knees
- Arch of feet

II. Spinal Regional Range of Motion Testing

The purpose of spinal regional range of motion testing is to determine potential somatic dysfunction in the components of the body that cause motion around the cardinal axes of motion (flexion, extension, side bending, and rotation). To perform this section of the exam, physicians should test active and passive range of motion in the cervical, thoracic, and lumbar spine in flexion, extension, side bending, and rotation. It is easiest to test the cervical and thoracic spine while the patient is seated and the lumbar spine while the patient is standing. Examiners should look for asymmetries when comparing left and right, as well as any significant increase or decrease in range of motion when compared to normal range of motion values. Importantly, normal range of motion values vary depending on the source (in some cases significantly), therefore, it is incumbent for physicians to use their clinical judgment in deciding whether an increase or decrease in range of motion represents somatic dysfunction or a healthy patient with an acceptable outlying range of motion value. Clinicians desiring specific values may reference chapter three in the second edition of *The Atlas of Osteopathic Techniques* (2012), which compares three different sources for normal spinal range of motion values.

III. Osteopathic Layer-by-Layer Palpation

The layer-by layer examination has eight components, which are:

1. Observation
2. Temperature
3. Skin topography and texture
4. Fascia
5. Muscle
6. Tendon
7. Ligament
8. Erythema friction rub

1. Observation

Before touching the patient, visualize the area being examined first for signs of somatic dysfunction. See if there are any visual signs of trauma, erythema, swelling, fullness, diaphoresis, abnormal hair patterns, nevi, follicular eruptions, etc.

2. Temperature

Metabolic changes from trauma, infection, or even chronic fibrotic effects may generate heat which can be sensed with the wrists or hands. To evaluate, place either the volar aspect of the wrist or dorsal aspect of the hypothenar eminence of the hand a couple inches above the skin being tested. Do this over the area of interest and over the paraspinal areas.

3. Skin topography and texture

Somatic dysfunction may cause an increase or decrease in the humidity, oiliness, thickening, roughening, etc. of the skin. This can be sensed as the pads of the fingers are applied to the area being examined, light enough that the fingernail beds do not blanch.

4. Fascia

Fascia may "bind" or tighten when somatic dysfunction is present. To sense this, place the hands over the area to be tested and apply just enough pressure that the fingernail beds blanch. Move the hands superiorly, inferiorly, left, right, clockwise, and counterclockwise, to evaluate for areas of ease or restriction.

5. Muscle

Acute and chronic muscle injuries may cause somatic dysfunction that can be deduced through palpation. Acute changes have a boggy feeling overlying the muscle, while the muscle itself may feel like it is contracted, rigid, or hard. Chronic changes feel ropey or stringy. To determine, place the hands over the area to be examined and apply pressure deeper than that applied to the fascia.

6. Tendons

Damaged tendons may undergo fibrous thickening, or changes in their elasticity. Palpate tendons from their bony attachments to their continuation with the muscle belly.

7. Ligaments

Ligaments can cause somatic dysfunction by being too lax, causing joint laxity, or too tight, causing joint restriction. Some ligaments are more amenable to palpation than others. If able, palpate ligaments in the area of concern.

8. Erythema friction rub

The purpose of this test is to discover paraspinal areas with autonomic changes that cause segmental dysfunction. To perform, place the pads of the second and third digits over the paraspinals and stroke downward two to three times. Evaluate for redness at each spinal segment.

IV. Intersegmental Motion Testing

Intersegmental motion testing refers to articulatory motion in the spinal facets or at any joint. Most often, the goal of motion testing is to obtain a specific diagnosis. In some instances, a specific diagnosis is unable to be ascertained (such as the standing flexion test). Providers should focus motion testing not only on the area of the patient's complaint, but also on other areas where the body may be compensating for the original somatic dysfunction. Intersegmental motion testing includes tests that aid in the diagnosis of Short Leg Syndrome and leg length inequalities such as individual lumbar spinal segment motion testing and the standing flexion test. The details on spinal motion testing as well as other joint motion testing is beyond the scope of this appendix. Those who desire to know more may reference chapter five of the second edition of *The Atlas of Osteopathic Techniques*, 2012.

Note: All information on performing an osteopathic structural exam has been taken from The Atlas of Osteopathic Techniques, Second Edition, by Alexander S. Nicholas and Evan A. Nicholas, 2012. Only a summary of chapters two through five is provided here. For a more thorough and complete understanding, please reference the textbook.

ACKNOWLEDGEMENTS

This paper would not have been possible without the help of my attending physician, John C. McDonald, DO, FACOI. Dr. McDonald was the one who decided this patient needed a more thorough structural examination. This led to the patient being correctly diagnosed, which gave me the opportunity to write this paper. Thank you for the opportunity, guidance, and lessons you taught me.

I would be amiss not to mention Kendi Hensel, DO, PhD, my advisor and the principle investigator for this paper. Thank you for your patience, feedback, and edits. You made this paper better than I ever could have alone.

REFERENCES

1. Knutson GA. Anatomic and functional leg-length inequality: A review and recommendation for clinical decision-making. Part I, anatomic leg-length inequality: prevalence, magnitude, effects and clinical significance. Chiropractic & Osteopathy. 2005;13:11. doi:10.1186/1746-1340-13-11.
2. Friberg, O. Clinical symptoms and biomechanics of lumbar spine and hip joint in leg length inequality. Spine (Phila Pa 1976). 1983. 8(6):643-51.
3. Rush, W.A., and Steiner, H.A. A study of lower extremity length inequality. The American Journal of Roentgenology and Radium Therapy. 1946. 56(5): 616-623.
4. McCaw, ST., Bates, BT. Biomechanical implications of mild leg length inequality. British Journal of Sports Medicine. 1991. 25:10–13.
5. Cummings, G., Scholz, J.P., Barnes, K. The effect of imposed leg length difference on pelvic bone symmetry. Spine. 1993;18:368–373.
6. Young, R.S., Andrew, P.D., Cummings, G.S. Effect of simulating leg length inequality on pelvic torsion and trunk mobility. Gait Posture. 2000. 11:217–23. doi: 10.1016/S0966-6362(00)00048-5.
7. Juhl, JH., Cremin, TM., Russell, G. Prevalence of frontal plane pelvic postural asymmetry – part 1. The Journal of the American Osteopathic Association. 2004. 104:411–21.
8. Giles, L.G.F. Lumbosacral facetal 'joint angles' associated with leg length inequality. Rheumatology and Rehabilitation. 1981. 20:233–238.
9. Giles, L.G.F., Taylor, J.R. Lumbar spine structural changes associated with leg length inequality. Spine. 1982. 7:159–162.
10. Vogel Jr., F. Short-leg Syndrome. Clinics in Podiatry. 1984. 1(3): 581-599.
11. Gofton JP, Trueman GE. Studies in osteoarthritis of the hip: Part II. Osteoarthritis of the hip and leg-length disparity. Canadian Medical Association Journal. 1971. 104(9):791–799.
12. Fisk, J.W., Baigent, M.L. Clinical and radiological assessment of leg length. New Jersey Medicine Journal. 1975. 81(540):477–480.
13. Dott, G.A., Hart, C.L., McKay, C. Predictability of sacral base levelness based on iliac crest measurements. Journal of the American Osteopathic Association. 1994. 94(5):383-390.
14. Caselli, M.A., Rzonka, E.C. Detecting and Treating Leg Length Discrepancies. Podiatry Today. 2002. 15(12).
15. Koczewski, P., Shadi, M. Lower limb inequality treatment with subtrochanteric femoral shortening osteotomy fixed with intramedullary nail. Polish Orthopaedics and Traumatology. 2012. 77:65-71.
16. Koczewski, P., Shadi, M., Napiontek, M., Marciniak, W. [Problems, obstacles, and complications of femoral lengthening with the use of the Italian modification Ilizarov device]. Chirugia Narzadow Ruchu Ortopedia Polska. 2000. 65(3):277-86.
17. Chou, R., Qaseem, A., Snow, V., Casey D., Cross, J.T. Jr., Shekelle, P., Owens, D.K., Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Annals of Internal Medicine. 2007. 147(7):478–91.
18. Amirdelfan, K., McRoberts, P., Deer, T.R. The differential diagnosis of low back pain: a primer on the evolving paradigm. Neuromodulation. 2014.17(Suppl 2):11–17.
19. Borczuk, P. An evidence-based approach to the evaluation and treatment of low back pain in the emergency department. 2013. 15(7):1-23.
20. Roy, D., March, L., Brooks, P., Blyth, F., Woolf, A., Bain, C., Williams, G., Smith, E., Vos, T., Barendregt, J., Murray, C., Burstein, R., Buchbinder, R. The global burden of low back pain: estimates from the Global Burden of Disease 2010 study. Annals of the rheumatic diseases. 2014. 73(6):968-974.

CTCA® **FORUM** 2017

As patients with cancer are living longer, how can primary care physicians provide comprehensive and coordinated patient care in partnership with oncology care teams?

Advancements in Cancer Management for Primary Care

For more information and to register, visit CTCAFORUMCME.ORG

Topics include:

- Managing treatment-related side effects of chemotherapy
- Managing comorbidities during cancer care
- Developing palliative care and survivorship plans
- And more!

Coming to the following locations:

Tulsa, OK*
Phoenix, AZ
Philadelphia, PA*
Atlanta, GA
Chicago, IL

Earn CME/CE Credit! 5 AOA Category 1-A CME credits anticipated at select locations.

Look what others are saying about past forums:

"A unique opportunity to learn about the latest updates in cancer screening and diagnosis."

"Excellent variety of topics pertinent to cancer management."

ACHLI
ACADEMY for CONTINUED HEALTHCARE LEARNING

Cancer Treatment Centers of America®

This activity is sponsored by the Academy for Continued Healthcare Learning and Cancer Treatment Centers of America.
*5 AOA Category 1-A CME credits anticipated at this location.

© 2016 Rising Tide

Painful Cutaneous Nodules

Dana Baigrie, DO, PGY2,[1] Lindsay Tjiattas-Saleski, DO, MBA, FACOEP,[2] & Bhatraphol Tingpej, MD[3]

[1]LewisGale Montgomery Hospital, Medical Education - Resident
[2]Palmetto Health Tuomey Medical Center
[3]Sumter Medical Specialists PA, Tuomey Medical Center, Infectious Disease

A 61 year old African American male presented to the emergency department with a chief complaint of a three week history of tender nodules on his ears and bilateral hands. He initially developed white-yellow nodules on his ears and then similar tender lesions appeared on his hands and toes. The patient noted the lesions often drained yellowish-white fluid if manipulated. Arthralgias of the hands and feet were also present for about two weeks. His joints were enlarged, warm and tender to palpation. He admitted to similar arthralgias in the past, but never to this severity. The patient denied fever, chills, vision changes, lesions in his mouth, or sick contacts at home. Dermatologic evaluation revealed skin-colored to white-yellow firm dermal and subcutaneous nodules with ulceration and bloody exudate involving the helix and antihelix of his right ear (Figure 1) and large skin-colored firm, mobile subcutaneous nodules involving his dorsal hands at the interphalangeal joints (Figure 2). There were also white-yellow papules with ulceration and erythema on the palmar surface of PIP and DIP (Figure 3). The nodules on the ear released a white chalky discharge when firm pressure was applied.

Past medical history is significant for hypertension, hyperlipidemia, and gout. He denies alcohol, tobacco or drug use. Patient reports that he takes no medications. Family history is non-contributory.

Laboratory studies in the emergency department included an elevated ESR of 67mm/hr (normal 0-30mm/hr), CRP >9.0mg/dL (normal <1.0mg/dL), WBC count of 14 (normal 3.5-10.5 billion cells/L), neutrophils 80.7% (normal 42-76%), and serum uric acid of 8.1 (normal 2-7mg/dL). His renal function and electrolyte panel were within normal limits.

FIGURE 1:

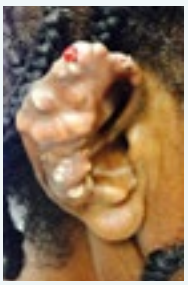


FIGURE 2:

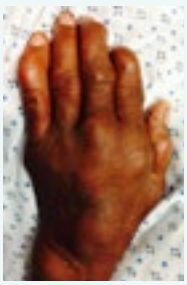


FIGURE 3:

QUESTIONS:

1. The most likely diagnosis?

A. Calcinosis cutis

B. Xanthomas

C. Rheumatoid nodules

D. Chronic tophaceous gout

2. What is the best diagnostic method?

A. Serum uric acid level

B. Lipid panel

C. Microscopic examination of aspirate or tissue sample

D. Imaging studies

3. What is the recommended initial treatment?

A. NSAIDS, colchicine, purine-free diet

B. Statin therapy and low-fat diet

C. IV antibiotics

D. Surgical excision of lesion

CORRESPONDENCE:
Lindsay Tjiattas-Saleski, DO | LRTJ55@yahoo.com

1877-5773X/$ - see front matter. © 2016 ACOFP. All rights reserved.

ANSWERS

1. What is the most likely diagnosis?

The correct answer is:

D) Chronic tophaceous gout

The differential diagnosis for gouty tophi includes xanthomas, rheumatoid nodules, and calcinosis cutis.[1] The diagnosis of chronic tophaceous gout can be made clinically with history and physical exam findings along with the presence of negatively birefringent needle-shaped crystals on microscopy. Xanthomas are dermal collections of lipids and may be a sign of underlying lipid disorder or gammopathy. They classically appear as firm yellow papules, nodules, or plaques distributed on the extensor surfaces of the hands, extremities, and/or buttocks.[1] Rheumatoid nodules appear in approximately 20% of patients with history of rheumatoid arthritis. The nodules are typically asymptomatic, firm, semi-mobile, and distributed over the extensor surface of joints spaces.[1] Calcinosis cutis or cutaneous calcification is due to a disruption in calcium pathways in the body and may occur due to medications, metastatic causes such as renal disease or sarcoidosis, infection, autoimmune disorders such as scleroderma, or idiopathic causes.[1]

2. What is the best diagnostic method?

The correct answer is:

C) Microscopic examination of aspirate or tissue sample

Identifying the presence of monosodium urate crystals under microscopy is the gold standard for diagnosis of gout. This diagnosis can be made via fine needle aspiration or skin tissue sampling.[2] The presence of intracellular needle-shaped crystals with negative birefringence on joint/lesion aspirate or tissue sample confirms the diagnosis of gout.[1] Hyperuricemia on serum uric acid laboratory studies is suggestive but not diagnostic for gout.[3] Lipid panel may be considered if evaluating for xanthomatous disease. The possibility of septic arthritis should be excluded with a gram stain and culture.[1] Imaging studies are typically not useful for diagnosing an acute gout flare.[1]

3. What is the recommended initial treatment?

The correct answer is:

A) NSAIDS, colchicine, purine-free diet

The treatment of an acute gout flare includes NSAIDs as first-line therapy as long as no contraindications such as renal failure or bleeding history are present.[1] Colchicine is used to treat both acute and chronic gout by decreasing swelling, reducing pain, and preventing future flare-ups.[1] Corticosteroids in oral, intravenous, intraarticular, or intramuscular form are an alternative pharmacologic treatment option in patients with medical contraindications to NSAIDs or colchicine such as those with renal disease.[3] Diets free of purine-rich foods including liver and fish as well as reducing alcohol consumption may also be of benefit to patients with gout. Xanthine oxidase inhibitors such as allopurinol are indicated, unless contraindications exist, for the long-term management of gout.[1,4]

CASE DISCUSSION

This patient was admitted to the hospital for further workup and management. Therapy with intravenous antibiotics and steroids was initiated as the diagnosis was unclear at time of presentation and was thought to possibly be infectious. During the course of his hospital stay, he reported improvement in symptoms. The chalky exudate (Figure 4) was sent for polarizing microscopy studies and revealed intracellular and extracellular needle-shaped negatively birefringent urate crystals and neutrophilia consistent with gout (Figure 5).

He was discharged on a regimen of allopurinol 100 mg daily, prednisone 40mg daily, colchicine 0.6mg PO daily, and cephalexin. 500 mg three times daily for seven days following discharge. Three weeks later, the patient reported improvement. His gout flare had clinically improved with tophi on the ears and extremities diminished in size (Figure 6,7,8). He did note continued arthralgias with erythema, swelling, and warmth in the affected joints, however improvement is anticipated with continued long-term control and medication compliance.

DIAGNOSIS

Chronic tophaceous gout

Gout is a chronic inflammatory disease that affects nearly 4% of the population in the United States.[5] In fact, it is the most common form of crystal-induced arthropathy, and the most common inflammatory arthopathy in men older than 40 years.[1] Gout is a deposition disease of metabolic origin caused by supersaturation of monosodium urate. Needle-like crystals are deposited into joints, connective tissue, and the kidneys. This deposition may lead to various clinical sequelae such as arthritis, tophi, and acute kidney injury.[1] Gout can transition through many phases including asymptomatic, acute, and chronic disease.[6] Environmental or genetic causes may contribute to development of hyperuricemia.[6] Men are more affected by gout than women, related to an estrogen-induced increase in urate clearance by the kidney.[7]

Primary forms of gout may be caused by inborn errors of purine metabolism or decreased excretion of uric acid by the kidneys. Secondary forms of gout are related to excessive cell turnover or secondary renal impairment.[2] Increased turnover may be related to diets rich in purines including proteins, fats, and sugary foods like soft drinks.[8] Under-excretion of uric acid by the kidneys accounts for up to 90% of gout cases.[2] Heavy alcohol intake and medications including diuretics, aspirin, and nicotinic acid may decrease excretion of uric acid and contribute to an acute attack.[8] Gout is also associated with hypertension, obesity, dyslipidemia, diabetes mellitus and insulin resistance.[7,9] Rare causes of hyperuricemia include tumor lysis syndrome, genetic conditions such as Lesch Nyhan syndrome, or malnutrition.[8]

Chronic untreated gout may lead to the development of tophi, a dermatologic manifestation involving collections of uric acid crystals that settle in soft tissues and joints. These skin lesions develop due to a foreign-body granulomatous reaction to the crystal deposits. Tophi are typically a clinical representation of long-standing gouty arthritis with average appearance 10 years after onset in approximately 10% of patients with gout.[1,10] The lesions often appear cream to yellow in color and are firm, mobile subcutaneous

FIGURE 4:

FIGURE 5:

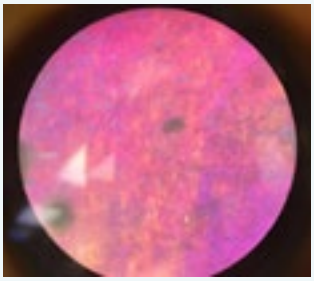


FIGURE 6:

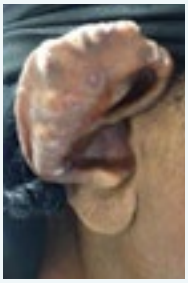


FIGURE 7:

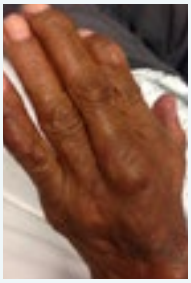
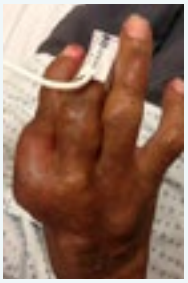


FIGURE 8:



nodules with erythematous overlying skin.[10] The drainage from lesions varies in appearance from a clear fluid to thick chalky discharge.[1] Tophi more commonly form in joints and cooler body surfaces such as the helix of the ear, fingers, toes, prepatellar bursa and olecranon.[11] Less common documented body sites of involvement include the eyelids, cornea, heart valves, and nasal cartilage.[10]

The presence of intracellular needle-shaped crystals with negative birefringence under polarized microscopy confirms the diagnosis of gout.[1] This diagnosis can be made via fine needle aspiration or skin tissue sampling such as shave or punch biospy.[2] Hyperuricemia on serum uric acid laboratory studies is suggestive but not diagnostic for gout.[3] Even with presence of urate crystals on microscopy, the diagnosis of septic arthritis should be excluded with a Gram stain and culture.[1] Imaging studies are not useful in the diagnosis of an acute gout attack, though may be useful in ruling out other diagnoses or identifying chronic changes associated with long-standing gout.[1]

Treatment of tophaceous gout involves implementation of dietary changes as well as pharmacologic and possibly surgical interventions.[12] Dietary changes such as eliminating purine rich foods and substituting for carbohydrate-rich and fatty foods may be difficult in patients with comorbidities such as diabetes and hyperlipidemia.[2] Medical treatment of chronic tophaceous gout includes long-term use of uric-acid lowering drugs such as allopurinol or probenecid.[8] Acute flares may be treated with colchicine, NSAIDs, or corticosteroids.[3] Comorbidities such as renal disease, hypertension, and diabetes must be considered before initiating medical treatment.[8] Corticosteroids in oral, intravenous, intraarticular, or intramuscular form are an alternative pharmacologic treatment option in patients with medical contraindications to colchicine such as those with renal disease.[3] Prior to the introduction of these gout medications approximately 60 years ago, chronic tophaneous gout would affect approximately half of patients with history of gout. However, this incidence has decreased significantly following the advent of allopurinol and colchicine.[7] Emphasizing the importance of long-term management of gout to patients is important. Complications of long-standing untreated gout may include secondary infection, urate nephropathy, renal stones, nerve impingement, or fractures in joints with tophi.[3]

REFERENCES:

1. Bolognia JL, Jorizzo JL, Schaffer JV. Dermatology. Third edition. Elsevier Saunders; 2012: 681-1547.
2. Perez-Ruiz, F., Castillo, E., Chinchilla, S.P., & Herrero-Beites, A.M. (2014). Clinical Manifestations and Diagnosis of Gout. Rheumatic Disease Clinics of North America, 40(2), 193-206.
3. Rothschild B. (n.d.). Gout and Pseudogout. Retrieved September 17, 2015, from http://emedicine.medscape.com/article/329958-overview.
4. Khanna D, Fitzgerald JD, Khanna PP. 2012 American College of Rheumatology Guidelines for Management of Gout Part 1: Systematic Nonpharmacologic and Pharmacologic Therapeutic Approaches to Hyperuricemia. Arthritis Care and Research. 2012; 64, 10:1431-1446.
5. Khanna P, Nuki G, Bardin T. Tophi and frequent gout flares are associated with impairments to quality of life, productivity, and increased healthcare resource use: Results from a cross-sectional survey. Health and Quality of Life Outcomes. 2012, 10:117
6. Neogi T. Gout. N Engl J Med. 2011; 364:443-452.
7. Hochberg M. Rheumatology. Sixth edition. Mosby, and imprint of Elsevier, 2015: 1549-1582.
8. Braun-Falco M, Hofmann C. Tophaceous gout in the finger pads. Clinical and Experimental Dermatology. 2010. 35, e22–e23.
9. Kharkar V, Kembre P, Mahajan S, Khopkar U. Multiple asymptomatic nodules in a middle-aged patient. Indian J Dermatol Venereol Leprol 2007;369-70.
10. Verma S, Bhargav P, Toprani T, Shah V. Multiarticular tophaceous gout with severe joint destruction: A pictorial overview with a twist. Indian J Dermatol 2014;59:609-611.
11. Zheng L, Han X. Cream-yellow and firm nodule in finger pad. Indian J Dermatol Venereol Leprol 2012;78:522.
12. Khandpur S, Minz A. Chronic tophaceous gout with severe deforming arthritis. Indian J Dermatol Venereol Leprol. 2010; 76, 1.

TEAMS WORK

WHERE HEALTH IS PRIMARY.

Health is Primary

Primary care teams of health professionals provide patients what they need when they need it in a coordinated setting.

Family doctors work closely with team members to keep their patients healthy.

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

Brought to you by
America's Family Physicians

#MakeHealthPrimary

Ecthyma

Timothy Potter, OMS IV,[1] Rob Danoff, DO, MS, FACOFP, FAAFP,[2] & Lindsay Tjiattas-Saleski, DO, MBA, FACOEP[3]

[1]Virginia College of Osteopathic Medicine - Blacksburg Campus
[2]Aria Jefferson Health - Langhorne, PA
[3]Palmetto Health Tuomey Medical Center

A mother brought her three month old son to the Emergency Department with a two day history of rapid onset of "lesions" on his left chest, left arm and face. The mother denies the patient had any trauma, fevers, chills, oral lesions, new medications or recent changes in formula. The patient does not have any significant past medical history. He is up to date on immunizations. Of note, his 11 year old sister presented with similar lesions on her posterior thigh/buttock region over the same time period. The only difference was that his sisters' lesions were pruritic.

Lesions on the three month olds' arm, chest wall and left cheek were as depicted in Figures 1-3.

QUESTIONS:

1. What is the moste likely diagnosis?

A. Candida

B. Ecthyma

C. Insect bites

D. Porphyria cutanea tarda

E. Venous stasis ulcers

2. What is the recommended plan of care?

A. Nystatin cream

B. Permethrin cream

C. Phlebotomy and low-dose hydroxychloroquine

D. Wound debridement, barrier creams and multilayered compression bandages

E. Wound debridement and topical mupirocin

FIGURE 1:

Left anterior-upper arm

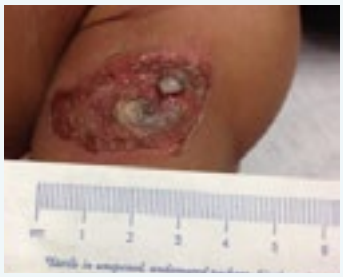


FIGURE 2:

Left cheek

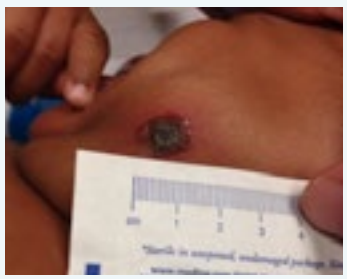
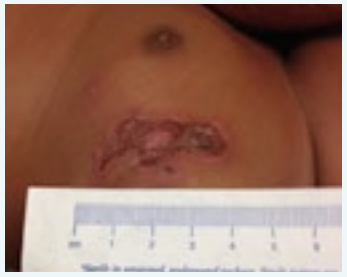


FIGURE 3:

Left anterior-lateral chest wall



CORRESPONDENCE:
Lindsay Tjiattas-Saleski, DO | LRTJ55@yahoo.com

1877-5773X/$ - see front matter. © 2016 ACOFP. All rights reserved.

ANSWERS

1. What is the most likely diagnosis?

The correct answer is:

B) Ecthyma

The patient's history combined with the characteristic appearance of the lesions points to ecthyma as the most appropriate diagnosis.[1] The lesion could have originally begun with an insect bite, but if so is now secondarily infected; ecthyma is a more correct answer.[1,2] Porphyria cutanea tarda is incorrect, as these lesions occur primarily on sun exposed areas.[1,3] Venous stasis ulcers are pruritic and typically occur on the lower extremities.[1] Candidiasis is unlikely as the lesions are not in the skin folds and lack the distinctive bright red exanthem, small pustules at the edges of the rash, or the characteristic pasty, white residue it may generate.[4]

2. What is the recommended plan of care?

The correct answer is:

E) Wound debridement and topical mupirocin

Treatment for ecthyma generally begins with debridement of the lesions so that antibiotics will better penetrate the skin and treat the underlying infection.[1,5] Choices of topical antibiotic include mupirocin three times daily for seven to ten days or retapamulin twice daily for five days.[1,5,6] For lesions that are extensive or resistant to initial treatment, oral antibiotics may be used.[1,7,8] Choices include dicloxacillin or cephalexin, 250 to 500 mg four times daily for ten days (clindamycin or erythromycin can be used for patients allergic to penicillins).[7,8] Phlebotomy and low-dose hydroxychloroquine are treatments for porphyria cutanea tarda.[1,3] Wound debridement, barrier creams and multilayered compression bandages are steps towards managing venous stasis ulcers.[7,11] Nystatin cream is applied topically for candida infection and permethrin cream is used in the treatment of scabies.[1,4,9]

THE BASICS OF IMPETIGO...WHAT IT IS, WHAT CAUSES IT, WHAT ARE THE TYPES:

Impetigo is a communicable skin infection that is more common in children, but can occur at any age.[10] It exists in three main forms including bullous, non-bullous and ecthyma.[10] Impetigo is contagious, and can be spread among individuals living in the same household.[11]

Ecthyma is an uncommon variant of the skin infection impetigo and is most commonly found on the distal extremities.[5,10] It consists of punched-out, ulcerative lesions with surrounding erythema.[5] While impetigo is most commonly caused by Staphylococcus aureus, the ecthyma variant is most often caused by group A Streptococcus.[5] It is worth noting, however, that both conditions can be caused by either organism.[2] While most frequently seen in children ages two to six years of age, this type of infection can be seen at any age.[11] Infection often occurs after minor skin injuries or conditions such as abrasions, dermatitis, and insect bites.[11] For this reason, it is often seen among the homeless population as well as individuals in third world countries without the ability to maintain proper hygiene.[11]

SYMPTOMS/DESCRIPTION

Both impetigo and ecthyma may cause mild pain and pruritis.[7] Infections are often found in areas of skin that have recently been injured due to scratching or an insect bite.[2] Scratching the lesions may spread the infection, and the development of satellite lesions is common due to autoinoculation.[5] The diagnosis of the three variants of impetigo is made clinically based on appearance of the lesions.[7] Those lesions exhibiting a honey-colored crust are characteristic of bullous and nonbullous impetigo.[7] Interestingly, these two forms of impetigo infection occur in the superficial epidermis and do not extend below the dermal-epidermal junction.[5]

By contrast, ecthyma is often referred to as deep impetigo because it extends into the dermis.[2] It begins with a small, pus-filled blister and red border, which eventually leaves a crusty ulcer underneath.[2] Ecthyma is characterized by purulent, shallow ulcers with a punched-out appearance.[7] Overlying the ulcer is a thick, brown-black crust and surrounding erythema.[7] Cultures of ecthyma lesions are indicated only when empiric antibiotic therapy fails to resolve the problem.[7] In this case, patients should have a nasal culture and wound culture performed to identify Methicillin Resistant Staphylococcus aureus.[7]

Important diagnoses to include in a differential include excoriated insect bites, Porphyria cutanea tarda, venous stasis and ischemic ulcers of the legs.[6] For any patient with a history of recent travel or relevant exposures, alternative diagnoses such as cutaneous anthrax and other potentially serious zoonotic infections must be considered.[12] Ecthyma, as a variant of impetigo, should not be confused with Ecthyma gangrenosum, a bacterial infection caused by Pseudomonas aeruginosa and most commonly seen in immunocompromised patients.[12] Ecthyma gangrenosum involves vesicles and pustules that hemorrhage and ulcerate into a necrotic eschar.[12]

TREATMENT

In order to treat both ecthyma and impetigo, the lesions must be debrided.[1,5] By removing the crusted exudate from the lesions, topical antibiotics are better able to penetrate the skin and treat the underlying infection.[5] Mupirocin has been shown to be highly effective against gram-positive bacteria, such as Staphylococcus aureus and group A streptococcus.[5] For effective treatment, mupirocin should be applied three times daily for a period of seven to ten days.[1,5] Alternatively, retapamulin or fusidic acid can also be used.[7,8] While penicillin is usually an effective oral agent, antistaphylococcal agents such as dicloxacillin, cephalexin, clindamycin, etc. may be necessary for extensive lesions or lesions that are resistant to treatment.[7,8]

Several measures can be taken to prevent infection.[11] Patients should be encouraged to practice good hygiene with use of soap and water, to avoid sharing towels and to wash their clothes regularly.[11] For individuals diagnosed with impetigo, family members of the individual should be checked for signs of infection.[6] If there are concerns regarding exposure to impetigo or ecthyma, further preventive measures such as benzoyl peroxide wash and ethanol or isopropyl gel for hands/involved sites can be taken.[6]

Timely treatment of impetigo generally leads to prompt recovery.[6] Failure to treat the infection can lead to more extensive spread of disease.[6] Lesions can progress to infections deeper in the skin and soft-tissues.[6] Complications of group A strep (GAS) induced impetigo include guttate psoriasis, scarlet fever and glomerulonephritis.[6] Recurrent infections can occur due to either failure to eradicate the pathogen or by recolonization.[6] Scarring may be seen upon healing of ecthyma lesions.[6]

REFERENCES

1. Bolognia, Jean, Joseph L. Jorizzo, and Julie V. Schaffer. Dermatology. Philadelphia: Elsevier Saunders, 2012. Web.
2. Berman, K. (2013, May 15). Ecthyma. Retrieved July 30, 2015, from http://www.nlm.nih.gov/medlineplus/ency/article/000864.htm
3. Singal, Ashwani K., Csilla Kormos–Hallberg, Chul Lee, Vaithamanithi M. Sadagoparamanujam, James J. Grady, Daniel H. Freeman, and Karl E. Anderson. "Low-Dose Hydroxychloroquine Is as Effective as Phlebotomy in Treatment of Patients With Porphyria Cutanea Tarda." Clinical Gastroenterology and Hepatology: 1402-409. Web. 7 Dec. 2015.
4. Aaron, Denise M., MD. "Candidiasis." Merck Manuals Consumer Version. Merck & Co., Inc. Web. 24 Jan. 2016. <http://www.merckmanuals.com/home/skin-disorders/fungal-skin-infections/candidiasis>.
5. Norman, R., & Nodine, S. (n.d.). Impetigo and Poor Hygeine. Journal of the American Osteopathic College of Dermatology, 12(1), 16-16. Retrieved July 19, 2015.
6. Wolff K, Johnson R, Saavedra A.P. (2013). Section 25. Bacterial Colonizations and Infections of Skin and Soft Tissues. Fitzpatrick's Color Atlas and Synopsis of Clinical Dermatology, 7e. Retrieved July 30, 2015 from http://accessmedicine.mhmedical.com.vcom.idm.oclc.org/content.aspx?bookid=682&Sectionid=45130158.
7. Dhar, A. (2013, May 1). Impetigo and Ecthyma. Retrieved July 30, 2015, from http://www.merckmanuals.com/professional/dermatologic-disorders/bacterial-skin-infections/impetigo-and-ecthyma
8. DermNet NZ: author: Vanessa Ngan, staff writer, 2003. Updated by Dr Jannet Gomez, Postgraduate student in Clinical Dermatology, Queen Mary University London, UK, January 2016.
9. "Medications." Centers for Disease Control and Prevention. Centers for Disease Control and Prevention, 09 Sept. 2015. Web. 24 Jan. 2016. <http://www.cdc.gov/parasites/scabies/health_professionals/meds.html>.
10. Pallin, Daniel, Nassisi, Denise. Skin and Soft Tissue Infections. Rosens Emergency Medicine. 8th Edition. Copyright 2014. Retrieved 9/2/2015 on VCOM Library Website
11. Usatine R.P., Smith M.A., Chumley H.S., Mayeaux E.J., Jr. (2013). Chapter 116. Impetigo. The Color Atlas of Family Medicine, 2e. Retrieved July 30, 2015 from http://accessmedicine.mhmedical.com.vcom.idm.oclc.org/content.aspx?bookid=685&Sectionid=45361173.
12. Orbuch, D., Kim, R., & Cohen, D. (2014). Ecthyma: A potential mimicker of zoonotic infections in a returning traveler. International Journal of Infectious Diseases, 29, 178-180. http://dx.doi.org/10.1016/j.ijid.2014.08.014

CALENDAR OF EVENTS

2017

January 13 - 15, 2017
Iowa ACOFP Midwinter Osteopathic Family Practice Conference
Des Moines, Iowa
www.ioma.org

January 19 - 22, 2017
Michigan ACOFP Mid-Winter Family Medicine Update
Bellaire, Michigan
www.maofp.org

February 2 - 5, 2017
Future Leaders Conference
Atlanta, Georgia
www.acofp.org

February 9 - 12, 2017
Maine Osteopathic Association Midwinter Conference
Portland, Maine
www.mainedo.org

February 10 - 12, 2017
Ohio ACOFP Annual Family Practice Review & Reunion
Dayton, OH
www.ohioacofp.org

March 16 - 19, 2017
ACOFP 54th Annual Convention & Scientific Seminar
Gaylords Palms Resort & Convention Center
Kissimmee, Florida
www.acofp.org

April 5, 2017
DO Day on the Hill
Washington, DC
www.osteopathic.org

April 19 - 23, 2017
Ohio Osteopathic Symposium
Columbus, Ohio
www.ohioacofp.org

April 27 - 30, 2017
Oklahoma Osteopathic Association
Norman, Oklahoma
www.okosteo.org

June, 2017
TOMA - Texas ACOFP Joint Annual Convention
Fort Worth, Texas
www.txacofp.org

June 9 - 11, 2017
Maine Osteopathic Association Annual Oceanside Convention
Rockport, Maine
www.mainedo.org

July 17 - 23, 2017
AOA House of Delegates
Chicago, Illinois
www.osteopathic.org

July/August, 2017
Florida ACOFP Annual Convention
Orlando, Florida
www.fsacofp.org

August 3 - 6, 2017
California ACOFP Annual Scientific Medical Seminar
Anaheim, California
www.acofpca.org

August 3 - 7, 2017
TOMA - Texas ACOFP Joint Annual Convention
San Antonio, Texas
www.txacofp.org

August 4 - 6, 2017
POFPS Annual CME Symposium
Hershey, Pennsylvania
www.poma.org

August 11 - 14, 2017
North Carolina Society ACOFP Annual Meeting
Carolina Beach, North Carolina
www.nc-acofp.org

October 7 - 11, 2017
OMED®17
AOA/ACOFP Osteopathic Medical Conference & Exposition
Philadelphia, Pennsylvania
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.

SEPTEMBER / OCTOBER 2016 ANSWERS: 1. B 2. C 3. B 4. A 5. A 6. A 7. C 8. A 9. A 10. D

acofp American College of Osteopathic Family Physicians

OBESITY: DIETARY MODIFICATIONS TO ACHIEVE A HEALTHY WEIGHT

Peter Zajac, DO, FACOFP, Author

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

Obesity can put you at risk for developing a number of conditions such as high blood pressure, diabetes, heart disease, and some forms of cancer. In addition to regular exercise and behavioral modification, eating a healthy well-balanced diet should be included in all obesity management approaches for a Body Mass Index (BMI) of 25.0 or higher. BMI is an index of weight-for-height commonly used to classify overweight and obesity in adult individuals. A BMI between 18.5 and 24.9 is considered a healthy weight. Overweight is characterized as a BMI equal to or more than 25.0 and 29.9. Obesity is a BMI equal to or more than 30.0. Healthy eating patterns support a healthy body weight, by balancing energy (caloric) intake to energy use, and can help prevent and reduce the risk of chronic disease.

DIETARY MODIFICATIONS INCLUDE:

- Your doctor should encourage you to eat a healthy well-balanced diet with whole grains, beans, fresh vegetables and whole fruits (instead of simple sugars and carbs), fat-free & low fat (1%) dairy, and protein foods (e.g. fish, lean meats, poultry, and nuts/seeds). If your doctor has no concerns about you doing exercise, you should also perform at least 150 min/week of physical activity (such as brisk walking) and muscle strengthening exercises over at least 2 days/week to achieve the appropriate weight loss and body conditioning.
- Avoid sugary soft drinks and fruit juices. Studies have shown that in overweight and obese middle-aged and older adults on a low calorie diet, drinking water before each main meal aided weight loss.
- A reasonable weight loss goal is 1-2 pounds per week. You can do this by eating 500-1,000 fewer calories each day (this could be as easy as two less sodas per day!). As with all chronic conditions, effective management of obesity requires a highly motivated individual and a committed team of health professionals including your Osteopathic Family Physician, nutritionists/dieticians, and other subspecialties.
- Weight-loss programs, approved by your doctor, can be encouraging, more successful and affordable led by specialty-trained staff. In spite of all the dietary strategies out there, weight management still comes down to the calories you take in versus those you burn off.
- Cutting calories is as simple as skipping high-calorie, low-nutrition items, substituting high-calorie foods for lower calorie options, and reducing portion sizes. At the beginning of a meal, take slightly less than what you think you will eat. Eat from plates, not packages or containers. Check food labels for nutritional facts, serving size, and number of calories per serving. Use a calorie counter as a helpful tool.
- Combining regular activity and healthy eating patterns will best help you achieve and maintain a healthy body weight.

MEDICAL CARE & TREATMENT OPTIONS:

If you have any questions about obesity, please contact your Osteopathic Family Physician. With a thorough history and physical exam, your doctor will help you determine which dietary changes along with any other treatment options will be best for you to achieve a healthy body weight. In case of any emergency, you should call your doctor or 911 right away.

SOURCE(S): ***Obesity and Dietary Modifications. Gov, Up-to-Date, and U.S. Centers for Disease Control & Prevention***

The Osteopathic Family Physician Patient Handout is a public service of the ACOFP. The information and recommendations appearing on this page are appropriate in many instances; however, they are not a substitute for medical diagnosis by a physician. For specific information concerning your personal medical condition, ACOFP suggests that you consult your family physician. This page may be photocopied noncommercially by physicians and other health care professionals to share with their patients.

For additional patient related educational material please visit our website at www.acofp.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

Over 30 AOA Category 1-A CME credits anticipated!

Pre-Con begins on March 15

acofp '17

MARCH 16 - 19, 2017

Kissimmee, Florida | Gaylord Palms Resort & Convention Center

Register online and make your hotel reservations this fall at acofp.org.