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EXAMS	LOCATIONS	POSTMARK DATE
Family Medicine / OMT Certification / OCC Cognitive Exam	**Electronic Testing** Regional Sites September 28, 2019	**April 1, 2019** *Late fee through June 1, 2019*
Family Medicine / OMT Certification / OCC Performance Evaluation Only	**AOA OMED Conference** Fall, 2019 exam dates TBD	**April 1, 2019** *Late fee through June 1, 2019*

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William Carey University College of Osteopathic Medicine

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William Carey University College of Osteopathic Medicine

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William Carey University College of Osteopathic Medicine

Kimia Etemadi
A.T. Still University - School of Osteopathic Medicine in Arizona

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

Kathryn Johnson
A.T. Still University - School of Osteopathic Medicine in Arizona

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

Nadia Khan
Michigan State University College of Osteopathic Medicine

Matthew Kon
William Carey University College of Osteopathic Medicine

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

Luiza Mnatsakanyan
William Carey University College of Osteopathic Medicine

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

Abi Moeller
A.T. Still University - School of Osteopathic Medicine in Arizona

Hennah Patel
William Carey University College of Osteopathic Medicine

Amena Payami
William Carey University College of Osteopathic Medicine

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

Mike Ramalia
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William Carey University College of Osteopathic Medicine

Diana Roy
William Carey University College of Osteopathic Medicine

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

Jordan Burr
A.T. Still University - School of Osteopathic Medicine in Arizona

Nicholas S. Tito
William Carey University College of Osteopathic Medicine

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

Nusrat Uddin
William Carey University College of Osteopathic Medicine

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2019 CALL FOR PAPERS

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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.

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

REVIEW ARTICLE TOPICS

- Disorders of Puberty: An Approach to Diagnosis and Management with an osteopathic component
- Chronic Kidney Disease: Detection and Evaluation with an osteopathic component
- Direct Primary Care: Emerging Practice Alternative with an osteopathic component
- Diagnosis and Management of Non-Melanoma Skin Cancer with an osteopathic component
- Update on Office-Based Strategies for the Management of Obesity with an osteopathic component

RESEARCH TOPICS

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

The content should include the following:

Abstract
Introduction
Methods
Results
Discussion
Conclusions
Acknowledgments

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EDITOR'S MESSAGE

Spring is in the Air

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

Welcome to another issue of the *Osteopathic Family Physician*. Spring is a time of renewal and rebirth and should be upon us soon given the recent groundhog revelation. The springtime traditions of Hanami in Japan, Whuppity Scoorie in Scotland, Baba Marta Day in Bulgaria to the burning of the Böögg on Sechseläuten in Switzerland all remind us of the coming seasonal change and perhaps give us pause to consider a virtual spring cleaning of our life and mind. Heavy stuff for an Editor's Message, I know.

On the "out with the old, in with the new" theme, this is the last year that there will be separate match systems for osteopathic medical students. Starting in 2020, there will be only one combined match program for all medical students in the United States applying for residency. This combined with changes in the Board certification process has created an air of excitement within the osteopathic family medicine community. I hope to have informational articles in the future to address these issues.

Speaking of issues, the articles within this month's issue bring to light some of the unique practices related to being an osteopathic physician and a medical student. Osteopathic manipulative treatment (OMT) is the most visible unique aspect of being a D.O. and our cover article helps provide some assistance in the use of OMT in patients with abdominal pain. The article on a patient-centered, holistic approach to eye conditions as well as the patient education handouts further demonstrates features making D.O.'s unique. Enjoy this issue and have a great start to your spring!

Burning of the Böögg on Sechseläuten.
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FROM THE PRESIDENT'S DESK

A Year in Review

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

Has it really been a year since Dr. Wiseman issued a gavel to the new President of the American College of Osteopathic Family Physicians?

Well, it started off with a blast, literally. Those of you who were there might recall the implosion of the building in downtown Austin. It was quite the show. Once the dust settled, we made our way home to the neighboring state of Oklahoma.

My first official trip as President started well. I made my way to Vallejo, CA. As I entered I was questioning whether I had found my way to a set used in the movie Top Gun. I later learned that in fact the campus is a decommissioned US Navy base. After the campus tour and visits with faculty and administration, I was treated to dinner with a number of medical students and afforded the opportunity to bring greetings and discuss the joys of osteopathic family medicine.

In the beginning of June my wife and I headed to the opposite corner of the US, Rockport, Maine. In addition to visiting with the Executive Committee of the state association and membership, I was treated to a lesson in lobster dissection and consumption, by none other than Dr. Buser.

From there our journey took us to Florida to meet with the **Board of Governors** for the annual Board strategic planning retreat. This year's retreat was a bit challenging, as Pete had announced his retirement, but a replacement had not been identified.

The House of Delegates was in mid-July, where many Governors also represent their state osteopathic medical societies. As the Delegate for the ACOFP, I presented testimony to the **AOA Board of Trustees** speaking against an amendment that appeared to make it more challenging for an osteopathic organization to gain a seat in the House than for a non-osteopathic group. The requirements should at least be the same, in my opinion.

The following week I took a return trip to Orlando for the **Florida State ACOFP Society** meeting. There I engaged with students, residents and faculty (COM and residency programs) in a variety of settings.

My next trip was to Hershey, PA. Yes, this is home to the giant chocolate company of the same name. I left Maine thinking that they had fed me well, but it is hard to compete with a suit case loaded with chocolate when headed home!

I took the long way home from Hershey, stopping along the way for a visit with Mike Park and the folks at Alston and Bird in Washington DC. There I was prepared for comments to be shared in the HHS headquarters regarding the opioid issues facing the United States.

The **ACOFP Search Committee** convened in Chicago to interview applicants who had applied for the Executive Director's position. Six individuals were interviewed initially, then three were invited for a second interview. It was through this process that our current Executive Director was hired.

As you are aware, **OMED** was in San Diego. That trip brought exciting news for me. An agreement was reached with the favored candidate for the Executive Director's position. Also, it was during this meeting that meaningful conversation with AOBFP about the future of OCC and certification began in earnest. Finally, the **ACOFP Foundation** articulated a proposal, which I will allow that body to reveal when the time is appropriate.

Some of the excitement was short lived, as Dr. DeLuca and I accepted an invitation to the **AAFP Congress of Delegates**. While there, the American Board of Family Medicine announced their pilot program for MOC, which subsequently lit up social media!

I attended one meeting in November in Wichita, where it was revealed that a proposed osteopathic medical school may be built. I was pleasantly surprised to encounter a pair of medical school classmates, who had completed residency and stayed there. Greetings were delivered from the ACOFP during lunch.

In December, it was off to Indianapolis. There, in addition to presenting comments to the state society meeting, I visited the Marian Campus. At this meeting students and faculty alike met with me in an informal setting for discussions about Osteopathic Family Medicine.

After the New Year, things became a bit hectic again.

The coolest trip of the year was to Des Moines, Iowa. Literally, the coolest, the temperatures were in single digits the whole weekend! To complete the blizzard-like conditions was the blowing snow! In addition to addressing the state society and installing a new president, I enjoyed visiting with Governor Cohen and Past President de Regnier.

The **AOA LEAD Conference** in Las Vegas afforded me the opportunity to visit with many of the state society leaders, once again, in addition to the AOA leadership. It also provided a chance to introduce our new executive director to similar folks from other specialty societies within the AOA.

I would like to wrap with a series of Thank Yous.

To Pete and now Bob: thanks for all of your dedication and hard work. You do amazing work running the office and keeping the Board on track.

To all of the state society executive directors: each of your respective states presents you with unique challenges. Thanks for all of your hard work. The same goes to the state officers. Your greetings, warmth and hospitality have been very much appreciated throughout the years.

To the Board of Governors, to the Committee Chairs and to the Committee members: thank you for your dedication to the ACOFP and to the osteopathic profession. Keep up the good work.

To Dr. DeLuca, thanks for your support and insight. Good luck and safe travels in YOUR year!

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

Rocky Mountain OPTI/Sky Ridge Medical Center
Neuromusculoskeletal Medicine + 1 Residency

Our program was established to enable physicians who have already completed a residency in an approved specialty to spend an extra year enhancing their skills in neuromusculoskeletal medicine and osteopathic manipulative medicine (NMM/OMM). Our goal is to develop highly trained physicians who can act as both clinicians and academicians. Our program places a significant emphasis on the integration of osteopathic manipulative medicine and the principles of primary care sports medicine. Our residents develop their Osteopathic clinical skills by providing inpatient care at Sky Ridge Medical Center and outpatient care at the Rocky Vista Health Center and other associated outpatient clinics.

Our program also includes such rotation choices as neurological surgery, occupational medicine, orthopedic spine surgery, podiatric medicine, primary care sports medicine, neurology, physical medicine and rehabilitation, rheumatology, musculoskeletal radiology, medical acupuncture, family medicine, integrative medicine, functional medicine, hospice and palliative care, internal medicine, obstetrics and gynecology and pediatrics. Academic development occurs through the Rocky Vista University College of Osteopathic Medicine in Parker, Colorado. Successful program completion will allow the physician to apply for the Neuromusculoskeletal Medicine/Osteopathic Manipulative Medicine certification examination.

Kenneth A. Ramey, DO, FACOFP serves as the program director and is a 1994 graduate of the Chicago College of Osteopathic Medicine. He is board certified in family medicine/osteopathic manipulative treatment, neuromusculoskeletal medicine/osteopathic manipulative medicine and has a certificate of added qualification in sports medicine. Dr. Ramey is a member of the medical staff at Sky Ridge Medical Center and has served as a team physician at the high school, college and semi-professional levels. He is an Associate Professor of OPP at Rocky Vista University and serves as the Director of the Sports Medicine and Osteopathic Manipulative Medicine Program at the Rocky Vista Health Center.

We have received ACGME Pre-Accreditation and would be honored to consider your application for our program. Please send a current CV, letter of interest and three letters of recommendation (including one from your residency director) to Dr. Ramey at kramey@rvu.edu. Please call Dr. Ramey at (720) 874-2421 if you need additional information.

"The purpose of Osteopathy is to make life a little more comfortable for the patient."

"What are the limits of Osteopathy? No one knows the limits of Osteopathy."

John Martin Littlejohn, DO

RESEARCH ARTICLE

Allopathic Supervision of Osteopathic Education: What Support is Needed?

Sarah J. James, DO[1]; Larissa Zakletskaia, MA[1]

[1] University of Wisconsin School of Medicine and Public Health, Department of Family Medicine and Community Health, Madison, WI

KEYWORDS:

Graduate Medical Education

Osteopathic Manipulative Medicine

Single Accreditation System

Abstract

Background: With the Accreditation Council for Graduate Medical Education implementation of a Single Accreditation System through which allopathic programs can obtain osteopathic recognition, it is vital to continue to support the development of resident osteopathic skills, which could be a challenge without properly trained faculty. The ability and confidence of allopathic and osteopathic faculty in providing Osteopathic Manipulation Treatment (OMT) supervision is unknown, but there is likely a need for faculty development in this area.

Methods: A 18-item survey to assess allopathic faculty confidence and concerns about faculty development in precepting OMT was completed by program directors and clinical coordinators of programs registered with the American College of Osteopathic Family Physicians.

Results: When comparing AOA (n=93) and AOA/ACGME accredited programs (n=80), significantly fewer respondents from AOA programs perceived their allopathic faculty as confident in their ability to precept OMT (36% vs. 50%, respectively, p<0.001). Despite concerns about allopathic faculty's ability to precept OMT, reported by 64% of AOA programs and 50% of AOA/ACGME programs, only a minority of programs had educational programs in place for allopathic faculty (26% and 41%, respectively). Respondents listed the four most important topics to include as part of faculty development in osteopathic skills as somatic function diagnosis, osteopathic treatment plan theory, muscle energy and myofascial techniques.

Conclusion: There was documented concern by respondents regarding their allopathic faculty's ability to precept OMT, suggesting the need for standardized education on OPP and OMT to better equip allopathic faculty to support osteopathic residents in OPP and maintain OMT skills.

INTRODUCTION

The use of osteopathic principles and practice (OPP) and osteopathic manipulation treatment (OMT) are factors that distinguish osteopathic physicians from their allopathic colleagues. OMT has been reported to be effective in a variety of conditions including back pain[1,2], headaches,[3] pregnancy related back and pelvic pain,[4] and pneumonia.[5] Many osteopathic medical school graduates have indicated that they would utilize OMT in their future practice.[6] Attainment of OMT and OPP knowledge is part of primary care osteopathic residency training in osteopathic(AOA) and dual(AOA/ACGME)-accredited residency programs. In these programs, residents indicated that having osteopathic mentors and support for OMT would influence them to continue its use in their future practice.[7] Therefore, it is important to have trained teaching faculty to provide education and supervision of OPP and OMT for these residents.

CORRESPONDENCE:

Sarah J. James, DO | Sarah.james@fammed.wisc.edu

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The American Osteopathic Association was added as a member of the American Council of Graduate Medical Education (ACGME) in 2014, creating a Single Accreditation System (SAS). The SAS allows ACGME-accredited residency programs to seek Osteopathic Recognition (OR), a desired designation for recruitment of osteopathic medical graduates. Through OR, osteopathic competencies have been added to ACGME accreditation standards for physician training. This added level of competency-based training will increase the need for faculty who can conduct such training. One criterion for attaining OR is to designate at least two osteopathically-educated faculty in these programs. While the need for faculty competent to continue the training of Doctor of Osteopathic Medicine (DO) residents is clear, the precise number needed is not.

Training additional clinicians to teach OMT is challenging. The competence of OMT precepting and preceptors is not clear. While standards exist for OMT-based knowledge acquisition and transfer of OMT skills at the medical-student level[8], no such criteria exist for residents. While DO residents are supervised by both board-certified DO and Doctor of Medicine (MD) faculty, there

is no formal accreditation certifying OMT skills for supervising physicians; skills may be less than they should be. Anecdotally, many DO faculty do not regularly use their OMT skills in practice, and many MD faculty are unfamiliar with OMT.[9]

The perceived need to train additional physicians to teach OMT, and concern about the quality of current residency-based OMT supervision is unknown. Residency leadership personnel are intimately involved in the day-to-day teaching and assessment of OMT and are in a unique position to offer input in OMT training and supervision; their views may serve as a proxy for direct assessment and inform future OMT-related training activities.

Therefore, we surveyed residency program directors and clinical coordinators of programs registered with the American College of Osteopathic Family Physicians. The purpose of this survey was threefold:

1) To determine the participants' confidence in the ability of their allopathic faculty to provide OMT supervision of osteopathic residents;

2) To determine whether participants are concerned about the quality of residency-based OMT supervision by allopathic faculty;

3) To assess current faculty development efforts for teaching and precepting OMT and their preferred content of a hypothetical standardized allopathic preceptor curriculum on OPP and OMT.

METHODS

Subjects and setting

We distributed a cross-sectional survey via paper and electronic format to residency program directors and clinical coordinators of programs registered with the American College of Osteopathic Family Physicians. One hundred and seventy AOA and 113 AOA/ACGME-accredited programs were surveyed. Programs ranged from two to thirty residents, with one to thirteen core faculty. The IRB determined this project exempt.

Instrument

We created an 18-item survey assessing program characteristics including AOA-accredited or AOA/ACGME-accredited status, numbers of faculty and residents, numbers of faculty who performed OMT, the perception of their allopathic faculty's confidence in their ability to precept OMT and their level of concern regarding allopathic faculty precepting OMT. Respondents were also asked about training provided to allopathic faculty in osteopathic education. Finally, respondents were asked to indicate the most important topics to introduce to allopathic faculty preceptors for faculty development in osteopathic skills to enable more effective supervision. Comments were solicited for methods of providing OPP/OMT education to allopathic faculty.

Procedures

Paper and online versions of this survey were distributed to the program directors and program coordinators for all programs registered with American College of Osteopathic Family Physicians Program (ACOFP). Paper versions were distributed to participants of the March 2014 ACOFP Program Director's workshop. A survey link was emailed twice to all 283 programs registered with ACOFP. If both the program coordinator and program director from an individual program completed surveys, one of the entries was randomly chosen. The same procedure was done for duplicate entries for participants completing both the paper and online versions. Respondents from four program surveys indicated that their program had not started taking residents yet; therefore they were removed from the data set. Also, the outliers of the highest and lowest number of residents were removed.

Data analysis

The analysis calculated summary statistics and compared responses between respondents from AOA only and AOA/ACGME-accredited programs using a chi-square test.

RESULTS

There was a 61% survey completion rate (173 of 283 surveys). Ninety-three (54%) were AOA programs and eighty (46%) were AOA/ACGME-accredited. There were about the same number of osteopathic residents in AOA and AOA/ACGME programs, as well as MD faculty. Both DO and MD faculty were more likely to perform OMT in AOA/ACGME programs compared to AOA-accredited programs. Additional characteristics of the responding programs are shown in *Table 1*.

When assessing respondents' perception of their allopathic faculty's confidence in the ability to precept OMT, 34% of AOA programs (n=93) and 53% of AOA/ACGME programs (n=80) answered positively ($p<0.001$). Most respondents (64%) from AOA programs reported concern regarding their allopathic faculty's ability to precept OMT and 50% of respondents from AOA/ACGME-accredited programs reported concern (P=.112).

As shown in *Table 1*, only 37% of AOA programs and 47% of AOA/ACGME programs had education in precepting OMT for their allopathic faculty. Also, 26% of AOA programs and 41% of AOA/ACGME-accredited programs reported providing education on OPP for MD faculty. Unfortunately, more respondents from AOA programs failed to respond to these questions.

The 17 most common topics suggested in training faculty supervisors are shown in *Table 2* - this table illustrates, in the order of importance, the topics that respondents thought would be adequate/necessary to improve an allopathic physician's ability to supervise/precept osteopathic residents/students. The top four, similar across program type, were somatic dysfunction diagnosis, osteopathic treatment plan theory, muscle energy and myofascial techniques. Participants were also asked to comment about possible forms of education delivery of this information. The common themes were: OMT clinic experience through mentoring by osteopathic faculty, regularly scheduled OMT didactic sessions with hands-on opportunities, and access to osteopathic literature through library access as well as journal club activities.

TABLE 1:

Program characteristics: Summary of educational environment characteristics of AOA and AOA/ACGME-accredited programs.
**p<0.01-indicates significance

CHARACTERISTIC	AOA ACCREDITED PROGRAMS (N=93)	AOA/ACGME ACCREDITED PROGRAMS (N=80)	P-VALUE
Mean No. of residents DO residents:	11.5 +9.7	9.0+5.7	N/A
MD residents:	0	13.5+8.9	
Total:	11.5 +9.7	22.4+9.8	
Mean No. core faculty DO faculty:	5.1 +5.2	2.3 + 1.3	NA
MD Faculty:	2.3 +1.5	2.7 + 0.8	
Total	7.4 +6.1	5.0 +1.7	
Mean No. of outside faculty preceptors	3.7 + 1.7	3.2 + 1.6	NA
% of programs that faculty perform OMT All DO faculty:	63% (53 out 84 respondents, missing=9)	83% (64 out of 77 respondents, missing=3)	0.005**
Any MD faculty:	9% (5 out of 56 respondents, missing=37)	28% (21 out of 74 respondents, missing=6)	0.006**
Education program on precepting OMT for MD faculty (%Yes)	37% (20 out of 54 respondents, missing=39)	47% (35 out of 74 respondents, missing=6)	0.247
Education program on OPP for MD faculty (%Yes):	26% (14 out of 54 respondents, missing=39)	41% (30 out of 74 respondents, missing=6)	p=0.094
If yes, mean number of hours:	49.8 + 110.6, range: 1-300	6.7 + 9.8, range: 1-45	

TABLE 2:

17 topics to improve allopathic physicians ability to supervise osteopathic residents/students (percent of responses). (N=173)

1. Somatic dysfunction diagnosis (69%)
2. Osteopathic treatment plan theory (61%)
3. Muscle energy (59%)
4. Myofascial release (55%)
5. Osteopathic Medicine History (47%)
6. Spinal motion (45%)
7. Anatomic landmarks (46%)
8. Lymphatic techniques (45%)
9. HVLA (36%)
10. Articulatory techniques (32%)
11. Facilitated positional release (31%)
12. Fryette's Principles (28%)
13. Still Techniques (24%)
14. Chapman's points (22%)
15. Visceral techniques (25%)
16. Facilitation (17%)
17. Craniosacral motion (12%)

DISCUSSION

In this study, we document concern of residency program directors and coordinators perception of their MD faculty's confidence and ability to precept residents in OMT. We found that a minority of programs provided faculty development in osteopathic skills although respondents provided many suggestions for topics and format for teaching this content to MD faculty. Interestingly, although AOA programs had more DO faculty on average when compared to AOA/ACGME-accredited programs, a lower number of DO faculty perform OMT when compared to DO faculty in AOA/ACGME-accredited programs. This difference is likely because the DO faculty in AOA/ACGME programs are generally responsible for the resident osteopathic education and instruction. Additionally, there were approximately the same number of MD faculty on average across program type, but those in AOA/ACGME-accredited programs were more likely to perform some OMT. These data suggest a need for additional support for both MD and DO residency faculty development in OPP and OMT.

The transition of graduate osteopathic training to the Single Accreditation System within ACGME will require better documentation of educational support of resident teaching in OPP and OMT. Use of the OSCE[10] and standards in trauma care[11] are good assessment tools of clinical and procedural competency, but there must be adequately trained faculty to supervise these assessments. Standardized faculty development in OPP and OMT would likely better equip allopathic faculty to support osteopathic residents in OPP and maintain OMT skills. Currently, there are no known studies on the effectiveness of training allopathic faculty on supervising OMT. Therefore, we need standard preceptor courses and subsequent studies to determine what is needed and how best to teach allopathic faculty to be adequate preceptors in a skill they may not perform themselves. Preceptor training courses to satisfy these needs could be modeled after courses based at osteopathic medical schools geared toward their local medical student preceptors, those done at the Society of Teachers of Family Medicine, or the one sponsored by the American Academy of Family Physicians.

Limitations of this study include indirect measurement of allopathic supervisor confidence in OMT precepting skills and only surveying AOA or AOA/ACGME accredited programs. We did not survey ACGME-only accredited programs. Future studies are needed as it is not known how ACGME-only programs view the need for osteopathic education and faculty development within their residencies. Future studies could be useful to address allopathic faculty directly regarding their concerns about precepting OMT as well as current exposure to OPP/OMT. Additional studies should also be directed toward osteopathic residents to assess their perceived needs to support their use of OPP/OMT.

CONCLUSION

During the transition to the ACGME Single Accreditation System, there is a need to continue to properly support graduate training in OPP and OMT. Currently, this study documented perceived concern by respondents regarding their allopathic faculty's ability to precept OMT. Also, education for allopathic faculty to enable more effective osteopathic resident supervision is currently limited and would likely better equip allopathic faculty to support osteopathic residents in continued development of OMT skills.

AUTHOR DISCLOSURES:
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REFERENCES:

1. Licciardone JC, Gatchel RJ, Aryal S. Recovery From Chronic Low Back Pain After Osteopathic Manipulative Treatment: A Randomized Controlled Trial. JAOA, March 2016: 116(3):144-55
2. Bigos S, Bowyer O, Braen G. Acute Low Back Problems in Adults. Rockville (MD): Agency for Health Care Policy and Research (AHCPR); 1994 Dec. (AHCPR Clinical Practice Guidelines, No. 14.) 3, Clinical Care Methods. Available from: https://www.ncbi.nlm.nih.gov/books/NBK52405/
3. Espi-Lopez GV, et.al. Do manual therapy techniques have a positive effect on quality of life in people with tension-type headache? A randomized controlled trial. European journal of physical & rehabilitation medicine. Aug 2016: 52(4):447-56
4. Hensel KL. Pregnancy Research on Osteopathic Manipulation Optimizing Treatment Effects: the PROMOTE study. American Journal of Obstetrics & Gynecology. Jan 2015: 212(1): 108.e1-9
5. Noll DR, et.al. Efficacy of osteopathic manipulation as an adjunctive treatment for hospitalized patients with pneumonia: a randomized controlled trial. Osteopathic Medicine and Primary Care. Mar 2010: 19;4:2
6. Baker HH, et.al. Osteopathic Medical Students Entering Family Medicine and Attitudes Regarding Osteopathic Manipulative Treatment: Preliminary Findings of Difference by Sex. Family Medicine. May 2017;49(5):374-377
7. Hempstead LK. Et.al. Resident and Faculty Attitudes Toward Osteopathic-Focused Education. JAOA, June 2017; 117(6):387-392
8. http://www.aacom.org/ome/councils/aacom-councils/ecop
9. Rubeor A, Nothnagle M, Taylor JS. Introducing osteopathic medical education in an allopathic residency. JAOA 2008; 108:404-408.
10. Dwyer T, et.al. How to set the bar in competency-based medical education standard setting after an Objective Structured Clinical Examination(OSCE). BMC Medical Education. Jan 2004; 16:1
11. Nousiainen MT, et.al. Resident education in orthopaedic trauma: the future role of competency-based medical education. Bone & Joint Journal, Oct 2016; 98-B(10): 1320-1324.

REVIEW ARTICLE

Current Hypogonadism Treatment Options

Steven H. Barag, DO, FACOFP[1]; Talin Meshefedjian, OMS[2]; Jay Yim, OMS[2]; Andrew Wilson, DO[3]

[1] Western University of Health Sciences - College of Osteopathic Family Medicine of the Pacific and Touro University College of Osteopathic Medicine, Pomona, CA

[2] Western University of Health Sciences - College of Osteopathic Medicine of the Pacific, Pomona, CA

[3] Dept. of Medicine, San Antonio Military Medical Center, Joint Base San Antonio-Fort Sam Houston, TX

KEYWORDS:

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Testosterone Treatment

Abstract: Treatment for hypogonadism is increasing, particularly in the senior population, but low testosterone levels are also on the rise in young men. Hypogonadism treatment represents a unique challenge to clinicians due to label warnings and the negative stigma of being diagnosed and treated for testosterone deficiency. Recent studies of testosterone formulations are showing promising data results of a reduction in adverse impacts. Recent studies have also shown that treating low testosterone is important to significantly reducing the associated risk of diabetes, obesity, metabolic syndrome, osteoporosis, and cardiovascular problems. Physicians need to consider these updated studies and testosterone formula options to educate patients and provide them with a safe and effective testosterone therapy.

INTRODUCTION

Male hypogonadism is defined as a clinical syndrome that results from failure of the testes to produce physiological levels of testosterone due to the disruption of one or more levels of the hypothalamic-pituitary-testicular axis.[1] Male hypogonadism affects between four and five million men in the United States.[2] It is associated with older males, but there is a growing prevalence of low testosterone levels in younger men. Testosterone levels tend to peak in adolescence and early adulthood, with levels declining by about 1 percent each year starting at age 30.[3] With testosterone concentrations declining as men age[4] and the increasing marketing campaigns raising awareness of hypogonadism symptoms, prescriptions for testosterone therapy have risen significantly increasing in recent years.[5]

In adult males, hypogonadism can impact them physically, mentally, and emotionally. Physical symptoms include erectile dysfunction, decreased bone and muscle mass, a decrease in beard and hair growth, and the development of breast tissue. Additionally, patients may become fatigued more easily, have reduced libido, and sometimes impaired focus and cognition. Patients exhibiting any one of these symptoms, combined with a total serum testosterone levels <300 ng/dL are diagnosed according to the endocrine guidelines as hypogonadal.[6]

If a repeat assay confirms low testosterone, luteinizing hormone (LH) should be measured to determine whether the cause is primary or secondary. The secondary cause is indicated by LH levels of <2 ng/mL while LH levels of >10 ng/mL indicates primary testicular failure. If the levels fall within a normal range, this suggests an age-related, decreased hypothalamic response to declining testosterone levels.[1]

This article will describe the risks and benefits of testosterone therapy along with identifying the varieties of testosterone replacement formulations currently available with the advantages and disadvantages of each identified.

RISKS AND BENEFITS OF TESTOSTERONE THERAPY

Testosterone replacement therapy provides many potential benefits to men with symptomatic hypogonadism. Studies have documented that treating low testosterone is important to significantly reduce the associated risk of diabetes, obesity, metabolic syndrome, osteoporosis, and cardiovascular problems.[7] These have been linked to the increase in fat mass and a decrease in muscle mass with declining testosterone. Testosterone replacement therapy has also been proven to impact a man's quality of life, by improving his mood and energy level.[8]

CORRESPONDENCE:
Steven H. Barag, DO, FACOFP | drbarag@gmail.com

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TABLE 1:

Potential improvements with testosterone replacement therapy.[9]

Libido
Erectile Dysfunction
Muscle strength and body composition
Mood
Cognition

The potential risks associated with testosterone replacement therapy also need to be considered in relation to the potential benefits. *See Table 2 for a list of these potential risks.*

TABLE 2:

Potential risks with testosterone replacement therapy.[9]

Erythrocytosis
Increases in prostate-specific antigen
Worsening of prostate disorders
Dermatologic
Worsening of existing obstructive sleep apnea
Suppression of Luteinizing Hormone
Decreased intra-testicular testosterone concentrations
Reduced spermatogenesis

In recent years, the U.S. Food and Drug Administration (FDA) has added warnings to testosterone products.

In 2009, the FDA required a box warning for transdermal gel products because of the risk of transference to women and children. In 2014, a warning on the increased events of venous thromboembolism was added to all testosterone products.[9] The following year, it was mandated that all testosterone manufacturers should include the increased risk of heart attack and stroke on the label[9] due to two concerning reports of increased CV risk with TTh. After publication, both of these studies received a significant amount of scrutiny. In the case of Vigen et al., it was originally reported that men who received TTh had an absolute rate of MI, stroke, or death of 25.7% compared with 19.9% in untreated men.[11] Soon after publication, it was discovered the authors had reversed the actual results, and the absolute rate of events was only 10.1% for men that received TTh and 21.2% in untreated men.[13] Additionally, the authors revealed they had miscategorized nearly 1,000 subjects (almost 10% of the patient population) of an all-male cohort to be women.

Considering that past studies suggested that low serum T level is associated with increased cardiovascular events, along with the growing warning label copy, it's understandable why patients may be hesitant to consider testosterone replacement treatment. However, recent studies of current testosterone formulations have shown promising data minimizing adverse impacts. One example is the retrospective cohort study of male veterans who received their medical care at the Veterans Health Administration (VHA) between December 1999 and May 2014. Their results concurred with a previous VA study. Shores et al.[14] analyzed data from seven VA medical centers. They found that TRT was associated with a significant decrease in all-cause mortality (HR: 0.61, CI 95%, P < 0.0001). While supporting the results of Shores et al., this study adds significantly to its conclusions both due to much larger sample size and also by more accurately identifying those who received and responded to the TRT.

The FDA added additional warnings to the testosterone package labeling in 2016 warning of the potential abuse of testosterone and other anabolic androgenic steroids. The potential adverse effects outlined by the FDA included stroke, depression, and aggression.

Additional evidence-based studies are needed so clinicians can better understand the potential risks of testosterone replacement therapy and resolve the significant uncertainty regarding the effect of testosterone replacement therapy on cardiovascular outcomes.

TESTOSTERONE REPLACEMENT THERAPY FORMULATIONS

Table 3 documents the variety of testosterone replacement formulations currently approved for use in the United States. Physicians need to consider multiple factors in choosing which formulation is best suited for their patient including ease of use and cost factors.

INJECTABLE FORMULATIONS

Intramuscular

Intramuscular injections have been used for years due to its efficacy and cost-effectiveness.

For intermediate acting therapies, there are two options. First, testosterone cypionate (Depo® testosterone) is dosed 100-200 mg every two weeks or 50-100 mg every week in the thigh or buttock.[13] Advantages include ease of home injections, infrequent treatment; a three-fold increase in testosterone within two days, affordability, and high efficacy. Disadvantages are fluctuating testosterone levels as testosterone levels gradually decrease until the next injection, and pain and irritation at the injection site.[12] Approximate cost is between $18.45 -$98.07. The second option is testosterone enanthate (Delatestryl®) with a 200 mg/mL dose every two weeks with an average cost of $23.46. Advantages include effective and affordable but in short supply.[13]

For long-acting treatment, the testosterone preparation available is testosterone undecanoate (Aveed®). Usual dosing is 750 mg initially, then 750 mg at four weeks, then 750 mg every ten weeks ongoing with the site of application is the buttock. The advantage is it's long-acting while disadvantages are that it needs to be administered in an office/hospital by a REMS-certified provider and there is a risk of pulmonary oil micro embolism and anaphylaxis. The cost is approximately $1,050 plus any fees associated with the cost of injection.[12]

INJECTABLE FORMULATIONS

INTRAMUSCULAR

INTERMEDIATE ACTING THERAPIES

TESTOSTERONE CYPIONATE (DEPO® TESTOSTERONE)[12,16]

- Dose: 100-200 mg every two weeks or 50-100 mg, weekly in thigh or buttock.
- Advantages: ease of home injections, infrequent treatment; three-fold increase in testosterone within two days, affordability, high efficacy.
- Disadvantages: fluctuating testosterone levels as testosterone levels gradually decrease until the next injection, pain and irritation at injection site.
- Average Cost: Between $18.45 -$98.07.

TESTOSTERONE ENANTHATE (DELATESTRYL®)[13, 17, 18]

- Dose: 200 mg/mL dose every two weeks
- Advantages: effective and affordable
- Disadvantages: short supply
- Average Cost: $23.46

FOR LONG-ACTING TREATMENT

TESTOSTERONE UNDECANOATE (AVEED®)[12]

- Dosing: 750 mg initially, then 750 mg at four weeks, then 750 mg every ten weeks ongoing, site of application the buttock.
- Advantages: long-acting
- Disadvantages: needs to be administered in an office/hospital by a REMS-certified provider, risk of pulmonary oil micro embolism and anaphylaxis.
- Average Cost: approximately $1,050 plus any fees associated with the cost of injection.

TRANSDERMAL FORMULATIONS

GELS

AndroGel® and Testim®[19]

- Dose: Available in 1% gels applied 50-100 mg daily on dry intact skin on the back, abdomen, upper thighs, and arms. AndroGel® is also available 1.62% gel in a metered dose pump.
- Advantages: steady serum testosterone concentration
- Disadvantages: risk of transfer, the need for a daily application, occasional skin irritation, inability of some to achieve normal T levels.
- Average Cost: $175 to 400 for generic and $480 to 550 for a brand name.

2% SOLUTIONS

Fortesta®[19]

- Dose: metered dosed pump, between 10-70 mg daily on dry, intact skin on the front and inner thighs.
- Advantages: Ease of application
- Disadvantages: possibility of redness/ irritation at the application site, acne
- Average Cost: from $160-400

AXIRON®[19]

- Dose: 30-120 mg per day on the dry, intact skin of the axilla.
- Advantages: ease of application, reduced risk of transfer
- Disadvantages: possibility of redness/ irritation, acne
- Average Cost: $260-1,200 per month.

PATCH

ANDRODERM® PATCH[19]

- Dose: between 2-6 mg daily, applied onto dry, intact skin of the arm or torso.
- Advantages: limited risk of transfer, no injection necessary.
- Disadvantages: About one-third of men report skin irritation; patch applied daily.
- Average cost: monthly $475-510.

ALTERNATIVE

NASAL Natesto®[20]	• Dose: is delivered with two pumps, one in each nostril, three times daily. • Advantages: minimal risk of secondary transference • Disadvantages: rhinorrhea, epistaxis, sinusitis, and nasal scabs • Average cost: $600-700 per month.
IMPLANTED SUBCUTANEOUS PELLET Tetopel®[22]	• Dose: 150-450 mg every 3 to 6 months into the subcutaneous fat of the buttock, lower abdominal wall, or thigh. • Advantages: no risk of transfer or need for daily treatment. • Disadvantages: extrusion infection, fibrosis at pellet sites. • Average cost: $150-175 plus the cost of pellet placement based on a dose of 150mg every three months.
BUCCAL Striant SR®[19, 23]	• Dose: 30 mg twice daily. Adheres to the depression in the gingiva superior to upper incisors, • Advantages: less invasive, levels normalize in 24 hours , • Disadvantages: frequent administration, gingival irratation • Average cost: The cost is $550-600 per month.
ORAL (TESTOSTERONE UNDECANOATE) Andriol®[24]	• Dose: 40-80mg, three times a day • Advantages: injections are not necessary, frequent dosing is required • Disadvantages: relatively low dose of testosterone delivered • Average cost: $200-300 per month

TRANSDERMAL FORMULATIONS

Gels

AndroGel® and Testim® are available in 1% gels applied 50-100 mg daily on dry, intact skin on the back, abdomen, upper thighs, and arms. The benefit of a steady serum testosterone concentration is balanced out by the risk of transfer, the need for a daily application, occasional reported skin irritation and the inability of some to achieve normal T levels.[21] The cost is $175 to 400 for generic and $480 to 550 for a brand name. AndroGel® is also available in a 1.62% gel in a metered dose pump.

A 2% solution, Fortesta® in a metered dosed pump, is dosed between 10-70 mg daily on dry, intact skin on the front and inner thighs.[22] Ease of application is the primary benefit countered by the possibility of redness/irritation at the application site, plus acne may occur. Costs range from $160-400.[22] Another 2% metered dosed pump application available is Axiron®, dosed 30-120 mg per day on the dry, intact skin of the axilla.[22] Primary advantages include ease of application and reduced risk of transfer with the possibility of redness/irritation and acne are potential side effects.[22] The range of cost is $260-1,200 per month.[19]

Patch

The first testosterone patch was developed for placement on the scrotal skin to maximize hormone absorptions. Due to skin irritation and adherence problems, the nonscrotal transdermal patch was invented with permeation enhancers to overcome the limited ability of nonscrotal skin to absorb testosterone.

The Androderm® patch is dosed between 2-6 mg daily and applied onto dry, intact skin of the arm or torso.[23] There is a limited risk of transfer and no injection necessary. About one-third of men do report skin irritation and the patch must be applied daily.[23] Average monthly cost is $475-510. Androderm® is currently the only transdermal testosterone patch available in the United States.[19]

ALTERNATIVES

Nasal

Natesto® is delivered with two pumps, one in each nostril, three times daily. There is a minimal risk of secondary transference with rhinorrhea, epistaxis, sinusitis, and nasal scabs as potential side effects (<4% for each respectively).[22] Cost ranges from $600-700 per month. According to a recent 90-day, randomized study in hypogonadal men, those treated with nasal gel showed statistically significant improvements in each of the five domains of erectile function and mood, both of which showed the most benefit by day 30, with smaller increases until day 90. An additional finding from this study showed that treatment with nasal gel successfully restored normal serum testosterone levels while maintaining mean luteinizing hormone (LH) and follicle stimulating hormone (FSH) concentrations within the normal reference range, which is important for retaining the natural pathways of hormones. This has the potential of preserving testicular volume and fertility, which are major issues caused by suppressed LH and FSH.[25]

Implanted Subcutaneous Pellet

Tetopel® is dosed in pellet form, 150-450 mg every 3 to 6 months. The pellets are implanted at a clinic or hospital by a trained provided under sterile conditions into the subcutaneous fat of the buttock, lower abdominal wall, or thigh.[22] There is no risk of transfer or need for daily treatment.[22] Risks include extrusion infection and fibrosis at pellet sites. The cost is $150-175 plus the cost of pellet placement based on a dose of 150mg every three months.[22] Moskovic et al. investigated a small series of young men with Klinefelter's who would be requiring lifetime T replacement and compliance with the Testopel® formulation was better than gels or injections.

Buccal

Striant SR® adheres to the depression in the gingiva superior to upper incisors, 30 mg twice daily. The advantage is that it is less invasive, levels normalize in 24 hours, but it requires frequent administration and there is gingival irritation reported. The cost is $550-600 per month.[19]

Oral (Testosterone undecanoate)

Andriol® is taken orally with a fat-containing meal, 40-80mg, three times a day and is available in many countries outside of the United States.[22] While injections are not necessary, frequent dosing is required and there is a relatively low dose of testosterone delivered.[22] The cost is $200-300 per month.[22] New self-emulsifying formulations are under development and expected to be available in the next 1-2 years.

MONITORING AND FOLLOW-UP

For all formulations of testosterone therapy outlined in this article, it is imperative for clinicians to have regular follow up visits with their patients to monitor serum T levels after ~3 months of initiating therapy. Additional labs should be ordered every six months to monitor PSA, hematocrit, gonadotropins - luteinizing hormone & follicle-stimulating hormone, and secondary testosterone metabolites; estradiol (E2) and dihydrotestosterone (DHT) are all key components to monitor the effects of the TTh and its impact on the hypogonadal-pituitary-gonadal (HPG) axis.[29] Practice guidelines for monitoring patients after initiation of TRT are available through the Endocrine Society at *https://www.endocrine.org/education-andpractice-management/clinical-practice-guidelines*.

CONCLUSION

The risks and benefits of testosterone replacement therapy need to be the focus of in-depth discussion with male hypogonadism patients when deciding on a treatment plan. Recent studies of new testosterone formulations and delivery methods have shown restoration of normal testosterone levels while maintaining normal LH, FSH, and hematocrit levels.[14,17] Most forms of testosterone are notorious for transference to spouses, children, and pets.[22] The new formulations, with the significantly reduced risk of secondary transference, afford much more consideration by clinicians and patients as an appropriate therapeutic option.

Lipshultz looked at testosterone replacement therapy patient satisfaction rates at Baylor via a survey based on patient recall and documented that the choice of therapy was heavily influenced by physician recommendation with 53, 31, and 17 % choosing injections, gels, and Testopel®, respectively.

New methods of delivery and dosing have proposed benefits by exploring bioavailability that mimics the body's physiological process of testosterone production. As physicians, it is important to educate patients on testosterone therapy options by informing them of the ease and safety of use based on evidence-based studies.

The continued growth of this two billion dollar testosterone market has led to the recent increase in direct to patient advertising and marketing. These stand-alone, for profit, low T clinics and men's health spas are exactly why proper counseling and education is critical for the patient to understand the best options, clinically.

It is important for family physicians to continue to explore, study, and consider all intramuscular, transdermal and novel treatment options to provide patients with evidence-based, safe and most effective testosterone therapy recommendation for their patients.

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REFERENCES:

1. Decaroli M, Rochira V. Aging and sex hormones in males. Virulence. 2016;8(5):545-57. doi:10.1080/21505594.2016.1259053.
2. Bhasin S, Cunningham GR, Hayes FJ, et al.: Testosterone therapy in men with androgen deficiency syndromes: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2010;95(6):2536–59. 10.1210/jc.2009-2354.
3. Finkelstein JS, Lee H, Burnett-Bowie SA, et al. Gonadal steroids and body composition, strength, and sexual function in men. N Engl J Med 2013; 369:1011.
4. Harman SM, Metter EJ, Tobin JD, et al. : Longitudinal effects of aging on serum total and free testosterone levels in healthy men. Baltimore Longitudinal Study of Aging. J Clin Endocrinol Metab.2001;86(2):724–31. 10.1210/jcem.86.2.7219.
5. Tan RS, Salazar JA: Risks of testosterone replacement therapy in aging men. Expert Opin Drug Saf.2004;3(6):599–606. 10.1517/14740338.3.6.599.
6. Bhasin S et al., Testosterone Therapy in Men with Androgen Deficiency Syndromes: An Endocrine Society Clinical Practice Guideline. Jour. Of Clinical Endo. & Metabolism. 2010.
7. Behre HM, Kliesch S, Leifke E, et al. Long-term effect of testosterone therapy on bone mineral density in hypogonadal men. J Clin Endocrinol Metab 1997; 82:2386.
8. Haider K, Haider A, Doros G, Traish A. Long-Term Testosterone Therapy Improves Urinary and Sexual Function, and Quality of Life in Men with hypogonadism: Results from a Propensity Matched Subgroup of a Controlled Registry Study. The Journal of Urology. 2017. doi:10.1016/j.juro.2017.07.039.
9. Bassil N, Alkaade S, Morley JE. The benefits and risks of testosterone replacement therapy: a review. Ther Clin Risk Manag 2009;5:427-48.
10. Decaroli M, Rochira V. Aging and sex hormones in males. Virulence. 2016;8(5):545-570. doi:10.1080/21505594.2016.1259053.
11. Vigen R, O'Donnell CI, Baron AE, et al. Association of testosterone therapy with mortality, myocardial infarction, and stroke in men with low testosterone levels. JAMA. 2013; 310 (17):1829-1836.
12. Finkle WD, Greenland S, Ridgeway GK, et al. Increased risk of non-fatal myocardial infarction following testosterone therapy prescription in men. Plos One. 2014; 29;9(1)76.
13. Morgentaler A, Lunenfeld B. Testosterone and cardiovascular risk: world's experts take unprecedented action to correct misinformation. Aging Male. 2014;17:63-65.
14. Testosterone and Cardiovascular Risk in Men: A Systematic Review and Meta-analysis of Randomized Placebo-Controlled Trial Haddad, Rudy M. et al.Mayo Clinic Proceedings , Volume 82 , Issue 1 , 29 – 3.
15. FDA. Topic Testosterone gel products: secondary exposure of children to topical testosterone products. PostMarket Reviews – Volume 2, November 3, 2009. www.fda.gov/Drugs/DrugSafety/DrugSafetyNewsletter/ucm189806.htm. Accessed March 28, 2015.
16. The Journal of Clinical Endocrinology & Metabolism, Volume 97, Issue 6, 1 June 2012, Pages 2050–2058, https://doi.org/10.1210/jc.2011-2591.
17. European Heart Journal, Volume 36, Issue 40, 21 October 2015, Pages 2706–2715, https://doi.org/10.1093/eurheartj/ehv346.
18. Food and Drug Administration. Drugs@FDA: FDA approved drug products. 2018; Retrieved from https://www.accessdata.fda.gov/scripts/cder/drugsatfda/.
19. Snyder PJ. Testosterone treatment of male hypogonadism, In A.M. Matsumoto and KA Martins (Eds.) UpToDate. 2018: Retrieved from www.uptodate.com.
20. American Society of Health-System Pharmacists. Testosterone enanthate injection. 2014; Retrieved from http://www.ashp.org/menu/DrugShortages/CurrentShortages/Bulletin.aspx?id=1045.
21. Ullah MI, Riche DM, Koch CA. Transdermal testosterone replacement therapy in men. Drug Design, Development and Therapy. 2014;8:101-112. doi:10.2147/DDDT.S43475.
22. Thirumalai, A., Berkseth, K., Amory, John, Treatment of Hypogonadism: Current and Future Therapies. F1000 Research. Doi:10.12688/f1000research.10102.1.
23. Pastore MN, Kalia YN, Horstmann M, Roberts MS. Transdermal patches: history, development and pharmacology. Alexander S, ed. British Journal of Pharmacology. 2015;172(9):2179-2209. doi:10.1111/bph.13059.

24. Rogol A, Tkachenko N, Bryson N. Natesto™, a novel testosterone nasal gel, normalizes androgen levels in hypogonadal men. Andrology. 2015;4(1): 46-54. doi:10.1111/andr.12137.
25. Connors, MD William et al. "Mp89-06 Preservation of Normal Concentrations of Pituitary Gonadotropins Despite Achievement of Normal Serum Testosterone Levels in Hypogonadal Men Treated with a 4.5% Nasal Testosterone Gel." The Journal of Urology, vol. 197, no. 4, 2017.
26. Moskovic DJ, Freundlich RE, Yazdani P, Lipshultz LI, Khera M. Subcutaneous implantable testosterone pellets overcome noncompliance in adolescents with Klinefelter syndrome. J Androl. 2012;33(4):570–3. doi: 10.2164/jandrol.111.013979
27. Ross RJM, Jabbar A, Jones TH, et al. Pharmacokinetics and tolerability of a bioadhesive buccal testosterone tablet in hypogonadal men. Eur J Endocrinol. 2004; 150(1) 57-63.
28. Hassan J, Barkin J. Testosterone deficiency syndrome: Benefits, risks, and realities associated with testosterone replacement therapy. Can J Urol. 2016; 23(Suppl 1), 20-30.
29. Rivas AM, Mulkey Z, Lado-Abeal J, Yarbrough S. Diagnosing and managing low serum testosterone. Proceedings (Baylor University Medical Center). 2014;27(4):321-324.
30. Pastuszak AW, Mittakanti H, Liu JS, Gomez L, Lipshultz LI, Khera M. Pharmacokinetic evaluation and dosing of subcutaneous testosterone pellets. J Androl. 2012;33(5):927–37. doi: 10.2164/jandrol.111.016295.
31. Rogol A, Tkachenko N, Bryson N. Natesto™, a novel testosterone nasal gel, normalizes androgen levels in hypogonadal men. Andrology. 2015;4(1): 46-54. doi:10.1111/andr.12137.

REVIEW ARTICLE

Symptomatic Approach to Gas, Belching and Bloating with OMT Treatment Options

Carly Gennaro, DO[1]; Helaine Larsen, DO[1]

[1]Good Samaritan Hospital Medical Center, West Islip, NY

KEYWORDS:

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Prevention and Wellness

ABSTRACT: Intestinal gas production is a normal physiologic progress. However, there are many pathophysiologic processes that can cause patients to experience bloating, abdominal pain, and distension from abnormal gas production or mobility. It is important for primary care physicians to understand the causes and mechanisms for both physiologic and pathologic gas and bloating in order to appropriately and effectively treat our patient population. This article will review the differential diagnosis of gas, bloating and belching, the necessary work-up, and the management of these disorders.

INTRODUCTION

Gas, bloating, and belching are common gastrointestinal (GI) symptoms reported in the primary care office. As many as 30% of the U.S. population experiences bloating symptoms, and most of these patients describe their symptoms as moderate to severe.[1] Common causes of these symptoms include aerophagia, gastroesophageal reflux disease (GERD), irritable bowel syndrome (IBS), small bacterial intestinal overgrowth (SIBO), and malabsorption. These disorders can lead to significant discomfort and pain. Once diagnosed, there are treatment options including dietary changes and medications that can provide relief and improve the patient's quality of life.

GAS PRODUCTION

Ninety-nine percent of gas in the intestinal tract consists of nitrogen (N2), oxygen (O2), carbon dioxide (CO2), hydrogen (H2) and methane. Swallowing is the primary cause of air in the stomach. Every time a person swallows he or she also ingest several milliliters of gas, comprised mostly of nitrogen and oxygen. Most of this gas is belched and usually does not make it to the duodenum.[1]

CORRESPONDENCE:
Carly Gennaro, DO | cgenna01@nyit.edu

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The primary cause of gas production in the colon is fermentation by colonic bacteria. Most people have about 100-200 milliliters of gas in our GI tract at any given time. Approximately 500 different species of bacteria reside within the colon, and nearly all of these species are anaerobes. Species of colonic bacteria differ between each individual depending on diet, antibiotic use, and how the patient was fed as an infant. The volume of gas increases after eating. Some food products that are incompletely digested within the small intestine such as lactose, fructose, sorbitol, legumes, fiber, and complex carbohydrates are broken down in the colon by colonic bacteria.[1]

TABLE 1:

Common causes of gas, bloating and belching.

– Aerophagia	– Lactose Intolerance
– Irritable Bowel Syndrome (IBS)	– Fructose Intolerance
– Small Intestinal Bacterial Overgrowth (SIBO)	– Celiac Disease

BELCHING

Every time a person swallows air is ingested. This air then travels down the esophagus through peristalsis and accumulates in the proximal stomach. When beverages with CO2 and bicarbonate are all ingested, larger volumes of gas accumulate. As the stomach becomes dilated with gas, stretch receptors are activated which triggers a vasovagal reflex. This reflex causes the lower esophageal sphincter and crural diaphragm to relax allowing intragastric air

to escape. This mechanism prevents the stomach from becoming damaged by excessive dilation.[2]

Many patients with GERD report increased belching. Transient lower esophageal sphincter (LES) relaxation is the major mechanism for both belching and GERD. Recent studies have shown that the number of belches is related to the number of times someone swallows air. These studies have concluded that patients with GERD swallow more air in response to heartburn and therefore belch more frequently.[3] There is no specific treatment for belching in GERD patients, so for now, physicians continue to treat GERD with proton pump inhibitors (PPIs) and histamine-2 receptor antagonists with the goal of suppressing heartburn and chest pain symptoms. Some patients who undergo fundoplication as a treatment for reflux will lose the ability to belch leading to bloating and dilation of the stomach and intestines.[2]

AEROPHAGIA

Aerophagia is the condition of excessive air swallowing and belching. Patients with this disorder can belch up to 20 times per minute. Stress can increase the frequency of belching. Aerophagia causes supragastric belching. There are two ways supragastric belching can occur. First, a patient can create negative intrathoracic pressure through inspiration against a closed glottis, allowing air to enter the esophageal body. Second, patients can bring air into the esophagus using their pharynx, palate and tongue. Supragastric belching or aerophagia usually does not occur with meals and does not have a scent or taste. It is considered a behavioral disorder exacerbated by anxiety. Treatment is usually behavior therapy or speech therapy to try to unlearn the belching behavior.[2]

FLATULENCE

Flatulence is flatus passed through the anus. For most people flatulence is normal and does not cause pain or discomfort. However, many people experience excessive bloating and pain. The normal amount of flatus passed each day is usually between 500 and 1500 mL.[4] In fact, most patients who complain of excessive flatus will still fall into this range. Physiologic gas can be caused by intake of lactose, fructose, sorbitol; indigestible starches in fruits, vegetables, and legumes; and carbonated beverages. Simethicone (Mylicon® and Gas-X®) is a common medication used for abdominal bloating but has not been shown to relieve excessive flatulence.[5] Simethicone works by changing the surface tension of gas bubbles allowing for easier breakdown. Beano®, a dietary supplement that contains the enzyme, alpha-galactosidase, is a commonly used over-the-counter medication for excess flatulence. The polysaccharides and oligosaccharides found in foods such as legumes, broccoli and brussels sprouts are metabolized and fermented by large intestinal flora to produce gases. The enzyme in Beano® breaks these complex sugars into simple sugars making them easier to digest with less gas production.[6]

IRRITABLE BOWEL SYNDROME (IBS)

IBS is abdominal pain or discomfort associated with altered bowel habits. It is the most commonly diagnosed GI disorder and accounts for about 30% of all GI referrals.[7] Criteria for IBS is recurrent abdominal pain at least one day per week in the last three months associated with at least two of the following: **1)** association with defecation, **2)** change in stool frequency, **3)** change in stool form. Diagnosis should be made using these clinical criteria and limited testing. Common symptoms are abdominal pain, bloating, alternating diarrhea and constipation, and pain relief after defecation. Pain can be present anywhere in the abdomen, but the lower abdomen is the most common location.[8] Abdominal bloating is a common complaint for the majority of these patients. Abdominal distension may also occur. The difference between bloating and distension is that bloating in the sensation of gassiness and fullness while distension is an actual increase in abdominal girth.[1] Studies have however shown that although patients with IBS feel gassy, they have a normal volume of gas in their intestinal tract compared to healthy individuals.[9] It is now believed that the cause of bloating and distension is due to impaired gas transit causing gas retention.[10]

There are three main types of IBS: IBS with predominant diarrhea, IBS with predominant constipation and IBS with mixed bowel habits. Patients should be encouraged to use the Bristol stool form scale *(Table 2)* to record stool consistency. When using the scale patients should not be on any medications to treat bowel habits.[8] Patients with constipation-variant IBS experience more abdominal distension due to prolonged transit time than those with diarrhea-variant IBS.[11]

TABLE 2:

Bristol stool form scale[8]

Type 1	Separate hard lumps, like nuts (hard to pass)
Type 2	Sausage-shaped but lumpy
Type 3	Like a sausage but with cracks on the surface
Type 4	Like a sausage or snake, smooth and soft
Type 5	Soft blobs with clear-cut edges
Type 6	Fluffy pieces with ragged edges, a mushy stool
Type 7	Watery, no solid pieces, entirely liquid

Gas related symptoms are commonly associated with food intolerance after eating poorly absorbable fermentable carbohydrate and polyols (FODMAPs). IBS patients may have a heightened sensitivity to poorly absorbable carbohydrates. These carbohydrates will be rapidly fermented by colonic bacteria leading to gas production, abdominal pain and flatulence.[12] It is important to obtain a full history of the patient's diet to try to determine which foods are exacerbating the patient's symptoms.

Patients with IBS may benefit from a diet low in FODMAPs *(Table 3)* and low in gas producing foods. Common gas producing foods include beans, onions, celery, carrots, raisins, bananas, apricots, prunes, brussels sprouts, wheat germ, pretzels, bagels, alcohol, and caffeine.[14]

TABLE 3:

FODMAPs[13]

FERMENTABLE	
Oligosaccharides	Wheat, barley, rye, onion, leek, garlic, shallots, artichokes, beetroot, fennel, peas, chicory, pistachio, cashews, broccoli, brussels sprouts
Disaccharides	Milk, custard, ice cream, and yogurt
Monosaccharides	Apples, pears, mangoes, cherries, watermelon, asparagus, sugar snap peas, honey, high-fructose corn syrup
Polyols	Apples, pears, apricots, cherries, nectarines, peaches, plums, watermelon, mushrooms, cauliflower, artificial sweeteners

Fiber supplementation is a common treatment for patients who experience constipation. However, some patients will experience increased bloating with fiber supplementation. It is recommended to start a low dose of psyllium fiber (soluble fiber) of one-half to one tablespoon per day in patients with IBS with constipation to avoid worsening of IBS symptoms. Insoluble fibers (such as bran) are more likely to cause increase bloating and flatulence.[8]

In patients who fail fiber therapy, polyethylene glycol (Miralax®) is recommended. Miralax® works as an osmotic laxative, improving constipation symptoms by causing more spontaneous bowel movements and lessening straining, but does not improve symptoms of bloating and abdominal pain.[15] Some patients will experience worsening cramping and bloating when using Miralax®. However, Miralax® is still preferred over lactulose and milk of magnesia for the use in chronic constipation and IBS with constipation as it has similar, if not greater efficacy, and has less side effects.

Pharmacologic stimulation of gut motility in IBS patients reduces gas retention and abdominal distension.[16] Commonly used prokinetics for IBS are linaclotide (Linzess®) and lubiprostone (Amitiza®). Linaclotide is a guanylate cyclase C agonist that works by increasing intestinal fluid secretion and motility. It is dosed once daily. The most common side effect is diarrhea. Lubiprostone activates type 2 chloride channels increasing intestinal fluid secretion and motility. It is dosed twice daily. Most common side effects are nausea and diarrhea.[8]

Antispasmodic agents can also be used on an as-needed basis. Hyoscyamine (Levsin®) and dicyclomine (Bentyl®) are commonly used anticholinergic agents. These medications may help patients with postprandial abdominal pain and bloating. Common side effects of these medications are dry mouth, dizziness, and blurry vision. Peppermint oil, which also has antispasmodic properties, has been shown to improve symptoms of bloating and pain.[17]

Psychosocial factors may also contribute to the development and exacerbation of IBS. Patients with IBS report more stressful events that non-IBS patients. Anxiety, sleep disturbance and somatic symptoms are independent risk factors for the development of IBS. Antidepressants, most commonly tricyclic antidepressants (TCAs), may be used to treat IBS. Independent of their psychiatric benefits, TCAs also decrease transit time, the time it takes ingested food to pass through the GI tract.18 Therefore, caution should be used when using TCAs in patients with predominant constipation. Selective serotonin reuptake inhibitors and selective serotonin-norepinephrine reuptake inhibitors have not yet been proven to improve IBS symptoms. There are many other medications used for the treatment of IBS, but this review article focuses on the treatment of bloating symptoms.

SMALL INTESTINAL BACTERIAL OVERGROWTH (SIBO)

There is a diverse population of microflora in the intestinal tract. A disruption in this microbiome can cause overgrowth of bacteria. The human body defends itself against overgrowth with gastric acid secretion, intestinal mobility, the ileocecal valve, immunoglobulins, and bacteriostatic pancreatic and biliary secretions. SIBO syndrome is usually due to disorders of these protective mechanisms including achlorhydria (due to chronic atrophic gastritis or long-term PPI use) and pancreatic exocrine insufficiency. Small intestine obstruction secondary to adhesions, tumors, and strictures can cause slow bowel transit with stasis of feces leading to dysmicrobia. Crohn's disease patients may have loss of ileocecal valve secondary to prior resection. In short bowel syndrome, patients may also have loss of the ileocecal valve, allowing large intestinal bacteria to colonize into the small bowel. The connective tissue disease, scleroderma affects the GI tract in most patients, leading to pseudo-obstruction. Patients with diabetes mellitus may experience delayed gastric emptying due to gastroparesis causing SIBO.[19]

Common symptoms of mild SIBO include bloating, flatulence, abdominal pain, and diarrhea. A patient with the more severe disease might have malabsorption.[19-20] It is very important to consider SIBO as a diagnosis in all patients with motility disorders and abnormalities of the small bowel. Microbial testing of jejunal aspirates is the gold standard for diagnosis.[19-20] The most common bacteria found in SIBO are Streptococci, Bacteroides, Escherichia, and Lactobacillus.[19] Clinically significant SIBO is diagnosed when bacterial counts exceed 10,000 organisms/mL on jejunal aspirate throughout endoscopy.[19] Because this test is invasive, expensive, and the bacterial overgrowth could be missed during aspiration, this test is not commonly used.[20] The most commonly used testing is by a hydrogen and methane breath tests.[20] Carbohydrate fermentation by colonic bacteria is the only source of hydrogen and methane gas production in the human body. During this test, lactulose, glucose or xylose are administered to the patient. The patient is then asked to exhale into a tube and hydrogen or methane is measured. In patients with SIBO, there will be an early peak in breath hydrogen or methane levels because the carbohydrate administered will be metabolized in the small bowel by colonic bacteria producing these gases.

Treatment of SIBO should start with treating the underlying disease. Antibiotics should cover the bacteria that are associated with causing SIBO. Rifaximin is a commonly used antibiotic as it has low GI absorption and covers gram positive, gram negative, aerobic and anaerobic bacteria.[19] Other options for treatment are ciprofloxacin, amoxicillin-clavulanate, and metronidazole plus sulfamethoxazole/trimethoprim.[20]

LACTOSE INTOLERANCE

Lactose intolerance occurs after ingestion of lactose in patients with lactase deficiency. When lactase is unavailable, lactose is not able to be digested and instead is fermented by bacteria in the colon. Common symptoms include abdominal pain, gas, bloating and diarrhea. Lactose intolerance is diagnosed by hydrogen breath testing and lactose ingestion.[21] The hydrogen breath test is more sensitive and specific than the lactose tolerance test and is widely available.

Acquired primary lactase deficiency is the most common cause of primary lactose malabsorption. At preschool age, most of the world's population begins to develop low intestinal levels of lactase. This is more common in Africans and Asians and less common in Caucasians. However, people who live in areas where cattle domestication has been a practice tend to better maintain their lactase levels.[22] Secondary lactose malabsorption is often caused by SIBO with increased fermentation of lactose in the small bowel leading to lactose intolerance symptoms. Many patients who self- diagnose themselves with lactose intolerance do not have impaired lactose digestion and many patients with impaired lactose digestion do not experience symptoms. Most patients with lactose intolerance can tolerate twelve grams of lactose (one cup of milk) in a single dose.[23]

Differentiating IBS from lactose intolerance is often difficult. These patients experience similar symptoms and patients with IBS often have hypersensitivity to lactose. 25% of patients with IBS also have defined lactase deficiency.[24] Either way, restriction of lactose in both groups may result in improvement of symptoms.

Dietary management is the first treatment for lactose intolerance. Patients may start with severe restriction and then work their way up to an amount of lactose they can handle. A total restriction is usually not necessary to avoid symptoms. Ice cream, yogurt and milk have high amounts of lactose, while cheese has lower lactose. Supplemental lactase enzymes, which are available over-the-counter, can be taken with food to help aid the digestion of lactose. These enzyme supplements cannot completely digest lactose and patients may still experience symptoms if lactose is ingested. In patients who avoid dairy, calcium and vitamin D should be supplemented and vitamin D levels should be monitored.[23]

FRUCTOSE INTOLERANCE

Fructose is a monosaccharide and is also found combined with glucose to make the disaccharide-glucose. It is commonly found in commercial sweeteners as high fructose corn syrup. The average ingests 11 to 54 grams of fructose per day, however, most people cannot fully absorb a load of 25 grams. As in lactose malabsorption, undigested fructose is digested by gut flora producing gas. Fructose intolerance is diagnosed by a fructose breath test.[25] Patients trying to avoid fructose should have a diet low in juices and fruits containing high net amounts of fructose-apples, pears, sweet cherries, prunes, dates, beverages sweetened with high fructose corn syrup, honey, and sorbitol containing gum and candy, as sorbitol can decrease fructose absorption.[26]

CELIAC DISEASE

Celiac disease, also known as gluten-sensitive enteropathy is an autoimmune disorder of the small intestine. It is caused by a reaction to gluten, a component of wheat protein. Exposure to gluten will cause mucosal inflammation and villous atrophy. Commonly known as a disease diagnosed in infancy, it is now being diagnosed in patients from 10 to 40 years old. Classic symptoms of untreated celiac disease are steatorrhea and flatulence.[27] However, more patients with celiac disease are reporting more atypical symptoms similar to IBS including abdominal pain, bloating and distension.[28] Most patients present with no signs of disease, but some patients present with signs of malnutrition including weight loss, stomatitis and easy bruising.[27]

IgA anti-tissue transglutaminase antibody is the preferred test for detection of celiac disease.[27] Endomysial antibody testing has higher sensitivity and specificity, but is also more expensive.[27] Antigliadin antibodies are no longer recommended for initial testing because of their low sensitivity and specificity.[27] Patients with positive IgA anti-tissue transglutaminase antibody serology should undergo small bowel biopsy.[27] Patients with a wheat allergy, a diagnosis separate from celiac disease, will have negative IgA anti-tissue transglutaminase antibody serologies but positive IgE serology and skin prick test to wheat.[29] Symptoms of a wheat allergy can include nausea, vomiting, indigestion and bloating. Other common symptoms are hives, cough, sneezing, asthma and anaphylaxis that are not seen in celiac disease.

The mainstay of treatment for celiac disease is adherence to a gluten free diet.[27] Patients should avoid wheat, rye, barley, and most beers. Rice, tapioca, soy, corn, potatoes and wine are safe to eat. Many patients with celiac disease may also develop lactose intolerance but this can be reversed once the intestines heal with gluten restriction.[27]

PROBIOTICS

Various studies have been performed that looked at the efficacy of probiotics in several gastrointestinal diseases. In IBS, multiple studies have shown that the probiotic Bifidobacterium infantis improves symptoms of IBS.[30,31] In a study that compared Lactobacillus GG to a low FODMAP diet in IBS, both were shown to aid in alleviation of symptoms.[32] Lactobacillus GG is sold under the brand name Culturelle®. In a study that compared B. infantis to both placebo and Lactobacillus, B. infantis was shown to be superior to both placebo and Lactobacillus in alleviating IBS symptoms.[31] B. infantis is sold under the brand name Align®. A systemic review of probiotics in lactose intolerance did not shown improvement in symptoms.[33] Probiotics in SIBO may relieve symptoms of abdominal pain but have not been shown to decrease the incidence of SIBO.[34]

OSTEOPATHIC MANIPULATIVE TREATMENT

Osteopathic manipulative treatment to the abdominal viscera is a useful way for primary care physicians to address abdominal bloating in the office. One commonly used technique for abdominal bloating and constipation is the mesenteric lift technique. The mesentery is the tissue that attaches the intestines to the abdominal wall. The goal of this technique is to improve blood flow and drainage of the vessels and lymphatics channels that course though the mesentery. This in turn will help restore normal intestinal motility. To perform this technique the patient lies supine with knees flexed. The physician places fingers medial to the anterior superior iliac spine (ASIS) and lifts the abdominal contents toward the umbilicus until a release is felt. This should then be repeated from the opposite lower quadrant. The physician should then apply traction from the left upper quadrant toward the umbilicus and then the right upper quadrant toward the umbilicus. Traction should be held until a release is felt.[35]

Another technique that can help with abdominal bloating is ganglion inhibition. The celiac ganglion, superior mesenteric ganglion, and inferior mesenteric ganglion carry sympathetic innervation to the intestines. This technique helps to normalize sympathetic innervation to the intestines. For this technique, the patient lies supine and the physician uses his fingers to apply pressure to the ganglion. The celiac ganglion is located inferior to the xyphoid process, inferior mesenteric ganglion under the umbilicus and just in between the two points is the superior mesenteric ganglion. Gentle pressure is applied by the physician's fingertips at all three points. As the patient exhales the physician allows his or her fingers to sink in, and while the patient inhales the physician resists. Pressure is held until a release is felt. This technique also helps to restore normal intestinal motility.[36]

CONCLUSION

Normal intestinal gas production is caused by bacterial metabolism in the colon. Belching is a normal physiologic process that can be exacerbated by excessive air swallowing and GERD. Symptoms of bloating are often caused by impaired GI transit such as in constipation and IBS. Foods high in FODMAPs and high gas producing foods can also cause the sensation of bloating. Lastly, bacterial overgrowth and malabsorption syndromes can cause increased gas production in the intestines. Treating gas and bloating starts by treating the underlying cause. Severe symptoms of abdominal pain and bloating caused by intestinal gas can be debilitating for many patients. Proper diagnosis and treatment can help patients to live more normal lives.

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No relevant financial affiliations

REFERENCES:

1. Lacy B, Gabbard S, Crowell, M. Pathophysiology, Evaluation, and Treatment of Bloating: Hope, Hype, or Hot Air. Gastroenterology & Hepatology. 2011, 7: 729-739 https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3264926/
2. Bredenoord AJ, Smout AJ. Physiologic and pathologic belching. Clin Gastroenterol Hepatol 2007; 5:772. http://www.cghjournal.org/article/S1542-3565(07)00189-9/fulltext
3. Bredenoord AJ, Weusten BL, Timmer R, et al. Relationships between air swallowing, intragastric air, belching and gastroesophageal reflux. Neurogastroenterol Motil 2005;17: 341-347.
4. Tomlin J, Lowis C, Read NW. Investigation of normal flatus production in healthy volunteers. Gut 1991; 32:665. http://gut.bmj.com/content/gutjnl/32/6/665.full.pdf
5. Friis H, Bodé S, Rumessen JJ, Gudmand-Høyer E. Effect of simethicone on lactulose-induced H2 production and gastrointestinal symptoms. Digestion 1991; 49:227.
6. Di Stefano M, Miceli E, Gotti S, Missanelli A, Mazzoccahi S, Corazza GR (January 2007). "The effect of oral alpha-galactosidase on intestinal gas production and gas-related symptoms". Dig. Dis. Sci. 52 (1): 78–83.
7. Drossman DA, Camilleri M, Mayer EA, Whitehead WE. AGA technical review on irritable bowel syndrome. Gastroenterology 2002; 123:2108. www.gastrojournal.org/article/S0016-5085(02)00481-X/pdf
8. Mearin F, Lacy BE, Chang L, et al. Bowel Disorders. Gastroenterology 2016.
9. Lasser RB, Bond JH, Levitt MD. The role of intestinal gas in functional abdominal pain. N Engl J Med 1975; 293:524.
10. Serra J, Azpiroz F, Malagelada JR. Impaired transit and tolerance of intestinal gas in the irritable bowel syndrome. Gut 2001; 48:14. http://gut.bmj.com/content/gutjnl/48/1/14.full.pdf
11. Agrawal A, Houghton LA, Reilly B, et al. Bloating and distension in irritable bowel syndrome: the role of gastrointestinal transit. Am J Gastroenterol 2009; 104:1998. http://www.ibs-care.org/pdfs/ref_176.pdf
12. Zhu Y, Zheng X, Cong Y, Chu H, Fried M, Dai N, Fox M. Bloating and Distention in Irritable Bowel Syndrome, The Role of Gas Production and Visceral Sensation After Lactose Ingestion in a Population with Lactase Deficiency. The American Journal of Gastroenterology. 2013, 108: 1516-1525. http://www.readcube.com/articles/10.1038/ajg.2013.198
13. Shepherd SJ, Lomer MC, Gibson PR. Short-chain carbohydrates and functional gastrointestinal disorders. Am J Gastroenterol 2013; 108:707
14. Hasler WL, Owyang C. Irritable bowel syndrome. In: Textbook of Gastroenterology, 4th ed, Yamada T (Ed), JB Lippincott, Philadelphia 2003. p.1817.
15. Chapman RW, Stanghellini V, Geraint M, Halphen M. Randomized clinical trial: macrogol/PEG 3350 plus electrolytes for treatment of patients with constipation associated with irritable bowel syndrome. Am J Gastroenterol 2013; 108:1508.
16. Caldarella MP, Serra J, Azpiroz F, Malagelada JR. Prokinetic effects in patients with intestinal gas retention. Gastroenterology 2002; 122:1748. http://www.gastrojournal.org/article/S0016-5085(02)00007-0/pdf
17. Gorard DA, Libby GW, Farthing MJ. Effect of a tricyclic antidepressant on small intestinal motility in health and diarrhea-predominant irritable bowel syndrome. Dig Dis Sci 1995; 40:86.

18. Ford AC, Moayyedi P, Lacy BE, American College of Gastroenterology monograph on the management of irritable bowel syndrome and chronic idiopathic constipation. Am J Gastroenterology 2014; 109 (Supp 1): S2-S26. http://gi.org/wp-content/uploads/2014/08/IBS_CIC_Monograph_AJG_Aug_2014.pdf
19. Bures J, Cyrany J, Kohoutova D, et al. Small intestinal bacterial overgrowth syndrome. World J Gastroenterol 2010; 16:2978-2986. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2890937/pdf/WJG-16-2978.pdf
20. Sachdev A, Pimentel M. Gastrointestinal bacterial overgrowth: pathogenesis and clinical significance. Therapeutic Advances in Chronic Disease; 2013; 4(5): 223–231. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3752184/pdf/10.1177_2040622313496126.pdf
21. Suchy FJ, Brannon PM, Carpenter TO, et al. National Institutes of Health Consensus Development Conference: lactose intolerance and health. Ann Intern Med 2010; 152:792. http://annals.org/aim/fullarticle/745834/national-institutes-health-consensus-development-conference-lactose-intolerance-health
22. Tishkoff SA, Reed FA, Ranciaro A, et al. Convergent adaptation of human lactase persistence in Africa and Europe. Nat Genet 2007; 39:31. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC2672153/
23. Misselwitz B, Pohl D, Fruhauf H, Fried M, Vavricka, Fox M. Lactose malabsorption and intolerance: pathogenesis, diagnosis and treatment. United European Gastroenterology Journal. 2013. 1(3): 151-159. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4040760/pdf/10.1177_2050640613484463.pdf
24. Tolliver BA, Herrera JL, DiPalma JA. Evaluation of patients who meet clinical criteria for irritable bowel syndrome. Am J Gastroenterol. 1994 Feb;89(2):176-8.
25. Ravich WJ, Bayless TM, Thomas M. Fructose: incomplete intestinal absorption in humans. Gastroenterology 1983; 84:26. www.gastrojournal.org/article/S0016-5085(83)80162-0/pdf
26. Skoog SM, Bharucha AE. Dietary fructose and gastrointestinal symptoms: a review. Am J Gastroenterol 2004; 99:2046. http://www.bashaar.org.il/files/101022005111814.pdf
27. Pelkowski T, Viera A. Celiac Disease: Diagnosis and Management. Vol 89, no 2. 2014. 99-105. https://www.aafp.org/afp/2014/0115/p99.pdf
28. Sainsbury A, Sanders DS, Ford AC. Prevalence of irritable bowel syndrome-type symptoms in patients with celiac disease: a meta-analysis. Clin Gastroenterol Hepatol 2013; 11:359.
29. Czaja-Bulsa G, Bulsa M. The natural history of IgE mediated wheat allergy in children with dominant gastrointestinal symptoms. Allergy, Asthma & Clinical Immunology 2014. 10:12 https://aacijournal.biomedcentral.com/articles/10.1186/1710-1492-10-12.
30. Whorwell PJ, Altringer L, Morel J, Bond Y, Charbonneau D, O'Mahony L, Kiely B, Shanahan F, Quigley EM. Efficacy of an encapsulated probiotic Bifidobacterium infantis 35624 in women with irritable bowel syndrome. Am J Gastroenterol. 2006;101(7):1582-1589. www.ibs-care.org/pdfs/ref_150.pdf
31. O'Mahony L, McCarthy J, Kelly P, Hurley G, Luo F, Chen K, O'Sullivan GC, Kiely B, Collins JK, Shanahan F, Quigley EM. Lactobacillus and bifidobacterium in irritable bowel syndrome: symptom responses and relationship to cytokine profiles. Gastroenterology. 2005;128(3):541-551. http://www.gastrojournal.org/article/S0016-5085(04)02155-9/pdf
32. Pedersen N, Andersen NN, Végh Z, Jensen L, Ankersen DV, Felding M, Simonsen MH, Burisch J, Munkholm P. Ehealth: Low FODMAP diet vs Lactobacillus rhamnosus GG in irritable bowel syndrome. World J Gastroenterol. 2014 Nov 21; 20(43): 16215–16226. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4239510/
33. Levri KM, Ketvertis K, Deramo M, Merenstein JH, D'Amico F. Do probiotics reduce adult lactose intolerance? A systematic review. J Fam Pract. 2005;54(7):613-619. https://www.mdedge.com/sites/default/files/Document/September-2017/5407JFP_AppliedEvidence3.pdf
34. Zhong C, Qu C, Wang B, Liang S, Zeng B. Probiotics for Preventing and Treating Small Intestinal Bacterial Overgrowth: A Meta-Analysis and Systematic Review of Current Evidence. J Clin Gastroenterol. 2017;51(4):300.
35. DiGiovanna E, Schiowitz S, Dowling D. An Osteopathic Approach to Diagnosis and Treatment. Third Edition. Lippincott Williams and Wilkins, 2005. 602-604
36. NYIT College of Osteopathic Medicine, Department of Osteopathic Medicine. 2013 http://koya.nyit.edu/Clinical_Applications/treatment_vis_abdomen.html

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REVIEW ARTICLE

Primary Care Approach to Eye Conditions

Sharanjit Kaur, DO[1]; Helaine Larsen, DO[1]; Alanna Nattis, DO[2]

[1] Good Samaritan Hospital Medical Center, Family Medicine, West Islip, NY
[2] Lindershurst Eye Physicians and Surgeons, PC, West Islip, NY

KEYWORDS:

Acute Angle-Closure Glaucoma

Chemical Burns

Conjunctivitis

Red Eye

Retinal Detachment

ABSTRACT: Many patients present to the primary care physician with complaints relating to the eye. While many are benign, others can be vision threatening. Performing a thorough history and physical can quickly assess the severity. More common, often benign conditions include conjunctivitis, keratoconjunctivitis sicca, blepharitis, subconjunctival hemorrhage, corneal abrasion, stye, chalazion, ectropion, entropion and episcleritis. Other more complex conditions include ptosis and cataracts. Vision-threatening conditions including uveitis, malignancies, retinal detachment, acute angle closure glaucoma, globe injuries and chemical burns require immediate recognition and referral to an ophthalmologist.

INTRODUCTION

Eye-related complaints make up 2-3% of primary care office visits.[1] Knowledge of how to respond when these patients present is fundamental for the family physician, as is recognizing when to refer to an ophthalmologist for further care. Family physicians should be able to recognize eye conditions that can lead to visual loss, therefore requiring urgent referral to the ophthalmologist.[2] A thorough history and physical is core in making a diagnosis and determining the urgency of the eye condition. History should focus on visual changes, duration of symptoms, presence or absence of a foreign body, history of trauma or recent eye surgery, and associated symptoms, such as a headache, nausea or ocular discharge. Nearly half of the eye problems that present to the family physician include conjunctivitis, keratoconjunctivitis sicca, and corneal abrasions.[3] More severe conditions include retinal detachment, acute angle closure glaucoma, mechanical globe injuries and chemical injuries.[4] Basic equipment such as a Snellen chart, a tonometer, a penlight, an ophthalmoscope, dilating drops and fluorescein stain are available to the primary care physician to aid in achieving the correct the diagnosis.

HISTORY AND PHYSICAL EXAMINATION

Initial evaluation should consist of questions relating to vision loss or change, foreign body sensation, photophobia and headache. If a patient complains of a foreign body sensation then corneal abrasion, retained foreign body or keratitis should be part of the differential diagnosis. A sandy sensation is often associated with keratoconjunctivitis sicca, blepharitis, or dry eye syndrome.[1] A thorough history of contact lenses use should be obtained focusing on the wearing schedule, overnight use, hygiene protocol, and swimming or showering while wearing a contact lens to rule out a corneal ulcer. If a patient is complaining of photophobia, it could be a sign of corneal involvement. A headache with associated eye pain points toward the diagnoses of acute angle-closure glaucoma, cluster headaches, iritis, and migraines.[1] When symptoms recur, a systemic inflammatory disease should be considered.

Comprehension of basic eye anatomy is pivotal for the primary care physician in order to perform a detailed and complete physical exam. The primary care physician should inspect the eyelid and sclera for inflammation, abrasions, hemorrhage, erythema or lesions. The upper eyelid should be evaluated and everted if corneal abrasion or retained foreign body is suspected. The eyelid and the periorbital region should be examined for rashes or vesicles.

Additionally, Woods Lamp can be utilized to evaluate for corneal abrasion or foreign body.[2] The conjunctiva should be evaluated for injection, which is indicative of inflammation or infection.[1] All patients complaining of eye pain should be assessed for visual disturbances *(Table 1)*. A Snellen chart should be used to assess visual acuity, having the patient read from a distance of 20 feet. Limitation of ocular motility should be ruled out by performing an exam of extraocular muscle function.

COMMON EYE CONDITIONS

One of the most common ophthalmologic diagnoses seen by the primary care physician is conjunctivitis.[3] The major causes of conjunctivitis can be divided between noninfectious and infectious.[5] *(Table 2)* Noninfectious causes include allergic, exposure, blepharitis, foreign body, subconjunctival hemorrhage, iritis, chemical burns,

CORRESPONDENCE:
Sharanjit Kaur, DO | skaur6103@yahoo.com

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and corneal abrasions.[5] Other conditions commonly seen in the primary care setting include strabismus, uveitis, carcinomas, entropion, ectropion, pterygium, stye, and chalazion *(Table 3)*.

EVALUATION AND MANAGEMENT OF COMMON OCULAR CONDITIONS

Viral conjunctivitis is most commonly caused by adenovirus and herpes, the former being highly contagious. Viral conjunctivitis is often associated with an upper respiratory infection and other generalized systemic symptoms such as a sore throat, fever, and headache.[3,5,6] Symptoms of adenoviral conjunctivitis include eye redness, lacrimation, watery discharge and blurred vision. These symptoms are usually mild and often self-limiting after one to two weeks. The treatment is often supportive care with cold compresses and artificial tears. Since viral conjunctivitis is easily transmissible, it is imperative to educate patients regarding strict hand and contact lens hygiene as well as the avoidance of sharing personal objects until their symptoms resolve completely. The primary care physician should refer the patient

TABLE 1:

Different causes of eye pain[3,6]

HISTORY	CAUSES
Photophobia	Keratitis, corneal abrasion, acute angle-closure glaucoma, migraine
Headache	Acute angle-closure glaucoma, migraine
Decreased vision	Optic neuritis, uveitis, cellulitis
Contact lens use	Corneal abrasion, keratitis, bacterial conjunctivitis, corneal ulcer
Foreign body sensation	Corneal abrasion, dry eye, keratitis, foreign body

TABLE 2:

Major causes of conjunctivitis[3, 6, 16]

CONJUNCTIVITIS	MOST COMMON CAUSES	SIGNS	SYMPTOMS
Viral	Adenovirus (most common), Herpes Simplex Virus, enterovirus, Coxsackievirus	Diffuse conjunctival injections, preauricular lymphadenopathy, lymphoid follicle on the eyelid	Mild to no pain, occasional discomfort with mild itching, watery or serous discharge, often starts of unilateral
Herpes zoster	Herpes zoster	Vesicular rash, uveitis, keratitis	Pain and tingling sensation precedes rash and conjunctivitis, unilateral
Bacterial: acute and chronic	Common pathogens (children): Streptococcus pneumonia (adults) Staphylococcus aureus	Edema, conjunctival injections	Mild pain, red eye with foreign body sensation, mild purulent discharge, bilateral glued eyes upon awakening
Bacterial: hyperacute	N. gonorrhoeae	Chemosis, possible corneal involvement	Severe pain, diminished vision, purulent discharge
Allergic (Figure 1)	Allergens	Conjunctival injection, cobblestone papillae under upper eyelid	Bilateral involvement, tearing, itching, watery discharge

to an ophthalmologist if the patient's symptoms do not resolve after a total of 10 days or if there is any suspicion of corneal involvement.[3,6]

Bacterial conjunctivitis is also highly contagious and usually spreads through direct contact with contaminated fingers. Bacterial conjunctivitis is usually unilateral and can be classified as hyperacute, acute or chronic. It usually consists of a greater amount of discharge and lid swelling than viral conjunctivitis. Neisseria gonorrhoeae is an important cause of hyperacute conjunctivitis. Those at risk include newborns who acquire the infection during delivery and young adults who acquire the infection during sexual activity.[6] The infection is usually sudden in onset and is characterized by copious, purulent discharge and severe pain. Patients also complain of some vision loss in the affected eye. Patients with a suspected diagnosis of Neisseria conjunctivitis should be referred to an ophthalmologist for aggressive management as it can quickly lead to vision loss secondary to corneal ulceration and perforation. Acute bacterial conjunctivitis has the classic symptoms of discomfort, blurry vision, and mucopurulent secretions with "sticky" eyelids upon awakening.[3,6] Symptoms usually last for less than seven days.

Staphylococcus aureus and Staphylococcus epidermidis are common etiologies of conjunctivitis in adults, while Streptococcus pneumonia and Haemophilus influenza tend to affect children.[6] There are various antibiotic eye drops available for treatment, and they are generally well tolerated *(Table 4)*. Chronic bacterial conjunctivitis occurs when symptoms last longer than four weeks with frequent relapses. The patient complains of sore eyelids and ocular discomfort with little discharge. Upon examination, the eyelids appear thickened, slightly inflamed and crusty.[3,6] The conjunctiva may appear normal or slightly erythematous. Bacterial culture is usually needed to identify the organism responsible for patients with chronic bacterial conjunctivitis.[5] Patients with this diagnosis typically require referral to an ophthalmologist for further management.

TABLE 3:

Common eye conditions[9]

CONDITION	SIGNS	SYMPTOMS	TESTS	TREATMENT
Entropion	An in-turned lower lid margin	Irritation, burning and foreign body sensation. Tearing results from lashes abrading the globe	Clinical diagnosis	Manually tape the lid away from the globe. Botulinum toxin injection Surgery is performed to correct the abnormality
Ectropion	An out-turned lower lid margin	Irritation, burning, and foreign body sensation. Tearing results from punctal malposition	Clinical diagnosis	Artificial tears, gel or ointment for lubrication Surgery is performed to correct the abnormality
Stye (Figure 2)	A painful, erythematous nodule on the skin surface or conjunctival surface of the lid	Painful nodule or pustule of the eyelid	Clinical diagnosis	Warm compresses and topical antibiotics drops (fluoroquinolones or polytrim) Incision and drainage if compresses and antibiotics fail
Chalazion (Figure 3)	A firm well demarcated nodule below the lid margin	Usually symptom free or minimally tender nodule of the lid	Clinical diagnosis	Early: warm compresses Intermediate: injection of triamcinolone Late: marsupialization of encysted meibomian gland
Pterygium	Fibrovascular growth extending from the conjunctiva onto the cornea	Symptom free Intermittent irritation, redness, mild visual disturbance	Clinical diagnosis	Artificial tears for lubrication Sunglasses to block UV light Surgical resection
Floppy eyelid syndrome	Usually unilateral or asymmetric	Irritation, burning, foreign body sensation and discharge	Clinical diagnosis	A fox shield is taped over the eye at night to prevent the lid rubbing on the pillow Surgery is necessary to tighten the upper and lower lids horizontally
Contact dermatitis	Acute: erythema and edema of the eyelid Chronic: scaling and lichenification	Generalized pruritis or painful eyelid	A thorough history of exposure Patch testing may be needed	Advised to avoid contact with suspected cause A topical corticosteroid such as fluorometholone 0.1% ophthalmic ointment

Chlamydial conjunctivitis is often seen in young sexually active adults. It presents very similarly to acute bacterial conjunctivitis, though it may be seen as smoldering chronic conjunctivitis in some cases. The common symptoms include ocular irritation, scant mucopurulent discharge, glued eyelids upon awakening and blurred vision. Patients do not respond well or fully to typical antibiotics that are prescribed for acute bacterial conjunctivitis. Bacterial culture (Giemsa stain) and ELISA testing can reveal the diagnosis of Chlamydial conjunctivitis. Treatment includes erythromycin ophthalmic ointment and oral therapy with azithromycin (single one gram dose) or doxycycline (100 mg twice a day for 14 days) to clear the infection. The patient's sexual partner should also be treated to prevent further infections and reinfection.[3,6]

TABLE 4:

Common ophthalmic antibiotics for acute bacterial conjunctivitis[3,5,6,16]

Trimethoprim/polymyxin B
Ofloxacin 0.3%
Azithromycin 1%
Besifloxacin 0.6%
Ciprofloxacin 0.3%
Erythromycin 0.5%
Levofloxacin 1.5%
Gentamicin 0.3%
Sulfacetamide 10%

Allergic conjunctivitis is seen in patients with an atopic disease, such as allergic rhinitis, eczema and asthma. Seasonal allergic conjunctivitis is often the most common type and it is related to specific environmental allergens. Symptoms include bilateral eye lacrimation, itching, and diffuse erythema *(Figure 1)*. Visual acuity is preserved and there is no corneal involvement. Large cobblestone papillae under the eyelid and chemosis may be present in severe cases.7 The primary care physician should educate the patient to avoid allergens and not to rub their eyes as this can worsen the condition. Over-the-counter oral antihistamines and topical histamine H1-Receptor antagonists can help alleviate symptoms. Acute allergic conjunctivitis is often self-limiting. [3,6]

Chronic allergic conjunctivitis, often referred to as vernal keratoconjunctivitis, is usually seen in patients age 3-25 years, with a history of asthma or eczema. It presents with chronic itching, photophobia, blurred vision, discoloration of the periorbital area and a thick, clear, stringy discharge. Everting the eyelids may reveal large flat papillae in severe cases of giant papillary allergic conjunctivitis. If the cornea appears hazy, ulcerated or symptoms fail to improve, the patient should be referred to an ophthalmologist for treatment. [3,6,7]

Keratoconjunctivitis sicca or dry eye is a condition caused by decreased tear production or poor tear quality. Some risk factors for the condition include advanced age, female sex, autoimmune conditions such as rheumatoid arthritis and Sjogren's syndrome,

FIGURE 1:

Allergic conjunctivitis

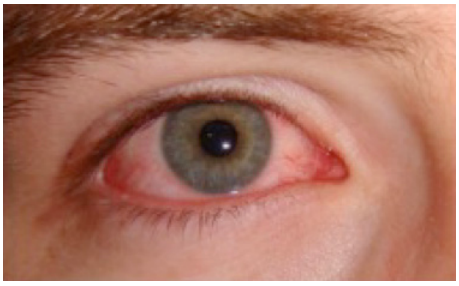


FIGURE 2:

Stye

FIGURE 3:

Chalazion

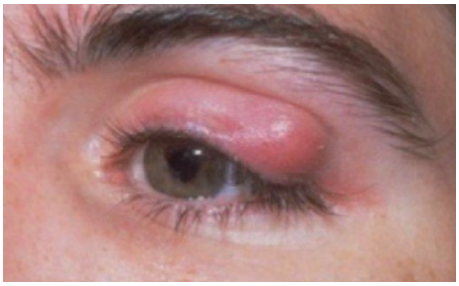
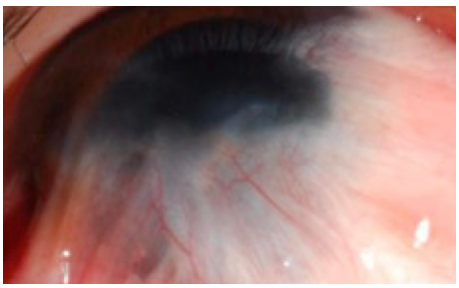


FIGURE 4:

Ocular surface burn with scar



as well as certain medications such as anticholinergics.[7] Diagnosis is usually made clinically, but certain diagnostic testing (i.e., tear osmolarity, tear break up time, and corneal fluorescein staining) can be used to facilitate the diagnosis.[3] Treatment can be initiated based on signs and symptoms. Treatment initially includes frequent use of artificial tears throughout the day and nightly application of lubricant ointments. Use of a humidifier

can also help decrease tear loss. If artificial tears and humidifiers are not efficacious, cyclosporine ophthalmic drops (Restasis ®, Allergan, Dublin, Ireland) or lifitegrast ophthalmic solution (Xiidra, Shire, Lexington, MA) may be used to increase tear production. Topical corticosteroids may also help in severe cases of dry eye. In general, if treatment beyond lubricants proves ineffective, the dry eye, the patient should be referred to an ophthalmologist.[3,7]

Blepharitis is a chronic inflammatory condition of the eyelid margins. If blepharitis is suspected, the patient should be evaluated for seborrheic dermatitis that is associated with scalp or facial flaking, as well as rosacea, which is associated with redness and swelling on the nose or cheeks.[3] The diagnosis of blepharitis is a clinical one. Treatment is supportive care such as eyelid hygiene, lid massage and warm compresses. When a patient does not respond to supportive care, topical erythromycin or bacitracin ophthalmic ointment can be used. In severe cases, oral antibiotics such as doxycycline or tetracycline may be considered.[7]

A corneal abrasion is a clinical diagnosis. Confirmatory tests such as fluorescein staining or Wood's lamp can be used.[8] A blanching pattern of staining suggests an abrasion or herpes virus (herpes simplex (HSV) or herpes zoster (HZV) infection. If corneal HSV or HZV is suspected, the patient should be referred to an ophthalmologist for appropriate treatment.[3] In the case of a corneal abrasion, the primary care physician should also check for foreign bodies under eyelids and in the conjunctival fornices. Treatment consists of antibiotic eye drops and/or ointment to prevent infection, supportive care, cycloplegics and pain control.[7] Steroids are contraindicated in corneal abrasions. If symptoms do not improve within 48 hours, the patient should be referred to an ophthalmologist.[8]

A subconjunctival hemorrhage occurs when a conjunctival blood vessel ruptures. It appears as a bright red patch in the subconjunctival space of the eye.[2] Subconjunctival hemorrhage is a clinical diagnosis and it is harmless and often requires no treatment. Supportive care with warm compresses and lubricants are the treatments of choice.[2] If pain is present, this should raise suspicion for foreign body and/or corneal involvement. Ophthalmologist referral is warranted if there is corneal involvement, history of blunt trauma, drainage, or persistent pain.[3]

Episcleritis is an inflammation of the superficial layers of episclera. It usually self-limited and resolves after two to three weeks. An in-depth investigation is needed if there are recurrent episodes. Treatment consists of supportive care and artificial tears, but in some cases may require a short course of topical steroids.[3]

Ptosis is defined as a drooping or falling of the upper eyelid. There are many etiologies of ptosis. Congenital ptosis results from a malformed levator muscle, while acquired ptosis may be due to the gradual thinning or disinsertion of the levator aponeurosis. For congenital or acquired ptosis, surgery is performed to tighten the levator aponeurosis or resect the levator muscle. Other important causes of ptosis include Horner's syndrome, third nerve palsy and myasthenia gravis. In patients with Horner's syndrome, the classic triad of miosis, ptosis and anhidrosis is seen.[9] If a Horner's syndrome is suspected, urgent referral to a neurologist and ophthalmologist is warranted for workup. In the case of an acute and painful Horner's syndrome, the patient should be sent for urgent neurologic and radiologic evaluation, as this could indicate a carotid dissection. The third (oculomotor) cranial nerve innervates all the extraocular muscles except the lateral rectus and superior oblique. Etiologies of the third nerve palsy include ischemic cranial mononeuropathy, vasculitis, compression of the third nerve by an aneurysm, tumor, or uncal herniation and trauma. Symptoms commonly seen are ptosis, diplopia, periorbital pain and headache. Magnetic resonance imaging of the brain with contrast is required when there is no obvious vascular risk factor. If symptoms are seen in young patients, or there is suspicion for an aneurysm, cerebral angiography may be necessary. Variable ptosis, or ptosis worse at the end of the day may be signs of ocular myasthenia. Myasthenia gravis and its ocular variant are autoimmune disorders of the neuromuscular junction. Patients may note that ptosis and symptoms of weakness improve after rest. A thorough workup including an acetylcholine receptor antibody titer, edrophonium chloride testing, nerve stimulation and chest computed tomography to rule out thymoma should be done. Patients with myasthenia should be referred to neurology for appropriate treatment.[9]

Strabismus can be esotropia or exotropia. Congenital esotropia is rare and occurs before the age of 6 months and accommodative esotropia occurs between two and four years of age.[9] Double vision and loss of depth perception occur initially. If strabismus is not treated, amblyopia may result which leads to blindness. In esotropia, one or both eyes deviate inward. In exotropia, one or both eyes have deviated outward.[9] All patients with strabismus should be referred to an ophthalmologist.

Uveitis is an inflammatory condition involving the uveal tract and can be classified as anterior uveitis and posterior. Most cases of anterior uveitis are acute in onset and have an idiopathic origin. The patient usually complains of redness, photophobia and pain. Nonocular symptoms such as back pain, joint stiffness, dysuria can occur if systemic disorders are the cause of uveitis.[9] On physical exam there is conjunctival injection and deposits on the posterior surface of the cornea. Floating inflammatory cells and protein in the anterior chamber are detectable with the slit lamp biomicroscope. Inflammatory cells are found in the iris surface.[9] In patients who are experiencing their first episode of unilateral and nongranulomatous anterior uveitis systemic workup is not necessary. Patients with recurrent episodes or bilateral granulomatous disease should have a systemic workup including a CBC, ESR, ANA, Lyme, RPR, and chest x-ray to rule out systemic disease.[9] Posterior Uveitis is usually acute and most commonly caused by toxoplasmosis.[9] Patients complain of decreased vision, floaters, redness, pain and photophobia. On physical exam, optic disc swelling and edema are observed. Inflammatory cells within the vitreous are known to cause a hazy view of the fundus of the eye.9 Retinal and choroid hemorrhages, exudates, and infiltrates can be noted during slit lamp biomicroscope examination.[9] All patient with uveitis should be referred to an opthalmologist within 24 hours.

The primary care physician may see galignant eyelid tumors such as basal cell carcinoma, squamous cell carcinoma and melanoma.. Basal cell carcinoma is the most common eyelid malignancy that appears in the lower and medial region and it appears as a pearly nodule.[9] If the lesion is located along the lid region, then eyelashes may be missing.

Basal cell carcinoma has a low potential to metastasize, but it can become locally invasive.[9] Surgical resection is the gold standard of treatment.[9] Options such as cryotherapy and radiation may be considered when surgery is not appropriate. Squamous cell carcinoma is less prevalent but more aggressive when compared to basal cell carcinoma. It is characterized by its erythematous, raised, scaly and central ulceration.[9] It occurs most frequently on the upper lid.

Actinic Keratosis can be the precursor lesion for this cancer.[9] The physician should palpate the preauricular and submandibular lymph nodes to detect potential metastases. The gold standard of treatment is surgical resection. Sebaceous carcinoma invades locally and spreads to lymph nodes.[9] It occurs in middle-aged to elderly patients and may mimic chalazion or blepharitis. It is known to be an aggressive tumor and metastasis to the lungs, liver and bone.[9] Melanoma is a rare eyelid tumor. When examining the eye, the physician should always evert the eyelid to look for any kind of conjunctival involvement.[9] If at a point in surveillance there is a change in the general appearance of the lesion, it warrants excisional biopsy of the lesion.

A cataract is a clouding of the eye's crystalline lens, and in most cases is age related. Patients complain of slowly progressive visual loss over months to years. Reduced color perception, monocular diplopia, and night-time glare are also common symptoms.[9] Cataracts are best evaluated after dilation of the pupil. The treatment of choice is surgical removal of the lens and placement of an intraocular lens implant. Over 1.5 million cataract surgeries are performed annually in the United States. Approximately 99% of patients obtain improved vision and quality of life after cataract surgery.[9]

OCULAR EMERGENCIES

Family physicians should be familiar with common signs and symptoms of some of the most common ocular emergencies. Ocular emergencies, if not recognized early, can lead to permanent vision loss and therefore warrant immediate attention. Some of the ocular emergencies include retinal detachment, acute angle-closure glaucoma, mechanical globe injuries and chemical injuries.[4,10] Physicians should begin with a thorough eye examination, which includes measurement of visual acuity, visual field testing, direct fundoscopic examination, and penlight examination of the anterior segment of the eye.

Retinal detachment occurs when the neurosensory layer of the retina is separated from the retinal pigment epithelium.[4] Risk factors include increased age, myopia, traumatic injury, family history, cataract surgery and a previous retinal detachment in the contralateral eye.[1] Patients with a retinal detachment may experience unilateral flashing lights and floaters in the affected eye. Severe vision loss may occur if the macula is involved in the detachment. If retinal detachment is suspected, the family physician should perform a dilated fundoscopic examination to visualize the detachment. If retinal detachment is suspected, the patient should immediately be referred to an ophthalmologist. Treatment of retinal detachment usually consists of surgery using laser photocoagulation to seal the retinal tear, then reattachment of the retina to the retinal epithelium. Untreated patients can have permanent and severe vision loss. Thus, it is very important for primary care physicians to have a high index of suspicion for retinal detachment based on the patient's signs and symptoms.[4,10]

A patient with unilateral eye pain and associated symptoms of a headache, nausea, vomiting should be considered to have acute angle-closure glaucoma until proven otherwise. Acute angle-closure glaucoma is a medical emergency in which the intraocular pressure rises rapidly, potentially leading to permanent vision loss within hours.[11] Risk factors associated with angle- closure glaucoma include older age, female sex, and Asian descent. Additionally, some commonly used medications are known to cause an increase in the intraocular pressure *(Table 5)*; therefore the primary physician should always thoroughly review the patient's medications. Physical examination typically shows a mid-dilated pupil, cloudy cornea and conjunctival injection.[12] Treatment of choice is a reduction of intraocular pressure using topical anti-hypertensive eye drops and laser peripheral iridotomy. The commonly used topical medications for this condition include 0.5% timolol maleate, 1% apraclonidine and 1% pilocarpine.[13]

Mechanical globe injuries and globe ruptures warrant immediate attention. Mechanical globe injuries occur when there is a full thickness rupture through the cornea and the sclera.[4] Globe rupture is often followed by blunt trauma to the eye. Patients with mechanical globe injuries present with eye pain, tearing, redness and decreased vision after trauma to the affected eye. If a patient presents with a history of blunt trauma to the eye, an exam with a penlight or slit lamp should be done to assess for a subconjunctival

TABLE 5:

Medications associated with acute-closure glaucoma [13,14]

CLASSES	MEDICATIONS
Adrenergic agonists	Ephedrine, phenylephrine
Anticholinergic agents	Ipratropium bromide, promethazine, botulism toxin
Cholinergics	Pilocarpine
Sulfa agents	Acetazolamide, topiramate
Antihistamines	Loratadine, diphenhydramine, cimetidine, ranitidine
Anticoagulants	Heparin

TABLE 6:

Roper-Hall Classification system[15]

GRADE	CORNEAL INVOLVEMENT	CONJUNCTIVAL LIMBUS	PROGNOSIS
I	Epithelial damage	No limbal ischemia	Good
II	Corneal haze, iris details visible	<1/3 limbal ischemia	Good
III	Total epithelial loss with stromal haze, iris details obscured	1/3 - ½ limbal ischemia	Guarded
IV	Cornea opaque, iris and pupil details obscured	> ½ limbal ischemia	Poor

hemorrhage, hyphema and irregular pupil. If a foreign body is visualized, it should not be removed.[4] A tetanus booster should be administered. A plastic or metal eye shield should be placed over the affected eye and patient should be immediately referred to an ophthalmologist. The primary care physician should educate the patient not to increase the eye pressure by coughing or straining. Computed tomography of the orbits is needed to evaluate for intraocular foreign bodies and fractures. Initial treatment consists of broad-spectrum antibiotics (i.e., ciprofloxacin, levofloxacin, moxifloxacin, ceftazidime). Removal of foreign bod(ies) and surgical repair by an ophthalmologist greatly reduces the risk of endophthalmitis if performed within the first 24 hours of injury.[4,10]

Chemical eye injury following exposure to acidic or alkaline compounds is another ophthalmologic emergency. Patients usually present with severe eye pain, redness, tearing, photophobia and decreased vision *(Figure 4)*.[10] The evaluating physician should try to identify the type and the amount of chemical involved. A thorough examination of the external eye is necessary; periocular burns should be identified and pH testing should be performed. The Roper-Hall classification system *(Table 6)* may be used to describe the extent of the injury.[10] The patient should immediately be referred to an ophthalmologist for treatment. Treatment involves placement of topical anesthetic followed by copious ocular surface irrigation using saline solution. The pH of the eye should be assessed after the irrigation. Continuous irrigation is needed until the pH of the affected eye is neutralized. Following irrigation, antibiotic eye drops are necessary; steroid drops and cycloplegics may also be used in certain cases. Ocular surface burns need close follow up with an ophthalmologist as early and late scarring can occur, leading to compromise of the ocular anatomy and possible vision loss.

CONCLUSION

Most primary care physicians are adequately equipped to manage common ophthalmologic conditions. A detailed history and physical will help tailor the differential diagnosis appropriately. The primary care physician should also be able to recognize the scenarios that warrant immediate referral to anophthalmologist.

AUTHOR DISCLOSURES:
No relevant financial affiliations

REFERENCES:

1. Pflipsen Matthew, Massaquoi Mariama, Wolf Suzzane. Evaluation of the Painful Eye. Am Fam Physician. 2016;93(12):991-998.
2. Galor A, Jeng BH. Red eye for the internist: when to treat, when to refer. Cleve Clin J Med. 2008;75(2):137-144.
3. Cronau H, Kankanala RR, & Mauger T. Diagnosis and Management of Red Eye in Primary Care. Am Fam Physician. 2010;(8):137-44.
4. Geltson D, Christopher. Common eye emergencies. Am Fam Physician. 2013;88(8):515-519.
5. Morrow L, Gary, Abbott L, Richard. Conjunctivitis. Am Fam Physician. 1998 Feb 15;57(4):735-746.
6. Foster A. Red eye: the role of primary care. Comm Eye Health.2005;(18):69-72.
7. Wirbelauer C. Management of the Red Eye for the Primary Care Physician. Am J Med. 2006;(119):302-306.
8. Wilson A, Stephen, Last Allen. Management of corneal abrasion. Am Fam Physician 2004;70:123-8,129-30.
9. Palay, David A., and Jay H. Krachmer.Ophthalmology for the primary care physician. Mosby, 1997.
10. Pokhrel K, Prabhat, Loftus A, Sanaz. Ocular Emergencies. Am Fam Physician. 2007;76:829-36
11. Distelhorst S, James, Hughes M, Grady. Open-Angle Glaucoma. Am Fam Physician. 2003;67:1937-44,1950.
12. Gupta Divakar, Chen P. Philip. Glaucoma. Am Fam Physician. 2016 Apr 15;93(8):668-674.
13. Quigley HA. Glaucoma. Lancet 2011; 377:1367.
14. Ah-kee Elliott Yann et al. "A Review of Drug-Induced Acute Angle Closure Glaucoma for Non-Ophthalmologists." Qatar Medical Journal 2015.1 (2015): 6.PMC. Web. 20 Jan. 2018.
15. Roper-Hall MJ. Thermal and chemical burns. Trans Ophthalmol Soc UK 1965;85:631-53.
16. Tarabishy B. Ahmad, Galor A, Jeng. Bacterial Conjunctivitis: A review for internists. Cleve Clin J Med. 2008;75(7):507-512

BRIEF REPORT

Autoimmune Anti-thyroid Encephalopathy: A Case of Steroid Responsive Hashimoto Encephalopathy

Joshua Mleczko, DO[1]; Daniel Pedersen, DO[2]

[1] Inspira Medical Center, Vineland, NJ

[2] Pedersen Family Medicine, Vineland, NJ

KEYWORDS:

Allergy/Immunology

Anti-thyroid

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Steroid Responsive Encephalopathy

Hashimoto

Hashimoto encephalopathy (HE) or steroid-responsive encephalopathy associated with anti-thyroid antibodies is a rare diagnosis but may be under-recognized among physicians. Although hyper or hypothyroidism is implied with the name Hashimoto, most of these patients are euthyroid. Nevertheless, most cases respond well with high-dose intravenous or oral steroids. The patient featured, SS, is a 46-year-old female who presented to our family medicine private practice in February for complaints of fatigue, word-finding difficulties, and generalized "mental fog." The patient's initial physical exam revealed prolonged relaxation phase of her bilateral patellar reflexes and poor accommodation in her right eye. The patient also exhibited a deficit in recall, in which she was only able to remember two out of the three objects on the mini-mental status exam. Her MRI showed numerous scattered T2/FLAIR hyperintensities in the cerebral matter, predominantly in the white matter. Her only lab abnormality was a mildly elevated anti-TPO antibody. CSF showed two oligoclonal bands. She was eventually diagnosed with HE eight months after presentation and responded very well to high-dose steroids for 3-4 months before relapsing, which ultimately required another round of steroid treatment. All in all, clinicians should check for the presence of anti-thyroid antibodies even if TSH is normal for patients presenting with either subtle or very profound, otherwise unexplained encephalopathy.

INTRODUCTION

Hashimoto encephalopathy (HE) or sometimes better classified as steroid-responsive encephalopathy associated with anti-thyroid antibodies (SREAT) is a rare disorder[1-3] that may be under-recognized, especially with primary care physicians. The first case of HE was diagnosed in 1966 and the existence of this disorder remains a topic of debate.[4] There are several theories to suggest that the pathophysiology stems from auto-immune complex mediated vasculitis, demyelination, and several more.[3] Although Hashimoto is in its name, these patients are euthyroid in a majority of cases.[1,3] Patients present with very varying clinical signs and symptoms, but a majority of cases improve with a brief course of high-dose steroids.[1-5] Because of this, it is important to make this difficult diagnosis.

CORRESPONDENCE:
Joshua Mleczko, DO | j.mleczko2@gmail.com

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The clinical manifestations of autoimmune disorders make up a large part of a primary care physician's practice, and vague symptoms such as fatigue and decreased concentration represent large and sometimes complicated differential diagnoses. Because of this, it can be difficult to decide where to start in the workup. Even worse, the history and physical exam may not be overwhelmingly helpful. The purpose of this article is to enlighten primary care physicians about this interesting diagnosis and to ensure that they consider this with otherwise unexplained encephalopathy. This case represents a case of HE with a very subtle presentation.

CASE PRESENTATION

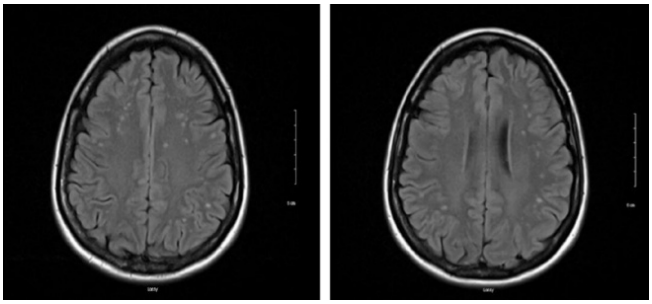
A 46-year-old female with no major past medical history presented as a new patient in our family medicine office in February with complaints of fatigue, progressive short term memory loss and word-finding difficulties for about one year. She first noticed the problem when she was deployed for the Air Force for four months from the prior year. Her symptoms fluctuated from day to day,

where cognition would be unpredictably worse on certain days. The patient had not seen a primary care provider for several years prior, as she stated that she had been healthy. The patient denied any head traumas, concussions, forgetting major events, falls, numbness, tingling, or weakness. She was adamant that she sleeps well, exercises regularly and eats a balanced diet. Her surgical history included a tubal ligation. She does not smoke tobacco, drink alcohol or use any illicit drugs. Her family history includes atrial fibrillation and hypertension in her mother. On physical exam, SS displayed prolonged relaxation phase of her bilateral patellar reflexes and poor accommodation in her right eye. The patient also exhibited a deficit in recall, in which she was only able to remember two out of the three objects registered earlier in the mini-mental status exam. The rest of her exam was completely normal.

Impressions of her MRI suggested numerous scattered T2/FLAIR hyper-intensities in the cerebral matter, predominantly in the white matter *(Figure 1)*. These were later classified to be non-specific, but according to the reading radiologist it can be seen in the setting of migraines, chronic small vessel ischemia, demyelinating disease, Lyme disease, vasculitides, prior insults such as infection, inflammation or trauma, as well as other etiologies. Lab work was all within normal limits. The Lyme panel only revealed one reactive KD 23 IgM. On follow up visit two weeks later, SS stated that there was no improvement to her memory or other symptoms and was sent to neurology at a tertiary care center nearby in Philadelphia. Repeat Lyme titers revealed reactive KD 23 IgM, KD 23 IgG and KD 41 IgG bands.

FIGURE 1:

Numerous scattered punctate T2/FLAIR hyper-intensities in the cerebral matter, predominantly subcortical white matter



At her initial neurology visit in April, SS displayed slowed mental searching capabilities and errors in delayed recall. Her deep tendon reflexes were also noted to be hyperactive with slowed relaxation. Labs including ammonia, heavy metals, lipids, sedimentation rate, CBC, coags, vitamin D, and hemoglobin a1c were all within normal limits, but her thyroid peroxidase antibodies were elevated at 23 IU/mL (almost 2.5 times normal). Her cerebral spinal fluid (CSF) was analyzed for Lyme, oligoclonal bands, cytology and protein electrophoresis. The fluid was positive for two oligoclonal bands, mildly low albumin and mildly high gamma globulin. At the patient's follow up visit one month later, SS started complaining of bilateral hip and knee pains. Consequently, she was sent for rheumatology workup as well as a neuropsychological (NP) evaluation. She was also seen by ophthalmology for visual evoked potential test to officially rule out multiple sclerosis.

Rheumatology had a low clinical suspicion of primary rheumatological disease. Regardless, SS was sent for x-rays of her cervical spine, hips and knees as well as extensive blood work, which was not available at the time of this write-up. Per the patient, this lab work was normal. X-rays of the hips and knees were normal, but the cervical spine showed degenerative disc changes at C5-C6. NP evaluation showed mild cognitive disorder with the largest deficits in auditory and visual attention, and executive and visuospatial functions. Recommendations included consideration of starting a psychostimulant as well as implementing compensatory strategies for memory, staying active, exercising, participating in psychotherapy like cognitive behavioral therapy, practicing mindfulness and a repeat NP evaluation in one year. SS did not tolerate the psychostimulant for more than two weeks, stating that she didn't like the way it made her feel. She felt like the medication made her feel jittery. In the interim, SS was evaluated by infectious disease in July who recommended a three week trial of doxycycline. She reported that her bilateral arthralgias did not improve and even worsened one week after stopping them.

The patient was seen by endocrinology in September. A thyroid ultrasound showed an inflamed thyroid gland and SS was subsequently started on 12.5 mcg of levothyroxine daily. Her repeat anti-TPO at this time was 587 IU/mL. The diagnosis of HE was made by neurology at this time. It was decided to treat the patient with a four-day course of high-dose solumedrol. After admission, she was discharged with a prednisone taper over one month. Repeat anti-TPO level during admission was 531 IU/mL. On follow up visit, SS reported resolution of her cognitive difficulties and was working again without issues. She still reported intermittent arthralgias in her knees and neck. She was re-evaluated by NP several weeks after her hospitalization, which showed resolution of her prior attention deficits.

The patient recently followed up with neurology, where she reported feeling well cognitively. She was able to work and think clearly without difficulty. Unfortunately, SS stated that her word-finding difficulties and "mental fog" returned in February of the following year. She was treated with five days of high-dose burst steroids and reports that her above symptoms, including her memory, almost completely resolved one week after.

DISCUSSION

Although rare, the diagnosis of Hashimoto Encephalopathy may be underdiagnosed and should be on the radar of any primary care doctor for any patient with symptoms resembling encephalopathy such as change in mental status, memory problems, "mental fog," and word-finding difficulties. The prevalence is about 2.1 in 100,000 patients.[1-3] HE predominantly affects women like other autoimmune disorders with a ratio of men to women of about 1:4, and has an average age of 52.[1]

HE is a steroid-responsive encephalopathy that is associated with autoimmune thyroiditis or more commonly with anti-thyroid peroxidase or anti-thyroglobulin antibodies. Average anti-TPO

antibody titers in the serum at the time of diagnosis is 900 IU/mL. The presence of antibodies has shown in the cerebral spinal fluid. Furthermore, serum antibody titers are always positive in the serum when CSF is positive for antibodies.[1] The clinical syndrome can vary from case to case but it usually lies on a spectrum ranging from stroke-like focal neurological deficits to a diffuse pattern that can cause impairments such as dementia, altered mental status, word-finding difficulties, hallucinations, confusion, etc.[1-3,5] Of note, a large percentage of patients with HE often suffer from altered cognitive functioning, seizures, and myoclonus. HE can present chronically with waxing and waning phases or acutely like delirium or rapidly progressive dementia.[3] Although Hashimoto is in the name of this disorder, hypo or hyperthyroidism is not a necessary criterion for the diagnosis. Most patients are euthyroid when the diagnosis is made. There also appears to be no correlation between the anti-thyroid level and the severity of the disease. Regardless, the presence of anti-thyroid antibodies is paramount to make this diagnosis.[3]

The pathophysiology of the disorder is not well understood, but some theories suggest autoimmune vasculitis or deposition of immune complexes as the cause of the signs and symptoms.[3] Another widely accepted explanation includes immune-mediated demyelination where anti-thyroid or TPO antibodies attacks neurons in the same way they destroy the thyroid gland.1 Patients often get near or complete resolution of their symptoms with high-dose steroids, even after years without treatment.[1-3,5] Immunomodulation with IV immunoglobulins, azathioprine, rituximab, plasma exchanges, or hydroxychloroquine also appears to be helpful in a handful of cases. Some of these above treatments were combined with steroids, but outcomes did not appear to differ.[1] To further complicate diagnosis and treatment, symptom relapse has been shown, as was evident with our above patient.

Brain MRI is sometimes negative, but up to 52% of cases have shown either cortical atrophy or nonspecific T2 signal abnormalities at the subcortical white matter. HE can also have non-specific findings on EEG in over eighty percent of cases. In many reported patients, EEG changes improved or resolved after proper treatment.[1]

As with any encephalopathy, it is important to rule out all other causes. The differential diagnosis in the above case with a physical exam and MRI findings included Lyme disease, multiple sclerosis, rapidly progressive dementia, stroke/transient ischemic accident, occult anxiety or depression, heavy metal toxicity, thyroiditis, or attention deficit hyperactivity disorder. With any syndrome including delirium or rapid dementia, other diagnoses such as Creutzfeldt-Jakob disease, infectious meningoencephalopathies, paraneoplastic encephalitis, tertiary syphilis, vitamin deficiencies, degenerative dementia, and cerebral vasculitis should be ruled out. To rule out all of these diagnoses, it is recommended to get a lumbar puncture, MRI with contrast, labs such as complete metabolic panel, complete blood count, urinalysis with culture, urine drug screen, ammonia, liver function tests, thyroid function, inflammatory markers, Lyme disease immunoglobulins, heavy metals such as lead and mercury, vitamin b12, and syphilis screen to rule out other causes of change in mental status, and an EEG.[3]

There were some limitations to this case report. As with many other private practice offices, patient information can be somewhat difficult to obtain from bigger, academic facilities and/or local specialists. To mitigate this weakness the patient featured in this report was contacted to corroborate the clinical course and to fill in any holes that may have presented themselves. It would have been particularly helpful to have data on the presence of anti-thyroid antibodies in her CSF because her HE diagnosis would have been made much quicker. It also appeared that the patient did not get an EEG study. It is unknown why this was never ordered. After a review of the literature, it is unclear which treatments are most efficacious for relapses. This should be a topic for further research. Regardless, the patient's diagnosis was made via a very systematic and logical clinical approach. In conclusion, clinicians should check for the presence of anti-thyroid antibodies even if the TSH is normal for patients presenting with either subtle or very profound, otherwise unexplained encephalopathy.

AUTHOR DISCLOSURES:
No relevant financial affiliations

INFORMED CONSENT:
The entirety of the case was reviewed with SS in our private practice office and she a gave informed consent to publish this case report.

REFERENCES:

1. Laurent C, Capron J, Quillerou B, et al. Steroid-responsive encephalopathy associated with autoimmune thyroiditis (SREAT): Characteristics, treatment and outcome in 251 cases from the literature. Autoimmunity Reviews. 2016;15(12):1129-1133. doi:10.1016/j.autrev.2016.09.008
2. Chang J-S, Chang T-C. Hashimoto's encephalopathy: Report of three cases. Journal of the Formosan Medical Association. 2014;113(11):862-866. doi:10.1016/j.jfma.2011.05.012.
3. Rubin, DI. Hashimoto encephalopathy. In: UpToDate, Aminoff, MJ & Ross, DS (Eds.), UpToDate, Waltham, MA, 2018
4. Brain L, Jellinek EH, Ball K. Hashimoto's disease and encephalopathy. Lancet 1966; 2:512
5. DynaMed Plus [Internet]. Ipswich (MA): EBSCO Information Services. 1995 - . Record No. 113943, Hashimoto thyroiditis; [updated 2014 Nov 17, cited 2018, Mar 29]; [about 19 screens]. Available from http://www.dynamed.com.ezproxy.rowan.edu/login.aspx?direct=true&site=DynaMed&id=113943. Registration and login required

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CLINICAL IMAGE

Ocular Surface Growth in 41-Year-Old Male

Leonid Skorin, Jr., DO, OD, MS, FAAO, FAOCO[1]; Emmalee A. Toldo, OD, MEd VFL[2]

[1] Mayo Clinic Health System, Albert Lea, MN
[2] Minnesota Eye Consultants, Minneapolis, MN

A 41-year-old male presents to the eye clinic for a long-standing growth on the nasal aspect of his left eye. He has noticed an increase in the size of the growth in the last six months. The patient reports that his left eye has some discomfort that began six months ago as well. When the eye gets uncomfortable, he uses artificial tears but notes minimal improvement in symptoms. He has no vision changes and his other eye is unaffected.

The patient had photorefractive keratectomy (PRK) refractive surgery for his nearsightedness in both eyes approximately 15 years ago. He experienced a good outcome from his PRK surgery and was asymptomatic. His current work as a landscaper includes exposure to dirt, dust, and dryness. He spends a lot of time outdoors and does not wear sunglasses. The patient also has a long-standing history of exposure to intense ultraviolet light while serving with the military in the desert in the Middle East. The patient has no other significant ocular or medical history.

On physical examination, the patient has an elevated, flesh-colored growth at the 9-o'clock perilimbal position of his left eye. It is wedge-shaped and extends from the conjunctiva approximately 1.5mm onto the cornea. There is no inflammation of the surrounding conjunctiva or sclera. No feeder vessels are present and there is no adjacent corneal thinning *(Figure 1 and Figure 2)*. The area is not painful to palpation and there is no pain on eye movement. His uncorrected visual acuity was 20/25 in each eye. Keratometry, which measures the anterior corneal curvature, indicates minimal corneal astigmatism. The remainder of the ocular health examination is unremarkable.

QUESTIONS

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

A. Limbal dermoid
B. Nodular episcleritis
C. Ocular surface squamous neoplasia
D. Pinguecula
E. Pterygium

2. What is the most likely cause of this condition?

A. Dust exposure
B. Environmental irritant exposure
C. Ultraviolet light exposure
D. Wind exposure
E. All of the above

3. What is the recommended treatment option?

A. Minimize exposure to dust, wind, and dryness
B. More frequent use of artificial tears
C. Surgical excision
D. Wear sunglasses when outdoors
E. All of the above

FIGURE 1:

Left eye growth, patient looking straight ahead

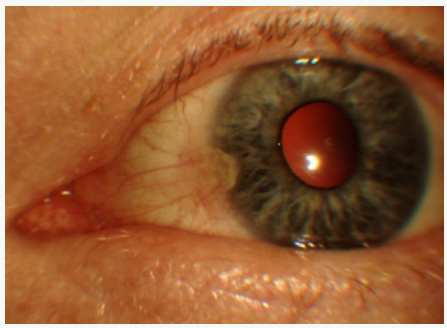
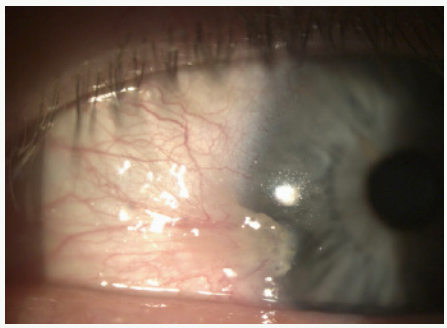


FIGURE 2:

Magnified view of left eye growth, patient looking temporally



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

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ANSWERS:

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

Correct Answer: E) Pterygium

Pterygia are a triangular or wing-shaped growth of elevated fibrovascular tissue that originates from the interpalpebral conjunctiva and grows onto the cornea.[1, 2] Pinguecula are yellow-white, flat or mildly elevated interpalpebral conjunctival lesions. They may have a similar appearance to pterygia, but they do not encroach onto the cornea and are normally asymptomatic.[1, 3] Ocular surface squamous neoplasia (OSSN) usually arises over a preexisting pinguecula. Like pterygia, ultraviolet light exposure is a known risk factor. Older age, smoking, and human papilloma virus or human immunodeficiency virus infection are other risk factors.[4] OSSN appears as a gelatinous epithelial thickening which may extend into the peripheral cornea with accompanying injection and prominent feeder vessels.[4] Nodular episcleritis, which is associated with collagen vascular disease, typically affects young adult females and has an acute presentation with more pain, redness, and an inflamed nodule overlying the sclera.[1, 3] Limbal dermoid is a congenital rounded whitish-yellow lesion. It is typically located at the inferotemporal limbus and there is no abnormal vascularization.[1, 3]

2. What is the most likely cause of this condition?

Correct Answer: E) All of the above

Prolonged exposure to any combination of the aforementioned environmental factors may lead to the development of a pterygium. Sunlight exposure and chronic irritation are classic contributors to pterygium formation.[3]

3. What is the recommended treatment option?

Correct Answer: E) All of the above

Choices A through C are the medical therapies that should initially be attempted. If they do not relieve symptoms, or if the lesion is threatening the visual zone, then surgical excision is recommended.[5] It is important to note that a pterygium will not resolve with the use of medical therapies and might even recur after surgical intervention.[5, 6]

DISCUSSION

A pterygium is an ocular surface disease that can cause varied problems for patients. It is described as a proliferative disorder resulting from an aberrant conjunctival wound healing response.[2, 7, 8] It is characterized by a wing-shaped growth of fibrovascular tissue onto the cornea.[2] It is caused by an altered limbal squamous epithelium with goblet cell hyperplasia.[2, 8] An advancing row of fibroblasts penetrates the cornea between Bowman's layer and the basement membrane of the overlying epithelium.[3] There is accompanying destruction of Bowman's layer of the cornea, affecting the cornea's clarity.[3] This has been shown to be mediated by ultraviolet light-induced matrix-metalloproteinases.[2] Pterygia are regarded as a benign lesion due to its slow growth, although they do tend to have local invasiveness and a high rate of recurrence if not properly removed during surgical intervention.[2]

Pterygia can be found worldwide. They are found more frequently in equatorial regions, at high altitude, and in highly reflective environments such as sand, snow, water, and concrete.[2, 3, 8] This is due to the increased levels of ultraviolet radiation encountered in these environments, which is a substantial risk factor for the development of pterygia.[2, 3, 8, 9] Exposure to hot, windy, dusty, or smoky environments is also a contributor.[10] There is a slight prevalence for older males which is likely related to a prolonged history of ultraviolet light exposure.[3, 11] Hereditary factors may also contribute to pterygium formation.[3, 10]

Pterygia are generally found on the nasal interpalpebral fissure, although simultaneous nasal and temporal involvement can occur.[9, 10] Temporal involvement alone is rare.[10] The affinity for the nasal limbus is thought to be due to reflection and refraction of sunlight in the nasal aspect of the cornea.[2, 10] Peripheral light focusing is a phenomenon where incidental light (commonly gathered at the temporal aspect of the eye) passes through the anterior chamber and focuses at the distal (nasal) limbus.[2] Damage to the limbal stem cells in this area are thought to be a main contributor to the aberrant wound-healing response and tissue changes in this ocular disorder.[2, 9]

Pterygia can cause several issues in affected patients. Changes in corneal topography and induced astigmatism can be seen as results of tissue proliferation.[2, 8] If the tissue encroaches into the visual zone then increased glare, decreased contrast sensitivity, obstruction of vision, corneal scarring, and subsequently reduced visual acuity can occur.[2,8] Chronic discomfort due to the inflammation and subsequent dry eye can cause problems for patients.[8,10] Photophobia, tearing, burning, red eye, and foreign body sensation are common patient complaints, as is the cosmetic concern.[8,10] Patients can also be asymptomatic. In very severe cases, ocular surface scarring can lead to more complications such as formation of symblepharon (cicatricial attachment of the conjunctiva of the eyelid to the conjunctiva of the eyeball), reduced ocular motility, and diplopia.[5, 8]

There are several different types of pterygium. Type I is a small primary pterygium which presents with minimal or no symptoms. It can be fibrous (parallel to the limbus), pinguecular (raised, 2-3 mm of stromal infiltration possible but no invasion of the cornea), or classical (apex invades 1-2 mm onto the cornea).[3] Type II pterygium, the most common type, is an advanced primary or recurrent pterygium.[3] There is no optical zone involvement. The cornea is invaded 2-4 mm and there is irritation with a reduction in vision due to irregular astigmatism.[3] Type III is an advanced primary or "malignant" recurrent pterygium.[3] There is more than 4 mm of corneal invasion and the optical zone is involved.[3] The patient has obvious symptoms of discomfort and there is always a reduction in vision. The "malignant pterygium" label is reserved for the rapid postoperative recurrence that may appear.[3]

TREATMENT

Conservative management is used for prevention, to control symptoms, and to minimize factors that promote pterygium progression. These would include avoiding sunlight exposure by

wearing wide-brimmed hats and sunglasses that filter ultraviolet light. If patients work or live in areas with significant dryness, wind, dust, or dirt exposure, then they should be encouraged to use topical lubricants such as artificial tears, ointments, and gels.[12, 13] Ocular decongestants such as naphazoline or tetrahydrozoline can also be used occasionally to reduce conjunctival swelling. These should not be used continuously due to secondary effects such as rebound hyperemia.[13] Topical nonsteroidal anti-inflammatory agents such as ketorolac or topical steroids such as loteprednol may be indicated for brief periods to more rapidly reduce inflammation and swelling when symptoms flare but should also be prescribed with caution due to secondary effects.[14] There is no known effective medical treatment to diminish or remove an established pterygium.[12, 15]

The definitive treatment is surgical.[8,15] Decisions to perform surgery are often based on a patient's cosmetic concerns, discomfort, or obstruction of vision.[2] Morphological features of the lesion that are considered markers of severity, such as visibility of the underlying episcleral blood vessels, should also be considered.[2, 9, 16] Medical indications for pterygium surgery include visual acuity loss, increased astigmatism, and encroachment of the optical zone.[5, 9,17] Ocular discomfort is an additional indication for surgery, although findings of disagreement between symptoms and signs could be indicative of possible corneal nerve damage.[9] When a pterygium is removed, the changes in astigmatism and corneal topography are often reversed, which may improve visual acuity.[5]

Pterygia often recur after excision; therefore, surgical excision should not be based on cosmetic appearance alone.[3, 15] Studies have found several risk factors that are associated with pterygium recurrence, including younger age at the time of surgery and increased or untreated postoperative inflammation.[5] A non-translucent, fleshy, higher-grade pterygium is also associated with increased recurrence rates.[5] Pterygium also tends to recur rapidly after excision. A 50% chance of recurrence within four months after surgery and a 97% chance of recurrence 12 months after surgery have been documented.[6] Shorter intervals between subsequent recurrence after surgical removal have also been documented.[6] This demonstrates the importance of choosing an effective surgery which has the least risk of recurrence.

There are many surgical techniques available. Different approaches have been attempted to decrease recurrence after excision while providing a safe and cosmetically appealing surgical outcome.[5,15] No surgical gold standard for safety and efficacy has been established.[5] Amniotic membrane graft and conjunctival autograft with fibrin glue or sutures have emerged as successful surgeries with decreased recurrence rates compared to historical surgical techniques.[5, 11] The use of fibrin glue was reported to have a statistically significant decrease in recurrence compared to suture use, as well as reduction in operation time and decreased postoperative inflammation.[5, 8, 11]

Some studies suggest that pterygia might have a propensity to evolve into precursors of ocular surface neoplasms.[2] The relationship between the two is not well understood; however, it has been suggested that ultraviolet radiation-induced mutations in tumor-suppressor genes play a role in their formation and therefore chronic ultraviolet light exposure is a shared etiology in both conditions.[2] As such, annual follow-up is recommended at a minimum or more frequently if there are concerning signs such as visual impairment, restriction of eye movement, or encroachment of the pupil.[2, 13]

This patient was diagnosed with a Type I pterygium with classical appearance, as it invaded 1.5mm onto the cornea and did not induce any irregular astigmatism. We discussed treatment options with our patient. He declined surgical intervention at the time. He will continue to use artificial tears with increased use in dry or dusty working environments. He will begin to wear sunglasses with ultraviolet light protection when he is outdoors. He has been scheduled to follow up in one year. The patient was advised to return sooner if the conservative therapy does not bring him adequate relief from his ocular discomfort or if he decides to proceed with surgical intervention.

AUTHOR DISCLOSURE:
No relevant financial affiliations

REFERENCES:

1. Bagheri N, Wajda B. Pterygium/Pinguecula. The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease. Seventh edition. Philadelphia: Wolters Kluwer. 2017;62-63.
2. Chui J, Coroneo MT, Tat LT, et al. Ophthalmic pterygium: A stem cell disorder with premalignant features. Am J Pathol. 2011;178(2), 817–827. doi:10.1016/j.ajpath.2010.10.037
3. Buratto L, Phillips RL, Carito G. Pterygium Surgery. Thorofare, NJ. SLACK Incorporated, 2000;3-47.
4. Sudesh S , Rapuano CJ, Cohen EJ, Eagle RC Jr, Laibson PR. Surgical management of ocular surface squamous neoplasms: The experience from a cornea center. Cornea. 2000;19(3)278–283.
5. Janson BJ, Sikder S. Surgical management of pterygium. Ocul Surf. 2014;12(2)112-119. doi:10.1016/j.jtos.2014.01.001
6. Hirst, LW, Sebban A, Chant D. Pterygium recurrence time. Ophthalmology. 1994;101(4)755-758. doi:10.1016/S0161-6420(94)31270-X
7. Di Girolamo N, Chui J, Coroneo MT, Wakefield D. Pathogenesis of pterygia: Role of cytokines, growth factors, and matrix metalloproteinases. Prog Retin Eye Res. 2004;23(2)195-228. doi:10.1016/j.preteyeres.2004.02.002
8. Fuest M, Mehta JS, Coroneo MT. New treatment options for pterygium. Expert Rev Ophthalmol. 2017;12(3)193-196. doi:10.1080/17469899.2017.1324297
9. Julio G, Lluch S, Pujol P, Merindano D. Ocular discomfort in pterygium patients. Optom Vis Sci. 2013;90(3)269–274. doi:10.1097/OPX.0b013e3182815b2a
10. Schwember J, Madrid L, Yori L. Rotatory conjunctival flap for pterygium removal: A simple and quick technique. Am J Cosmet Surg. 2017;34(4):183-186. doi:10.1177/0748806817705809

11. Todorovic D, Vulovic T, Sreckovic S, et al. Updates on the treatment of pterygium. Serb J Exper Clin Res. 2016;17(3), 257-262. doi:10.1515/sjecr-2016-0012
12. Hoffman RS, Power WJ. Current options in pterygium management. Int Ophthalmol Clin. 1999;39(1)15 -26.
13. Rapuano CJ. Color Atlas & Synopsis of Clinical Ophthalmology. Wills Eye Institute. Cornea. Second Edition. Philadelphia: Lippincott Williams & Wilkins. 2012;42-43.
14. Frucht-Pery J, Siganos CS, Solomon AT, et al. Topical indomethacin solution versus dexamethasone solution for treatment of inflamed pterygium and pinguecula: a prospective randomized clinical study. Am J Ophthalmol. 1999;127:148-152.
15. Farjo QA, Sugar A. Pterygium and conjunctival degenerations. In: Yanoff M, Duker JS (eds). Ophthalmology. Third edition. New York, NY: Mosby Elsevier. 2009;248-250.
16. Labbé A, Gheck L, Iordanidou V, et al. An in vivo confocal microscopy and impression cytology evaluation of pterygium activity. Cornea. 2010;29(4):392-399. doi:10.1097/ICO.0b013e3181bd44ce
17. Hirst L. Pterygium Surgery. In: Alberts DM, Lucarelli MJ (eds). Clinical Atlas of Procedures in Ophthalmic and Oculofacial Surgery. Second Edition. New York, NY: Oxford University Press. 2012;180-194.

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Samer Shaja, DO

Ronald Januchowski, DO, FACOFP, Editor • Paula Gregory, DO, MBA, CHCQM, FAIHQ, Health Literacy Editor

Gas (flatulence or belching) is very common and is not harmful. Flatulence is gas that is released by the rectum. Belching (or burping) is gas that is released from the mouth. The buildup of gas or food contents within the digestive tract can lead to an upper body fullness feeling often described as bloating. Gas is commonly caused by swallowing air; you swallow air into your stomach when you eat food or drink fluids. Gas can also form as a byproduct of bacteria in your intestines when digesting food. Some foods that can increase gas being formed include high fiber foods such as: beans, broccoli, lentils, asparagus, peas, onions, cabbage and whole grain foods. Other foods, such as dairy products or carbonated drinks, can also cause a lot of gas to form. Poorly fitting dentures can cause gas as a lot of saliva (with air bubbles in it) is swallowed. Chewing gum or sucking hard candy can also produce swallowed gas.

POSSIBLE CAUSES:

- Carbonated beverages such as soda or beer
- Chewing gum or sucking hard cand
- Improperly fitted dentures
- Beans
- Drinking dairy
- Eating food too fast

MEDICAL CARE AND TREATMENT OPTIONS:

Gas, bloating and belching are not usually due to medical problems but rather; they are symptoms that are often due to the way your body normally works.

WHEN TO SEEK EMERGENCY ATTENTION?

- If you experience chest pain, sweating, shortness of breath, painful breathing or dizziness/lightheadedness while having gas pain you should seek emergency medical attention.
- If you have nausea, vomiting, persistent or worsening abdominal pain, bright red stools, dark black or sticky/tarry stools or vomiting of a coffee ground-like material. The dark black material in the vomitus or stool may be blood.
- If it has been longer than usual since your last bowel movement and you are no longer passing gas through your rectum. This is when the normal flow of material through the digestive tract is blocked.

SOURCE(S): *American College of Gastroenterology; National Institute of Diabetes and Digestive and Kidney Diseases; International Foundation for Functional Gastrointestinal Disorders*

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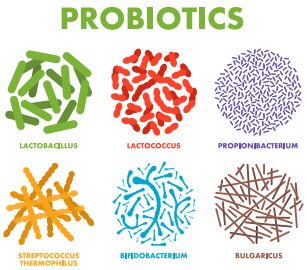
PROBIOTICS: WHAT ARE THEY? WHAT DO THEY DO?

Sandi Aung, DO

Ronald Januchowski, DO, FACOFP, Editor • Paula Gregory, DO, MBA, CHCQM, FAIHQ, Health Literacy Editor



Probiotics are the good "gut bacteria" that are made to be similar to the bacteria already in your digestive tract. Having too much of the "bad" bacteria in your body can cause an imbalance, leading to all types of health problems such as fatigue, constipation, diarrhea, weight gain, and all varieties of chronic health problems.

DIFFERENT PROBIOTIC FORMS

Probiotics come in several forms, including the more familiar form of nutritional supplement pills, but they also exist in probiotic food products such as certain type of yogurts, kefir, cheeses, lactobacillus milk products, cheeses, or fermented foods like sauerkraut, and kimchi.

A probiotic dietary supplement can aid your body in maintaining health in a variety of ways. However, not all probiotics are the same, and your body may respond to different forms of probiotics and different strains of bacteria in different ways. For example, if you're lactose-intolerant, avoid taking probiotics in the form of dairy products. Common strains include Lactobacillus species, Bifidobacteria, Saccharomyces boulardii and Bacillus coagulans.

SAFETY OF PROBIOTICS

Probiotics are safe in the amounts you normally find in food. In general, most healthy adults can safely add foods or dietary supplements that contain probiotics to their diets. Some individuals might experience gas (flatulence), but that generally passes after a few days. Remember, probiotics are not all the same, and your body may react differently to different strains and how you ingest it.

WHAT DO PROBIOTICS DO?

Probiotics are believed to balance our digestive tract to keep a healthy balance between the good and bad bacteria. In our daily lives, many factors including stress, poor sleep, nutritional imbalance, antibiotic overuse, other medications, emotional and environmental stress can all cause a shift towards the bad bacteria predominating our digestive tract.

When our digestive tract is healthy, it does its job more effectively by filtering toxins, and eliminating harmful bacteria, chemicals, and other waste products more efficiently and promote healthy immune system and reduce some common health issues such as bloating, diarrhea, antibiotic-associated diarrhea, Helicobacter pylori infection and promote healthy gut transit.

Research on probiotics also has shown it may protect us from allergic reactions and autoimmune diseases such as Rheumatoid arthritis, Ulcerative colitis, Irritable bowel syndrome, and lessen opportunistic infections in general.

Talk to your family doctor if beginning a probiotic supplement is right for you. Your osteopathic physician can help in deciding the right form of probiotics, and guide you towards healthy digestive tract options.

SOURCE(S): *The Centers for Disease Control and Prevention; Medicinenet.com; MayoClinic.org*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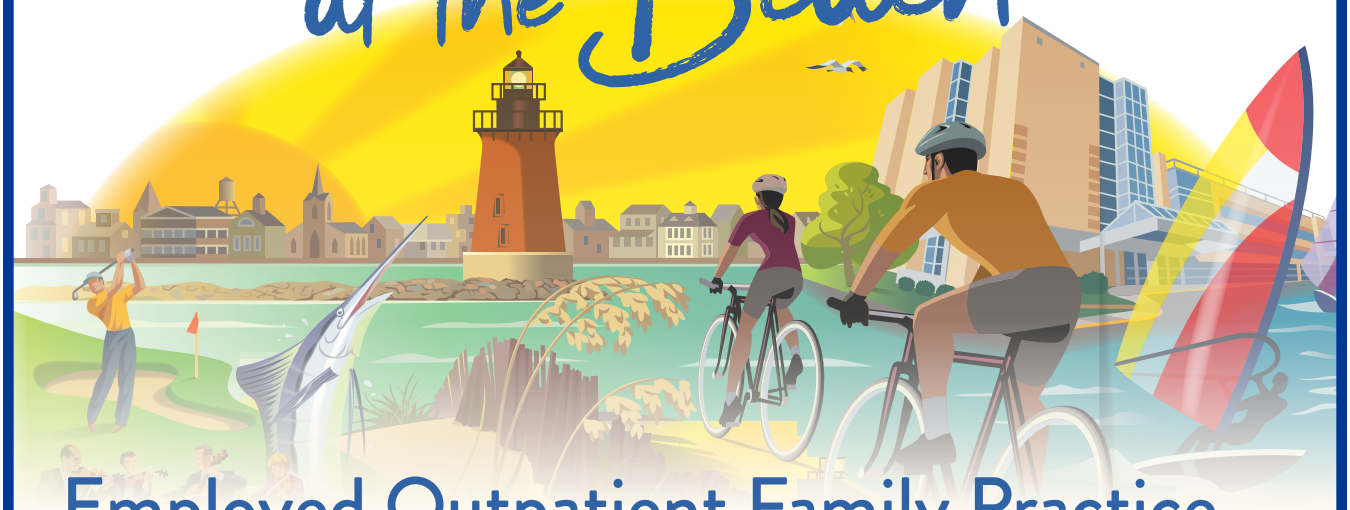

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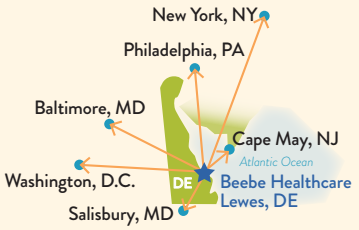

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