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Midwinter Osteopathic Family Practice Conference
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Des Moines, IA

JANUARY 19-20, 2024
2024 Winter Family Medicine Update
Michigan ACOFP
Belville, MI
https://maofp.org/event-5364371

JANUARY 18-21, 2024
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https://www.msacofp.org/2024-winter-family-medicine-update

FEBRUARY 9-11, 2024
2024 ACOFP Faculty Development and Program Directors' Workshop
Virtual
https://www.acofp.org/acofpimis/Acofporg/ContentAreas/Events/FDPD_Registration.aspx

APRIL 4–7, 2024
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New Orleans, LA

SEPTEMBER 19–22, 2024
OMED 2024
American Osteopathic Association
San Diego, CA

APRIL 2–6, 2025
ACOFP 62nd Annual Convention & Scientific Seminars and 75th Anniversary
Palm Springs, CA

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This giving season, join the 300+ donors who have contributed to the future of family medicine.

ACOFP member contributions directly support early career osteopathic family physicians by providing grants and resources to reduce the financial burden of osteopathic board certification and encourage early career leadership within the profession. New this year, the ACOFP Foundation has also established an Auxiliary Fund, which supports scholarships for osteopathic medical students, as well as leadership training and development for family medicine residents.

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(Receiving the grant) meant more than just the monetary donation and grant for my licensure, but it really brought me into the ACOFP family, and that is what we are here, is a family. It significantly helped me financially to not have the burden of testing and travel, especially coming out of medical school."

Stephanie Kidd, DO, MBA

EDITOR'S MESSAGE

A Traumatic Event

Paula Gregory, DO, MBA, FACOFP

Each day, physicians go to war against innumerable problems that patients have either self-created or that were created by the breakdown in the body on the macro or micro level. All of our bodies are aging, and systems are likely to fail. Over the course of our careers, we have witnessed many traumas and dramas in caring for our patients, traumas that also affect us. We often don't take time to process a bad outcome, and our patients' troubles do affect how we feel. At times, the doctor feels worse than the patient. Yet, we continue to show up to treat people.

Recently, with all the violence in the news, some stories stand out. One young physician took her own life and her child's. It's possible this could have been prevented. Students and young physicians are taught to accept scenarios in which things cannot be changed and move on to the next patient. We know that there are others who need care; their needs are pressing.

As healthcare providers, we are at daily risk of mental trauma ourselves. Repeated trauma changes our genetic makeup and can, over time, be damaging to our bodies. Additionally, changes from stress generate increased cortisol production. Mental health struggles can accelerate stress eating, cause lack of energy due to fatigue, and create burnout symptoms.

Adding to this stress is the fact that physical violence in healthcare settings is accelerating. Statistics show that this violence is not exclusive to large hospital emergency rooms. Regardless of the size or status of the healthcare setting, physicians must be aware of the possibility of violence. Literature in this area has mainly focused on experiences of violence against nurses (e.g., https://pubmed.ncbi.nlm.nih.gov/32175613/), while there are fewer studies on physicians. Aggression in the workplace has been associated with somatic injuries as well as with psychological consequences, such as burnout, post-traumatic stress disorder, depression, and anxiety.

In the workplace, we face daily mental traumas during which we feel that our advice and intercession should be helping; however, we know some situations are beyond our control. We would benefit from debriefing to discuss what went right, what could be changed or not, and how people are doing, even after an event that we consider routine. Debriefing our staff on community incidents and future challenges is equally important to ensure they are coping and feeling safe. A good program of mental health first aid can help create a safety net and help staff recognize issues in their coworkers and patients.

We have wonderful patients who are like family. Perhaps there are missed opportunities to understand deeply what they may be feeling. The patients of a family with an incident of violence may be hesitant to speak openly due to fear of not getting care themselves, and so things that should be discussed are often left out. Patients are often marginalized in a small community, as our staff sometimes feel that we need to know about an incident that they know happened. Our medical school training teaches us to be nonjudgmental and to offer humane treatment. We know from our interactions that choices are made for many reasons. Younger people who experience situations such as bullying or childhood abuse are at risk. We are less likely to interact with a physically healthy adolescent unless they have a vaccine or sports physical.

We must care for ourselves, our colleagues, and our patients. If we feel the need to talk to someone, it's okay. As we work to improve and maintain health and wellness for our patients and staff, we must "put our own oxygen mask on first" to do our best work. This will allow us to be the best physicians we can be for our patients, while maintaining our own mental health and well-being.

FROM THE PRESIDENT'S DESK

Honoring the Leadership of Women

David J. Park, DO, FAAFP, FACOFP *dist*.

In this issue of *Osteopathic Family Physician*, there is a focus on women's health. We all know that the landscape of women's health is a mosaic of complex needs, which include reproductive health, maternal care, functional and lifestyle considerations, and other gender-specific medical conditions. As scientific knowledge continues to expand in these areas, it is our obligation to keep current with new evidence-based practices as lifelong learners in family medicine. We are privileged with our ability to provide compassionate, holistic care, reinforced by our osteopathic approach that embraces the diverse dimensions of women's lives.

As osteopathic family physicians, we hold a sacred responsibility to take care of women at every stage of life. I am proud of the great strides we have made these past few decades in women's health, but what excites me even more is our path into the future. The evolution of technology beckons us with promises of transformative impact. The integration of electronic health information, virtual monitoring, and telehealth offers us unprecedented tools to engage in women's health in many innovative ways. Imagine a healthcare world where information is readily available whenever and wherever we are, enabling us to intervene before issues escalate, and where patients are empowered to take ownership of their well-being through real-time data and remote support. Not to mention the flexibility and convenience of access this offers busy women with multiple life responsibilities! I believe we are in the evolutionary process of increasing access for patients, decreasing in-person costs, and improving health outcomes with the integration of technology. However, we must also be vigilant in advocating for ourselves and the members on our healthcare team to ensure a good quality of life and job satisfaction by decreasing the stresses of new mandates from employers and regulatory bodies of government.

While our commitment to improving our patients' health is essential, so is our active involvement in leadership. There are many ways for you to involve yourself in this endeavor and one great way is to become more active in ACOFP. Visit the ACOFP website to learn more about the many ways you can contribute to the profession of osteopathic family medicine. There have been many female physician leaders in ACOFP who have exemplified this and have been great role models. They have been instrumental in advancing our mission by promoting excellence in osteopathic family medicine through education, leadership, and advocacy. It is within this context that we honor several of them who have served as presidents of our organization. The past presidents you will see spotlighted in this issue have worked to ensure that the voices and needs of women are elevated within our profession, and we celebrate them.

Let us be inspired by the leadership of our past, with a bright outlook for the future as we continue to incorporate new technologies, innovation, and knowledge in medicine to enhance the future of healthcare and wellness of our patients and ourselves.

Together, let us achieve new heights!

Professionally Yours,

David J. Park, DO, FAAFP, FACOFP *dist*.
2023–24 ACOFP President

PAST PRESIDENTS SPOTLIGHT:
A Legacy of Women

THIS NEW FEATURE HIGHLIGHTS THE LEGACY OF ACOFP PAST PRESIDENTS.

Jan D. Zieren, DO, MPH, FACOFP *dist.*

TERM OF PRESIDENCY: 2009-2010

THEME OF PRESIDENCY: TIME
I made "time" the theme. My focus was on relationships, connections, and collaboration.

ACCOMPLISHMENTS:

- Had the ACOFP Board meet with AOBFP to improve the relationship between us
- Invited Dr. Karen Nichols (incoming first female elected president for the American Osteopathic Association) to the ACOFP Board retreat to strengthen our connection while we discussed several AOA topics that impacted the ACOFP
- Attempted collaboration efforts over the year by meeting with Dr. Lori Heim (incoming second female elected president for the American Academy of Family Physicians), as our organizations had several mutual goals

Nicole Heath Bixler, DO, MBA, FACOFP *dist.*

TERM OF PRESIDENCY: 2020-2022

THEME OF PRESIDENCY: We Can D.O. It! This theme did not really come to fruition because of COVID, showing that a theme isn't the most important thing, but rather adaptability to what is needed at the time for the organization.

ACCOMPLISHMENTS:

- COVID response and the adaptability of our organization to,not only survive, but thrive
- Formulating the Task Force on Racism and Health that eventually morphed into the DEI Advisory Group, which helped formulate the blueprint for our organization's efforts in diversity, equity,and inclusivity
- Leading the initiative to reflect on our board and committee structure and spearhead change to modernize our governance to more adequately reflect our diverse membership
- Being a role model to other female physicians to demonstrate that you can DO it all . . . physician, mother, spouse, daughter, mentor, and leader!

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

TERM OF PRESIDENCY: 2014-2015

THEME OF PRESIDENCY: Enhanced state society and student chapter services and enhanced support for quality reporting and reimbursement

ACCOMPLISHMENTS:

- Task-force work leading to new state chapters in Alabama and Oregon and a regional society for Pennsylvania/Delaware and Maryland, development of distressed state society programs for financial support for state societies in need, and four national calls/webinar "conversations with the president," in which state society and student chapter leadership were invited to attend
- Regular CME education for members on quality improvement and enhanced reimbursement and continued work with the ACOFP Quality Metrics program (EHR-based quality reporting)
- Represented the ACOFP and other specialty societies and AOA affiliate state societies bringing forward concerns with the single accreditation system proposal. Our united efforts resulted in improved communication and assurances that DO training, residency programs, and program directors would continue to be respected and osteopathic distinctiveness would continue to thrive

REVIEW ARTICLE

AN OSTEOPATHIC APPROACH TO URINARY INCONTINENCE INCLUDING BIOPSYCHOSOCIAL ASPECTS AND MULTIPLE MODALITIES FOR A HOLISTIC APPROACH TO OPTIMIZE ONGOING CARE

Nicholas Trivelas, PGY-1[1]; Kirby Slaughter[2]; Kenneth Zaremski, DO[1]; Carol Kirila, DO[1]

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KEYWORDS

Urinary incontinence

Self-image

Socialization

Osteopathic manipulative therapy

Pelvis

ABSTRACT

Urinary incontinence is a nuanced and stigmatized condition that causes significant challenges for a large number of people in the United States and imposes a large financial and community burden. We provide an overview of major categorizations of incontinence by type as well as potential etiologies. We discuss how this condition impairs self-image, interferes with socialization, and can lead to depression and isolation; these elements inter-relate with access to care and implementation of the therapeutic options, further exacerbating patient suffering. We recognize the key components of patient evaluation regarding history and physical examination. Medicinal, surgical, and assistive device use are reviewed. Osteopathic manipulative treatments addressing the structures of the pelvis are also reviewed in detail. We illustrate how these techniques can be used to optimize outcomes. Utilizing a holistic approach to mitigate the multiple challenges that this condition presents can lead to greater success, reduced distress, and improved patient satisfaction.

INTRODUCTION

Osteopathic family physicians are often the first to evaluate urinary incontinence, which is common and affects both men and women. Urinary incontinence has a similar severity of impact on the quality of life as Alzheimer's disease, Parkinson's disease, and stroke. The prevalence is estimated to affect over 50% of adult women in the United States and 1% to 39% of men worldwide.[1,2] The impact of urinary incontinence is far reaching and additionally affects family, loved ones, and caregivers.[3] The direct and indirect costs of urinary incontinence in the United States have been estimated to be $25 billion for patients over 65 years.[4,5] The direct financial cost in the United States including diagnostic procedures, treatments, medications, devices, and personal products has been estimated to be over $16 billion per year. Indirect costs to consider include complications and disabilities, loss of productivity, reduced quality of life, as well as caregiving and nursing-home placement.[3,6,7] The emotional cost, however, is often overlooked and it is important to consider how it affects not only the patients, but also their families. Common etiologies of urinary incontinence include age-related changes, pregnancy and childbirth, obesity, neurologic conditions, and certain medications. This article will review current treatment options for the various forms of incontinence, including osteopathic manipulative treatment. It will also incorporate the emotional consequences of incontinence and discuss the importance of a holistic approach to best serve these patients.

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CATEGORIES OF URINARY INCONTINENCE

Urinary incontinence is typically categorized by pathophysiology and clinical presentation. The six most common types are stress, urge, reflex, overflow, functional, and mixed.

Stress urinary incontinence (SUI) is the leakage of urine during periods of increased intra-abdominal pressure such as standing up or lifting, coughing, sneezing, or laughing. It arises from mechanisms that cause structural insufficiency to the pelvic floor or the internal and/or external sphincters.[8-10] Etiologies for SUI include urethral hypermobility, intrinsic sphincter deficiency secondary to neurologic compromise (including postprostate surgery), lack of tissue bulk from estrogen deficiency, and medications.[11-19]

Urge urinary incontinence (UUI) is when the patient experiences the sensation of needing to void immediately followed by leakage of urine. It is related to external cues such as hearing the sound of running water or washing hands. It shares overlapping symptoms

with overactive bladder arising from insufficient relaxation and overactivity of the detrusor muscle leading to uninhibited bladder contractions.[20] Fascial strains inducing tension across the detrusor muscle magnify overactivity.[21,22] Etiologies for these include idiopathic neurologic disorders, bladder abnormalities, increased and altered bladder microbiome, medications that increase bladder contractility, or obstruction.[18,19,23-26]

Overflow incontinence results from incomplete bladder emptying, with symptoms of urinary leakage or dribbling, weak or intermittent urinary stream, hesitancy, frequency, and/or nocturia. This form of incontinence can be thought of as a cup that has been overfilled.

Urine retention can be caused by detrusor muscle underactivity or bladder outlet obstruction (BOO). Causes include impaired contractility from smooth muscle damage from acute severe or chronic sustained overdistention of the bladder or fibrosis.[27] Fibrosis also restricts bladder distension, resulting in maximal filling with low volume. Additional causes of impaired contractility include low estrogen state, peripheral neuropathy (due to diabetes mellitus, vitamin B12 deficiency, alcoholism), spinal cord damage, or spinal detrusor efferent nerve damage from conditions such as multiple sclerosis and spinal stenosis.[28-31] Causes of BOO include external compression of the urethra from fibroids, Fowler's syndrome (urethral sphincter relaxation disorder), advanced pelvic organ prolapse, overcorrection of the urethral tract from prior pelvic floor surgery, regional scar formation, benign prostatic hypertrophy (BPH), constipation, and medications.

Reflex incontinence is the disruption of the neurologic pathways involved in controlling the bladder.[32,33] Afferent signals from the bladder sent through the pelvic nerve activate spinobulboreflex pathways that ascend to the brain.[34,35] Cortical efferent signals pass through the pontine micturition center (PMC) and stimulate sacral parasympathetic outflow to the bladder activating the detrusor and then inhibiting sympathetic urethral sphincter contraction.[36] Neuronal damage from multiple sclerosis, stroke, or Parkinson's disease removes cortical inhibition resulting in detrusor overactivity and decreased bladder filling.[37,38] Patients with spinal cord injuries proximal to the sacral segments also lose connections for voluntary voiding, but over time, afferent C-fiber nerves provide feedback-based autonomic micturition.[39,40] However, this emerged pathway does not maintain the sequential activation of sphincter relaxation and detrusor muscle activation. Therefore, bladder contraction occurs simultaneously with sphincter contraction and interferes with bladder emptying.[36] Similarly, damage to the pelvic (parasympathetic), hypogastric (sympathetic), or pudendal (somatic) nerves may result in reflex incontinence.[36]

While other types of urinary incontinence occur due to problems with the bladder or urinary system, functional incontinence is primarily caused by difficulties with mobility or cognition. It can affect people of all ages, but it is more common in elderly individuals and/or those with medical disabilities.[2] Common causes include arthritis, dementia, Parkinson's disease, stroke, or other limitations that make it difficult to move quickly or easily.[41-47]

Mixed urinary incontinence (MUI) most commonly combines urge and stress symptoms. While various mechanisms can result in MUI, it often results from conditions that impair function of both the bladder and urinary sphincter, such as neurologic disorders like spina bifida, spinal cord injury, prostate surgery, or pelvic radiation.[48] An interesting cascade can result from pudendal nerve impairment, causing pelvic floor laxity and uterine prolapse, leading to bladder-neck funneling, thus stimulating urethral afferent nerves to trigger reflex detrusor contractions.[49] The effects of an overflow stretched bladder can result in increased intra-abdominal pressure and activate stress incontinence. The overstretched bladder may also stimulate minor detrusor muscle contractions stimulating urgency.

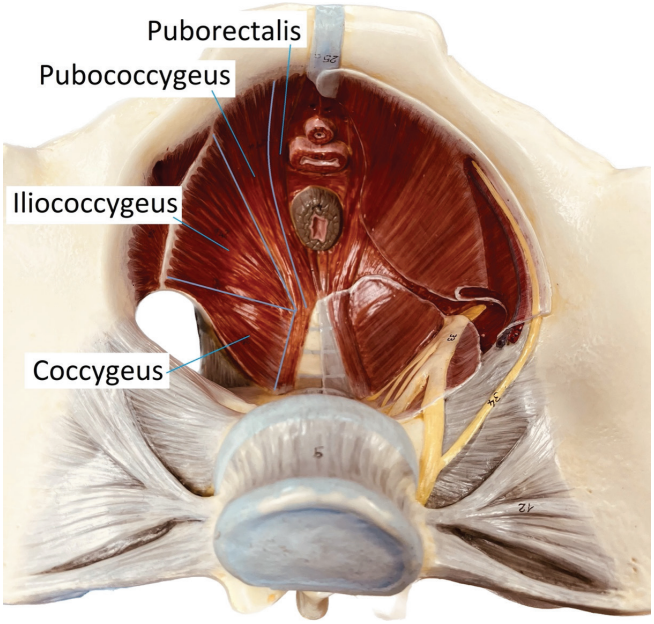
EVALUATION

A thorough history is the most important element for an accurate diagnosis. Validated brief questionnaires (available from *Ann Intern Med*, volume 145 on page 932-5) have sensitivities and specificities approaching 80% for distinguishing stress from urge incontinence in adult females.[50] Use of a voiding diary that tracks time of urination, estimated volume, symptoms associated with each void/leakage, any precipitating events, and large fluid intake, as well as measured urinary output, is often recommended.[51] It is important to recognize that the spectrum of gender expression may influence evaluations and treatment plans. A detailed genitourinary surgical and social history (including abuse) helps inform the physical examination and underlying causes. Considering the nature of the condition, patients are generally embarrassed and hesitant to fully disclose their experiences. Maintaining an open, compassionate, and nonjudgmental approach to this delicate and personal matter benefits accuracy of the information and completeness of the obtained history.

A complete history and physical examination can elucidate other contributing factors and diagnoses. The focused portion of the physical examination should include a complete urogenital, neurologic, and regional osteopathic structural evaluation. The osteopathic evaluation would include the regions of the lumbar spine, pelvis and sacrum, pelvic floor muscles and fascia, uterine/prostate mobility, and proximal lower extremities (Figure 1). Mobility and restriction of the pelvic structures can be superficially assessed via transabdominal palpation. However, they are best evaluated via transvaginal and transrectal examination, keeping in mind the importance of having a chaperone present.[52] Urodynamic tests have not been found to be very beneficial but may be warranted when the diagnosis is in question and/or treatment response is insufficient.[53]

FIGURE 1:

Model depicting muscles of the pelvic floor



Patients often present with a narrow perception of options. After failure or perceived failure of management, reviewing a treatment algorithm can help keep patients engaged in pursuing appropriate care.[54]

PSYCHOLOGICAL COMPONENT

Urinary incontinence is a very stigmatized condition that can prevent individuals from seeking medical care.[55,56] It is estimated that 75% of women affected by urinary incontinence have not sought medical care.[56-58] Men are half as likely to seek care compared to women.[59,60] Some research has found that Hispanic and Black women are less likely to seek medical treatment for urinary incontinence, but these findings have been inconsistent.[64,65] Friends and relatives of those affected, as well as physicians, may tend to normalize urinary incontinence following childbirth, also contributing to low levels of treatment-seeking among patients.[61] Another source of anxiety and hesitation is the cost of transportation and healthcare visits, as well as possible surgical interventions.[62,63]

The shame and embarrassment that often accompany urinary incontinence impairs people's ability to engage in day-to-day activities. The deterioration in quality of life for people with urinary incontinence is largely due to the secrecy that often surrounds it. This can lead to social withdrawal as they increasingly avoid activities outside their home where they may not have ready access to bathroom facilities.[75] Yip et al[75] found that patients with daily urinary incontinence reported more feelings of social isolation. The authors have observed family members and patients fabricating excuses to avoid outings or to leave activities abruptly to change clothes. This highlights the importance of addressing urinary incontinence in addition to any other causes of anxiety that may accompany leaving the house and attending social activities.

People with urinary incontinence report higher levels of anxiety and greater impairments to their quality of life with severity correlating with urinary incontinence symptom severity.[70,71] Depression is similarly correlated, with MUI having the strongest association.[72-74] These results illustrate the importance of considering both physical health and mental health when treating urinary incontinence.

Gascón et al[76] found that 41.9% of participants had sexual dissatisfaction, demonstrating the impact of incontinence on intimate relationships. The researchers hypothesized that fear of urine loss during intercourse partially contributed to low levels of sexual desire. This also suggests the influence of altered self-image with urinary incontinence.[77]

In some instances, it may become untenable for the patient to do activities of daily living due to their urinary incontinence. Thereafter, a caregiver is essential for obtaining food, assisting with toileting, and washing soiled clothes. However even if a patient has a caregiver, they may feel uncomfortable asking for help with these activities, especially when they have full mental capacity. These issues are potentially more challenging when the caregiver is the patient's adult child.[78]

It is imperative that physicians consider the potential association of incontinence with abuse. This abuse may include coercion, chastisement, and/or neglect.[79] Many factors are known to increase the risk of elder and patient abuse, including care dependence, low social support, and social isolation.[79] Urinary incontinence is associated with many of these factors, especially in older adults who are disproportionately affected by this condition where caregivers may not fully understand the patient's condition. Patient abuse may arise due to the emotional stress caused by the amount of physical labor required to care for the person with incontinence. There may be frustration with the nonlinear nature of recovery for those with urinary incontinence and even a belief that the incontinence is a conscious choice.[79] All of this highlights the importance of educating both the patient and the caregiver in order to help minimize the risk of elder and patient abuse.

MANAGEMENT OPTIONS

The following sections offer various treatment modalities as options for part of a holistic approach. Guidelines from various professional societies also offer additional support and may be cited when working to obtain resources and coverage for patients.[80-82]

Cognitive and Behavioral Interventions

Cognitive and behavioral interventions can reduce anxiety and/or depression and can influence incontinence as well. Although there has been very little research on the effectiveness of cognitive behavioral therapy (CBT) on the incidence of urinary incontinence, it has been shown to improve the quality of life for

patients with urinary incontinence.[91] Lifestyle changes that may help to reduce the symptoms of urinary incontinence include fluid management, weight loss, smoking cessation, and long-term moderate physical activity.[92-97] Patient home exercises may include bladder training and Kegel exercises; a 40% decrease in weekly incontinence was seen following six weekly bladder training sessions for functional or neurologic urinary incontinence.[97,98] Cavkaytar et al[99] found that Kegel exercises improved the symptoms of nearly 70% of participants with SUI and 40% of participants with MUI.

Pelvic Floor Physical Therapy

Pelvic floor therapy has been shown to be an effective treatment option for various types of urinary incontinence, including stress incontinence, urge incontinence, and mixed incontinence. Pelvic floor therapy includes exercises that strengthen the muscles of the pelvic floor and may reduce cystocele associated urethral malpositioning. Urethral sphincter strengthening may include Kegel exercises or other forms of resistance training.[100,101] Therapy may also include biofeedback techniques to help individuals learn how to correctly engage or relax their pelvic floor muscles and/or bladder. Therapy may also target incontinence-associated pelvic floor disorders such as uterine prolapse and pelvic tissue strain. Physical therapists specializing in pelvic floor disorders may provide better guidance on the most appropriate treatment options for an individual's specific needs.

Osteopathic Manipulative Treatment

Some patients are diagnosed with interstitial cystitis when their symptoms are due to myofascial imbalance.[102] Regional mechanical tension transmitted into the bladder fascia and muscle creates the possibility for both poor and/or early emptying. This tension can lead to urgency from increased nerve sensitivity or the tension-related misperception of a distended bladder. These concepts are also supported by other related studies: patients who have undergone treatment for prostatitis sometimes feel persistence of symptoms due to fascial strains resulting from their pathology.[21,22] Therefore, a quality physical examination should be performed to identify myofascial influences on urinary incontinence.[102]

Osteopathic treatment for urinary incontinence primarily focuses on improving tissue dysfunctions and imbalances of the musculoskeletal system. It addresses associated regional tissue disturbances with the goal of improving the function of the pelvis and associated regions. Osteopathic manipulative therapy (OMT) is a technique that often uses gentle manual forces to release restricted joint motion, and release problematic tension in the muscles and fascia. It may also be chosen to improve inhibited muscle tone, increase regional blood flow, and improve organ function. A systematic review of five clinical trials found OMT to be statistically significant and clinically relevant for women suffering from lower urinary tract symptoms (LUTS).[103]

When considering structures to evaluate and treat, the authors recommend checking regions that appear to be distant but have fascial connections directly to the bladder. These include the psoas and obturator internus muscles, and the medial and median umbilical ligaments. The pubococcygeus and iliococcygeus muscles directly support the bladder, and their fascia is contiguous with the bladder. The superficial and deep transverse perineal muscles influence pelvic pressure as well.

The following are techniques that would be beneficial to consider: sacral articulatory technique, myofascial release (MFR) of the pelvic region, doming the pelvic diaphragm, bladder MFR, uterus/prostate MFR, and psoas stretches.[104] For the deeper bladder-associated structures, transrectal or transvaginal MFR may be more effective for the pelvic floor, prostate, uterus, and ovary treatments. To augment the results, these can be performed bimanually with concomitant transabdominal pelvic-tissue engagement.

Sacral articulation: The dysfunction-related malpositioning of the sacrum and two halves of the pelvis will impact the tension of the muscles that cross the region. Additionally, when the positional relationships of the bones change, there is direct alteration of fascial tension due to the movement of boney fascial anchor points. Articulatory technique is an easy and effective option for sacral dysfunction correction. The hands can be stacked overlying the sacrum as demonstrated in Figures 2A and 2B. With slight anterior pressure, the sacrum can be rocked in any direction that is needed to engage restrictions to its motion. The restriction is then rhythmically engaged and disengaged at a rate of approximately 1 to 1.5 seconds each, or in time with the patient's breathing, until the palpation of motion restriction resolves.

FIGURE 2A:

Sacral ART demonstrated on a prone patient; physician's wrist splitting the top hand's third and fourth digits

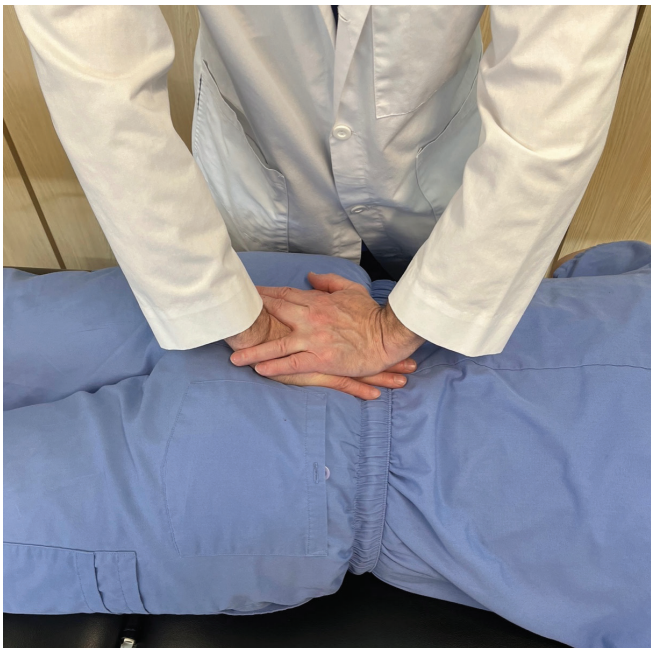
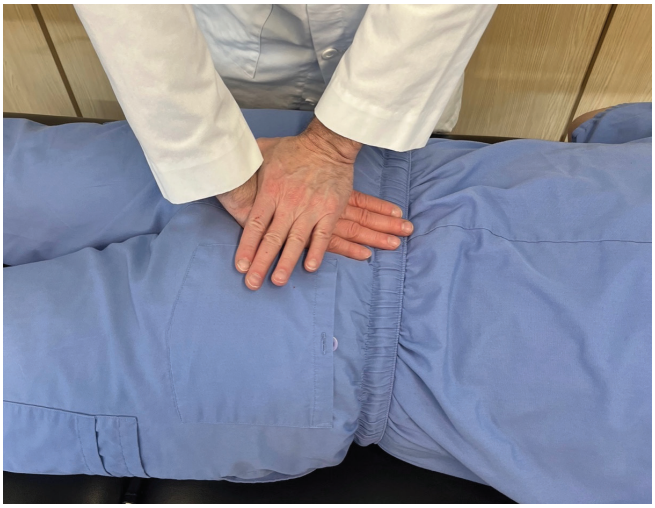


FIGURE 2B:

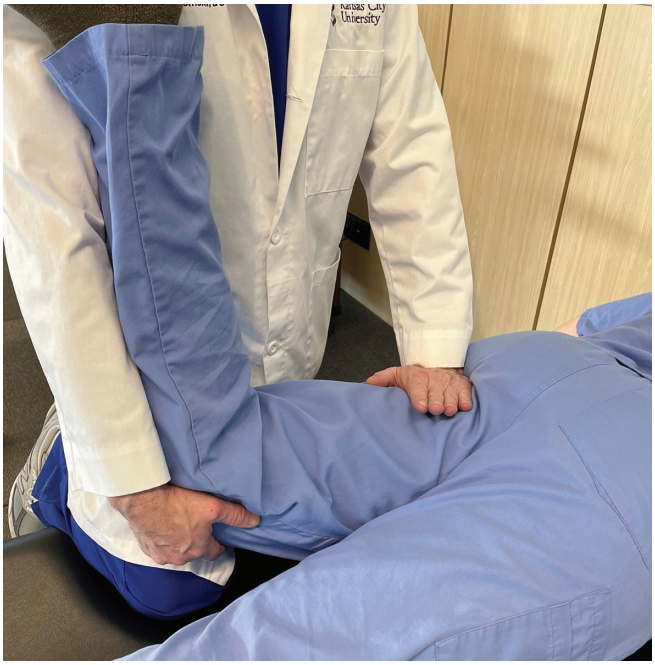
Alternate hand position for sacral ART demonstrated on a prone patient: physician's top hand lays perpendicular to the hand contacting the patient



Psoas stretches: While the bladder does not rest upon the psoas, portions of the ureter lay across this muscle, which may create a point of bladder traction as a result of tension within the psoas muscle and its fascia. The fascia of the inferior abdominal iliacus and psoas muscles is contiguous with the bladder and thereby also creates influence. While these psoas tissues may be evaluated and treated using a transabdominal approach, longitudinal stretching can be effective alone. A prone approach (Figure 3) can be utilized to gently engage the restriction to hip extension, with or without adding patient muscle contraction.

FIGURE 3:

Psoas stretch demonstrated on a prone patient



MFR to the pelvic bowl as an entire unit: The fascia of the pelvic bowl can be treated as a single unit. The superior aspects of that bowl can be influenced with suprapubic and lumbosacral junction hand placements as demonstrated (Figures 4A and 4B). Attention should be paid to the restrictions of motion of the fascia that is inferior and deep to those contact points. Thereafter, a direct restriction engagement or indirect tension balancing can be used to reduce resting tension and improve balance to the regional and bladder-related tissues.

FIGURE 4A:

MFR to the pelvic bowl demonstrated on a supine patient

FIGURE 4B:

MFR to the pelvic bowl demonstrated on a pelvic model

Transvaginal or transrectal direct stretching or MFR to the pelvic floor: Though the tissues can be influenced by distal contact, the authors find that greater and more immediate benefits are achievable through more direct palpation of the dysfunctional tissues. Several muscles of the pelvic floor are contactable through the rectum or vagina including the puborectalis (Figure 5A), pubococcygeus and iliococcygeus (Figure 5B), and coccygeus muscles (Figure 5C). Direct myofascial release is thereafter easily achievable through facing the treating palm towards the dysfunctional tissue, flexing the proximal or distal interphalangeal joint to 90° and then very gently pulling inferiorly until the muscles and fascia return to normal texture and tension.

FIGURE 5A:

Transrectal or transvaginal MFR to the pelvic floor demonstrated on a pelvic model with the physician's distal interphalangeal (DIP) joint bent to contact the puborectalis muscle

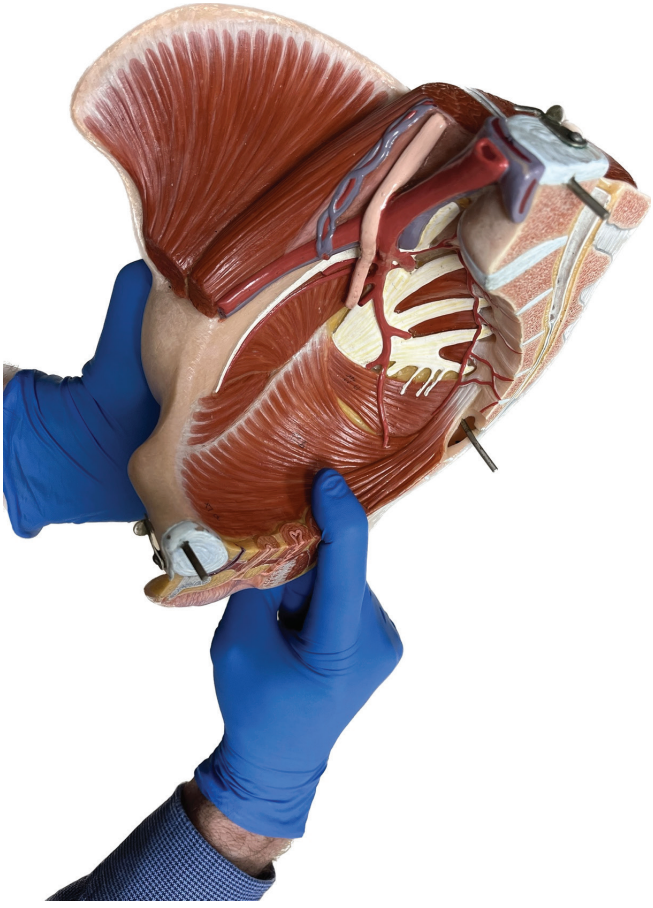


FIGURE 5B:

Transrectal or transvaginal MFR to the pelvic floor demonstrated on a pelvic model. with the physician's proximal interphalangeal (PIP) joint bent to contact the pubococcygeus and iliococcygeus muscles

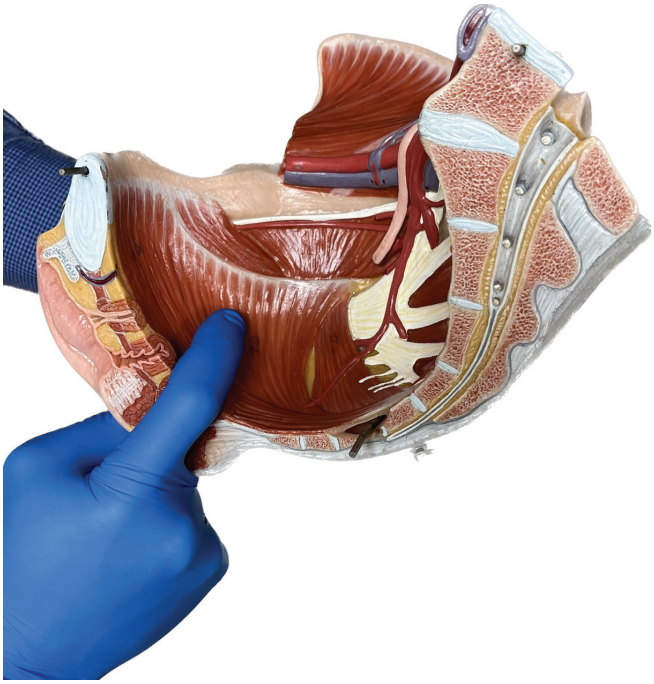
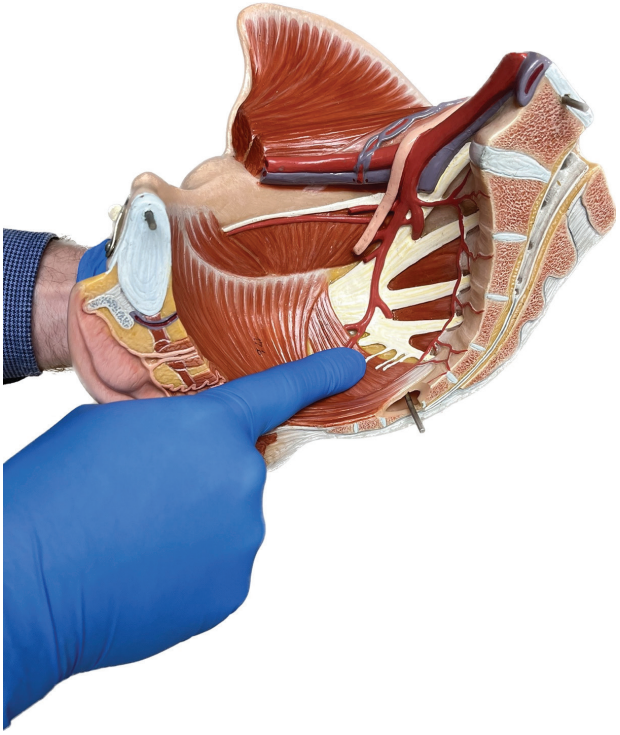


FIGURE 5C:

Transrectal or transvaginal MFR to the pelvic floor demonstrated on a pelvic model with physician contacting the coccygeus muscle



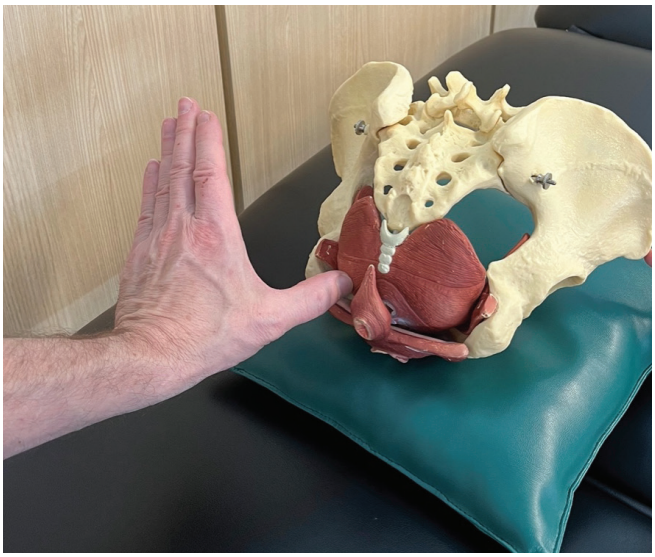
Doming of the pelvic diaphragm: Regional stretching of the pelvic diaphragm can be achieved externally from a prone, lateral recumbent, or supine position and is generally performed one side at a time. The center of the ischiorectal fossa is located approximately 1 inch posterior and medial to the most inferior aspect of the ischial tuberosity. Directly superior deep pressure is applied and held at this location (Figures 6A and 6B). Upon patient deep inspiration, the pelvic floor can be felt as it moves inferiorly, and a stretch is created around the physician contact point. During patient exhalation, deeper pressure is applied, and further anterior placement is achieved. Patient respiration is again used to create additional stretching. This pattern and sequence are followed until normal muscle and fascial tension are restored.

FIGURE 6A:

Doming of the pelvic diaphragm demonstrated on a prone patient

FIGURE 6B:

Doming of the pelvic diaphragm demonstrated on a pelvic model



Transabdominal MFR to the bladder: With transabdominal palpation, the bladder's lateral and superior fascia can be assessed (Figure 7), and with the same contact, can be treated with direct or indirect MFR.

FIGURE 7:

Transabdominal bladder MFR demonstrated on a supine patient

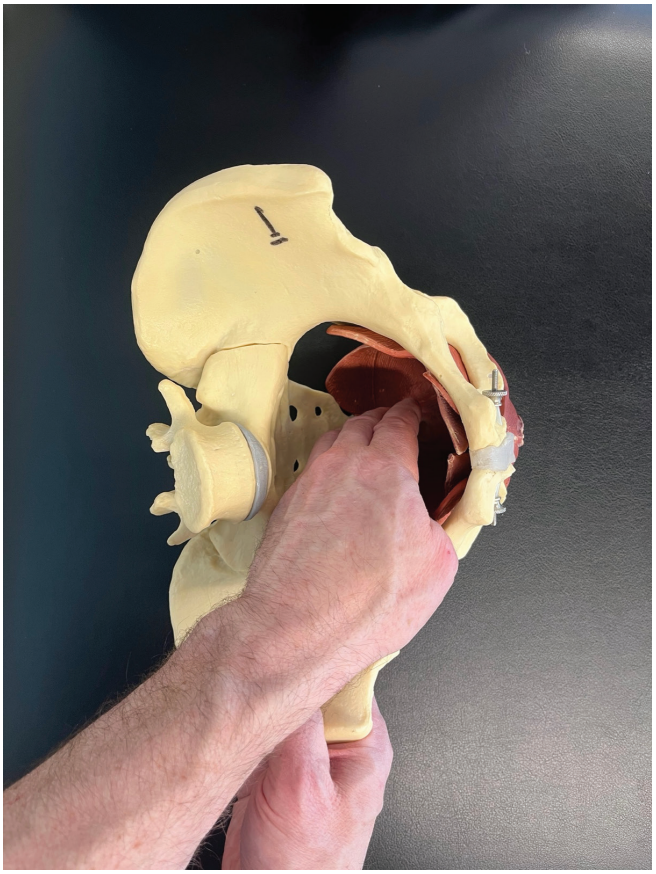
Transabdominal MFR to the prostate, uterus, or ovaries: At times, these structures represent the origin of fascial stress upon the bladder. While these structures and their supporting fascia are more easily palpated and directed for treatment through a rectal/vaginal (or with abdominal bimanual) approach, they can still be addressed transabdominally as demonstrated in Figures 8A and 8B. Thereafter, restriction of motion can be assessed and treated with direct or indirect MFR.

FIGURE 8A:

Transabdominal prostate MFR demonstrated on a supine patient

FIGURE 8B:

Transabdominal prostate MFR demonstrated on a pelvic model



Medications

The effectiveness of medication management in urinary incontinence can vary depending on the type and severity of the incontinence, as well as the individual patient. Medication is not always the best choice for everyone. For optimal results, pharmacology is best considered in combination with other approaches in this discussion. The following are some general considerations.

Antimuscarinic drugs, such as oxybutynin and tolterodine,[105-108] have been shown to be effective in reducing UUI in women and men.[105,108] These drugs relax the bladder detrusor muscle and allow for increased filling. This can decrease the frequency of the perceived need to urinate and reduce urgency of urination by decreasing neurologic signaling of bladder tension. Common side effects include dry mouth, constipation, and blurred vision and antimuscarinic agents are contraindicated with glaucoma.[109-112]

Beta-3 agonists, such as mirabegron, are effective in reducing UUI in both women and men. These drugs also provide detrusor muscle relaxation. They are generally well tolerated, but can cause common side effects such as headache, hypertension, and urinary tract infections.[113-115]

Estrogen therapy can be effective, particularly for UUI.[116] However, it may not be effective for all women and can have side effects such as vaginal bleeding and breast tenderness.[117,118] For those with estrogen contraindications, local estrogen cream may still be appropriate and effective.[119,120]

Alpha-adrenergic agonists, such as pseudoephedrine, can reduce SUI. This medication class tightens the muscles in the urethral sphincter and prostate, which can help improve control over urine flow. However, they can also have side effects such as increased blood pressure, anxiety, insomnia, and urinary retention.[121-125]

Assistive Medical Devices

There are several medical assistive devices that can be used in the management of urinary incontinence. Absorbent products include liners, pads, and briefs. They can be disposable or reusable, and come in a range of sizes and absorbency levels.[83] Catheters can be inserted into the bladder through the urethra or a small incision in the abdomen and can be used on a temporary or permanent basis, depending on the underlying condition. External collection devices are worn on the penis or around the vulvar labia to collect urine.[84] Urethral inserts are small tampon-like devices that are inserted into the urethra to help block the flow of urine. They can be used on a temporary basis, such as during physical activity or social events.[85] In patients with cystocele-related SUI, pessaries are inserted into the vagina to support the bladder and urethra.[86] These devices work well when minimal intervention is required or as part of a multifaceted approach.

Surgical Procedures

Patients with cystocele-related SUI may benefit from bladder taping or utilization of a sling or mesh implant to support the bladder and urethra.[87] Bladder augmentation or cystoplasty can be performed to increase filling volume.[88] Patients with urethral sphincter insufficiency may benefit from an artificial urinary sphincter.[89] Urethral stenosis may be treated with dilatation, urethrotomy, or urethroplasty.[90]

CONCLUSIONS

Urinary incontinence presents many challenges for patients, their support systems, and their care teams. It presents both complex diagnostic and treatment challenges that often require heightened awareness to the social, emotional, psychological, and physical components. This article highlights the importance of a biopsychosocial approach to care, which often requires regular re-evaluation of initial therapy and the engagement of patients in the follow-up process. This can be enhanced by reassurance that options of additional or alternative modalities are available. With compassion and persistence, effective management may be found that can greatly improve patients' lives.

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REVIEW ARTICLE

DIAGNOSIS AND MANAGEMENT OF ECTOPIC PREGNANCY: A COMPREHENSIVE REVIEW FOR THE OSTEOPATHIC PHYSICIAN

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KEYWORDS

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ABSTRACT

Ectopic pregnancy (EP) is a serious obstetric complication that can be life-threatening. Age-adjusted incidence of ectopic pregnancy is roughly 15.8 pregnancies per 1000.[1] Despite this seemingly low value, EP remains one of the largest contributors to maternal mortality in the first trimester. While some patients require urgent surgery, there is a role for medical and conservative management in patients who are hemodynamically stable. In cases where medical or conservative management is appropriate, family physicians can choose to manage this condition with collaboration from the patient's obstetrician. Particularly for residents in the 50% of US counties with no obstetrician,[2] it is imperative for family doctors to diagnose EP quickly and accurately.

BACKGROUND

Ectopic pregnancy (EP) is defined as any pregnancy in which a fertilized ovum implants outside of the uterus.[3] Most EPs implant in the fallopian tube, accounting for over 90% of EPs. Implantation can occur outside the fallopian tube as well. Roughly 3% of EPs implant on an ovary, 2% to 4% are classified as interstitial, and the remaining 1% are classified as abdominal, cervical, intramural, or cesarean section scar EPs.[4] Heterotopic pregnancy is an extremely rare cause of EP in which intrauterine pregnancy and EP occur simultaneously; this accounts for an estimated 1 in 30,000 pregnancies.[5] Due to the lack of national surveillance in the United States, prevalence data are difficult to ascertain; however, according to the American College of Obstetricians and Gynecologists (ACOG), it is estimated that EPs account for 2% of pregnancies in the United States. Despite only representing a small percentage of pregnancies, ruptured EPs are a major health concern and a leading contributor to maternal mortality.[6] Currently, EPs are responsible for up to 10% of pregnancy-related deaths and are the number one contributor to maternal mortality in the first trimester.[7]

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The most significant risk factors for EP include previous tubal surgery, sterilization, previous EP, history of pelvic inflammatory disease, and current use of an intrauterine device.[8] History of EP is one of the strongest risk factors, as patients with a previous EP have a 10% to 20% increase in risk of recurrent EP in future pregnancies.[9,10] Those with previous tubal surgeries are more likely to go on to develop a future EP with an odds ratio of 8.8.[11] Lack of risk factors should not be considered reason to exclude EP as a diagnosis, as up to 50% of patients presenting with EPs have no clinical risk factors.[12] Classic symptoms of EP are not sensitive or specific for EP alone, and patients may present with a variety of signs and symptoms. The most common presenting symptoms include abdominal pain and vaginal bleeding, while other symptoms such as amenorrhea, breast tenderness, nausea, syncope, or frequent urination may also be seen.[13-15] A syncopal episode in a patient with a possible EP could indicate the ectopic has ruptured and that the patient may need urgent surgical intervention.[7] The symptoms are not specific to EP, thus the differential may be large. The differential diagnosis includes appendicitis, urinary tract infection, ovarian torsion, normal intrauterine pregnancy (IUP), implantation bleeding, spontaneous abortion, or tubo-ovarian abscess.

In the primary care setting, the key differentiation among patients with a possible EP is hemodynamic stability. Those that are hemodynamically stable and at minimal risk for rupture may be worked up and treated in the outpatient setting by a primary care provider. Unstable patients with signs such as hypotension and tachycardia potentially need surgical intervention, will require urgent inpatient care, and should be transferred to the emergency department.

DIAGNOSIS

A hemodynamically stable female patient of reproductive age presenting to their primary care physician with signs and symptoms of an EP should first receive a urine pregnancy test in the office. If the urine pregnancy test yields a positive result, stable patients in whom there is suspicion for EP may continue to be worked up in the outpatient setting. The next step is to obtain an urgent quantitative beta-human chorionic gonadotropin (β-hCG) and perform transvaginal ultrasonography.

QUANTITATIVE β-HCG

Quantitative measurements are used to evaluate β-hCG growth trends and determine if the baseline value is above or below the discriminatory value. The discriminatory zone is a serum hCG value at which an IUP can be visualized. Each institution may have slight variations in their exact definition of discriminatory value, but in most cases, it is a β-hCG level between 1500 and 2000 mIU/mL.[7,12,16] If the quantitative β-hCG level is below the discriminatory level, the recommendation is to follow up with a repeat quantitative β-hCG in 48 hours. In normal IUP, the β-hCG level will typically double every 48 hours, but viable pregnancy can see β-hCG increase as little as 35% in 2 days.[17] In 2 days, if the repeat β-hCG results are now >1500 mIU/mL, a transvaginal ultrasound can be completed. If the level is still <1500 mIU/mL and declining, spontaneous abortion should be considered. If β-hCG is <1500 mIU/mL and increasing, the serum β-hCG should be repeated in another 48 hours.[18]

ULTRASONOGRAPHY

Following ACOG guidelines, any reproductive-aged female showing signs and symptoms of EP with positive serum β-hCG requires a diagnostic transvaginal ultrasound.[5] Generally, ultrasound cannot be used to rule out an EP, but results can confirm the diagnosis.[19,20] There are three possible results of transvaginal ultrasonography: confirmed EP, indeterminate IUP, or normal IUP.[8,12,18] Early in the course of an IUP, an ultrasound finding referred to as the "double ring sign" may be appreciated, which refers to the decidua parietalis and decidua capsularis forming around the gestational sac.[21] While this cannot rule out an EP, it does provide supportive evidence favoring an IUP. Detection of ectopic cardiac activity is the most accurate result for confirming an EP, with a likelihood ratio >100.[18]

It is best to use the results of both quantitative β-hCG and transvaginal ultrasonography rather than relying on one test independently. A quantitative β-hCG >1500 and transvaginal ultrasonography showing no IUP has a sensitivity ranging from 67% to 100% and specificity of 100% for diagnosing EP.[18]

MANAGEMENT

Once an unruptured EP has been identified, there are three potential methods to manage the EP: expectantly, medically, or surgically. Options become limited with additional complications such as ruptured EP, clinical instability, or hemodynamic instability—all situations which would require emergent surgical intervention. Assuming no complications exist and that the patient is stable, the decision to treat the patient medically or surgically should be made by the patient with information provided by their physician.[12]

Additionally, it is important to note that consideration of patient preferences and limitations may play a role in treatment options. Medical management with methotrexate is less expensive than surgery; in addition to being less invasive. A retrospective review found the direct costs of methotrexate-treated EPs were roughly 24% of the direct costs for treatment using laparoscopy.[22] However, β-hCG levels typically fall faster with surgical intervention.[23] Some studies indicate a higher success rate with surgical management as well.[18] Surgery may be a better option for patients who have little access to medical facilities for lab testing or for those with financial insecurity, and especially when there is concern regarding patient compliance for follow-up.

Expectant Management

Despite the limited patient population eligible for expectant management, this treatment option should not be overlooked as some EPs may resolve on their own.[24] Expectant management has been shown to be effective only in those patients with a low or decreasing β-hCG level.[25,26] However, the exact cutoff has been debated, with some studies citing a safe level of <1000 mIU/mL,[25] and others reporting efficacy as high as 3000 mIU/mL.[26] Patients who present with an initial β-hCG level of 200 mIU/mL or less have spontaneous resolution of the EP 88% of the time.[12] Most importantly, there should be a measured plateau or decline of β-hCG levels to safely consider expectant management.[27,28] Patients should be educated on the risks of expectant management and the warning signs of EP rupture; the treating physician should ensure the patient has access to emergency medical treatment and knows how to access this care in the case of an emergency.[27]

To ensure patient safety, the patient must be willing to return for close follow-up monitoring to confirm resolution of the EP. Follow-up appointments with the patient managed expectantly should begin at the 48-hour mark to reassess β-hCG levels and patient symptoms. If β-hCG levels are declining from baseline and the patient remains asymptomatic, expectant management may be continued. Once β-hCG decline has been proven, expectant management can continue as weekly quantitative β-hCG measurements.[27] Each week, measurements are expected to decline by at least 15%. If this is not seen, the patient should undergo follow-up in 48 hours and cessation of expectant management should be considered.[26] These weekly follow-up appointments should continue until β-hCG is no longer detectable.[18] At any point during expectant management, if the β-hCG level has risen or the patient is now experiencing symptoms of potential rupture, expectant management should no longer continue.[26]

Medical Management

Medical treatment of an EP can be managed by many specialties, including a primary care or family physician.[12] Those patients who do not qualify for expectant management as described above can often be medically managed using methotrexate.[28] Patients should be treated with methotrexate if they have a positive pregnancy test and a confirmed extrauterine pregnancy. These patients should be hemodynamically stable and should not show signs of imminent decline, impending or active fallopian tube rupture, or other complications that can be seen with EPs.[12] Other relative contraindications include embryonic cardiac activity, size greater than 4 cm, and maternal liver disease.[29] A baseline complete blood count (CBC) and comprehensive metabolic panel (CMP) should also be acquired to rule out contraindications to methotrexate such as renal insufficiency, liver disease, anemia, or leukopenia.[12,29] Rh D factor status for the patient should be determined and, if the patient is Rh D-negative, administration of Rh D immune globulin is required.[30] As always, a complete medical history should be taken to ensure safe use as described by the Food and Drug Administration (FDA) drug label.[29]

Once the decision to pursue medical management has been made, the physician must decide between a single- or multiple-dose regimen of methotrexate.[12] A single dose of methotrexate has been shown to be more effective in patients with an initial β-hCG level <5000 mIU/mL, with increased treatment failure rates above this β-hCG threshold.[31,32] Linear increase in risk of treatment failure is seen with increasing β-hCG, as risk of failure increases by 0.12% for each unit increase in β-hCG.[32] Women who received single-dose methotrexate treatment reported fewer side effects compared to those who received multiple-dose methotrexate treatment.[28] Additionally, better fertility outcomes were reported when women received a single dose as opposed to a multidose regimen.[33]

While there are several medications used to medically terminate pregnancies, methotrexate is the only medication approved by the FDA for treatment of EPs.[34] Methotrexate works as a folate antagonist, which prevents the rapidly dividing cells of the embryo from synthesizing new DNA, thereby preventing growth. The cells are no longer able to divide, so the pregnancy will not progress.[29] Methotrexate should be administered intramuscularly, with the hope that this is a one-dose treatment, with a dosing of 1.0 mg/kg or 50 mg/m^2.[30] The patient should return for β-hCG measurement in 2 to 4 days; if the level has risen, or has fallen <15% by day 4, an additional dose of methotrexate should be administered at the same dose.[30] If a second dose is needed, patients should return for β-hCG on days 7, 11, and 14.12 If a β-hCG decline >15% is noted at any point, no further methotrexate doses are indicated and the patient should return for weekly monitoring as described with expectant management.[12,30] Alternative to intramuscular delivery, oral methotrexate may also be used as a 4-day treatment course on days 0, 2, 4, and 6 at 1.0 mg/kg.[30] Patients should return for weekly monitoring until the EP has fully resolved, demonstrated by a nondetectable β-hCG.[18] This typically occurs in 4 to 6 weeks but can take up to 8 weeks to confirm success.[14,18]

Successful treatment with methotrexate is not guaranteed, and the success rate is estimated to range from 70% to 95%. The factor that most significantly indicates likelihood of methotrexate failure is higher baseline β-hCG level.[5,35] In the case that β-hCG does not diminish appropriately by day 7, surgical management should be considered and discussed with the patient. Conversion to surgical management of the EP should always be considered as an alternative and should be pursued if the patient shows signs of decline, hemodynamic instability, or new symptoms.[23]

Surgical Management

With most EPs, surgical intervention is not necessary for successful treatment. Typically, only 10% of patients will need surgical management.[36] However, it may be critical in saving the patient's reproductive function and potentially their life. Typically, surgical management of an EP is implemented when the patient fails medical management or is unable to complete medical management due to other medical contraindications such as hemodynamic instability.[37]

There are, however, certain situations that may indicate immediate surgical referral for patient safety. If an ultrasound demonstrates a visualizable embryo outside of the uterus with clear cardiac activity, the patient should receive urgent surgical management to prevent potential morbidity.[12]

Surgical management typically involves a salpingectomy (complete removal of the fallopian tube) or a salpinx-sparing salpingotomy (removal of gestational sac only). The latter is preferred, especially in cases in which preservation of fertility is a concern.[2] Despite surgical management, there is a chance of persistent EP; it is estimated that in nearly 8% of salpingostomy cases, patients still have the EP postoperatively.[2,11] Surgical management also requires follow-up to monitor β-hCG levels to confirm decline until no longer detected. However, β-hCG levels typically return to undetectable levels after surgery.[37]

OSTEOPATHIC PERSPECTIVE

Providing care to rural and underserved communities is a mainstay of osteopathic values and many patients may experience barriers to receiving proper maternity care. Data from ACOG show there are only 5.39 residency-trained ob-gyns per 10,000 reproductive-aged women, and almost one-half of counties in the United States do not have any ob-gyns.[2] Family physicians are essential in bridging gaps in care in these underserved areas. Family doctors practicing in rural areas provide 42% of total healthcare and, despite decreasing numbers, also provide the majority of obstetrical care.[38] Women in small rural towns have an 80% higher maternal mortality rate than those in urban locations. With EP continuing to be a top cause of maternal death, the role of family physicians in diagnosing and medically treating EPs is imperative.[39]

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BRIEF REPORT

OSTEOPATHIC MANIPULATIVE TREATMENT FOR CHRONIC BRONCHIECTASIS: A CASE REPORT

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KEYWORDS

Bronchiectasis

Lymphatics

Obstructive lung disease

Inflammation

Pneumonia

ABSTRACT

Bronchiectasis is an uncommon chronic obstructive lung disease caused by chronic airway inflammation leading to irreversible bronchial dilation and destruction of medium-sized airways. In this case, we treated a bronchiectasis patient with a combination of osteopathic manipulative treatment (OMT) techniques including myofascial release and lymphatic techniques as adjunctive treatments in addition to other pharmacologic and nonpharmacologic therapies; OMT was shown to improve subjective symptoms of airway congestion as well as reduce frequency of emergency room visits and hospital admissions for this patient.

INTRODUCTION

Bronchiectasis is a chronic lung disease characterized by persistent lifelong widening of bronchial airways and weakening of mucociliary transport mechanism, owing to repeated infection, thus contributing to bacterial invasion and mucus pooling throughout the bronchial tree due to neutrophilic invasion.[1] Associated comorbid illnesses include bronchitis, hemoptysis, pleuritis, and recurrent bacterial pneumonia. Chronic airway inflammation appears to have an inhibitory effect on lymphangiogenesis contributing to lymphatic blockage and stasis. The pathophysiology of lymphatic vasculature changes in the setting of lung disease is not well understood yet.[2] By directing osteopathic manipulative treatment (OMT) to dysfunctional organ systems, movement of lymphatic fluids throughout the body is encouraged using noninvasive passive soft-tissue movements and release of underlying tissue restrictions. In 2010, Noll et al[3] published "Efficacy of Osteopathic Manipulation as an Adjunctive Treatment for Hospitalized Patients With Pneumonia: A Randomized Control Trial." Known as the Multicenter Osteopathic Pneumonia Study in the Elderly (MOPSE), this study showed a significant reduction of length-of-stay, decreased duration of intravenous antibiotics, and decreased respiratory failure or death when OMT was performed. MOPSE definitively laid the groundwork for more research in the area of respiratory diseases and OMT.

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CASE REPORT

A 59-year-old female presented to her osteopathic physician with complaint of recurrent chest congestion and shortness of breath. Her symptoms have been chronic and ongoing for several years. Her medical history included bronchiectasis, reactive airway disease, anemia of chronic disease, morbid obesity, diabetes mellitus type 2, immunoglobulin (Ig)G subclass deficiency, chronic pain syndrome, generalized anxiety disorder, and recurrent pneumonia and *Mycobacterium avium* infection. She had recurrent hospitalizations due to her symptoms: two admissions in 2021, four admissions in 2020 (when she started receiving OMT), six admissions in 2019, andseven admissions in 2018. Her past surgical history was notable for laparoscopy, carpal tunnel surgery of bilateral wrists, and frenectomy. Her family history was noncontributory to her current symptoms. Her social history is unremarkable for alcohol, tobacco, or drug use. Her medications included tramadol extended-release 100 mg, two tablets daily; alprazolam 0.5 mg, one tablet every 6 hours as needed for anxiety; benralizumab 30-mg injection every 8 weeks; acetazolamide 150 mg, one tablet daily; and armodafinil 150 mg, one tablet daily. Her vital signs at the most recent visit were unremarkable (116/72 mm Hg, 97.9 °F, 79 bpm, 18 rpm, 97% O_2 saturation in room air, body mass index [BMI] 28.3 kg/m^2). Cardiovascular examination showed +2 left lower extremity edema. Neurologic examination revealed cranial nerves 2-12 grossly intact, 5/5 muscle strength, 2/4 deep tendon reflexes of the bilateral upper and lower extremities, and notably reduced sensation of the left lower extremity. Pulmonary function testing and other laboratory data and imaging were not available from the patient.

Management of this patient was multifaceted and employed chest physiotherapy to improve airway clearance, pharmacologic treatments such as benralizumab (an immunosuppressant) and acetazolamide (correcting underlying metabolic alkalosis) to minimize recurrent infection and to maintain airway patency, and OMT. As described previously,4 a comprehensive examination of 10 body regions of somatic dysfunction—head, cervical, thoracic, lumbar, sacrum, pelvis, lower extremity, upper extremity, ribs, and abdomen—was performed. Affected areas included cervical, thoracic, ribs, and lower extremity regions. The following somatic dysfunctions and structural abnormalities were identified over the course of several visits: 1) C2-C7 RRSR, 2) T1-T5 NSLRR, 3) left rib 1 exhalation dysfunction, 4) right ribs 2-5 inhalation dysfunction, 5) left ribs 6-10 exhalation dysfunction, 6) right ribs 11-12 exhalation dysfunction, and 7) +1 to +2 bilateral lower extremity edema. Osteopathic manipulative medicine was employed as an adjunctive treatment modality for this patent's somatic dysfunction and bronchiectasis, in addition to pharmacologic therapies. Pharmacologic therapy was provided by the patient's primary care physician and pulmonologist. Treatments were performed twice monthly for 2 years. The autonomic dysfunctions were treated with OA release to normalize parasympathetic tone of the vagus nerve and rib raising.[3] Biomechanical considerations were directed toward the thoracic and lumbar spines and included myofascial release of paraspinal musculature as well as muscles of exhalation, specifically targeting the scalenes, pectoralis minor, serratus anterior, latissimus dorsi, and quadratus lumborum. Lymphatic techniques were directed toward symptomatic relief. Fascial torsions owing to hypertonic musculature can directly decrease lymphatic flow through the fascia by compressing lymphatic vessels. Other treatment techniques used include the splenic pump, effleurage, and petrissage. Treatment techniques were applied in a cephalad to caudad fashion.

Frontal/temporal/mandibular drainage, mandibular distraction (the Galbreath technique), pectoral traction, Miller thoracic pump, pedal pump (also called the Dalrymple pump), and lower extremity effleurage/petrissage treatment techniques were performed.[5-7]

Use of OMT in this patient was shown to subjectively improve her symptoms of airway congestion as well as objectively reduce the frequency of emergency room visits and hospitalizations. However, despite subjective relief and decreased hospitalizations, objective improvement via pulmonary function testing[8] (PFT; Figure 1)—specifically FEV1/FVC—did not consider the subjective and symptomatic benefits derived from OMT. These treatments provided relief and were targeted to affected autonomic, biomechanical, and lymphatic dysfunctions.4 FEV1/FVC represents the proportion of the patient's vital capacity that they are able to expire in the first second of forced expiration compared to the full forced vital capacity.

FIGURE 1:

Pulmonary function test data

Component Ref Range & Units	8 d ago (3/2/22)	1 yr ago (11/25/20)	2 yr ago (7/30/19)	2 yr ago (3/25/19)	3 yr ago (1/29/19)
FVC Pred 0.05 - 9.99 Liters	3.06	2.96	3.10	3.14	3.01
FVC Pre 0 - 12 Liters	3.13	3.10	2.78	2.36	2.69
FVC %Pre Pred 0 - 300 %	103	105	90	75	89
FEV1 Pred 0.05 - 9.99 Liters	2.41	2.35	2.46	2.50	2.40
FEV1 Pre 0 - 12 Liters	2.71	2.69	2.44	2.07	2.32
FEV1 % Pre Pred 0 - 300 %	112	115	99	83	97
FEV1/FVC Pred 1 - 99%	79	80	80	80	80
FEV1/FVC Pre 0 - 12%	86	87	88	88	86
FEF25-75% Pred 0 - 12 L/sec	2.22	2.21	2.33	2.35	2.29
FEF25-75% Pre 0 - 12 L/sec	4.23	4.34	4.15	3.29	3.60
FEF25-75% %Pre Pred 0 - 300%	190	197	178	140	157
FEF50% PRE 0 - 12 L/sec	5.03	5.8	6.44	6.02	4.68
FET100% PRE Sec	7.94	9.04	8.16	7.22	8.39

DISCUSSION

Bronchiectasis is a chronic lung disease characterized by persistent and lifelong widening of the bronchial airways and weakening of the mucociliary transport mechanism, owing to repeated infection. It contributes to bacterial invasion and mucus pooling throughout the bronchial tree due to neutrophilic invasion including *H. influenzae* and *P. aeruginosa*. Other contributory pathogens include *Moraxella catarrhalis*, *Streptococcus pneumoniae*, *Staphylococcus aureus*, and other Gram-negative bacteria and, less commonly, nontuberculosis *Mycobacteria species*.[9,10]

The lymphatic system allows for the flow of nutrients toward, and waste away from, individual cells. It is a passive system whose function can be influenced by external forces. Lymph is a substance that leaks out of arterial capillaries, into the interstitium, and into single-cell lymphatic vessels. Primary cells of lymph are lymphocytes, which are clear in color, contain proteins, salts, and large particles (such as bacteria and viruses) prior to filtration through a lymph node or organ.[11]

Lymphatic channels perfuse most tissues of the body. Simple squamous epithelium cells of lymphatic capillaries allow great permeability of fluids from the interstitium back into the lymphatic system. Capillaries flow into larger channels, which eventually drain into the venous system via the right lymphatic duct or thoracic duct. The right upper extremity, right chest, and right upper quadrant of the abdomen drain via the right lymphatic duct. The remaining portions of the body drain into the thoracic duct.

The functions of the lymphatic system include maintaining fluid balance in the body, purification and cleansing of tissues, defense, and nutrition. Direct external pressure on lymphatic channels increases the flow of lymph. The mechanism of lymphatic flow occurring through lymph channels is influenced by three factors: 1) interstitial fluid pressure: forces fluid into lymphatic capillaries, 2) intrinsic lymphatic pumps: channels with valves similar to the venous system that form sections that function independently and smooth muscle in the channel walls that contract when distended, allowing one to pump to the next, and 3) ionic gradients, whereby fluid goes toward higher concentrations of ions.

Specific functions with regard to applications to bronchiectasis include production of immunologic cells and antibodies, clearing of waste from fighting infections, and filtering toxins. Clearance of exudates and inflammatory mediators form the interstitium results in facile fluid movement in the lymphatics. Remnants of proinflammatory mediators lead to persistent inflammation and delayed healing. Other offending agents include fibroblasts, plasma proteins, platelets, transforming growth factor-β (TGF-β: transforms present monocytes into macrophages), and fibroblast growth factor.[7] Continued presence of these substances leads to scarring, fibrosis, and chronic inflammation. Lymphatics are the predominant means to remove inflammatory exudates and promote the healing phase of the inflammatory process.[7]

Diaphragms are muscular and fascial structures giving shape and form to the body.[7] The eight diaphragms that contribute to lymphatic flow and its obstruction are the diaphragma sellae, tentorium cerebelli, suboccipital diaphragm, thoracic inlet diaphragm, respiratory diaphragm, pelvic diaphragm, popliteal diaphragm, and plantar fascia. The respiratory diaphragm is an important external pump. Respiration causes lymph to move via pressure gradients, by which negative pressure causes fluid to move toward it and positive pressure pushes it away. The pelvic diaphragm works in synchrony with the abdominal diaphragm to maximize flow of interstitial fluid and lymph, relaxing with contraction of the abdominal diaphragm and springing back with exhalation, producing a mechanical pump for lymphatics in the pelvis and lower abdomen.

Prior to treating any lymphatic issues (i.e., edema), the diaphragms described previously must be opened. The ideal order is opening the: 1) thoracic inlet, 2) respiratory (thoracoabdominal) diaphragm, 3) pelvic (presacral) diaphragm, and, 4) popliteal diaphragm. Once the diaphragms are open, fluid mobilization can be performed. This order is utilized to promote maximum drainage of lymphatic flow clearance and to prevent any additional congestion. Failure to open the diaphragms in this manner can be counterproductive and exacerbate an already underlying problem by causing backup of fluids that cannot flow through the preceding closed diaphragm.

The consequence of decreased lymphatic function is edema: buildup of excess interstitial fluid caused by either too much fluid going into the interstitium or too little getting out. Conditions that overload the interstitium override the absorptive capabilities of the lymphatic system. Excessive interstitial fluid increases interstitial pressure and collapses lymph capillaries resulting in further edema and congestion. Edema also causes dilation of lymph capillaries causing valves not to function and the intrinsic pump to shut down. Conditions of increased venous pressure are associated with increased capillary filtration rates and tend to produce edema. These include congestive heart failure (both systolic and diastolic),incompetent heart valves, venous obstruction (i.e., bronchiectasis), and gravity. Conditions that decrease osmotic gradients across the capillary, such as cirrhosis and starvation, decrease lymphatic function and lead to edema.

Inadequate drainage, from conditions such as posttraumatic or postsurgical scarring, may lead to edema. Edema causes compression, not only of lymphatic structures but also vascular and neurologic structures, leading to decreased functional ability. Edema causes further fluid stasis, which causes buildup of waste products and decrease in nutrient delivery, and can further affect bioavailability of drugs and hormones, decreasing their respective functions.

Treatment goals of lymphatic techniques include opening myofascial pathways at transitional areas (i.e., diaphragms) of the body, normalizing diaphragmatic motions, increasing pressure differentials to augment fluid flow beyond normal levels, and mobilizing targeted tissue fluids into the lymphatic system.[4-7] Emphasis on removing impedances to lymphatic flow and improving and augmenting the flow of lymph is apparent. Techniques that remove somatic dysfunction, which causes decreased efficiency of respiration, diaphragmatic excursion or motion, or fascial torsion, will improve lymphatic function. The goal is to have a balanced well-functioning system without edema. The low-pressure lymphatic system is dependent on motion and drainage of lymph for optimal function. As a result of OMT, the patient reported significant symptomatic relief at subsequent visits as well as decrease in the number of emergency room visits and subsequent hospital admissions.

This case is applicable to the osteopathic family physician in that treatment techniques used to manage symptoms—in addition to chest physiotherapy and pharmacologic treatment—do not require a large amount of time to perform and can be done in the office. Additionally, use of OMT in the treatment of this patient may allow for longer intervals between follow-up appointments. The techniques discussed here will also have application to other respiratory illnesses such as chronic obstructive pulmonary disease (COPD), reactive airway disease, and other pulmonary diseases characterized by recurrent mucus plugging and airway obstruction.

CONCLUSION

Bronchiectasis is a chronic lung disease characterized by persistent and lifelong widening of bronchial airways and weakening of the mucociliary transport mechanism, owing to repeated infection. Bronchiectasis contributes to bacterial invasion and mucus pooling throughout the bronchial tree.[1] Recurrent inflammation, due to inflammation and mucus trapping, results in airway dilation and dysfunction of the mucociliary system. Sequelae include persistent cough, dyspnea, and excessive sputum production. Many of these symptoms are largely due to mucus trapping and dysfunction of lymphatic channels in lung parenchyma. OMT provides a passive mechanical method to promote mechanical airway clearance by disrupting mucus plugging and occlusion within the lungs. By considering the intersectionality of these simultaneous conditions in addition to a patient's goals of care (maximize functional capacity and decrease hospitalization), OMT directed at autonomics, biomechanics, and lymphatics/circulation can aid in achievement of this patient's treatment goals.

TABLE 1:
Summary of myofascial release (MFR) and lymphatic techniques

TECHNIQUE	DIRECT OR INDIRECT	ACTIVE OR PASSIVE	MECHANISM OF ACTION	ABSOLUTE CONTRAINDICATIONS*	RELATIVE CONTRAINDICATIONS*
MFR	Direct or indirect	Passive	• Light, moderate, or heavy force that engages fascia vs deeper tissue with constant pressure; piezoelectric changes relax and release restricted tissues (direct) • Guiding fascia along the path of least resistance until free movement is achieved (indirect)	• Treatment directly over fracture or dislocation • Serious vascular compromise • Local malignancy or infection	• Vascular compromise • Malignancy • Infection • Severe osteoporosis or osteopenia • Acutely injured muscles • Patient intolerance
Lymphatics (extension of MFR)	Direct	Passive	• Mechanical compression via physician's force leads to mobilization of lymphatic fluid	• Necrotizing fasciitis	• Acute hepatitis • Mononucleosis Malignancy • Deep venous thrombosis • Severe heart failure

*Patient refusal or lack of somatic dysfunction is always a contraindication.

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BRIEF REPORT

PREVOTELLA DENTICOLA DACRYOCYSTITIS AND ABSCESS IN A CHILD WITH GOLDENHAR SYNDROME

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KEYWORDS

Prevotella denticola

Dacryocystitis

Goldenhar syndrome

Preseptal cellulitis

ABSTRACT

Dacryocystitis is the inflammation of the nasolacrimal sac and is due to obstruction of the nasolacrimal system or lacrimal stagnation, leading to favorable environments for infection. Any disruptions to the nasolacrimal pathway can increase the risk of dacryocystitis. Here is a unique case of *Prevotella denticola* dacryocystitis that progressed into cellulitis and abscess formation in a 7-year-old female with a medical history of Goldenhar syndrome, recurrent dacryocystitis, and periodontal disease. The patient presented with standard dacryocystitis symptoms—tenderness to palpation, erythema, and edema to the medial canthus. The patient was treated empirically with vancomycin, ceftriaxone, and clindamycin. She was discharged home on clindamycin with symptomatic improvement and plans for dacryocystorhinostomy. Goldenhar syndrome is a rare congenital craniofacial malformation disorder due to aberrant morphogenesis in structures derived from the first and second branchial arches. *P. denticola* is an anaerobic gram-negative bacillus significant to the oral microbiome. There is currently limited research suggesting the relationship between Goldenhar syndrome and dacryocystitis complicated by *Prevotella denticola*. Based on the patient's medical history, we predict that her complex presentation was secondary to the dissemination of *Prevotella* from her oral cavity. This case report emphasizes the importance of gram-negative anaerobic coverage in complicated dacryocystitis in patients with nasolacrimal defects.

INTRODUCTION

Dacryocystitis, the inflammation of the nasolacrimal sac, is commonly caused by obstruction of the nasolacrimal duct—either due to the backup or stasis of tears.[1] The nasolacrimal system starts with lacrimal tear production at the main lacrimal gland, drains into the common canaliculus, travels through the valve of Rosenmüller, and into the lacrimal sac.[2] From there, lacrimation continues inferiorly into the nasolacrimal duct, through the valve of Hasner, and into the nasal cavity.[2] There is increased risk for dacryocystitis infections in the setting of abnormalities or alterations to the nasolacrimal system.

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The etiology of dacryocystitis is characterized as acute, chronic, congenital, or acquired. There is a bimodal distribution of causes, with newborns and infants susceptible to acute and congenital cases, while adults over 40 years are prone to chronic and acquired situations.[1] The most causative organisms are gram-positive *Staphylococcus aureus*, coagulase negative *Staphylococcus*, and *Streptococcus pneumoniae*. *Haemophilus influenzae* is the most causative gram-negative organism.[1] Chronic dacryocystitis stems from long-term obstruction secondary to systemic diseases like Wegener's granulomatosis or sarcoidosis, tumor-like lesions, and mechanical obstruction.[3] Acquired dacryocystitis is caused by repeated trauma to the nasoethmoid bones, surgeries (endonasal and endoscopic procedures), or thickening of facial structure and bones.[4]

Congenital dacryocystitis is an obstruction in the distal nasolacrimal duct at the valve of Hasner.[1] It is detected when the amniotic fluid buildup from the womb fails to flow through the nasolacrimal system after delivery. Any congenital malformations to the craniofacial morphogenesis can increase risk of obstruction to the nasolacrimal duct. Goldenhar syndrome, also known as oculo-auriculo-vertebral (OAV) syndrome, is a rare congenital disorder defined as the malformation of the structures derived from the

first and second branchial arches during development, including the eyes, mouth, lip, tongue, palate, ear, maxilla, and mandible.[5] Because of the multiorgan involvement, OAV syndrome can lead to increased susceptibility to infection, inflammation, and issues in the craniofacial area.

Dacryocystitis is a clinical diagnosis based on the patient's history and physical exam, specifically the ocular exam—including normal visual acuity, absence of pain in extraocular movements, location of tenderness, and lack of ophthalmalgia. Individuals tend to endorse an erythematous, edematous, and tender mass at the medial canthus of the eye with additional nasal dorsum involvement.[6] The inflammatory symptoms of dacryocystitis follow the path of the nasolacrimal system; therefore, if erythema or edema is present beyond the nasolacrimal system, alternative diagnoses must be considered. When suspected clinically, dacryocystitis is confirmed by culturing the purulent fluid from the abscess. Radiologic imaging is not required for diagnosis, but computerized tomography (CT) scans can rule out more severe cases of orbital cellulitis.[7] Complication of dacryocystitis can be detrimental, with significant sequelae of orbital cellulitis, lacrimal fistulas, brain abscesses, cavernous sinus thrombosis, meningitis, and death.[1]

Uncomplicated dacryocystitis is treated conservatively with warm compresses and Crigler massage, a manual decompression of the nasolacrimal duct. In acute settings, Crigler massage is not warranted due to increase in hydrostatic pressure from the massage, causing a vulnerable environment for the spread of infection. Broad-spectrum antibiotics that cover gram-positive and gram-negative organisms, especially antistaphylococcal agents, are considered in the acute phase.[1] Initially, intravenous (IV) therapy of penicillin and cephalosporins are indicated. Vancomycin is the drug of choice in the setting of methicillin-resistant *Staphylococcus aureus* (MRSA) while clindamycin should be considered in the setting of anaerobic coverage.[6] In recurrent cases of dacryocystitis, ophthalmologists should be consulted for potential nasolacrimal probing, a highly successful technique used to irrigate the nasal cavity. In more advanced cases, invasive procedures like balloon dacryoplasty, nasolacrimal intubation, or nasolacrimal stenting are available.[3] Definitive therapy is a dacryocystorhinostomy, a new pathway for the tears to flow through the nasolacrimal sac and into the nose.[3]

CASE PRESENTATION

A 7-year-old Caucasian female with a past medical history of Goldenhar syndrome with mild dysmorphic features, type 1 Duane retraction syndrome, prematurity, and extensive dentoalveolar concern presented to inpatient pediatrics with bilateral infraorbital erythema with pronounced edema in the medial aspect of the right eye that exhibited significant tenderness to palpation and cellulitis-like symptoms (Figure 1). The patient denied any fevers or recent sick contacts. Immunizations were up to date. Her foster mother reported that the patient had a history of lacrimal duct infections that were similar to this presentation but less tender. The most recent episode happened the week prior, and the patient was managed with a 7-day course of amoxicillin. Her social history is significant for substance abuse in her biological mother, neglect and malnourishment by her biological parents, and involvement of the Department of Social Services (DSS).

On admission, the patient was afebrile with vitals showing signs of tachycardia at 127 beats per minute and tachypnea at 24 breaths per minute. The patient's initial labs were significant for signs of inflammation and infection with an elevated C-reactive protein of 12.3 mg/dL (reference range: 0.0-0.6 mg/dL), elevated erythrocyte sedimentation rate of 66 mm/h (reference range: 0-20 mm/h), and leukocytosis at 14.4 x10^3/µL (reference range: 4.5-13.5 x10^3/µL). Aside from her facial erythema and edema, the patient's physical exam was unremarkable. During the first few hours, the patient had a temperature of 102.5°F (39.2°C) that was decreased to 98.1°F (36.7°C) with acetaminophen and ibuprofen.

Dacryocystitis was suspected, and IV ceftriaxone and vancomycin were prophylactically started while awaiting blood culture results. The patient was not adequately responding to the antibiotic therapy; therefore, vancomycin was switched to IV clindamycin for anaerobic coverage. Despite antibiotics and warm compresses, the patient continued to experience progressively worsening right-sided infraorbital tenderness.

FIGURE 1:

(Left) Bilateral infraorbital erythema with notable edema medial to the right eye

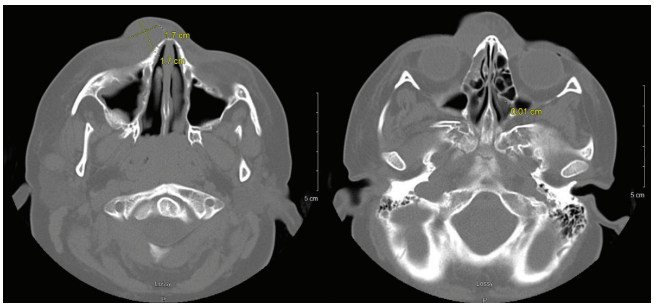
(Middle) Postrupture of abscess and purulent drainage

(Right) Prior to discharge: significant improvement of erythema, edema, and tenderness

Ophthalmology and otolaryngology services were both consulted and recommended a CT scan without contrast of the sinuses. It showed prominent soft tissue density along the right nasal bridge and frontal process of the maxilla that extended along the right premaxillary soft tissues and periorbital region (Figure 2). Most of the infection appeared to be localized over the lacrimal duct without orbital involvement. The intraconal and extraconal fat planes were preserved with no evidence of postseptal extension of the soft tissue density, but moderate circumferential mucosal thickening of the right maxillary sinus with occlusion of its ostium was shown. Additionally, focal dehiscence and disruption of the anterior alveolar cortex were visible and associated with an unerupted right paramedian maxillary tooth.

FIGURE 2:

CT without contrast of sinuses



CT showed prominent soft tissue density along the right nasal bridge and frontal process of the maxilla measuring 1.7 x 1.7 cm that extended along the right premaxillary soft tissues and periorbital region—right greater than left.

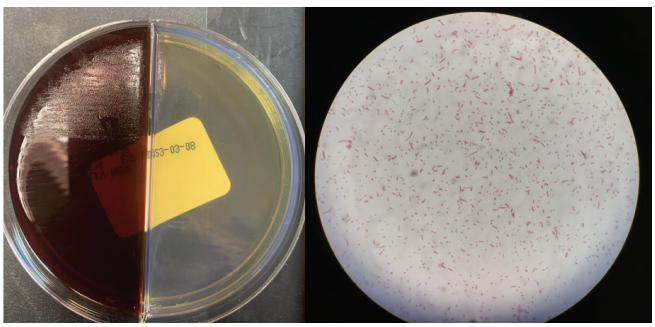
Moderate applied warm compress pressure was placed over the infected area leading to the rupture of the abscess and purulent drainage. Initial blood culture showed moderate gram-positive cocci and wound cultures exhibited preliminary results of anaerobic isolates. After the abscess ruptured, the patient responded to the antibiotic therapy, and her mother reported that the patient was back to her baseline—without nasolacrimal tenderness or purulent drainage. Therefore, the patient was discharged home with a 7-day course of oral clindamycin with plans to consider ocular ductal plasty reconstruction or dacryocystorhinostomy of her left ductal duct with her ophthalmologist in the future.

The patient's abscess cultures macroscopically showed growth of pinpoint colorless colonies on Laked Brucella Blood agar with Kanamycin and Vancomycin (LKV) but had a notable absence of growth on Bacteroides Bile Esculin agar (BBE) under anaerobic conditions for 48 hours. Microscopically, gram stains of isolated colonies revealed short gram-negative coccobacilli rods. Prevotella denticola was confirmed on matrix-assisted laser desorption/ionization-time of flight (MALDI-TOF) mass spectrometry (Figure 3).

(Left) Light growth of pinpoint colorless colonies on LKV agar and absence of growth on BBE agar under anaerobic conditions for 48 hours. (Right) Gram-negative coccobacilli rods seen on isolated colonies. Several organisms show morphologic elongation and ballooning degeneration, likely attributable to cell wall–active antimicrobials at the time of sample collection.

FIGURE 3:

Macroscopic and microscopic lab results



DISCUSSION

This patient has a notable medical history of Goldenhar syndrome, recurrent dacryocystitis, and dentoalveolar concerns and presented to the hospital with acute dacryocystitis and preseptal cellulitis. We believe that the *P. denticola* infection complicated her acute dacryocystitis due to her history of craniofacial anomalies and extensive periodontal disease.

The patient's Goldenhar syndrome presented with mild dysmorphic facial features at birth—a narrow palpebral fissure and a right-sided preauricular skin tag. Her Goldenhar syndrome is complicated by right esotropia, also known as type I Duane retraction syndrome. The craniofacial malformations associated with Goldenhar syndrome are secondary to disrupted migration and proliferation of the mesenchymal and neural crest cells of the first and second branchial arches. This is believed to be secondary to a disruption in the embryonic vascular supply between weeks 4 and 8 of gestation.[5] Risk factors for Goldenhar syndrome are sporadic and multifactorial, incorporating chromosomal aberrations and environmental causes. Reported maternal conditions associated with Goldenhar syndrome include maternal diabetes, multiple gestations, assisted reproductive techniques, hormonal therapy, maternal hypothyroidism, vasoactive drug use, tobacco use, cocaine abuse, and malnourishment.[5] Upon further review, we found that the patient had a history significant for these risk factors.

There is limited research discussing the association between lacrimal gland abnormalities and Goldenhar syndrome. When documented, the association is hypothesized to be related to dysembryogenesis. During the seventh week of gestation, the nasolacrimal system forms as a cord of ectodermal cells extending from the nasal cavity.[8] Throughout gestation, this system develops and canalizes. The last portion of the nasolacrimal system to recanalize is the inferior meatus, the most common site of obstruction in lacrimal defects.[2] When reported, lacrimal anomalies related to Goldenhar syndrome were found in the form of obstruction to the nasolacrimal duct or common canaliculus.[8] We believe the disruption of embryonic vascular supply during early gestation contributes to nasolacrimal malformations associated with Goldenhar syndrome.

Dacryocystitis has been reported to potentially be complicated by more serious infections like orbital cellulitis, meningitis, lacrimal abscesses, and cavernous venous sinus thrombosis.[1] This patient's case was complicated by a lacrimal abscess. With *S. aureus*, *S. pneumoniae*, and *H. influenzae* being the most causative organisms for dacryocystitis, the patient was started on broad-spectrum, empiric gram-positive, and gram-negative coverage of vancomycin and ceftriaxone. However, the patient's symptoms were not improving, so vancomycin was switched to clindamycin, an antibiotic that covers both MRSA and anaerobes. Anaerobic coverage was crucial in this scenario as her culture grew *P. denticola*, supporting the severity of the patient's presentation. *Prevotella* spp. produces beta-lactamase, which contributes to its virulent antimicrobial resistance.[9] Although susceptibility patterns of *Prevotella* spp. are limited, most are susceptible to piperacillin-tazobactam, imipenem, meropenem, tigecycline, and metronidazole.[9] Intrinsic resistance of *Prevotella* spp. to beta-lactam antibiotics coupled with recurrent treatment of dacryocystitis with amoxicillin may contribute to the development of opportunistic infection through altered

homeostasis of the natural oral microbiome.

The *Prevotella* spp. is classified as an anaerobic gram-negative bacilli and is one of the core mucosal anaerobes of the oral, respiratory, and gastrointestinal microbiome.[10] This species contributes to the production of bacterial biofilm in the oral cavity and can be involved in chronic inflammation and the progressive destruction of periodontal tissue.[11] When the mucosal microbiome is compromised, the reduction or overabundance of *Prevotella* spp. can cause inflammatory infections.[10] An increase in *Prevotella* spp. is seen in periodontitis, bacterial vaginosis, and low-grade systemic inflammation due to the mucosal spread of the bacteria throughout the body.[10]

In terms of periodontal infections, the patient had an extensive dentoalveolar procedure done 2 years ago with dental restorations. She had eight tooth extractions and 12 pulpotomies with stainless steel crowns. Evidence-based research has supported the gut-retina microbiome axis hypothesis, describing the relationship between the oral microbiome and ocular infections. Periodontal disease is known to have an association with inflammatory disease states. At this time, although the exact pathophysiologic mechanism is unknown, current literature supports three hypotheses including transmigration, pro-inflammatory molecule production, and hematogenous spread throughout the nasolabial triangle.[12] The nasolabial triangle, bound by the right and left oral commissures to the nasal bridge, is highly vascularized and full of intricate anastomoses, which can easily communicate infections between the oral cavity and the nasolacrimal system.[12] We suspect that *P. denticola* from the patient's oral cavity disseminated via this route to her nose and laid dormant until it had the opportunity to disrupt the nasal microbiome and proliferate.

CONCLUSION

We discussed a 7-year-old female with a medical history notable for Goldenhar syndrome, multiple dental surgeries, and recurrent dacryocystitis infections previously treated with amoxicillin, who presented acutely for bilateral infraorbital edema, erythema, and tenderness. She was hospitalized for acute dacryocystitis complicated by *P. denticola*, and she was successfully treated with IV ceftriaxone and clindamycin for broad-spectrum and anaerobic coverage. We hypothesized that her anatomic anomalies associated with Goldenhar syndrome, her history of periodontal disease, and her chronic history of dacryocystitis contributed to the spread of *P. denticola* to her lacrimal sac. We could not find any case reviews or reports describing acute dacryocystitis complicated by *P. denticola* infection in patients with Goldenhar syndrome and periodontal disease. Furthermore, there is limited research regarding Goldenhar syndrome and dacryocystitis complicated by opportunistic organisms. This case accentuates the significance of considering gram-negative and anaerobic coverage in complicated acute recurrent dacryocystitis for successful clinical outcomes, especially in those with developmental malformations of the craniofacial region.

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CLINICAL IMAGE

A CASE OF METASTATIC ANAL CANCER WITH MULTIPLE CUTANEOUS LESIONS

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CASE REPORT

A 56-year-old female presents with multiple painless but enlarging cutaneous nodules. The lesions rapidly developed on her face, torso, and legs (Figures 1 and 2). There was no associated warmth or fluctuance, and there did not appear to be any surrounding cellulitic changes or trauma. The patient denied any further systemic symptoms or similar previous episodes. Her past medical history was significant for: acid reflux, anxiety, depression, arthritis, constipation, hepatitis C, migraines, pancreatitis, sleep apnea, liver nodules, and anal squamous cell carcinoma (SCC). She had a 25-pack-year smoking history. Her past surgical history was significant for cholecystectomy and hysterectomy. She was also previously treated with combined chemoradiation for anal SCC.

About a year prior, the patient presented to the emergency department with a chief complaint of 6 weeks of constipation refractory to oral laxatives and suppositories. This was accompanied by lower abdominal and rectal pain with episodes of loose stool and blood per rectum. Upon physical exam, a perianal lesion was diagnosed as an external hemorrhoid and she was successfully treated for constipation and discharged. A month later, she presented again with alternating diarrhea, constipation, and pain per rectum. She was admitted for recurrent hematochezia and pain. An anal mass was observed and biopsied, with a final diagnosis of anal SCC with human papillomavirus (HPV) etiology. At computed tomography (CT) staging, the tumor was 7 cm in the craniocaudal dimension and 4 cm from the anal verge below the dentate line, and it was extending into the right ischioanal fossa with associated lymphadenopathy. Therapy included combined radiation and chemotherapy, but surgery was not indicated.

FIGURE 1:

Cutaneous nodule on face

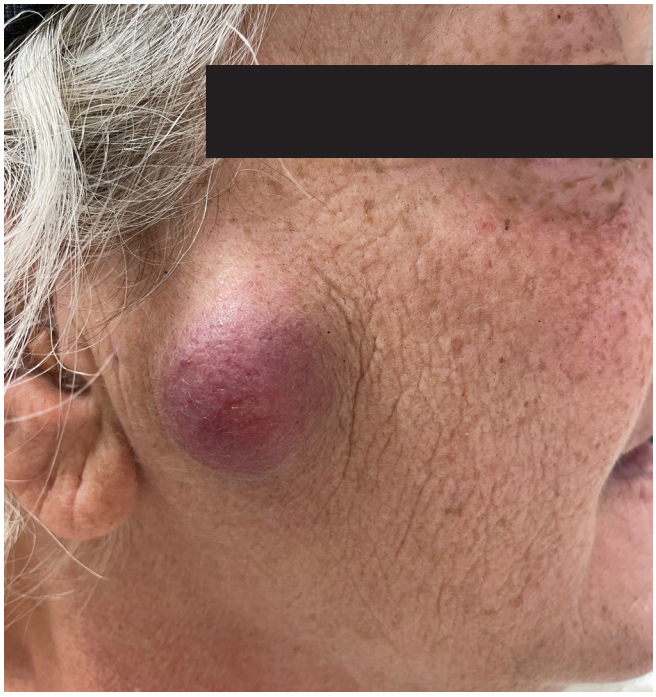
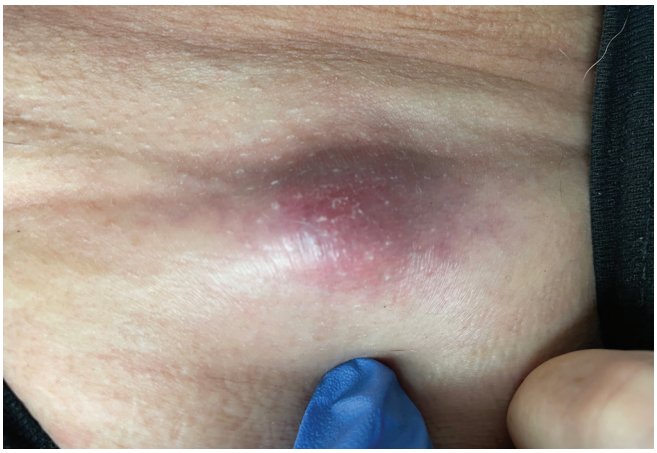


FIGURE 2:

Cutaneous nodule on torso



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We would like to acknowledge our kind brave patient.

QUESTIONS

1. What is the most likely diagnosis for the cutaneous nodules, given the history presented above?

a. Erythema nodosum

b. Lymphadenopathy

c. Metastases

d. Nodular lymphangitis

e. Sporotrichosis

Correct answer:
c. Metastases

When taking the patient's history and presentation into consideration, the lesions are most concerning for metastases. The lesions are not painful, do not appear to be infectious, and are fixed on exam. While this may have started as lymphadenopathy, the distant and pervasive spread is more indicative of a metastatic process. Furthermore, her exposure history to a fungus or bacteria to cause sporotrichosis, erythema nodosum, or nodular lymphangitis was not revealed in her history and is unlikely.

2: What is your next step in management?

a. Incision and drainage

b. Itraconazole

c. Oral antibiotics

d. Referral for biopsy

e. Topical steroid cream

Correct answer:
d. Referral for biopsy

Due to the clinical suspicion for metastases, a biopsy should be performed to characterize the histology and etiology of the tissue. Care should be taken when considering the region of the body that is being biopsied, history of present illness, and acuity of postbiopsy follow-up. In this case, a periauricular lesion was referred to an ENT for biopsy due to the sensitive nature of nearby neurovascular and bleeding risks, while the other lesions were referred to general surgery given the high suspicion for metastasis. There were no indications of infection in the area, so incision and drainage, antibiotics, or antifungals would not be appropriate. The lesions were painless, nonpruritic, and otherwise not impacting the patient at the time of presentation, so topical steroids are also not indicated.

DISCUSSION

This patient presented with a very uncommon and aggressive form of metastatic anal SCC caused by HPV 16. Anal cancer is a rare gastrointestinal cancer and is distinct from the more common colorectal cancers. Specifically, anal cancer makes up an estimated 3% of gastrointestinal cancers with 9760 new diagnoses a year.[1,2] Over 80% of anal cancer cases are due to SCC.[1] Squamous cells are ubiquitous in the skin and lining of hollow organs, like the anal canal below the dentate (or pectinate) line.[1] HPV infections are implicated for 93% of anal cancers causing SCC.[1] The strain of HPV can influence severity and especially devastating is HPV 16.[3,4] Screening and early diagnosis are pivotal to lessen the occurrence of anal cancer and its potential metastasis.

Rectal pain, bleeding, and pruritus are the most common presenting symptoms of anal cancer.[1] Anywhere from 50% to 80% of anal cancers are misdiagnosed due to their presentation mimicking benign processes like hemorrhoids, constipation, and diarrhea.[5-7] Therefore clinical suspicion should increase if these more benign diagnoses are refractory to initial standards of care. Women have higher rates of anal cancer than men (of the new cases per year, 3180 occur in men and 6580 in women).[2] While women are screened for HPV more frequently than men in a gynecologic setting with cervical Pap tests, anorectal screening should be included if the patient is a high-risk patient, positive for high-risk HPV types, or if cancer is suspected.[4] This can be done with an anal Pap test with cytology or digital rectal exam (DRE) if a lesion is observed.[2] Those who have a history of other cancers caused by HPV (cervical, oropharyngeal, penile, etc), have undermanaged HIV, are immunocompromised, practice anal receptive intercourse, or have received radiation to the pelvic area are at higher risk for developing anal cancer.[1]

Colonoscopy/anoscopy and biopsy serve to definitively diagnose anal cancer.[1] The location of the original lesion relative to the dentate line at the time of diagnosis determines patterns of lymph-node drainage and influences metastasis and treatment.[1] It is speculated that both lymphatic and hematologic systemic circulation can influence metastasis. Limited cases of metastasis from anal cancer are identified in the literature and include: the cranial bone and brain,[8-10] lymph nodes (including pelvic[11] and mediastinal locations[12]), paravertebral tissue,[13] liver,[14] and cutaneous nodules at the labia majora.[15] While SCC contributes to skin cancer as a primary lesion, metastasis to the skin is not typical.[16,17] To date, there were no documented cases of multiple, pervasive, metastatic cutaneous lesions in the literature.

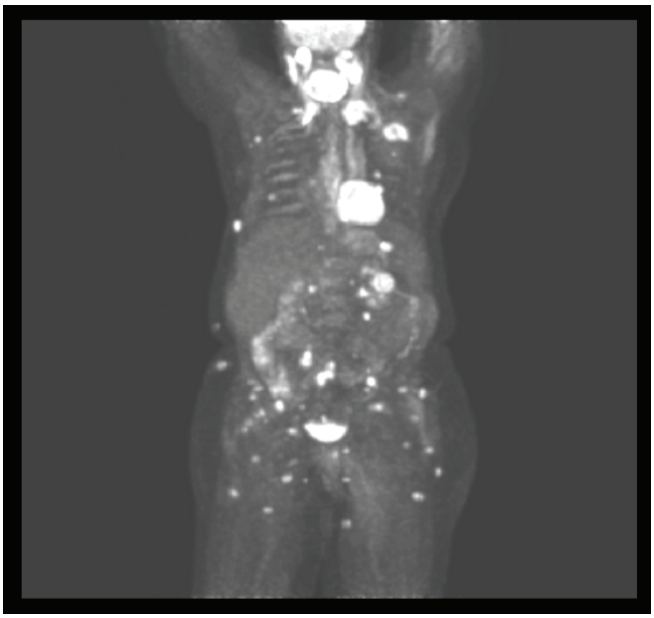
If anal cancer is diagnosed, there are standard treatment approaches that usually yield a positive prognosis and outcome for the primary lesions but success may be limited at later metastatic stages. There has been a transition away from surgical resection to utilizing anal-sparing chemoradiation therapy for initial treatment (either 5-fluorouracil and mitomycin C or 5-fluorouracil and cisplatin), which is often successful.[1,18] Little is known on the rate and timing of metastasis, with one study indicating 9% of anal cancers develop distant metastases.[19] Local recurrence is more common at 17%.19 For local recurrence, failure to respond to chemoradiation, or failure of local excision for superficial SCC, abdominoperineal resection (APR) should be considered.[1,18] Limited information exists for distant metastases due to its rare occurrence, but combined chemoradiation therapy is often reinitiated.[18,19] Recently, some developments in genome sequencing have improved survival and personalized treatment.[3] Though strides have been made to treat anal cancer, the HPV vaccine has made surmountable headway in prevention.

The ubiquity of HPV vaccination has lessened the overall occurrence of neoplasms caused by HPV. With regard to anal cancer, HPV is implicated in over 90% of cases with the p16 subtype the culprit for most infections.[1,18] The HPV vaccine is recommended for routine vaccination at age 11 or 12 years (but can be started as early as 9 years) and for everyone through the age of 26 years.[20] Some adults may opt for the vaccine up to the age of 45 years, based on discussion with their physician.[20] A recent study found that anal SCC rates are declining among vaccine-eligible adults compared to the incidence of disease in older populations, which continues to rise.[21]

When biopsied, the cutaneous lesions on this patient were revealed to be invasive moderately-to-poorly differentiated SCC involving the lateral and deep margins and invading the lymphovascular space. These were determined to be metastases from the primary lesion, and were invisible in sites ranging from the periauricular, lingual, inguinal, thigh, and back regions. A subsequent positron emission tomography (PET) scan, often used to assess the extent of disease when metastases are suspected, revealed innumerable cutaneous, subcutaneous, and muscular foci of F-fluorodeoxyglucose (FDG) activity indicating metastasis (Figure 3) with numerous areas of adenopathy. She was also profoundly hypercalcemic at this time. The patient began palliation and pain management as the lesions became exceedingly painful after biopsy and was properly referred to oncology for further chemotherapy and possible palliative radiation. During her course of diagnosis, remission, and recurrence, she had socioeconomic challenges to seeking care and was eventually lost to follow-up. The patient's disease sequela, comorbidities, and social determinants of health yielded a poor prognosis with a life expectancy of only about 6 months.

FIGURE 3:

PET scan revealing metastases



SUMMARY

Anal cancer is rare and its insidious symptoms can make diagnosis difficult. Moreover, mislabeling the diagnosis as colorectal cancer can impact reporting and subsequent prevalence and focus in medical literature. For family physicians, encouraging HPV vaccination, remaining vigilant to risk factors, and documenting accurate diagnoses can significantly lessen this disease burden on our patients.

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CLINICAL IMAGE

BILATERAL LOWER EXTREMITY RASH IN A 34-YEAR-OLD MAN

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CASE REPORT

A 34-year-old Caucasian male with no past medical history presented to the emergency department for a bilateral lower-leg rash that began 3 days prior. The rash was not painful or pruritic. The area of involvement had expanded and the rash had darkened since onset (see Figures 1 and 2). The patient denied any trauma to the area or contact with any irritants. No fevers were reported. He denied any similar rash in the past. He reported that he works at a large retailer and his job requires him to stand most of the day, in addition to walking, climbing, and performing physical work.

On examination, he was obese and hypertensive with a blood pressure level of 172/98 mmHg. His skin examination revealed a rash on his bilateral lower extremities that was nonpalpable, nonblanching, and nontender. It cut off abruptly at the sock line and spared the feet. It was not warm to palpation and there were no areas of open wounds or drainage. There was no mucous-membrane involvement or involvement of the palms or soles. There was no edema to the lower extremities.

FIGURE 1:

Right leg

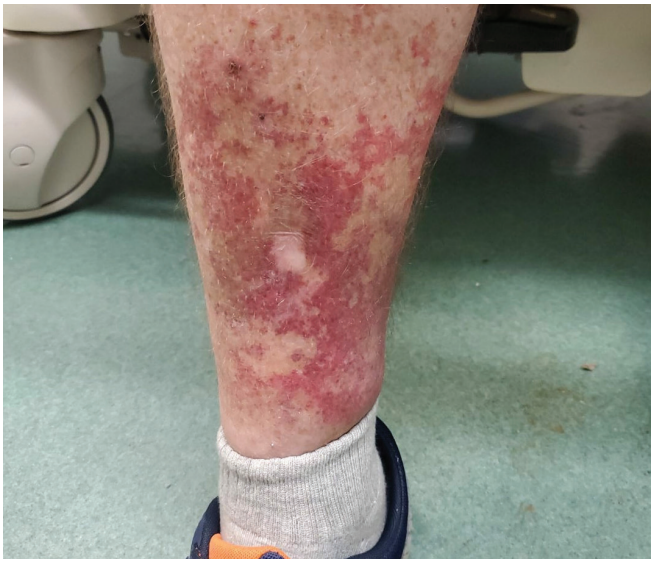
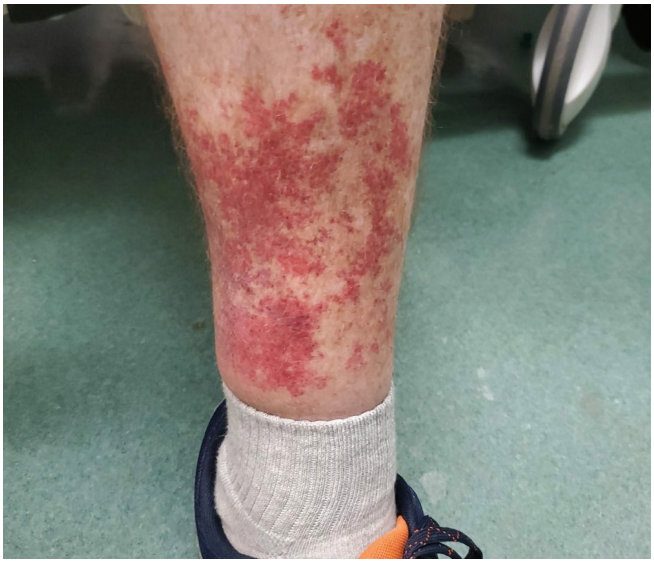


FIGURE 2:

Left leg



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QUESTIONS

1. This presentation aligns most with which diagnosis?

a. Cellulitis

b. Exercise-induced vasculitis (EIV)

c. Immunoglobulin A (IgA) vasculitis

d. Immune thrombocytopenia

Correct answer:
c. Exercise-induced vasculitis (EIV)

This patient presents with a lower-leg erythematous lesion, sparing below the sock line after prolonged standing, consistent with EIV.[1-5] Although EIV typically occurs in people over the age of 50 years who golf or hike in warm weather,[2,3] cases have been reported in younger patients.[6,7] The patient's history of prolonged standing with his job, possibly in combination with being obese, are risk factors for development of EIV.[8] Other types of vasculitis typically are not confined to the lower legs. IgA vasculitis would usually show signs of renal involvement. Immune thrombocytopenia presents with increased bleeding, and the palpable purpura is not typically present in crops in dependent body regions, like it is with EIV.[9] Cellulitis would present with erythema and tenderness to palpation, and the skin involved would be hot when palpated.[10]

2: What is the first-line treatment for the patient's diagnosis?

a. Intravenous (IV) antibiotics

b. Oral colchicine

c. Oral prednisone taper

d. Supportive care including leg elevation, compression stockings, nonsteroidal anti-inflammatory drugs (NSAIDs), and antihistamines

Correct answer:
c. Oral prednisone taper

Due to the clinical suspicion for metastases, a biopsy should be performed to characterize the histology and etiology of the tissue. Care should be taken when considering the region of the body that is being biopsied, history of present illness, and acuity of postbiopsy follow-up. In this case, a periauricular lesion was referred to an ENT for biopsy due to the sensitive nature of nearby neurovascular and bleeding risks, while the other lesions were referred to general surgery given the high suspicion for metastasis. There were no indications of infection in the area, so incision and drainage, antibiotics, or antifungals would not be appropriate. The lesions were painless, nonpruritic, and otherwise not impacting the patient at the time of presentation, so topical steroids are also not indicated.

2: One proposed mechanism for the patient's pathology is:

a. Intravenous (IV) antibiotics

b. IgA-mediated tissue damage

c. Immune-mediated platelet destruction leading to dysfunctional clotting cascade

d. Local bacterial tissue infection

Correct answer:
a. Dysfunctional local tissue temperature regulation

Although the pathophysiology of EIV is not well understood, one proposed mechanism is dysfunctional local temperature regulation.[4] EIV is known to take place commonly in warm climates.[1-3] Some literature suggests that increased cutaneous blood flow in warm temperatures, in combination with dysfunctional venous return, can lead to extravasation of red blood cells resulting in purpuric or petechial lesions on the lower legs.[4,8] IgA-mediated destruction of tissues is the mechanism of IgA vasculitis that is seen on biopsy.[9] Local bacterial tissue infection is consistent with cellulitis.[10] Immune-mediated platelet destruction leading to a dysfunctional clotting cascade is consistent with a diagnosis of immune thrombocytopenia.[9]

DISCUSSION

EIV is a benign and self-limited cutaneous small-vessel vasculitis that has been found to occur after prolonged exercise and is especially prominent in warmer temperatures.[2,3,11] The vasculitis generally presents with nonblanching, purpuric, petechial, or erythematous lesions on the bilateral lower legs, although blanchable lesions have been reported.[4,5,9] In some patients, the lesions can be noted on the feet or thighs, although the sock area is usually spared, with clear demarcation.[3,6,7,13-15]

The lesions can be asymptomatic, but they are often pruritic, painful, and have a burning sensation.[8-10] Some lesions are seen with lower-leg edema.[4,8,13] EIV is typically a self-limited condition that resolves in less than 10 days, but recurrence is common.[1,2,8] It is commonly seen in golfers, hikers, and persons who participate in other activities that include prolonged walking or running.[1,3,5,13,14] It is reported most in women and people over the age of 50 years.[1-3,8]

The pathophysiology of EIV is not fully understood. Some studies suggest that diminished thermoregulation might contribute to development of EIV.[4] Raising the core body temperature results in increased cutaneous blood flow and vasodilation, causing an increase in venous blood volume to the extremities. If the venous system becomes overfilled due to the presence of edema or venous insufficiency, this might potentially lead to damaged vasculature and extravasation of red blood cells, causing EIV.[4] Increased adipose tissue in the legs may also alter thermoregulation causing an increased core temperature and precipitating EIV through thermal damage to blood vessels.[8] Additionally, venous filling time is reduced with prolonged exercise, subsequently decreasing the efficacy of venous return and also causing stasis and red blood cell extravasation.[8]

Although EIV is self-limited and benign, it can be difficult to differentiate its presentation from systemic etiologies of cutaneous small-vessel vasculitis. The differential diagnosis for cutaneous small-vessel vasculitis includes several autoimmune conditions including systemic lupus erythematosus and antinuclear cytoplasmic antibody vasculitis.[9] Infectious etiologies and adverse drug effects should also be ruled out. Pertinent review of systems should be performed to assess for systemic vasculitis, fever, weight loss, fatigue, arthralgias, hematuria, abdominal pain, paresthesias, weakness, shortness of breath, cough, hemoptysis, and sinusitis.[9] Although EIV can be diagnosed clinically using a patient's history and physical examination, some literature suggests that skin biopsy should be performed to rule out systemic pathologies.[9-12,14] If biopsy is performed, the histologic findings may vary depending on at what point in the disease the sample was obtained. The sequence of events leading to EIV suggested by timed biopsies is a multistep cascade that begins with immune complex deposition and results in neutrophil damage to vessels.[16,17] If underlying systemic disease cannot be ruled out with physical examination and review of systems, laboratory testing, including complete blood count, creatinine levels, erythrocyte sedimentation rate, liver function tests, and urinalysis, should be completed.[9]

The first-line treatment for EIV is supportive care, including rest and leg elevation.[9,11,12] If the patient's lesions are symptomatic, topical steroids and NSAIDs are indicated.[9,11,12] Some patients find that the use of compression stockings and lighter clothing decreases recurrence.[1-3,8,11] A short taper of systemic steroids, oral colchicine, or dapsone can be trialed for ulcerated or severe lesions.[2,8,9,11] If the vasculitis persists despite these treatments, immunosuppressants can be attempted.[9,11] If the patient has progressed to needing oral prednisone and immunosuppressants, there should be a full workup performed for systemic etiology of cutaneous small-vessel vasculitis, and specialist referral should be considered.[9,11]

Although EIV is a benign and self-resolving condition, awareness of the disease is important because it can be easily misdiagnosed as a more serious condition prompting unnecessary workup and treatment. The physician should rely on a thorough history and physical examination while looking for signs of systemic disease and considering more serious pathologies.

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PATIENT EDUCATION HANDOUT

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Osteopathic Manipulative Treatment for Obstetrics

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Pregnancy, or gestation, is defined as the period in which a fetus develops inside a female's uterus. There are physiologic changes that occur as a consequence of pregnancy, which include cardiovascular changes (such as an increase in cardiac output), an increase in smooth muscle relaxation secondary to progesterone, and musculoskeletal changes. More importantly, these musculoskeletal changes, which include an increase in lumbar lordosis, forward neck flexion, and joint laxity, can result in several complaints with lower-back, pelvic and neck pain being the most prevalent. Osteopathic manipulative medicine (OMM) can play a vital role to help alleviate these conditions throughout a woman's pregnancy course.

HOW OMT CAN HELP

OMT, otherwise known as osteopathic manipulative techniques, are methods utilized to treat somatic dysfunctions, which are impairments in the function of different parts of the body including skeletal, joint, myofascial structures and their related vascular, lymphatic, and neurologic elements.[1]

Cervical soft-tissue OMT: In order to relieve strain to the cervical region, secondary to patients' necks being in forward flexion, an osteopathic physician may apply cervical soft-tissue techniques to the area of concern. Upon diagnosing a somatic dysfunction, a physician may apply soft force and traction to the muscles in your neck until relaxation or a "release" is felt. The physician will then reassess the area to ensure the "dysfunction," or the key point causing the neck pain is relieved.[2]

Seated forward-leaning thoracic spine articulator: In this technique, the physician stands in front of the patient using their knees to stabilize the patient on the table. The physician then asks the patient to cross her arms and lean forward against the physician while they wrap arms around the patient, contacting key articular components of the thoracic spine. The patient is drawn forward and various movements are introduced into this technique such as side bending and rotation to release the tension and increase the mobility of the patient's spine.[3]

Pubic decompression: This technique involves alternating relaxation and contraction of muscles. The patient's hips and knees are flexed and held together while the patient tries to pull them apart against the physician's counterforce. This is done multiple times with the patient's knees sequentially spread apart at each interval. The goal of this manipulation is to relieve any strain in the pelvic diaphragm and the pubic symphysis.[3]

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Hyperemesis Gravidarum in Pregnancy

Cindy Kim, PGY-1

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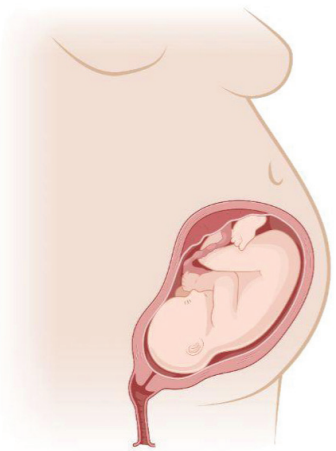

Hyperemesis gravidarum is an extreme form of morning sickness during pregnancy that causes nausea and severe vomiting more than 3 to 4 times per day, which can cause weight loss and changes in your electrolytes. It is seen in approximately 0.3% to 3% of pregnancies and is the most common cause of hospital admissions during the first part of pregnancy. The severe nausea is thought to be caused by high levels of human chorionic gonadotropin (hCG) and estrogen, which are hormones released in your body during pregnancy. Some risk factors include being a first-time mother, having multiple pregnancies, being overweight, having hyperemesis gravidarum in an earlier pregnancy, and presence of trophoblastic disease, which involves abnormal growth of cells in the uterus. It is recommended to take prenatal vitamins containing folic acid for 1 month before conception to prevent nausea and vomiting during pregnancy. Hyperemesis gravidarum has not been shown to cause significant harm to the fetus.

SYMPTOMS

Symptoms of hyperemesis gravidarum usually occur during 4 to 6 weeks of pregnancy and peak between 9 to 13 weeks. Listed below are some common signs and symptoms you can expect to experience

- Severe nausea and vomiting
- Aversion to foods
- Weight loss of 5% or more of prepregnancy weight
- Dehydration (urinating less often than normal, having dark-colored urine, feeling dizzy when standing up)
- Headaches
- Extreme fatigue
- Low blood pressure, fast heart rate

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TREATMENT OPTIONS

Mild cases of hyperemesis gravidarum can be managed with rest, hydration, antacids, and avoidance of triggers such as certain odors, heat, humidity, noise, or flickering lights. Eating frequent small meals every 1 to 2 hours that include high-protein snacks and avoiding spicy or fatty foods may help. You can also try homeopathic remedies such as consuming ginger ale/tea or wearing an acupressure band, which applies pressure on a certain part of your wrist to reduce nausea. More severe cases may require hospitalization and intravenous fluids to provide hydration, fix electrolyte imbalances, and replenish low vitamins and minerals. Restoring vitamin B1 levels is especially important to prevent a serious neurologic disease called Wernicke encephalopathy. You may be given medications such as vitamin B6 (pyridoxine), metoclopramide, antihistamines, or antireflux agents to treat severe nausea and vomiting. If you have persistent vomiting and weight loss that is uncontrolled by medications, you may be given nutrition through a feeding tube into your stomach.

WHEN TO CONTACT YOUR OSTEOPATHIC FAMILY PHYSICIAN

It is important to understand the difference between morning sickness and hyperemesis gravidarum because severe cases of hyperemesis gravidarum can cause dehydration, electrolyte imbalances, malnutrition, or unintended weight loss that may require intravenous fluids and nutritional support at the hospital. Please see your osteopathic family physician or obstetrician/gynecologist if you have any of these concerning symptoms during your pregnancy. In case of emergency, please call 911 or go to your nearest emergency department.

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