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WINTER 2025

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JUN 6-8 2025 — Future Leaders Conference, Chicago, IL

DEC 5-7 2025 — Future Leaders Conference, Chicago, IL

EDITOR'S MESSAGE

It Takes a Team

Paula Gregory, DO, MBA, FACOFP

This edition of OFP focuses on topics of interest to readers, including the childhood obesity epidemic, the care of transgender patients, and the treatment of trigger finger. All are important to the physician of today. The osteopathic profession and family osteopathic physicians are far richer for the collaboration and articles seen in OFP.

Family medicine physicians must know more, recognize more, and treat more complex and ever- changing disease states. Osteopathic physicians are often the only resource for the community and must be well versed in all aspects of medicine, frequently working without other specialties nearby. That is why it is especially important to recognize the work of the journal as it educates us on changes in medicine, with topics of interest to the osteopathic family physician, serving as a solid resource for our readers.

Our peer reviewers and editorial committee have been impactful in advising and ensuring the content is applicable and contains information for our daily practice. They spend many hours reviewing and collaborating with the editors and authors to bring the most up-to-date information to this journal.

The editors of our journal have advised and mentored countless contributors over the years of the journal's publication. Please join with me in recognizing the past editors, the editorial committee, and the peer reviewers who work to ensure that the journal has content relevant for the physician.

The past 75 years of ACOFP have shown that our leaders steered the community, and we are indeed standing on the shoulders of giants as we look forward to the future. Thanks to the previous leaders of the ACOFP.

The future of osteopathic medicine is a path that embodies principles that have withstood the test of time. The esteemed journal editors and writers, who dedicate time and knowledge so that we are ready to face the challenges of practice and leadership, should be recognized for their efforts and steadfastness.

We are enriched daily due to these wonderful role models. It is with gratitude and renewed hope in the osteopathic profession that we look forward to many more journal articles and issues. The future is bright for us, as many wonderful physician writers, peer reviewers, and student writing fellows are dedicated to educating us.

LETTER TO THE EDITOR

Vaccines and Knowledge: Silent Guardians Against HPV

What We Know About HPV and What We Need to Know About HPV

To the OFP Editor:

Student Doctors Anna-Taylor Harbin and Alexa Lane provided a useful overview of human papillomavirus in the OFP Patient Education Handout (published in the Fall 2024 issue). They also raised some important questions concerning HPV for family physicians.

Which HPV-related cancers can be screened for? While the authors argue that cervical cancer is the only HPV-related cancer that can be screened for, and it is currently the only HPV cancer screening included in the preventive medicine guidelines, there are other HPV-related cancers that should be screened for in high-risk populations.

There is a screening method for HPV-related rectal cancer called the anal Pap smear (also known as anal cytology or anal rectal cytology (ARC).[1] This test involves collecting cells from the anus using a swab, like a cervical Pap test. The collected cells are then examined under a microscope for abnormalities that could indicate precancerous changes or cancer.[2]

If abnormal cells are detected, further tests, such as a digital anal rectal examination (DARE) or high-resolution anoscopy, may be recommended to examine the anal canal more closely and potentially treat any precancerous lesions.

Esophageal cancer from HPV virus can also be tested for by esophagoscopy and biopsy.[3] While the test is too intrusive to be a screening test for the public, there are times when this should be pursued.[4]

Noting that we do not treat men who have HPV, even though HPV is associated with penile cancers in men,[5] raises more questions.[6] And the issue of whether HPV infection is sexually transmitted or sexually activated is one that our patients will ask about and could direct future research.[7-8]

Tyler Cymet, DO, FACOFP

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SOURCES

1. Howard Brown Health Center. (2019). Anal dysplasia screening guidelines. https://howardbrown.org/wp-content/uploads/2019/08/Howard_Brown_Health_Anal_Cancer_Screening_Guidelines-2019.pdf

2. National Cancer Institute. (2024.) Anal cancer advances open door to screening and prevention. https://www.cancer.gov/news-events/cancer-currents-blog/2024/anal-cancer-screening-self-collected

3. National Cancer Institute. (2024.) Esophageal cancer screening (patient version). https://www.cancer.gov/types/esophageal/patient/esophageal-screening-pdq

4. Weaver, B. A., & Cymet, T. C. (2011). Human papillomavirus: Every answer raises more questions. *Journal of Osteopathic Medicine*, 111(s32), ii-2.www.cancer.gov/types/esophageal/patient/esophageal-screening-pdq

5. Bailey, J., & Cymet, T. C. (2006). Planning for the HPV vaccine and its impact on cervical cancer prevention. *Comprehensive therapy*, 32, 102-105.

6. Cymet, T. C. (2006). The secret sits. AOA's Women and Wellness, 106, 2-3.

7. Cymet, T. (2007). HPV prevalence and transmission. JAMA, 298(1), 38-38.

8. Cymet, T. (2007). Talking about HPV has its challenges. Journal of Family Practice, 56(2), 112-113.

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FROM THE PRESIDENT'S DESK

Preparing for the Challenges and Opportunities Ahead

Brian A. Kessler, DO, DHA, FACOFP *dist.*

As we progress in 2025, I am privileged to share my reflections in this special edition of the *Osteopathic Family Physician*. This issue marks the 75th anniversary of the American College of Osteopathic Family Physicians (ACOFP), a significant milestone that celebrates our enduring legacy and calls us to reaffirm our commitment to the principles that define osteopathic family medicine: compassion, service, and a holistic approach to care that considers each patient's physical, emotional, and social well-being.

For 75 years, ACOFP has supported osteopathic family physicians by serving as a beacon of advocacy, education, and innovation. Our organization has empowered physicians to meet the needs of their patients and communities while upholding the values of integrity and holistic care. The commemorative timeline and features in this issue illustrate the evolution of ACOFP and its transformative impact on our profession. They remind us of the tireless efforts of those who came before us and challenge us to honor their legacy by addressing the opportunities and obstacles of today.

The articles in this issue highlight the adaptability and commitment of osteopathic family physicians to addressing the full spectrum of healthcare. Discussions on inclusive care for transgender patients, approaches to managing childhood obesity, and the application of osteopathic principles to postpartum pelvic girdle pain illustrate the broad impact of our profession. Reviews of treatments for trigger finger and the use of statin therapy over the long term emphasize the importance of integrating evidence-informed practices into clinical care. These contributions underscore our shared mission to provide personalized patient-centered care that upholds the values of osteopathic medicine.

The observances we celebrate in March further align with the principles of equity, inclusivity, and safety that guide our profession. International Women's Day, Women's History Month, and International Transgender Day of Visibility highlight the importance of providing equitable healthcare to all individuals, regardless of their background or identity. These occasions inspire us to continually refine our approaches to patient care and ensure we treat each person respectfully and with dignity. Patient Safety Awareness Week emphasizes our responsibility to maintain the highest trust, quality, and safety standards in every patient interaction.

For those of us engaged in academic medicine, Match Day on March 21st represents a defining moment as we celebrate the next generation of osteopathic physicians stepping into their residency training. This milestone underscores our duty to serve as mentors, educators, and role models for these future leaders of osteopathic family medicine. By imparting values and practical skills, we ensure that they will continue to uphold and advance the mission of our profession.

National Doctors' Day on March 30th allows us to reflect on the privilege and responsibility of being a physician. This day provides an opportunity to honor our predecessors' contributions, celebrate our colleagues' achievements, and recommit to delivering hope and healing to the individuals and families we serve. It reminds us of our patients' profound trust in us and the transformative impact of our work on their lives.

As we commemorate ACOFP's 75th anniversary, we must also look to the future with resolve and purpose. The challenges we face today, from addressing healthcare disparities to navigating technological advancements, present unique opportunities for osteopathic family physicians to lead with empathy, expertise, and creativity. We must advocate for policies that promote patient-centered care and ensure all individuals have access to the resources necessary to achieve optimal health outcomes. By uniting our efforts, we can strengthen the foundation for future generations of osteopathic physicians.

We must deepen our community engagement and build collaborative partnerships to advance our impact. Osteopathic family physicians bring a unique perspective to healthcare by understanding the holistic needs of patients within the context of their families and communities. By working closely with community organizations, health systems, and policymakers, we can address the root causes of health disparities and develop solutions that improve outcomes for all. These collaborations enhance our ability to meet diverse populations' needs while reinforcing osteopathic family medicine's role as a leader in compassionate and comprehensive care.

The upcoming ACOFP Annual Convention in Palm Springs will allow us to celebrate our collective achievements, foster connections, and energize our shared mission. A highlight of the event will include the inauguration of Dr. Gautam Desai as the next ACOFP President. Dr. Desai's dedication, vision, and leadership will guide our organization toward continued excellence and innovation. His work reflects our values and sets the stage for a vibrant and impactful future for ACOFP and osteopathic family medicine.

I want to express my deepest gratitude to each of you for your dedication and contributions to our profession. Your work upholds the pillars of osteopathic family medicine and ensures that our mission continues to thrive. Together, we will advance the health and well-being of the individuals and communities we serve and build a stronger foundation for the next generation of osteopathic physicians.

I wish you a fulfilling and prosperous year ahead. Let us embrace this milestone to honor our past, strengthen our present, and shape a future where osteopathic family medicine continues to lead with integrity and purpose.

Warm regards,

Brian A. Kessler, DO, DHA, FACOFP dist.
2024-2025 President, American College of Osteopathic Family Physicians

REVIEW ARTICLE

Caring for Transgender Persons: Gender in Clinical Decisions, Tools, and Labs

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KEYWORDS

Transgender

Gender affirmation

Gender transition

Preventative screening

ABSTRACT

Individuals who identify as transgender (TGD) who are taking gender-affirming hormone therapies (GAHT) may experience physiologic changes that impact the interpretation of laboratory results, diagnostic tools, and risk calculations. This review provides guidance for healthcare practitioners on navigating these challenges to ensure appropriate and safe patient care. Key considerations include interpreting standard laboratory results and vital signs, alongside recommendations for preventative cancer screening and cardiovascular risk assessments. Additionally, there are some key considerations when using the CHA2DS2-VASc score in TGD patients with atrial fibrillation, as well as when interpreting a QT/QTc interval. Hormone therapies may also influence a patient's risk of venous thromboembolism (VTE), necessitating vigilant monitoring and avoidance of supraphysiologic hormone levels. Renal function estimations in patients on GAHT require careful consideration, with alternative equations potentially offering more accurate assessments. Finally, pulmonary function testing poses challenges, highlighting the need for gender-affirming approaches in interpretation. Overall, this comprehensive review underscores the importance of individualized care and shared decision-making in TGD healthcare.

BACKGROUND

A person who identifies as transgender (TGD) is someone whose sex assigned at birth (defined as one's chromosomal makeup and generally assigned based on external genitalia) is incongruent with the gender they identify with (defined by the societal characteristics of being a man or woman).[1] Patients experiencing psychologic stress due to this mismatch are said to be experiencing gender dysphoria.[1] TGD patients can transition and express their gender in a variety of means including social, legal, surgical, and medical via gender-affirming hormone therapies (GAHT). A TGD person may pursue all, none, or a combination of these therapy types. Transitions are often discussed in terms of patients going from male to female (MtF) or female to male (FtM), although patients who identify as nonbinary may also use these means to algin with their gender identity.[1] In terms of GAHT, there are various approaches to how these medications are titrated, all centered around the patient's satisfaction with secondary sex characteristics and safety.[1] Common medications used for this type of hormone replacement therapy (HRT) include estradiol and testosterone. Obtaining a detailed history of a patient's transition is crucial for providing appropriate care.

Laboratory tests, diagnostic tools, health screenings, and risk calculations differ based on one's sex assigned at birth. Surgical or medical transitioning can alter physiology, affecting the accuracy of these laboratory values, screenings, tools, or scores when the patient's sex assigned at birth is used. This review aims to review the use of such tools in patients on GAHT and discuss recommendations on when to use birth sex vs gender identity.

INTERPRETATION OF STANDARD LABORATORY RESULTS AND VITAL SIGNS

Common laboratory tests may be affected by sex steroids or body size.[2] Accurate interpretation of these results, considering GAHT, is essential in providing accurate evidence-based care. Table 1 summarizes major changes in common laboratory values affected by GAHT. There is no significant impact on vital signs in TGD patients using GAHT.[3,4]

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TABLE 1: Common laboratory tests and effects of GAHT

Laboratory Test	Persons on Masculinizing HRT	Persons on Feminizing HRT
Hematology		
Hemoglobin, hematocrit, red blood cells (RBC) [3-5]	**Initial:** increase at initiation; stabilizing at 6 months **Long term:** maintains for at least 5 years **Typical levels:** same as cisgender males	**Initial:** decrease at initiation; stabilizing at 3 months **Typical levels:** same as cisgender women
Platelets[3]	No change	**Initial:** increase from baseline; no absolute change
Mean corpuscular volume (MCV), white blood cells (WBC)[3,5]	No change	No change
Basic Metabolic Panel		
Chloride[3]	**Long term:** decrease with use up to 3-5 years	No changes
Sodium[3]	No change	**Initial:** decrease at initiation; stabilizing at 3-12 months **Long term:** maintains for at least 5 years
Glucose[3]	No change	**Initial:** increase at initiation for 6 months **Long term:** returns to normal after 6 months
Renal Function Tests		
Creatinine[3,4]	**Initial:** increase at initiation; stabilizing at 6 months	**Long term:** decrease at 12 months
Albumin[3]	No change	**Initial:** decrease at initiation **Long term:** decrease continues for up to 5 years
Liver Function Tests		
Alanine aminotransferase (ALT)[3,4,6]	**Initial:** increase beginning at 48 months **Long term:** continued for at least 5 years	No changes
Alkaline phosphatase (ALP)[3,4,6]	No change	**Initial:** decrease at initiation **Long term:** return to normal levels at 5 years
Aspartate aminotransferase (AST), total bilirubin[3,4,6]	No change	No change
Lipid Panel		
Low-density lipoprotein (LDL)[3,4]	**Long term:** relative increase through 5 years	No changes
High-density lipoprotein (HDL)[3,4]	**Long term:** relative decrease through 24 months	**Long term:** relative increase through 5 years
Total cholesterol, triglycerides[3,4]	No change	No change

For persons on GAHT, laboratory values that change with HRT use (Table 1) typically return to normal range around 10 weeks after discontinuing estradiol and 11 weeks after stopping testosterone therapy.[3]

PREVENTATIVE SCREENING

When assessing an individual's need for adult preventative care, consider their use of GAHT and any gender-affirming surgeries.[7] Gender dysphoria does not affect cancer risk, but can decrease the likelihood of screening for birth sex organs due to both provider and patient factors.[7] Some patients might find screenings gender affirming, such as mammograms for patients on feminizing HRT, while patients on masculinizing HRT might experience physical and emotional discomfort during Pap smears.[10] When screening patients who have undergone gender affirmation, it is important to get a thorough organ inventory to assess their risks. Table 2 summarizes recommendations on which patients should receive gender-related preventative screenings.

Cardiovascular Risk (Primary Prevention)

Current cardiovascular risk scores do not account for the impact of GAHT on patients. Cardiovascular risk assessment tools include the Framingham Risk Score, Reynolds Risk Score, SCORE (Systematic COronary Risk Evaluation), and QRISK/JBS, which all use gender or sex to stratify cardiovascular risk.[11]

GAHT may contribute to poor cardiovascular health in TGD people because of the potential cardiovascular effects of these treatments, such as an increased risk for venous thromboembolism (VTE) among TGD women taking estradiol.[12] Studies have identified a higher risk of CVD or stroke in TGD women on GAHT compared with cisgender counterparts.[10,13] However, these studies often do not quantify the individual's baseline cardiovascular risk before GAHT, nor differentiate the type, dose, or duration of GAHT utilized.

The American Heart Association (AHA) suggests coronary artery calcium (CAC) as a tool to classify cardiovascular risk in individuals with borderline or intermediate estimated 10-year risk, as part of shared decision-making.[11] CAC is recommended in adults ages 40 to 75 years with LDL 70 to 189 mg/dL and 5% to 19.9% 10-year atherosclerotic cardiovascular disease (ASCVD) risk, or those with <5% risk and a family history of premature ASCVD.[14] In a small cohort of TGD individuals (n = 47) on GAHT, the CAC score was similar to their cisgender age-matched counterparts.[15] Table 3 summarizes how the CAC score can inform statin use.

TABLE 2: Preventative health screening in transgender patients

	Persons on Masculinizing HRT	Persons on Feminizing HRT
Cancer		
Breast[7]	Without bilateral mastectomy: follow guidelines for cisgender females With chest reconstructive surgery: yearly chest wall and axillary exams	GAHT 5-10 years: screening mammography every 2 years starting at age 50 years No GAHT: no routine screening
Cervical[7,8]	Intact cervix: annual Pap smear over the age of 21 years Consider pretreatment with vaginal estrogen to obtain adequate sample Total hysterectomy, no history of precancerous cervical legions or cervical cancer: no screening needed	No screening needed
Colon[7]	Follow guidelines for cisgender patients	Follow guidelines for cisgender patients
Prostate[7]	No screening needed	Follow guidelines for cisgender males
Endometrial[7]	Follow guidelines for cisgender patients	Follow guidelines for cisgender patients
Ovarian[7]	Follow guidelines for cisgender patients	Follow guidelines for cisgender patients
Osteoporosis Screening[9]		
	Recommended for patients who are not compliant with hormone therapy, or who develop risks for bone loss	Consider bone mineral density (BMD) testing at baseline. Individuals at low risk: start screening at age 60 years
Human Papillomavirus (HPV) Screening[8]		
	Follow guidelines for cisgender patients	No screening needed
Abdominal Aortic Aneurysm (AAA) Screening		
	No formal recommendation	No formal recommendation

TABLE 3: CAC score interpretation[14]

Absolute CAC Score	10-Year ASCVD Risk	Statin Benefit
≥100	>7.5%	Recommended
≥300	Higher than 7.5%	High intensity
≥1000	Need for aggressive LDL lowering	High intensity

Although there is no consensus on how to utilize cardiovascular risk assessment tools in TGD patients, considering the type and duration of hormone therapy is important. Suggested strategies for quantifying risk with binary cohort equations include using sex assigned at birth, affirmed gender, or an average of both, though these may overestimate or underestimate actual risk. Alternatively, the CAC score may provide a more accurate cardiovascular risk assessment and inform therapy interventions better. Due to the lack of data in the TGD population, shared decision-making about potential cardiovascular risk and primary prevention is crucial.

Atrial Fibrillation

The CHA2DS2-VASc risk score has the best evidence for predicting thromboembolic risk in people with atrial fibrillation requiring anticoagulation.[16] The sex category criteria apply one point to female sex as a risk factor, but do not account for estrogen use or TGD females on GAHT.

Nielsen et al. evaluated the impact of female sex as a risk modifier for stroke in atrial fibrillation and confirmed an excess risk among females. The risk was more pronounced in those with at least two nonsex-related risk factors.[17] The effect of female sex was notably higher stroke risk in individuals with a CHA2DS2-VASc score of 2, 4, or 5 compared to cisgender males.[17] While the study did not evaluate the impact of estrogen therapy on event risk, it suggests that hormone therapy in MtF patients may warrant consideration, especially in those with additional risk factors.

Decreasing testosterone levels have been linked with increased incidence of atrial fibrillation.[18] In cisgender males older than 55 years, there was a correlated increase in atrial fibrillation risk with each standard deviation decrease in testosterone levels.[18] Additionally, individuals who achieved therapeutic testosterone levels with replacement therapy had a lower risk of atrial fibrillation compared to those with subtherapeutic levels.

QT/QTc

GAHT may influence QTc interval duration and risk for dysrhythmias, particularly if titrated to match levels in cisgender patients.[18] There is limited direct evidence to delineate appropriate changes in QTc changes in TGD patients on GAHT. Studies in cisgender patients on HRT have been used to inform the risks of QTc changes. The available studies also used different cutoffs for QTc prolongation.

The approach to hormone therapy in persons on feminizing hormones mirrors HRT in agonadal and menopausal cisgender females. Research in postmenopausal cisgender females taking HRT demonstrates that estrogen therapy is associated with QTc lengthening and progesterone limits the QTc prolongation.[18]

Effects of endogenous testosterone on repolarization are suspected to be responsible for the shorter QTc in cisgender males.[18] In hypogonadal cisgender males on testosterone HRT, normal testosterone levels reduce the QTc changes and risk of Torsade de Pointes (TdP).[18]

In patients receiving GAHT, it is recommended to avoid supraphysiologic levels of hormones and monitor for QTc changes before and during GAHT. Also, one should consider the individual's overall risk for dysrhythmias and avoid QTc-prolonging medications, especially in patients with baseline QTc prolongation.

VTE Risk

The use of exogenous hormones impacts the risk of VTE and is correlated to the patient's comorbidities, estrogen formulation (type and route), and duration of GAHT.[19,20]

Prediction and risk stratification tools for pulmonary embolism (PE) include the Pulmonary Embolism Rule-out Criteria (PERC), Wells Score for PE, revised Geneva score (RGS), Pulmonary Embolism Severity Index (PESI), simplified PESI (sPESI), and the Hestia criteria.[21] The Wells-PE and RGS do not consider sex, gender, or hormone use as risk factors. However, the PERC incorporates hormone use, assigning one risk point for use of oral contraceptives, HRT, or estrogen use in male or female patients, thus accounting for anyone on GAHT.

The PESI, sPESI, and Hestia criteria are used alongside clinical judgement to help identify patients suitable for outpatient treatment of PE.[21] The PESI assigns 10 points (out of a possible 125) to male gender, which may affect its accuracy in patients taking GAHT. In contrast, the sPESI and Hestia criteria do not rely on gender to inform the clinical decision, potentially making them more accurate for clinical decision.

Nephrology

Literature on assessing renal function in TGD individuals is inconsistent or based upon small observational studies.[22] Although glomerular filtration rate (GFR) can be measured directly, it would be impractical for routine monitoring. Therefore, we utilize estimates of renal function based on surrogate biomarkers including serum creatinine and serum cystatin-C.

Creatinine clearance is dependent on gender and muscle mass, hence changes in serum creatinine. GAHT affects muscle mass, thereby impacting creatinine levels. Serum creatinine changes over time in patients on GAHT, with a significant increase at 6 months in individuals taking testosterone and a decrease at 12 months in those taking estrogen.[23]

Kidney Disease Improving Global Outcomes (KDIGO) recommends the use of the eGFRcr-cys equation to estimate a patient's GFR, which is an average of the eGFRcr and eGFRcys equations. However, both equations may be inaccurate in the TGD population.[22] An alternative for assessing renal function in TGD individuals is the KDIGO CKD EPI eGFRcys equation. Although gender is a variable in this equation, the results are less affected by gender.[22] Another alternative is the European Kidney Function Consortium (EKFC) cystatin equation, which does not differentiate results based on gender. It was found to be unbiased and more accurately reflects directly measured GFR compared to the KDIGO CKD EPI eGFRcys equation.[22,24]

Considering the limitations of the KDIGO CKD EPI equations, it is most appropriate to utilize the EKFCcys calculation to obtain the most accurate assessment of renal function for all patients, as this eliminates confounders relating to gender or muscle mass. Table 4 details which ideal body weight (IBW) equation and gender to use for patients on GAHT using the Cockcroft-Gault equation.[25]

TABLE 4: IBW and gender recommendations for the Cockcroft-Gault equation[25]

Less than 6 months	IBW and gender: based on sex assigned at birth
At least 6 months	IBW and gender: based on gender identity

PULMONARY TESTING

Currently, the American Thoracic Society recommends identifying and respecting gender identity, but utilizing sex assigned at birth for the gender reference range during pulmonary function testing (PFT).[26,27] Altering the gender reference range when interpreting spirometry results can significantly alter the diagnosis of restrictive and obstructive lung disease, potentially leading to misdiagnosis and inappropriate treatment in TGD patients.[26]

PFT uses a cisgender algorithm for lung function estimation. Providers inconsistently apply female and male reference ranges for TGD patients when interpreting PFT. Using male predicted values for a female-sized body can result in pseudorestriction, while the opposite can mask true restriction.[28]

Lung function in patients who start GAHT earlier in puberty will likely align more closely to the lung function of their affirmed gender.[26] However, further research is needed to define the effects of prepubertal gender-affirming hormones on lung size in this population.[27]

Hormones play a role in the development of obstructive sleep apnea in cisgender individuals. Consideration should be given to repeating a sleep study in a patient who starts GAHT.[27]

Individuals who use chest wall binders report shortness of breath, chest pain, scarring, and rib fracture.[27] Up to 50% of binder users report respiratory complaints, yet only approximately 20% seek medical care.[2] Binder use may alter the outcomes of physiologic pulmonary testing.

CONCLUSION

This review highlights the intricate landscape of caring for patients undergoing GAHT and emphasizes the need for further research across multiple domains. Nuanced guidelines that consider both sex assigned at birth and gender identity are essential in providing comprehensive recommendations. Continued investigation and innovation are crucial for advancing towards optimal care and support for this underserved population.

LITERATURE SEARCH AND DATA SOURCES

Current disease state guidelines were utilized to identify commonly recommended scoring systems that included references to gender or hormone therapy. PubMed and Google Scholar were searched using these scoring systems and their associated disease states as primary search terms in addition to the terms gender, female, male, transgender, and hormones.

REFERENCES

1. Deutsch M. *Guidelines for the Primary and Gender-Affirming Care of Transgender and Gender Nonbinary People*. San Franscico, CA: UCSF; 2016.
2. Cheung AS, Lim HY, Cook T, et al. Approach to interpreting common laboratory pathology tests in transgender individuals. *J Clin Endocrinol Metab*. 2021;106(3):893–901. doi: 10.1210/clinem/dgaa546
3. Allen AN, Jiao R, Day P, Pagels P, Gimpel N, SoRelle JA. Dynamic impact of hormone therapy on laboratory values in transgender patients over time. *J Appl Lab Med*. 2021;6(1):27–40. doi: 10.1093/jalm/jfaa192
4. Fernandez JD, Tannock LR. Metabolic effects of hormone therapy in transgender patients. *Endocr Pract*. 2016;22(4):383–388. doi: 10.4158/EP15950.OR
5. Greene DN, McPherson GW, Rongitsch J, et al. Hematology reference intervals for transgender adults on stable hormone therapy. *Clin Chim Acta*. 2019;492:84–90. doi: 10.1016/j.cca.2019.02.011
6. Humble RM, Greene DN, Schmidt RL, et al. Reference intervals for clinical chemistry analytes for transgender men and women on stable hormone therapy. *J Appl Lab Med*. 2022;7(5):1131–1144. doi: 10.1093/jalm/jfac025
7. Sterling J, Garcia MM. Cancer screening in the transgender population: a review of current guidelines, best practices, and a proposed care model. *Transl Androl Urol*. 2020;9(6):2771–2785. doi: 10.21037/tau-20-954
8. Peitzmeier SM, Reisner SL, Harigopal P, Potter J. Female-to-male patients have high prevalence of unsatisfactory Paps compared to non-transgender females: implications for cervical cancer screening. *J Gen Intern Med*. 2014;29(5):778–784. doi: 10.1007/s11606-013-2753-1
9. Hembree WC, Cohen-Kettenis PT, Gooren L, et al. Endocrine treatment of gender-dysphoric/gender-incongruent persons: an Endocrine Society Clinical Practice Guideline [published correction appears in *J Clin Endocrinol Metab*. 2018;103(2):699]. doi: 10.1210/jc.2017-02548] [published correction appears in *J Clin Endocrinol Metab*. 2018;103(7):2758–2759. doi: 10.1210/jc.2018-01268]. *J Clin Endocrinol Metab*. 2017;102(11):3869–3903. doi: 10.1210/jc.2017-01658
10. Coleman E, Radix AE, Bouman WP, et al. Standards of care for the health of transgender and gender diverse people, Version 8. *Int J Transgend Health*. 2022;23(suppl 1):S1–S259. doi: 10.1080/26895269.2022.2100644
11. Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *Circulation*. 2019;140(11):e596–e646. doi: 10.1161/CIR.0000000000000678
12. 12. Caceres BA, Streed CG Jr, Corliss HL, et al. Assessing and addressing cardiovascular health in LGBTQ adults: a scientific statement from the American Heart Association. *Circulation*. 2020;142(19):e321–e332. doi: 10.1161/CIR.0000000000000914
13. Streed CG Jr, Beach LB, Caceres BA, et al. Assessing and addressing cardiovascular health in people who are transgender and gender diverse: a scientific statement from the American Heart Association [published correction appears in Circulation. 2021;144(6):e150]. *Circulation*. 2021;144(6):e136–e148. doi: 10.1161/CIR.0000000000001003
14. Orringer CE, Blaha MJ, Blankstein R, et al. The National Lipid Association scientific statement on coronary artery calcium scoring to guide preventive strategies for ASCVD risk reduction. *J Clin Lipidol*. 2021;15(1):33–60. doi: 10.1016/j.jacl.2020.12.005
15. Duro T, McClain M, Aragon KG, et al. Risk assessment and coronary artery calcium scoring in transgender and gender-diverse individuals receiving gender-affirming hormone therapy. *Endocr Pract*. 2023;29(4):229–234. doi: 10.1016/j.eprac.2022.12.007
16. January CT, Wann LS, Alpert JS, et al. 2014 AHA/ACC/HRS guideline for the management of patients with atrial fibrillation: a report of the American College of Cardiology/American Heart Association Task Force on practice guidelines and the Heart Rhythm Society [published correction appears in Circulation. 2014;130(23):e272–e274]. *Circulation*. 2014;130(23):e199–e267. doi: 10.1161/CIR.0000000000000041
17. Nielsen PB, Overvad TF. Female sex as a risk modifier for stroke risk in atrial fibrillation: using CHA2DS2-VASc versus CHA2DS2-VA for stroke risk stratification in atrial fibrillation: a note of caution. *Thromb Haemost*. 2020;120(6):894–898. doi: 10.1055/s-0040-1710014
18. Grouthier V, Moey MYY, Gandjbakhch E, et al. Sexual dimorphisms, anti-hormonal therapy and cardiac arrhythmias. *Int J Mol Sci*. 2021;22(3):1464. doi: 10.3390/ijms22031464
19. Rosendale N, Goldman S, Ortiz GM, Haber LA. Acute clinical care for transgender patients: a review. *JAMA Intern Med*. 2018;178(11):1535–1543. doi: 10.1001/jamainternmed.2018.4179
20. Shatzel JJ, Connelly KJ, DeLoughery TG. Thrombotic issues in transgender medicine: a review. *Am J Hematol*. 2017;92(2):204–208. doi: 10.1002/ajh.24593
21. American College of Emergency Physicians Clinical Policies Subcommittee (Writing Committee) on Thromboembolic Disease: Wolf SJ, Hahn SA, et al. Clinical Policy: Critical Issues in the Evaluation and Management of Adult Patients Presenting to the Emergency Department With Suspected Acute Venous Thromboembolic Disease. *Ann Emerg Med*. 2018;71(5):e59–e109. doi: 10.1016/j.annemergmed.2018.03.006
22. Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2024 Clinical Practice Guideline for the Evaluation and Management of Chronic Kidney Disease. *Kidney Int*. 2024;105(4S):S117–S314. doi: 10.1016/j.kint.2023.10.018
23. Humble RM, Imborek KL, Nisly N, Greene DN, Krasowski MD. Common hormone therapies used to care for transgender patients influence laboratory results. *J Appl Lab* Med. 2019;3(5):799–814. doi: 10.1373/jalm.2018.027078

24. Pottel H, Björk J, Rule AD, et al. Cystatin C-based equation to estimate GFR without the inclusion of race and sex. *N Engl J Med*. 2023;388(4):333–343. doi: 10.1056/NEJMoa2203769

25. Webb AJ, McManus D, Rouse GE, Vonderheyde R, Topal JE. Implications for medication dosing for transgender patients: a review of the literature and recommendations for pharmacists. *Am J Health Syst Pharm*. 2020;77(6):427–433. doi: 10.1093/ajhp/zxz355

26. Roth KA. Spirometry for transgender and nonbinary patients. *Ann Am Thorac Soc*. 2023;20(7):944–946. doi: 10.1513/AnnalsATS.202301-033IP

27. Turner GA, Amoura NJ, Strah HM. Care of the transgender patient with a pulmonary complaint [published correction appears in *Ann Am Thorac Soc*. 2021;18(8):1432]. *Ann Am Thorac Soc*. 2021;18(6):931–937. doi: 10.1513/AnnalsATS.202007-801CME

28. Foer D, Rubins D, Almazan A, Wickner PG, Bates DW, Hamnvik OR. Gender reference use in spirometry for transgender patients. *Ann Am Thorac Soc*. 2021;18(3):537–540. doi: 10.1513/AnnalsATS.202002-103RL

REVIEW ARTICLE

Childhood Obesity: Updates on Current Treatments

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ABSTRACT

Rates of obesity among adults and children in the United States have been on the rise for decades and continue to increase based on recent studies. This is due to a combination of individual, environmental, and socioeconomic factors. Most recent studies indicate that 19.7%, or approximately 14.7 million children and adolescents, are affected by obesity. Addressing and preventing obesity in this population requires a comprehensive approach that includes counseling on healthy diet and exercise, behavioral interventions, possible pharmacotherapy, and, in some cases, surgical referral. A team-based approach is recommended, involving physicians, exercise physiologists, physical therapists, dietitians, diet assistants, psychologists, and social workers. While therapeutic lifestyle changes are the primary focus of treatment, providers must also consider socioeconomic factors, mental health, treatment of comorbidities, and familial factors in their treatment plans. Primary care providers should be familiar with all treatment options, including pharmacotherapy and when to refer for bariatric surgical consultation. This article aims to summarize the risks and consequences of childhood obesity, outline the approach the osteopathic family physician can take to treat the pediatric patient with obesity, and provide updates on the latest guidance and recommendations available.

INTRODUCTION

Obesity arises from a complex combination of factors, including various individual, environmental, and socioeconomic influences.[1] The prevalence of obesity among adults and children has been on the rise for decades[2] and continues to rise, according to the latest studies.[3] Between 2017 and March 2020, the rate of obesity among children and adolescents in the United States was 19.7%, indicating that approximately 14.7 million youths aged 2 to 19 years were affected by obesity.[3] This is a stark increase from the 16.9% of children with obesity in 2009 to 2010.[4] As osteopathic family physicians are particularly well suited to provide comprehensive care through a holistic treatment approach for all patients, this article aims to summarize the risks and consequences of childhood obesity, outline the approach the osteopathic family physician can take to treat the pediatric patient with obesity, and provide updates on the latest guidance and recommendations available.

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DEFINITION

Obesity is characterized by an excessive accumulation of body fat, typically assessed in adults through body mass index (BMI), calculated by dividing body weight in kilograms by the square of height in meters. Obesity is characterized by a BMI equal to or greater than 30.0 kg/m^2, while severe obesity is indicated by a BMI equal to or greater than 40.0 kg/m^2.[5] BMI serves as a widely recognized screening tool for obesity in children over 6 years old and is endorsed with Grade B evidence by the US Preventive Services Task Force (USPSTF).[6,7] For children, BMI is adjusted based on percentiles for age and sex: normal weight (5th to 84.9th percentile), overweight (85th to 94.9th percentile), obesity (≥95th percentile to 120% of 95th percentile), class 2 obesity (>120% of 95th percentile), and class 3 obesity (>140% of 95th percentile).[8] Although BMI has limitations, it remains the simplest method for identifying obesity.[9] Notably, there is no universally accepted definition of obesity in children under 24 months[10]; however, those at risk can be identified using World Health Organization (WHO) weight-for-length charts.[11]

COMORBIDITIES/COMPLICATIONS

Many adults with obesity have other serious chronic diseases. Examples include 58% of US adults with obesity have high blood pressure, a risk factor for heart disease, and about 23% of adults with obesity have diabetes.[3]

Similar patterns can be seen in children as numerous potential consequences have been identified, including cardiovascular disease, type 2 diabetes mellitus, asthma, obstructive sleep apnea, musculoskeletal disorders, nonalcoholic fatty liver disease, attention deficit/hyperactivity disorder, conduct disorder, depression, learning disability, developmental delay, and lower executive function.[12-20] Moreover, childhood obesity has been linked to a higher risk of cancer, particularly multiple myeloma, colorectal, uterine corpus, gallbladder, kidney, and pancreatic cancers in young adults who were obese as children.[21,22] Children with obesity also face elevated risks of discrimination and bullying.[23] Additionally, these health challenges often persist into adulthood, as adolescents with obesity are five times more likely to have obesity as adults, with approximately 80% maintaining their weight status into adulthood.[24]

DEMOGRAPHICS AND RISK FACTORS

Childhood and adolescent obesity stem from a complex interplay of genetic, behavioral, and environmental factors. Healthcare providers should prioritize monitoring patients with predisposing factors for obesity, such as parental obesity, inadequate nutrition, reduced physical activity, sedentary lifestyles, insufficient sleep, high consumption of sugary drinks and fast food, bedroom television, low family income, and food insecurity.[25,26] It is crucial for clinicians to also consider risk factors in younger children, including maternal/gestational diabetes, gestational hypertension, maternal smoking, excessive gestational weight gain, and rapid infant growth.[25,27]

When reviewing the statistics on obesity prevalence by patient demographics, it should be noted that some groups are disproportionately affected more than others. Obesity prevalence among children and adolescents aged 2 to 19 years varied by age, race/ethnicity, and family income. Rates increased with age, ranging from 12.7% among those aged 2 to 5 years to 22.2% among those aged 12 to 19 years. Highest prevalence was observed among Hispanic (26.2%) and non-Hispanic black (24.8%) youths, followed by non-Hispanic white (16.6%) and non-Hispanic Asian (9.0%) youths. Obesity rates generally decreased with higher family income levels. Overall, obesity prevalence did not significantly differ between boys (20.9%) and girls (18.5%), except among specific age groups and racial/ethnic categories. Among US girls, obesity prevalence was highest among non-Hispanic Black girls (30.8%). Among US boys, obesity prevalence was highest among Hispanic boys (29.3%). Boys had higher obesity rates than girls among those aged 6 to 11 years and among non-Hispanic Asian youths and those from higher-income families. Conversely, among non-Hispanic black youths, obesity prevalence was lower in boys compared to girls.[3]

The American Academy of Pediatrics (AAP) guidelines published in 2023 acknowledge that obesity is a multifaceted chronic disease whose risk and treatment challenges are impacted by adverse childhood experiences (ACEs) and social determinants of health (SDoH). The guidelines also highlight the fact that obesity does not affect all demographics equally, requiring providers to be aware of the impact of factors like racism on management of childhood obesity.[28]

Patterns of obesity prevalence can vary based on sociodemographic characteristics, such as age, race/ethnicity, and family income. When looking at the effect income can have on childhood obesity, research has shown obesity prevalence increases as family income decreases with obesity noted in 11.5% of US children with family income more than 350% of the Federal Poverty Level (FPL), 21.2% with family income 130% to 350% of FPL, and 25.8% with family income 130% or less of FPL.[3]

TREATMENT

The aim of treating children and adolescents with overweight or obesity is to establish healthy habits and lifestyle patterns that will ideally continue into adulthood, thereby preventing future health issues, enhancing quality of life, and boosting body image and self-esteem.[29] The treatment strategy should be patient-centered and holistic, encompassing nutrition, physical activity, behavioral changes, and, in certain cases, pharmacotherapy or bariatric surgery. For some patients, an interdisciplinary team-based approach might be most effective.[30] The USPSTF has found that a comprehensive, team-based method involving primary care clinicians, exercise physiologists, physical therapists, dietitians, diet assistants, psychologists, and social workers can be beneficial.[6]

A primary responsibility of the clinician is to educate patients and their families, encouraging them to adopt healthy lifestyles.[14] Although there is no single diet recommended for all children, certain general guidelines can help reduce obesity. Consumption of sugar-sweetened beverages, processed foods, fast food, candies, snacks, cakes, animal products, whole milk, and refined grains is linked to higher obesity rates.[31] Clinicians should inform patients and their parents about these associations to provide general dietary guidance. Conversely, diets low in sugar and fat but high in fruits, vegetables, whole grains, fish, nuts, legumes, and yogurt are less likely to be associated with obesity.[31]

Behavioral Approach

Behavioral interventions have proven effective in improving weight management for children and adolescents. To be effective, behavioral interventions should combine education on physical activity and nutrition, promote

healthy behaviors through goal-directed change, and be delivered frequently over an extended period.[6,32]

Effective behavioral interventions must also be personalized and specific. Healthcare providers should use their initial assessment of the patient's dietary and physical activity history to identify gaps relative to recommended behaviors. They should also identify any barriers that have previously prevented the patient from achieving a healthy weight and assess the readiness of the patient and their family to commit to change.[33] This information can be effectively gathered through motivational interviewing, a communication style in which the provider asks questions to understand and strengthen the patient's commitment to change.[34]

Providers should use a family-centered and nonstigmatizing approach that acknowledges obesity's biologic, social, and structural drivers. Motivational interviewing should be utilized when appropriate during the evaluation process. A referral to intensive health behavior and lifestyle treatment should be offered by providers to patients aged 6 years or older and may be considered in those aged 2 to 5 years. Research has found that greater contact hours lead to more effective treatments, being most beneficial when including at least 26 hours of multicomponent treatment over 3 to 12 months.[6]

In many cases, intensive treatment may not be available. If this is the case, providers should increase the intensity of weight-management support through collaboration with resources in the community to support nutrition, food insecurity, physical activity, and other SDoH. Some examples include food provision programs, local parks, and other recreation programs.[28]

Physical activity recommendations for children are based on the amount of time and level of intensity (see Table 1).[35] Encouraged activities should be age-appropriate, varied, and enjoyable for the child.[36] Priority should be given to activities that develop fundamental movement skills (such as running, kicking, throwing, catching, jumping, and balancing), as children who master these skills are more likely to remain physically active as they grow older.[37] Additionally, healthcare providers should focus on reducing sedentary time by limiting nonacademic screen time and replacing it with physical activity whenever possible.[33]

For children who are not meeting the recommended levels of physical activity, families should be provided with examples of age-appropriate activities that match the child's skill level and intensity requirements. The duration and frequency of these activities should be gradually increased in small increments until the recommended amount is reached.[35]

TABLE 1: Physical activity recommendations for children[35]

Age	Activity Amount	Activity Intensity*
3 to 5 years old	>180 minutes/day	Any intensity; some moderate to vigorous
5 to 17 years old	>60 minutes/day	Moderate to vigorous intensity; activity type should include bone/muscle strengthening 3 days/week

*Activity intensity is rated as light, moderate, or vigorous. Light is defined by MET (metabolic equivalent of task) <3 (e.g., walking, playing catch). Moderate is defined by MET 3 to 6 (e.g., jogging, yardwork). Vigorous is defined by MET >6 (e.g., running, ice skating, jumping rope).

Dietary recommendations for children and adolescents, including calorie intake and diet composition, vary by age and gender. The Dietary Reference Intake Calculator for Healthcare Professionals can help estimate calorie and nutrient needs. It is also important to specifically address the intake of sugar-sweetened beverages (such as soda, fruit drinks, and sports drinks), which are high in calories and added sugars.[38] If it is determined that the dietary calorie intake is excessive, the focus should be on specific behaviors that increase calorie intake (e.g., avoiding soda) rather than setting strict calorie limits (e.g., consuming less than 2000 calories per day).

The provider should establish weight targets as a way to monitor improvement in weight over time. This goal can be shared with patients and their families, but providers should be mindful not to prioritize weight change over behavior change. The target for children and adolescents will vary based on a number of factors, such as age and BMI percentile.[6,32]

Adherence to a behavioral intervention is considered crucial for its success.[25] Follow-up visits should be scheduled based on the patient's readiness to change and the type of counseling provided. Typically, visits are scheduled monthly during the initial stages of weight management but may be more frequent, such as weekly, for more intensive interventions. Weight targets should be reassessed every 3 to 6 months. If there is no progress toward the weight target within 6 to 12 months, the patient should be evaluated for alternative treatment options or referred to a weight management specialist. If weight loss exceeds recommended rates, the patient should be screened for an eating disorder and referred for appropriate care.[6,32]

Pharmacotherapy

When lifestyle modifications and behavioral interventions are insufficient to manage obesity, medications can be considered as an additional therapy (see Table 2). Until recently, only three medications were approved for weight loss in adolescents: phentermine for children over 16 years old, and orlistat and liraglutide for children aged 12

and older.[39-41] However, in 2022, once-weekly semaglutide and combination phentermine/topiramate were approved in adolescents as well.[42,43]

Phentermine is a sympathomimetic amine first approved in 1959. It is a DEA Schedule IV stimulant agent approved for short-term use (12 weeks). Some patients may lose about 5% of their body weight. Phentermine causes an increase in release of norepinephrine from the hypothalamus resulting in hunger suppression and a slight increase in energy expenditure. It is approved for patients >16 years of age.[39] Phentermine should not be used with overactive thyroid or uncontrolled high blood pressure or seizure disorder. It is contraindicated in patients with history of cardiovascular disease, glaucoma, agitated states, drug abuse, or within 14 days of monoamine oxidase inhibitor use.

Combination of phentermine and topiramate is also approved as a DEA Schedule IV drug. Some patients may lose an average of 5% to 10% of their body weight. It is approved for patients ≥12 years of age.[44] The mechanism of weight loss with topiramate is unclear. Topiramate has an inhibitory action on glutamate neurotransmission. Glutamate stimulation of the lateral hypothalamus increases food intake. Also, topiramate has been associated with decreased levels of neuropeptide Y, a potent appetite-stimulating neurohormone. Phentermine/topiramate combination should be discontinued with acute myopia and secondary angle glaucoma and should not be used with glaucoma or hyperthyroidism. Topiramate can cause birth defects so phentermine/topiramate should not be started until a pregnancy test is negative. Thereafter, the FDA recommends women use effective contraception and have monthly pregnancy tests during treatment with phentermine/topiramate.

Orlistat is a gastrointestinal lipase inhibitor that impairs digestion of dietary fat. Lower doses are approved over the counter. Some patients may lose about 5% of their body weight. It is approved in patients ≥12 years of age.[40] Potential side effects include oily discharge with flatus from the rectum, especially after fatty foods, and it may promote gallstones and kidney stones. There is also potential for malabsorption of fat-soluble vitamins (A, D, E, K). As such, patients should take a multivitamin daily. It is contraindicated in chronic malabsorption syndrome and cholestasis.

Semaglutide is a weekly injectable GLP-1 receptor agonist. At lower doses (1 mg per week), semaglutide is indicated to lower blood sugar among patients with type 2 diabetes mellitus, while it is approved for treatment of obesity at doses up to 2.4 mg. Some patients may lose up to 15% of their body weight with the higher dose. Semaglutide results in increased insulin secretion inhibition of glucagon release and gluconeogenesis. There is also delayed gastric emptying and reduced appetite. Semaglutide is approved in patients ≥12 years of age with an initial BMI in the ≥95th percentile for age and sex.[42]

Similar to semaglutide, liraglutide is a GLP-1 agonist also approved for obesity in patients ≥12 years of age with an initial BMI in the ≥95th percentile for age and sex.[41] The difference, in this case, is the medication is injected daily as opposed to weekly. Potential side effects are similar to semaglutide.

Despite the limited pharmacologic options for treating obesity in children, clinicians play a crucial role in managing the impact of medications on a patient's weight. When treating other conditions in pediatric patients with overweight and obesity, it is important to avoid obesogenic medications when possible. The Endocrine Society's 2016 guidelines provide recommendations for pharmacologic treatments to minimize further weight gain due to medication side effects.[45]

TABLE 2: Medications approved for weight loss in pediatric patients[29]

Medication	Minimum Age for Use	Mechanism of Action	Potential Side Effects
Phentermine	>16 years of age	Sympathomimetic amine; increases norepinephrine release from the hypothalamus; suppresses hunger and increases resting energy expenditure	Dry mouth, headache, elevated blood pressure or heart rate, tremor, insomnia
Phentermine/ topiramate	≥12 years of age	Phentermine	Phentermine
Orlistat	≥12 years of age	Topiramate has an inhibitory action on glutamate neurotransmission and has been associated with decreased levels of neuropeptide Y	Paresthesia, dizziness, dysgeusia, insomnia, constipation, dry mouth
Liraglutide	≥12 years of age	Inhibits gastric and pancreatic lipases, reducing absorption of fat	Oily discharge with flatus from the rectum; increased risk of gallstones and kidney stones; may cause malabsorption of fat-soluble vitamins (A, D, E, K)
Semaglutide	≥12 years of age	GLP-1 receptor agonist (weekly)	Nausea, hypoglycemia, diarrhea, constipation, vomiting, headache, decreased appetite, dyspepsia, fatigue, dizziness, abdominal pain, increased lipase, and renal insufficiency

Bariatric Surgery

Although bariatric surgery is not studied as extensively in adolescents as it is in adults, children with obesity and comorbid conditions who have not responded to comprehensive behavioral interventions might be candidates for surgical or device therapy. The AAP suggests considering bariatric surgery for patients with Class 2 obesity (BMI ≥35 kg/m^2, or 120% of the 95th percentile for age and sex, whichever is lower) with significant comorbid conditions, and for patients with Class 3 obesity (BMI ≥40 kg/m^2, or 140% of the 95th percentile for age and sex, whichever is lower).[46] Adolescent patients pursuing bariatric surgery face various challenges, including lack of insurance approval,[47] limited provider knowledge,[48] and insufficient access to tertiary care facilities equipped for pediatric bariatric surgery.[49] In 2022, the American Society for Metabolic and Bariatric Surgery (ASMBS) published updated guidelines with further evidence supporting the benefits of bariatric surgery in this population.[50]

CONCLUSION

Although bariatric surgery is not studied as extensively in adolescents as it is in adults, children with obesity and comorbid conditions who have not responded to comprehensive behavioral interventions might be candidates for surgical or device therapy. The AAP suggests considering bariatric surgery for patients with Class 2 obesity (BMI ≥35 kg/m^2, or 120% of the 95th percentile for age and sex, whichever is lower) with significant comorbid conditions, and for patients with Class 3 obesity (BMI ≥40 kg/m^2, or 140% of the 95th percentile for age and sex, whichever is lower).[46] Adolescent patients pursuing bariatric surgery face various challenges, including lack of insurance approval,[47] limited provider knowledge,[48] and insufficient access to tertiary care facilities equipped for pediatric bariatric surgery.[49] In 2022, the American Society for Metabolic and Bariatric Surgery (ASMBS) published updated guidelines with further evidence supporting the benefits of bariatric surgery in this population.[50]

REFERENCES

1. Jastreboff AM, Kotz CM, Kahan S, Kelly AS, Heymsfield SB. Obesity as a disease: The Obesity Society 2018 Position Statement. *Obesity (Silver Spring)*. 2019;27(1):7–9. doi: 10.1002/oby.22378
2. Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among U.S. adults, 1999-2000. *JAMA*. 2002;288(14):1723–1727. doi: 10.1001/jama.288.14.1723
3. Stierman B, Afful J, Carroll MD, et al. National Health and Nutrition Examination Survey 2017–March 2020 prepandemic data files development of files and prevalence estimates for selected health outcomes. *Natl Health Stat Report*. 2021;158. doi: 10.15620/cdc:106273
4. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010. *JAMA*. 2012;307(5):483–490. doi: 10.1001/jama.2012.40
5. Centers for Disease Control and Prevention. Adult obesity facts. https://www.cdc.gov/obesity/php/data-research/adult-obesity-facts.html#:~:text=Many%20U.S.%20adults%20have%20obesity&text=The%20prevalence%20of%20obesity%20increased,BMI)%20of%2030.0%20or%20higher. Accessed June 17, 2024.
6. US Preventive Services Task Force. Screening for obesity in children and adolescents: US Preventive Services Task Force recommendation statement. *JAMA*. 2017;317(23):2417–2426. doi: 10.1001/jama.2017.6803
7. United States Preventive Services Taskforce. High body mass index in children and adolescents: interventions. Draft recommendation: high body mass index in children and adolescents: interventions. https://www.uspreventiveservicestaskforce.org/uspstf/draft-recommendation/high-body-mass-index-children-adolescents-interventions#fullrecommendationstart. Accessed June 17, 2024.
8. Gulati AK, Kaplan DW, Daniels SR. Clinical tracking of severely obese children: a new growth chart. *Pediatrics*. 2012;130(6):1136–1140. doi: 10.1542/peds.2012-0596
9. Brown CL, Perrin EM. Obesity prevention and treatment in primary care. *Acad Pediatr*. 2018;18(7):736–745. doi: 10.1016/j.acap.2018.05.004
10. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011-2012. *JAMA*. 2014;311(8):806–814. doi: 10.1001/jama.2014.732
11. Daniels SR, Hassink SG; Committee on Nutrition. The role of the pediatrician in primary prevention of obesity. *Pediatrics*. 2015;136(1):e275–e292. doi: 10.1542/peds.2015-1558
12. Umer A, Kelley GA, Cottrell LE, Giacobbi P Jr, Innes KE, Lilly CL. Childhood obesity and adult cardiovascular disease risk factors: a systematic review with meta-analysis. *BMC Public Health*. 2017;17(1):683. doi: 10.1186/s12889-017-4691-z
13. Bacha F, Gidding SS. Cardiac abnormalities in youth with obesity and type 2 diabetes. *Curr Diab Rep*. 2016;16(7):62. doi: 10.1007/s11892-016-0750-6
14. Caprio S, Santoro N, Weiss R. Childhood obesity and the associated rise in cardiometabolic complications. *Nat Metab*. 2020;2(3):223–232. doi: 10.1038/s42255-020-0183-z
15. Peters U, Dixon AE, Forno E. Obesity and asthma. *J Allergy Clin Immunol*. 2018;141(4):1169–1179. doi: 10.1016/j.jaci.2018.02.004
16. Kumar S, Kelly AS. Review of childhood obesity: from epidemiology, etiology, and comorbidities to clinical assessment and treatment. *Mayo Clin Proc*. 2017;92(2):251–265. doi: 10.1016/j.mayocp.2016.09.017
17. Pollock NK. Childhood obesity, bone development, and cardiometabolic risk factors. *Mol Cell Endocrinol*. 2015;410:52–63. doi: 10.1016/j.mce.2015.03.016
18. Africa JA, Newton KP, Schwimmer JB. Lifestyle interventions including nutrition, exercise, and supplements for nonalcoholic fatty liver disease in children. *Dig Dis Sci*. 2016;61(5):1375–1386. doi: 10.1007/s10620-016-4126-1
19. Quek YH, Tam WWS, Zhang MWB, Ho RCM. Exploring the association between childhood and adolescent obesity and depression: a meta-analysis. *Obes Rev*. 2017;18(7):742–754. doi: 10.1111/obr.12535
20. Rollins BY, Riggs NR, Francis LA, Blair CB. Executive function and BMI trajectories among rural, poor youth at high risk for obesity. *Obesity (Silver Spring)*. 2021;29(2):379–387. doi: 10.1002/oby.23064

21. Sung H, Siegel RL, Rosenberg PS, Jemal A. Emerging cancer trends among young adults in the USA: analysis of a population-based cancer registry. *Lancet Public Health*. 2019;4(3):e137–e147. doi: 10.1016/s2468-2667(18)30267-6

22. Berger NA. Young adult cancer: influence of the obesity pandemic. *Obesity (Silver Spring)*. 2018;26(4):641–650. doi: 10.1002/oby.22137

23. Beck AR. Psychosocial aspects of obesity. *NASN Sch Nurse*. 2016;31(1):23–27. doi: 10.1177/1942602X15619756

24. Simmonds M, Llewellyn A, Owen CG, Woolacott N. Predicting adult obesity from childhood obesity: a systematic review and meta-analysis. *Obes Rev*. 2016;17(2):95–107. doi: 10.1111/obr.12334

25. O'Connor EA, Evans CV, Burda BU, Walsh ES, Eder M, Lozano P. Screening for obesity and intervention for weight management in children and adolescents: evidence report and systematic review for the US Preventive Services Task Force. *JAMA*. 2017;317(23):2427–2444. doi: 10.1001/jama.2017.0332

26. Frongillo EA, Bernal J. Understanding the coexistence of food insecurity and obesity. *Curr Pediatr Rep*. 2014;2(4):284–290. doi: 10.1007/s40124-014-0056-6

27. Patro Golab B, Santos S, Voerman E, et al. Influence of maternal obesity on the association between common pregnancy complications and risk of childhood obesity: an individual participant data meta-analysis. *Lancet Child Adolesc Health*. 2018;2(11):812–821. doi: 10.1016/S2352-4642(18)30273-6

28. Hampl SE, Hassink SG, Skinner AC, et al. Clinical practice guideline for the evaluation and treatment of children and adolescents with obesity. *Pediatrics*. 2023;151(2):e2022060640. doi: 10.1542/peds.2022-060640. Erratum in: Pediatrics. 2024;153(1):e2023064612. doi: 10.1542/peds.2023-064612

29. Cuda S, Censani M, O'Hara V, et al; Obesity Medicine Association. Pediatric obesity algorithm. https://obesitymedicine.org/resources/obesity-algorithm/pediatric-obesity-algorithm/. Accessed June 26, 2024.

30. Styne DM, Arslanian SA, Connor EL, et al. Pediatric obesity—assessment, treatment, and prevention: an Endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2017;102(3):709–757. doi:10.1210/jc.2016-2573

31. Liberali R, Kupek E, Assis MAA. Dietary patterns and childhood obesity risk: a systematic review. *Child Obes*. 2020;16(2):70–85. doi: 10.1089/chi.2019.0059

32. Spear BA, Barlow SE, Ervin C, et al. Recommendations for treatment of child and adolescent overweight and obesity. *Pediatrics*. 2007;120(suppl 4):S254–S288. doi: 10.1542/peds.2007-2329F

33. Hagan JF, Shaw JS, Duncan PM. Bright Futures: *Guidelines for Health Supervision of Infants, Children, and Adolescents*. 4th ed. American Academy of Pediatrics; 2017.

34. Miller WR, Rollnick S. Motivational Interviewing: *Helping People Change*. 3rd ed. Guilford Press; 2013.

35. 2018 Physical Activity Guidelines Advisory Committee. 2018 Physical Activity Guidelines Advisory Committee Scientific Report. U.S. Department of Health and Human Services, 2018. https://health.gov/sites/default/files/2019-09/PAG_Advisory_Committee_Report.pdf. Accessed June 26, 2024.

36. Lipnowski S, Leblanc CM; Canadian Paediatric Society, Healthy Active Living and Sports Medicine Committee. Healthy active living: physical activity guidelines for children and adolescents. *Paediatr Child Health*. 2012;17(4):209–212. doi: 10.1093/pch/17.4.209

37. Jaakkola T, Yli-Piipari S, Huotari P, Watt A, Liukkonen J. Fundamental movement skills and physical fitness as predictors of physical activity: a 6-year follow-up study. *Scand J Med Sci Sports*. 2016; 26(1):74–81. doi: 10.1111/sms.12407

38. U.S. Department of Agriculture and U.S. Department of Health and Human Services. Dietary Guidelines for Americans, 2020-2025. 9th ed. December 2020.

39. Ryder JR, Kaizer A, Rudser KD, Gross A, Kelly AS, Fox CK. Effect of phentermine on weight reduction in a pediatric weight management clinic. *Int J Obes (Lond)*. 2017;41(1):90–93. doi: 10.1038/ijo.2016.185

40. Chanoine JP, Hampl S, Jensen C, Boldrin M, Hauptman J. Effect of orlistat on weight and body composition in obese adolescents: a randomized controlled trial. *JAMA*. 2005;293(23):2873–2883. doi: 10.1001/jama.293.23.2873. Erratum in: *JAMA*. 2005;294(12):1491.

41. Kelly AS, Auerbach P, Barrientos-Perez M, et al. A randomized, controlled trial of liraglutide for adolescents with obesity. *N Engl J Med*. 2020;382(22):2117–2128. doi: 10.1056/NEJMoa1916038

42. Weghuber D, Barrett T, Barrientos-Pérez M, et al. Once-weekly semaglutide in adolescents with obesity. *N Engl J Med*. 2022;387(24):2245–2257. doi: 10.1056/nejmoa2208601

43. Center for Drug Evaluation and Research. FDA approves treatment for chronic weight management in Pediatric Pati. U.S. Food and Drug Administration. June 27, 2022. https://www.fda.gov/drugs/news-events-human-drugs/fda-approves-treatment-chronic-weight-management-pediatric-patients-aged-12-years-and-older. Accessed June 17, 2024.

44. Kelly AS, Bensignor MO, Hsia DS, et al. Phentermine/topiramate for the treatment of adolescent obesity. *NEJM Evid*. 2022;1(6):10.1056/evidoa2200014. doi: 10.1056/evidoa2200014

45. Apovian CM, Aronne LJ, Bessesen DH, et al. Pharmacological management of obesity: an endocrine Society clinical practice guideline. *J Clin Endocrinol Metab*. 2015;100(2):342–362. doi: 10.1210/jc.2014-3415. Erratum in: J Clin Endocrinol Metab. 2015;100(5):2135–2136. doi: 10.1210/jc.2015-1782

46. 46. Armstrong SC, Bolling CF, Michalsky MP, Reichard KW. Pediatric metabolic and bariatric surgery: evidence, barriers, and best practices. *Pediatrics*. 2019;144(6):e20193223. doi: 10.1542/peds.2019-3223

47. Inge TH, Boyce TW, Lee M, et al. Access to care for adolescents seeking weight loss surgery. *Obesity (Silver Spring)*. 2014;22(12):2593–2597. doi: 10.1002/oby.20898

48. Vanguri P, Lanning D, Wickham EP, Anbazhagan A, Bean MK. Pediatric health care provider perceptions of weight loss surgery in adolescents. *Clin Pediatr (Phila)*. 2014;53(1):60–65. doi: 10.1177/0009922813500848

49. Bolling CF, Armstrong SC, Reichard KW, Michalsky MP. Metabolic and bariatric surgery for pediatric patients with severe obesity. Pediatrics. 2019;144(6):e20193224. doi: 10.1542/peds.2019-3224

50. Eisenberg D, Shikora SA, Aarts E, Sent al. 2022 American Society for Metabolic and Bariatric Surgery (ASMBS) and International Federation for the Surgery of Obesity and Metabolic Disorders (IFSO): indications for metabolic and bariatric surgery. *Surg Obes Relat Dis*. 2022;18(12):1345–1356. doi: 10.1016/j.soard.2022.08.013

BRIEF REPORT

Osteopathic Consideration of Pelvic Girdle Pain in the Postpartum Patient: A Case Study

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KEYWORDS

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ABSTRACT

Pelvic girdle pain (PGP) during and after pregnancy can present diagnostic and therapeutic challenges. In our case, a 31-year-old Asian American woman developed PGP and lower back pain that was not relieved 3 months' postpartum. She was treated with over-the-counter analgesics and physical therapy for 12 weeks with minimal and temporary improvement. The patient was offered OMT and examined nine months' postpartum. A full osteopathic structural exam was performed along with treatment, and exercises were recommended. OMT was focused on sacroiliac and pelvic techniques. A significant reduction in pain was reported posttreatment, followed by complete pain resolution, indicating great benefit of incorporation of OMT into the treatment plan.

INTRODUCTION

Pelvic girdle pain (PGP) is a common issue during pregnancy, affecting 15% to 25% of pregnant individuals, with a higher prevalence of 20% to 30% overall and up to 50% in some cases.[1] It typically arises from increased uterine pressure, lumbar lordosis, and relaxation of pelvic ligaments.[1] Specifically, PGP is a type of pain that affects the pelvic region, typically centered around the joints connecting the hip bones to the spine and surrounding muscles.[2] Symptoms include lower back pain, especially between the posterior iliac crest and the gluteal fold, and around the sacroiliac joint, which can radiate to the thighs and hips and worsen with weight bearing.[3] PGP diagnosis is primarily clinical, involving physical examinations to assess pain and mobility in the pelvic region.[2] Imaging, such as ultrasound or magnetic resonance imaging (MRI), is occasionally used, especially if a diagnosis is unclear, but physical exams remain central for most cases.[2] Regarding delivery methods, studies have found mixed results. Some suggest that women who experience PGP during pregnancy may have lingering symptoms postpartum, but there is limited evidence specifically comparing pain outcomes between those who deliver vaginally and those who have cesarean sections.[2] Common management options encompass nonpharmacologic approaches like heat therapy, physical therapy, braces, osteopathic manipulative techniques, and pharmacologic interventions such as acetaminophen.[4] While most cases resolve within 12 weeks' postdelivery, approximately one in four women may experience chronic postpartum pain.[5] Various risk factors contribute to PGP development, including prior low back pain, pelvic trauma, stress, multiparity, and low job satisfaction, while nonrisk factors include contraceptive use, time since past pregnancy, height, weight, and smoking.[6] Persistent pain may necessitate individualized exercise prescription following guidelines.[6] In our study, we considered the use of osteopathic treatment and a continuous exercise program for the treatment of PGP in postpartum patients.

CASE DESCRIPTION

The patient is a 31 year-old gravida 1 para 1 Asian American woman who presented with PGP and persistent lower back pain despite multiple trials of Tylenol and nonsteroidal anti-inflammatory drugs three months' postpartum after her first vaginal delivery. The patient was questioned to evaluate potential risk factors, including prior low back pain, pelvic trauma, stress, multiparity, and low job satisfaction, all of which she denied. The patient reported that the lower back pain occurred

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randomly during daily activities and denied any consistent correlation with specific times or positions. Osteopathic structural exam and treatment was performed by multiple OMS-IIs under osteopathic manipulative medicine (OMM) faculty member supervision. Examination of the pelvis revealed bilateral tenderness of the sacroiliac joint, a positive standing flexion test on the right, a superior left anterior superior iliac spine (ASIS) with a superior right posterior superior iliac spine (PSIS), pubic bones of the same height, and a shorter leg on the left. Diagnosis of a right anterior innominate somatic dysfunction was made. Further testing demonstrated a positive hip drop test on the right side with a rib hump on the right thoracic-lumbar region T11-L2. Examination of the patient's lower thoracic and lumbar regions were also conducted and revealed hypertonic muscles at thoracolumbar junction R>L, as well as three lumbar somatic dysfunctions: L1-L5 neutral rotated right side bent left, L5 flexed rotated right side bent right, and L3 flexed rotated right side bent right. The patient was negative for the straight leg test and Thomas test.

METHODS AND TREATMENT APPROACH

The patient was treated with muscle energy technique for the right anterior innominate somatic dysfunction[7] and articulation technique for sacroiliac joints. Following the treatment, the patient's pelvis was reexamined with the findings of more symmetrical ASIS and PSIS bilaterally, thus indicating improvement of the somatic dysfunction following the osteopathic treatment. A total of three treatment sessions were performed weekly by the same physician.

FIGURE 1: Examples of recommended stretches.

The patient was reevaluated 1 month after undergoing osteopathic treatment and maintaining a routine of persistent core exercises to assess the long-term effects of the interventions. Upon examination, bilateral tenderness of the sacroiliac joint was no longer present, and the ASIS and PSIS were symmetrical bilaterally, indicating the resolution of the right anterior innominate somatic dysfunction identified during the initial visit. Furthermore, the previously noted R>L hypertonic muscles at the thoracolumbar junction had resolved, as had the somatic dysfunctions at L3 and L5. Although the L1-L5 neutral, rotated-right, side-bent-left somatic dysfunction was still present, it was significantly reduced. Overall, the patient demonstrated marked improvement, including reduced lower back pain, a more symmetrical pelvis bilaterally, and improved postural balance.

DISCUSSION

OMT has been widely advocated for improving biomechanical function and, thus, for improving motion, with potential benefits such as pain reduction.[8] Analysis of current reports suggests that osteopathic treatments may produce clinically relevant benefits for postpartum women with lower back pain.[8] The benefits of core stability exercises have been implicated in postpartum women with lower back pain.[9,10] The two muscles most commonly affected and of primary concern are the transverse abdominis and multifidus, as persistent pain in these muscles has been associated with an increased chance of back pain recurrence.[9] Maintaining correct and stable posture is very important and aids in relieving pain, while improper posture may exacerbate pain or create new injuries.[9] Overall, current literature on persistent lower back pain in postpartum patients has reported significant effects in favor of osteopathic treatments for addressing pain and functional status in postpartum patients.[8] However, conclusions such as the aforementioned have been called into question upon further investigation. In their systematic review and meta-analyses conducted on lower back pain in pregnant and postpartum patients, H. Franke et al. have examined a multitude of studies exploring the effects of osteopathic treatments among both pregnant and postpartum patients.[8] While statistical analysis demonstrated a significant medium-sized effect on decreasing pain and increasing functional status in women during pregnancy, there is "low-quality evidence" that OMT had a significant effect on decreasing pain and increasing functional status in postpartum women with lower back pain.[8,11] Findings for postpartum women were largely attributed to inconsistencies in the study methods utilized by the examined studies. Multiple studies suggest that hormonal changes during pregnancy can contribute to postpartum PGP. Pregnancy-related hormonal changes,

such as increases in relaxin, estrogen, and progesterone levels, are potentially linked to ligament hyperlaxity and joint instability, contributing to lumbopelvic pain.[17] After the postpartum period, relaxin levels drop significantly from 126.2 to 19.1.[17] Relaxin hormones can alter ligament mechanics due to their collagenolytic effects by releasing matrix metalloproteinases (MMPs),[13] collagenase,[14] and plasminogen activator.[15] During pregnancy, the increased levels of relaxin may cause permanent changes to the ligaments in the pelvic joints, leading to postpartum PGP. This is supported by evidence that relaxin is also involved in nonpregnancy-related fibrotic diseases.[16] If relaxin causes fibrotic changes and hardening of ligaments in incorrect positions leading to somatic dysfunction, then osteopathic manipulative techniques could be effective in correcting the somatic dysfunctions caused by the influx of relaxin during pregnancy. Biomechanical instability is a common cause of PGP during pregnancy. As the uterus enlarges and the compensatory lordosis of the lumbar spine increases, musculoskeletal strain is placed on the pelvic region. This strain is exacerbated by pelvic rotation around a fulcrum at the second sacral segment, which increases in tandem with the lordosis. Additionally, the center of gravity shifts anteriorly, producing further strain on the lumbar spine and sacroiliac joints.[18] During pregnancy, the sacroiliac joints become increasingly lax under the hormonal influence of relaxin, further contributing to instability and strain on the lower back and pelvis. Previous studies have shown that relaxin can cause fibrotic changes.[19] We hypothesize that the elevated levels of relaxin during the first three trimesters of pregnancy may induce fibrotic changes in the ligaments of the pelvic region. When relaxin levels decrease postpartum, these fibrotic changes may persist, potentially leading to permanent alterations in the lumbar spine and pelvic joints that were subjected to increased lordosis and rotation during pregnancy. These changes may result in altered permeability and function of the lumbar spine and pelvic region, contributing to somatic dysfunction and pain. In cases where somatic dysfunction specifically affects the pelvic region, OMT may be the only effective intervention to restore proper alignment and relieve pain.

TABLE 1: Relaxin levels during pregnancy and postpartum (first trimester, second trimester, third trimester postpartum)

	First Trimester	Second Trimester	Third Trimester	Postpartum
Relaxin (pg/mL)	129.7 5.8*	122.6 6.6*	126.2 7.7*	19.0 9.1†

*Statistically significant compared with postpartum value, P < .001.

†Statistically significant compared with the first trimester value, P < .001. (From Marnach ML, Ramin KD, Ramsey PS, Song SW, Stensland JJ, An KN. Characterization of the relationship between joint laxity and maternal hormones in pregnancy. Obstet Gynecol. 2003;101(2):331-5.)[20]

Furthermore, only three major studies were considered in examining postpartum pain due to the larger sample sizes of those studies. It is noteworthy that while the emphasis on the lack of sufficiently consistent and precise evidence for the effective treatment of lower back pain in postpartum women is important to keep in mind, it is even more important to report that, in general, there are still insufficient studies examining the effectiveness of osteopathic treatments on postpartum women.[11] While the effects of osteopathic treatment in reducing pain are well known, more research on the effects of osteopathic treatment in reducing lower back pain in postpartum women specifically still needs to be conducted, as current literature has focused more on pregnant women than on postpartum women. For this reason, authors aimed to demonstrate the effects of osteopathic treatment on a 31-year-old Asian American woman who presented with lower back pain that was not relieved 3 months' postpartum.

CONCLUSION

The presented case shows that OMT and a regimen of core exercises are suggested to play a critical role in reducing PGP in postpartum women. This is particularly important given the potential for ligamentous hardening and fibrotic changes caused by increased relaxin hormone levels during pregnancy. Although current studies have shown limited research and reports of the effects of OMT on postpartum women, our case shows that OMT in PGP reduction needs to be considered and studied more thoroughly, particularly in the postpartum population. This could potentially ensure that these women receive adequate support and care to treat their lower back pain. As a result, OMT could have a lasting impact on these patients' lives.

REFERENCES

1. Tanner H. Craniosacral therapy for pregnancy-related pelvic girdle pain. Herman & Wallace Pelvic Rehabilitation Continuing Education. https://hermanwallace.com/blog/craniosacral-therapy-for-pregnancy-related-pelvi c-girdle-pain
2. Vermani E, Mittal R, Weeks A. Pelvic girdle pain and low back pain in pregnancy: a review. *Pain Practice*. 2010;10(1):60–71. doi: 10.1111/j.1533-2500.2009.00327.x
3. Wiezer M, Hage-Fransen MAH, Otto A, et al. Risk factors for pelvic girdle pain postpartum and pregnancy related low back pain postpartum; a systematic review and meta-analysis. *Musculoskelet Sci Pract*. 2020;48:102154. doi: 10.1016/j.msksp.2020.102154
4. Kanakaris NK, Roberts CS, Giannoudis PV. Pregnancy-related pelvic girdle pain: an update. *BMC Medicine*. 2011;9(1). doi: 10.1186/1741-7015-9-15

5. Gutke A, Lundberg M, Östgaard HC, Öberg B. Impact of postpartum lumbopelvic pain on disability, pain intensity, health-related quality of life, activity level, kinesiophobia, and depressive symptoms. *European Spine Journal*. 2010;20(3):440–448. doi: 10.1007/s00586-010-1487-6

6. Albert HB, Godskesen M, Korsholm L, Westergaard JG. Risk factors in developing pregnancy related pelvic girdle pain. *Acta Obstetricia et Gynecologica Scandinavica*. 2006;85(5):539–544. doi: 10.1080/00016340600578415

7. DiGiovanna EL, Amen CJ, Burns DK. *An Osteopathic Approach to Diagnosis and Treatment, 4th ed*. Wolters Kluwer; 2021.

8. Franke H, Franke J-D, Belz S, Fryer G. Osteopathic manipulative treatment for low back and pelvic girdle pain during and after pregnancy: a systematic review and meta-analysis. *Journal of Bodywork and Movement Therapies*. 2017;21(4):752–762. doi: 10.1016/j.jbmt.2017.05.014

9. Chaudhry S, Siddiqui FR, Shah IH. Effectiveness of core stabilization exercises along with postural correction in postpartum back pain. *Rawal Medical Journal*. 2013;38(3):256–259.

10. Saleh MSM, Botla AMM, Elbehary NAM. (2019). Effect of core stability exercises on postpartum lumbopelvic pain: a randomized controlled trial. *J Back Musculoskelet Rehabil*. 2019;32(2):205–213. doi: 10.3233/BMR-181259

11. Ruffini N, D'Alessandro G, Cardinali L, Frondaroli F, Cerritelli F. Osteopathic manipulative treatment in gynecology and obstetrics: a systematic review. *Complement Ther Med*. 2016;26:72–78. doi: 10.1016/j.ctim.2016.03.005

12. Daneau C, Abboud J, Marchand A-A, et al. Mechanisms underlying lumbopelvic pain during pregnancy: a proposed model. *Front Pain Res* (Lausanne). 2021;2:77398. doi: 10.3389/fpain.2021.773988

13. Qin X, Garibay-Tupas J, Chua PK, Cachola L, Bryant-Greenwood GD. An autocrine/paracrine role of human decidual relaxin. I. Interstitial collagenase (matrix metalloproteinase-1) and tissue plasminogen Activator1. *Biol Reprod*. 1997;56(4):800–811. doi: 10.1095/biolreprod56.4.800

14. Granström LM, Ekman GE, Malmström A, Ulmsten U, Woessner JF. Serum collagenase levels in relation to the state of the human cervix during pregnancy and labor. *Am J Obstet Gynecol*. 1992;167(5):1284–1288. doi: 10.1016/s0002-9378(11)91701-3

15. Koay ES, Too CK, Greenwood FC, Bryant-Greenwood GD. Relaxin stimulates collagenase and plasminogen activator secretion by dispersed human amnion and chorion cells in vitro. *J Clin Endocrinol Metab*. 1983;56(6):1332–1334. doi: 10.1210/jcem-56-6-1332

16. Yuan S, Guo D, Liang X, Zhang L, Zhang Q, Xie D. Relaxin in fibrotic ligament diseases: its regulatory role and mechanism. *Front Cell Dev Biol*. 2023;11:1131481. doi: 10.3389/fcell.2023.1131481

17. Marnach ML, Ramin KD, Ramsey PS, Song S-W, Stensland JJ, An K-N. Characterization of the relationship between joint laxity and maternal hormones in pregnancy. *Obstet Gynecol*. 2003;101(2):331–335. doi: 10.1097/00006250-200302000-00021

18. Seffinger MA. Foundations of Osteopathic Medicine: Philosophy, Science, Clinical Applications, and Research. Wolters Kluwer; 2018. 19. Ritchie JR. Orthopedic considerations during pregnancy. *Clin Obst Gynecol*. 2003;46(2);456–466. doi: 10.1097/00003081-200306000-00024

19. Ritchie JR. Orthopedic considerations during pregnancy. *Clin Obst Gynecol*. 2003;46(2);456–466. doi: 10.1097/00003081-200306000-00024

CLINICAL IMAGE

Rare and Recurrent Tendon Ruptures in the Context of Long-term Statin Therapy

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CASE REPORT

Statins are the drug of choice to help lower cholesterol levels. Adverse effects, although infrequent, are usually linked to dose increases and improve with medication reduction/cessation. The growing population of patients prescribed statin medications and the nature of their use for lifetime cardiovascular disease (CVD) risk reduction have supported interest in studies focused on the long-term impacts of prolonged statin therapy. While the mechanism is not fully understood, statins have been linked to fibrous changes that contribute to tendinopathy and rupture.

An 86-year-old male presented with successive ruptures of his semitendinosus and latissimus dorsi, two rare tendinous injuries. He maintained an active lifestyle, engaging in regular aerobic exercise and supplemental machine weightlifting, before this unexpected occurrence while moving boxes in his home. His medications included long-term pravastatin (80 mg) and warfarin (2 mg), without history of recent changes.

A review of published literature reveals growing support of the causative relationship of long-term statin treatment and the occurrence of rare tendon ruptures. Clinical indication for medication changes also appears to be supported due to the possibility of patient benefit. Although limited to one patient, this case study provides a unique educational opportunity to update information on a common medication as well as supports further research into the prevalence of this adverse effect and its link to statin therapy that may impact recommended best practices.

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HISTORY

An 86-year-old male presented to the clinic with a chief complaint of pain, swelling, and bruising behind his left knee that had occurred suddenly 1 week prior while moving boxes in his home. The exertion involved was within the patient's normal physical behavior as he maintained an active lifestyle, engaging in regular aerobic exercise and supplemental machine weightlifting. He reported no history of injury or surgery to the extremity. His past medical history included hyperlipidemia and atrial fibrillation, for which he regularly took pravastatin (80 mg) for 20 years and warfarin (2 mg) for 15 years, without a history of recent changes. His cholesterol levels were checked annually and remained well maintained with medication and lifestyle modifications.

Upon initial assessment, the patient's vitals were the following: blood pressure 120/76 mmHg, heart rate 80 bpm, and respiratory rate 12 breaths per minute. His physical exam revealed edema, ecchymosis, and a palpable defect of the posterior left thigh just superior to and along the medial edge of the popliteal fossa (Figure 1). He demonstrated reduced active/passive range of motion (ROM) with left knee flexion/extension as well as reduced strength in left knee flexion. Palpation of the knee joint localized an exaggerated tender point at the point of insertion of the left semitendinosus near the pes anserinus. A diagnosis of left semitendinosus tendon rupture was made clinically, and the patient was returned home on supportive care.

Six weeks later, the patient returned with another spontaneous injury, causing weakness, swelling, and ecchymosis in his left upper extremity (Figures 2 and 3). He had been moving boxes when he felt a "pop" and pain in his axillary region. A tender point was assessed near the humeral insertion point of the left latissimus dorsi muscle. He demonstrated weakness in shoulder adduction and extension. A clinical diagnosis of a tendon rupture to his latissimus dorsi was made.

FIGURE 1

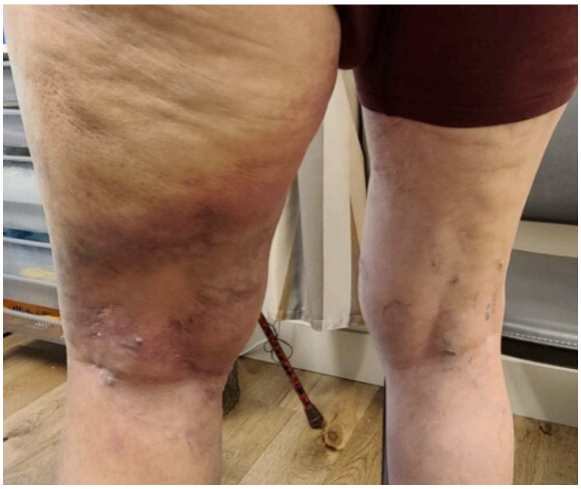


FIGURE 2

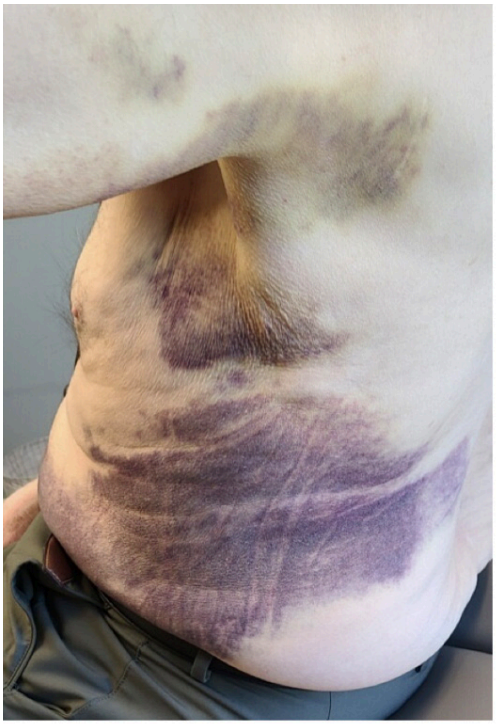
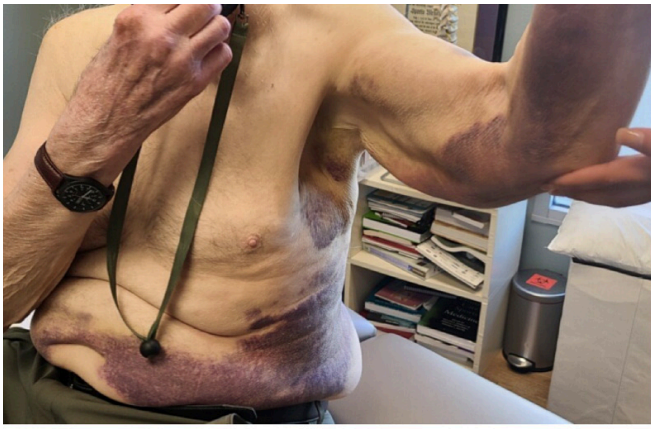


FIGURE 3



QUESTIONS

1. Which of the following are considered reliable ways to diagnose a tendon rupture?

a. Magnetic resonance imaging (MRI)

b. Clinical evaluation

c. Ultrasound

d. All of the above

Correct Answer:
d. All of the above

Special testing, active and passive exams, and assessment of palpable tissue defects have shown high sensitivity and specificity when it comes to diagnosing the occurrence of a tendon rupture and offer important information required before even considering imaging. While studies that have quantified these statistics tend to be isolated to select regions of the body, e.g., the Achilles and biceps tendons, due to the efficacy and accessibility, it is widely accepted that clinical diagnosis of tendon rupture is sufficient.[1,2] The physical exam findings for a latissimus dorsi rupture were well described by George M, et al. in the Journal of the American Academy of Orthopaedic Surgeons, and further demonstrate how a diagnosis can be made in a clinical setting, especially in the case of a complete tear.[3] Similar studies have quantified a higher level of specificity achieved through ultrasound imaging, although the sensitivity remains equivalent. MRI has been shown to provide more detailed information regarding soft tissue injuries; however, due to the high cost and other barriers to access, it is rarely used as a test to confirm tendon rupture.[1]

2. Which of the following is the most common side effect of statin therapy?

a. Hepatic dysfunction

b. Myalgia

c. endinopathy

d. Renal dysfunction

Correct Answer:

b. Myalgia

Statins have been shown to have only low rates of adverse effects, assessing these effects through randomized trials and controlling to a placebo. The most commonly reported adverse reaction was myalgia, while a few experienced more severe effects including tendinopathy, hepatic dysfunction, and renal dysfunction. Reports, however, are more common in clinical practice than data would predict, possibly due to patient awareness of side effects or the effects of comorbidities that are frequently excluded from more controlled studies.[4]

3. When are myalgia adverse effects of statins expected to present themselves with respect to therapy initiation or dose increase?

a. Within 24 hours (dependent upon half-life of the specific statin)

b. Within 1 week

c. Within 1 month

d. Within 6 months

Correct Answer:

c. Within 1 month

On average, studies have shown that myalgia symptoms present 1 month after statin therapy initiation or an increase in dose. Strengthening this temporal relationship, it was found that these symptoms improved 2 weeks after discontinuing the medication. The reoccurrence of these symptoms upon statin drug rechallenge was also found to be 2 weeks. Due to the strong evidence surrounding short-term statin adverse reactions, the timing of symptom onset is a factor currently accepted as one that can strengthen or reduce the likelihood of a pathology's association with the medication.[4]

DISCUSSION

Statins are the drug of choice to help lower cholesterol levels in patients with hyperlipidemia, with the goal of lowering risk of atherosclerotic cardiovascular disease (ASCVD) development and the associated morbidity and mortality. In 2018, statins were prescribed to 92 million patients, with this number growing significantly in recent years.[5] Statins act through inhibition of HMG-CoA (3-hydroxy-3-methylglutaryl–coenzyme A) reductase (HMGR), an enzyme required for the biosynthesis of cholesterol.[6] Reducing levels of intrahepatic cholesterol synthesis also leads to an increase in low-density lipoprotein (LDL) receptor turnover, supporting the removal of this "bad cholesterol" from the bloodstream.[7]

The popularity of this medication is assuredly due to its infrequent association with side effects, as well as its impact on patient ASCVD risk reduction. On average, if adverse effects do occur, it is usually within the first month of treatment or within 1 month of a dose increase, and they typically improve with medication reduction or cessation. As use of statin medications has increased over the last 2 decades, the clinical occurrence of tendon rupture in those undergoing statin therapy has brought the possibility of a causal relationship to the attention of the scientific community. While the mechanism of action is not fully understood, studies have shown disruption of tendon matrices in vitro as well as increased release of matrix metalloproteinase (MMP)-1 and MMP-13 in the context of statin exposure.[8] These fibrous changes are believed to contribute to tendinopathy and an increased risk of tendon rupture.

Tendon ruptures are relatively uncommon injuries and are rarer still in larger tendon structures. Most commonly they are seen in individuals who suddenly and significantly increase their activity level. Larger tendon ruptures, if they occur, are usually seen in professional athletes following escalation of already intense training routines. Chronic conditions such as diabetes, hyperparathyroidism, and poor conditioning can contribute to a patient's increased risk of tendon rupture. Medications such as fluoroquinolone antibiotics and corticosteroid injections have strong causal relationships with tendon ruptures. As this is a well-known risk, it is routinely considered in the medical decision-making process.

A review of published literature finds an expanding collection of studies supporting a strong connection between statin therapy and tendinopathy. In addition to a plethora of clinical cases demonstrating this correlation,[9-14] a nationwide population-based cohort study looking at over 590,000 participants demonstrated a greater risk of tendinopathy in statin users when compared to nonusers.[15] A smaller retrospective study investigating 104 patients with ages ranging from 22 to 78 years also demonstrated that there was a two-times greater risk of distal biceps tendon rupture in those undergoing statin therapy.[16] While some speak to the connection between the two, another cross-sectional study using ultrasound imaging to support contrasting claims of no correlation concluded that there was no evidence of negative impact of statin therapy on the Achilles tendon structure.[17] One review even proposed that a period of missed statin therapy may contribute as a risk factor to increase the chance of a patient experiencing a re-tear of their rotator cuff—although statin influence on rotator cuff injuries has generally contrasted all other tendinopathy trends and has seemed to delineate a separate area of study.[18]

Statins play an important role in reducing CVD in patients with hyperlipidemia, and patient treatment must balance considerations of risks and benefits. While connections to tendon rupture appear significant, some studies have shown discontinuation of statin therapy to return risk levels to baseline.[9,14] In patients who maintain an elevated ASCVD risk score, a change to medications that function through separate mechanisms may reduce the risk of tendon injury in those shown to experience this adverse effect from statin therapy. Alirocumab and evolocumab are monoclonal antibodies that bind and inhibit proprotein convertase subtilisin/kexin type 9 (PCSK9), an enzyme that degrades hepatic LDL receptors, ultimately elevating levels of plasma LDL-C. A comparative study addressing the efficacy and safety of PCSK9 inhibitors actually found them to be the

most effective lipid-lowering agent while also avoiding common statin-related side effects.[19] Additional testing, including measurement of apolipoprotein (B) (Apo(B)) and lipoprotein(a) (Lp(a)) levels, has been shown to provide a more complete assessment of a patient's total ASCVD risk. Clinical consideration of these components can help physicians better assess patient risks and guide pharmacologic de-escalation, limiting unnecessary exposure to potential harm from medications.[20-24]

CONCLUSION

It is important to consider all the risks and benefits of treatment before prescribing any new medications to a patient. When statins are prescribed, it is expected that adverse reactions (most commonly myalgias) will typically present within the first month of either medication initiation or a dosage increase. However, as the prevalence of statin therapy surges in the population, the likelihood of clinically encountering a serious adverse event will rise in conjunction. Studies have shown that long-term statin exposure may have a disruptive effect on tendon matrices. This puts forth a possible explanation underlying the mechanism of injury behind the tendon ruptures that have been noted as an infrequent but severe side effect. It is important to appreciate this connection in the context of the growing population being treated with statins as well as the prolonged therapeutic timelines. In the setting of tendinopathy and rupture, physicians are reminded of the importance of a thorough assessment and including statin adverse effect in the differential, even outside of the context of a recent dose adjustment.

Awareness of this adverse outcome can encourage physicians to include this differential etiology in clinical encounters with unusual tendinopathy and adjust their plan accordingly. Steps must be taken to ensure that injury does not lead to a lasting impact on activity and quality of life. In younger patients who experience tendinopathy in the setting of statin therapy who also maintain an elevated ASCVD risk score, switching to a medication with a different mechanism of action (such as a PCSK9 inhibitor) will allow for continued primary prevention while reducing the risk of further tendon injury. For patients >75 years old, guidelines are less clear as there is no definitive evidence that statin therapy can prevent future coronary artery disease (CAD) or death.[25,26] However, the risk of patient harm with statins has been clearly demonstrated. This balance of risk vs benefit in this population is important to consider in the shared decision-making process and may be aided by further workup through assessment of Apo(B) and Lp(a), helping to guide possible pharmacologic de-escalation.

REFERENCES

1. Amendola F, Barbasse L, Carbonaro R, et al. The acute Achilles tendon rupture: an evidence-based approach from the diagnosis to the treatment. *Medicina*. 2022;58(9):1195. doi: 10.3390/medicina58091195
2. Zwerus EL, van Deurzen DFP, van den Bekerom MPJ, The B, Eygendaal D. Distal biceps tendon ruptures: diagnostic strategy through physical examination. *Am J Sports Med*. 2022;50(14):3956–3962. doi: 10.1177/03635465221129874
3. George MS, Khazzam M. Latissimus dorsi tendon rupture. *J Am Acad Orthop Surg*. 2019;27(4):113–118. doi: 10.5435/JAAOS-D-17-00581
4. Newman CB, Preiss D, Tobert JA, et al. Statin safety and associated adverse events: a scientific statement from the American Heart Association. *Arterioscler Thromb Vasc Biol*. 2019;39(2):e38–e81. doi: 10.1161/ATV.0000000000000073
5. Matyori A, Brown CP, Ali A, Sherbeny F. Statins utilization trends and expenditures in the U.S. before and after the implementation of the 2013 ACC/AHA guidelines. *Saudi Pharm J*. 2023;31(6):795–800. doi: 10.1016/j.jsps.2023.04.002
6. Istvan ES, Deisenhofer J. Structural mechanism for statin inhibition of HMG-CoA reductase. *Science*. 2001;292(5519):1160–1164. doi: 10.1126/science.1059344
7. Ness GC, Zhao Z, Lopez D. Inhibitors of cholesterol biosynthesis increase hepatic low-density lipoprotein receptor protein degradation. *Arch Biochem Biophys*. 1996;325(2):242–248. doi: 10.1006/abbi.1996.0030
8. Eliasson P, Dietrich-Zagonel F, Lundin AC, Aspenberg P, Wolk A, Michaëlsson K. Statin treatment increases the clinical risk of tendinopathy through matrix metalloproteinase release—a cohort study design combined with an experimental study. *Sci Rep*. 2019;9(1):17958. doi: 10.1038/s41598-019-53238-7
9. Moniri NH, Momary KM, McMahon T, Nayee E. Statin-associated Achilles tendon rupture and reproducible bilateral tendinopathy on repeated exposure. *Mayo Clin Proc*. 2018;93(10):1531–1532. doi: 10.1016/j.mayocp.2018.08.005
10. Carmont MR, Highland AM, Blundell CM, Davies MB. Simultaneous bilateral Achilles tendon ruptures associated with statin medication despite regular rock climbing exercise. *Phys Ther Sport*. 2009;10(4):150–152. doi: 10.1016/j.ptsp.2009.01.003
11. Kearns MC, Singh VK. Bilateral patellar tendon rupture associated with statin use. *J Surg Case Rep*. 2016;2016(5):rjw072. doi: 10.1093/jscr/rjw072
12. 12. Pullatt RC, Gadarla MR, Karas RH, Alsheikh-Ali AA, Thompson PD. Tendon rupture associated with simvastatin/ezetimibe therapy. *Am J Cardiol*. 2007;100(1):152–153. doi: 10.1016/j.amjcard.2007.02.068
13. Bove A, Orabona N, De Matteo V, et al. Spontaneous bilateral quadriceps tendon rupture treated with polyethylene terephthalate tape augmentation: report of two consecutive cases. *Jt Dis Relat Surg*. 2022;33(3):666–672. doi: 10.52312/jdrs.2022.764
14. Gowdar SD, Thompson PD. Multiple tendon ruptures associated with statin therapy. *J Clin Lipidol*. 2020;14(2):189–191. doi: 10.1016/j.jacl.2019.12.001
15. Kwak D, Moon SJ, Park JW, Lee DH, Lee JI. Effects of statin treatment on the development of tendinopathy: a nationwide population-based cohort study. *Orthop J Sports Med*. 2023;11(7):23259671231167851. doi: 10.1177/23259671231167851
16. Savvidou C, Moreno R. Spontaneous distal biceps tendon ruptures: are they related to statin administration? *Hand Surg*. 2012;17(2):167–171. doi: 10.1142/S0218810412500153

17. de Sá A, Hart DA, Khan K, Scott A. Achilles tendon structure is negatively correlated with body mass index, but not influenced by statin use: a cross-sectional study using ultrasound tissue characterization. *PLoS One*. 2018;13(6):e0199645. doi: 10.1371/journal.pone.0199645

18. Lee S, Lee N, Shin SJ. Relationship of missed statin therapy and 10-year atherosclerotic cardiovascular disease risk score to retear rate after arthroscopic rotator cuff repair. *Am J Sports Med*. 2023;51(8):1988–1996. doi: 10.1177/03635465231175476

19. Zhao Z, Du S, Shen S, et al. Comparative efficacy and safety of lipid-lowering agents in patients with hypercholesterolemia: a frequentist network meta-analysis. *Medicine (Baltimore)*. 2019;98(6):e14400. doi: 10.1097/MD.0000000000014400

20. Kim K, Ginsberg HN, Choi SH. New, novel lipid-lowering agents for reducing cardiovascular risk: beyond statins. *Diabetes Metab J*. 2022;46(4):517–532. doi: 10.4093/dmj.2022.0198

21. O'Donoghue ML, Rosenson RS, Gencer B, et al. Small interfering RNA to reduce lipoprotein(a) in cardiovascular disease. *N Engl J Med*. 2022;387(20):1855–1864. doi: 10.1056/NEJMoa2211023

22. Rhainds D, Brodeur MR, Tardif JC. Lipoprotein(a): when to measure and how to treat? *Curr Atheroscler Rep*. 2021;23(9):51. doi: 10.1007/s11883-021-00951-2

23. Jang AY, Lim S, Jo SH, Han SH, Koh KK. New trends in dyslipidemia treatment. *Circ J*. 2021;85(6):759–768. doi: 10.1253/circj.CJ-20-1037

24. Ahmad M, Sniderman AD, Hegele RA. Apolipoprotein B in cardiovascular risk assessment. CMAJ. 2023;195(33):E1124. doi:10.1503/cmaj.230048.

25. Nanna MG, Abdullah A, Mortensen MB, Navar AM. Primary prevention statin therapy in older adults. *Curr Opin Cardiol*. 2023;38(1):11–20. doi: 10.1097/HCO.0000000000001003

26. Lazris A, Roth AR. Overuse of statins in older adults. *Am Fam Physician*. 2019;100(12):742–743.

PATIENT EDUCATION HANDOUT

SEXUAL AND REPRODUCTIVE HEALTH

Masculinizing Gender-Affirming Hormone Therapy (GAHT) in Adults

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WHAT IS MASCULINIZING GAHT?

Masculinizing gender-affirming hormone therapy (GAHT) is the use of hormones to develop masculine physical attributes. The individual must have gender incongruence or gender identity that does not match birth sex, which is typically female. This is independent of whether or not they tell other people about it, dress differently, or change their name.

FERTILITY CONSIDERATIONS PRIOR TO INITIATING GAHT

Prior to beginning GAHT, the individual should have a discussion with their healthcare provider about their desire to have biological children. The long-term effects on fertility and ability to become pregnant are not well understood. Discussion should address options to preserve fertility such as freezing eggs, sperm, or embryos. Other options could include fostering, adoption, and coparenting. It is also important to understand that testosterone is not a reliable method to prevent pregnancy.

HEALTH RISKS FOR GAHT

- Heart disease and heart attacks
- High blood pressure
- Weight gain
- High cholesterol or lipid profile
- Increased red blood cell count

CONTRAINDICATIONS FOR GAHT

- Pregnancy or trying to become pregnant
- Current lactation or breast/chest feeding
- Uncontrolled high blood pressure
- Polycythemia (elevated red blood cell count)

TYPICAL TESTOSTERONE REGIMEN

- Injection of 20 to 200 mg testosterone every 1 to 2 weeks
- Application of one to eight pumps of testosterone gel daily

PHYSICAL EFFECTS OF TAKING TESTOSTERONE

EFFECT	TIMING
Acne	Onset within 1 month and peak after 2 years
• Increased hair on the body and face (can be permanent) • Male pattern baldness (can be permanent) • Decrease in fat and increase in muscles	Onset within 6 months and peak after 5 years
• Deepening of voice (can be permanent) • Reduced or no periods • Increased sexual desire • Increased size of clitoris (can be permanent) • Sexual dysfunction such as genital dryness or pain with sex	Onset within 1 month and peak after 2 years

WHAT MONITORING WILL BE DONE WITH ADMINISTRATION OF MASCULINIZING GAHT?

Levels of estrogen and testosterone will be monitored regularly with the goal of achieving similar levels of hormones present in the desired gender. The targeted range for testosterone level is generally 400 to 700 ng/dL. In the first year, laboratory tests will be done every 3 months. After stable dosing is achieved, frequency may decrease to one to two times per year. Other monitoring will include hemoglobin level and cholesterol. It is also important to receive regular screenings indicated for one's age and body parts, including breast cancer and cervical cancer.

SOURCES

Coleman E, Radix AE, Bouman WP, et al. Standards of care for the health of transgender and gender diverse people, Version 8. *Int J Transgender Health*. 2022;23(suppl 1):S1–S259. doi: 10.1080/26895269.2022.2100644

Hembree WC, Cohen-Kettenis PT, Gooren L, et al. Endocrine treatment of gender-dysphoric/gender-incongruent persons: an Endocrine Society Clinical Practice Guideline. *J Clinl Endocrinol Metab*. 2017;102(11):3869–3903. doi: 10.1210/jc.2017-01658

UCSF Gender Affirming Health Program, Department of Family and Community Medicine, University of California San Francisco. Deutsch MB, ed. *Guidelines for the Primary and Gender-Affirming Care of Transgender and Gender Nonbinary People; 2nd edition*. transcare.ucsf.edu/guidelines

PATIENT EDUCATION HANDOUT

HEALTH AND WELLNESS

Causes, Symptoms, and Management of Sleep Apnea

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Sleep apnea is a common sleep disorder characterized by pauses in breathing during sleep. These pauses can last from seconds to minutes and can occur multiple times throughout the night. Sleep apnea can affect individuals regardless of age, gender, or ethnicity, though it occurs more frequently in men than women.

CAUSES OF SLEEP APNEA

Obstructive sleep apnea is the most common type of sleep apnea and occurs when the upper airways become blocked while sleeping, which partially or completely stops airflow. This is commonly caused by obesity, large tonsils, nasal congestion, or drinking alcohol before falling asleep.

Central sleep apnea is less common and occurs when the brain does not send the correct signals required to breathe. Common causes include heart failure, neuromuscular disorders, and chronic opioid use.

SYMPTOMS OF SLEEP APNEA

- Loud snoring
- Gasping, choking, or pauses in breathing during sleep witnessed by a partner
- Excessive daytime sleepiness
- Morning headaches
- Irritability or mood changes
- Difficulty concentrating
- Dry mouth or sore throat upon waking

MANAGEMENT OF SLEEP APNEA

It is important to diagnose and treat sleep apnea to prevent serious complications, including high blood pressure and stroke. A sleep study, or polysomnography, monitors breathing and brain activity while sleeping to diagnose sleep apnea.

The preferred treatment for obstructive sleep apnea is positive airway pressure (PAP) therapy. This involves wearing a mask connected to a machine that delivers airflow to keep the airway open during sleep.

Other options include oral appliances, which are not as effective, hypoglossal nerve stimulation, and surgery in serious cases. Lifestyle changes can also be implemented to reduce risk of developing and improve symptoms of sleep apnea, including:

- Weight loss
- Alcohol avoidance
- Tobacco avoidance
- Side sleeping
- Elevating the head of the bed
- Nasal decongestant use

Figure 1

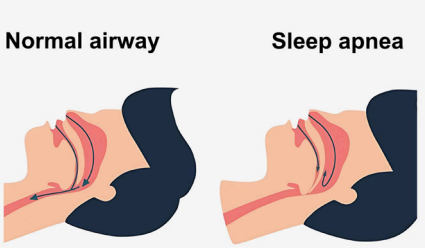
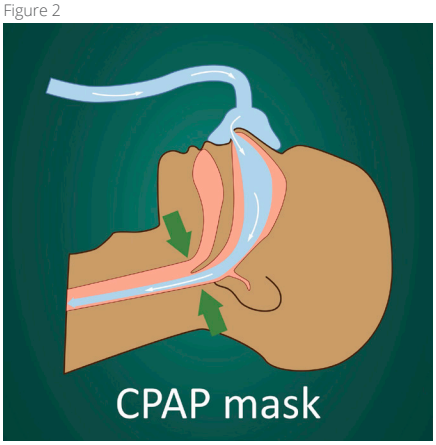


Figure 2



SOURCES

Epstein LJ, Kristo D, Strollo PJ Jr, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med.* 2009;5(3):263–276.

Patil SP, Ayappa IA, Caples SM, Kimoff RJ, Patel SR, Harrod CG. Treatment of adult obstructive sleep apnea with positive airway pressure: an American Academy of Sleep Medicine Clinical Practice Guideline. *J Clin Sleep Med.* 2019;15(2):335–343. doi: 10.5664/jcsm.7640

Qaseem A, Holty JEC, Owens DK, et al. Management of obstructive sleep apnea in adults: a clinical practice guideline from the American College of Physicians. *Ann Intern Med.* 2013;159(7):471–483. doi: 10.7326/0003-4819-159-7-201310010-00704

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REVIEW ARTICLE

Injection Options for the Treatment of Trigger Finger: A Review of Current Literature

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KEYWORDS

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ABSTRACT

Introduction: Stenosing tenosynovitis of the flexor tendon, more commonly known as trigger finger (TF), is an ailment characterized by inflammation of the A1 pulley. This inflammation can eventually lead to pain and the inability to manipulate the digit. While surgical release is considered the gold standard for TF treatment, corticosteroid injection is often trialed before proceeding with surgery. It is an effective treatment for those who do not want to undergo surgery. This review aims to investigate the current literature regarding TF injection options and techniques to identify best practices and current gaps in research that warrant further investigation.

Methods: A clinical review was conducted using the keywords "trigger finger," "stenosing tenosynovitis," "injection," and "treatment." Articles discussing surgical treatment or other pathologies aside from TF were excluded. Some articles outside the search parameters were included to provide scientific and clinical context.

Discussion: There are several gaps in the current literature regarding TF treatment. Studies have shown that local anesthetic in conjunction with corticosteroid does not decrease pain associated with injection. This warrants an investigation into the continued use of local anesthetic with TF injections despite the known chondrotoxic effects. Studies have also shown mixed results regarding use of ultrasound-guided injections and long-term patient outcomes, which could benefit from repeat studies with larger sample sizes. Furthermore, the efficacy and cost-benefit of orthobioligic injectate options, such as platelet-rich plasma, require further research. Finally, further investigation of preventative treatments, such as osteopathic techniques, would benefit the field.

INTRODUCTION: ETIOLOGY, PATHOPHYSIOLOGY, AND TREATMENT PROTOCOLS

Stenosing tenosynovitis of the flexor tendon, more commonly known as trigger finger (TF), is an ailment characterized by inflammation of the A1 pulley. The cause of this inflammation is unclear, but it is hypothesized to be associated with repetitive movements, trauma, stress, and degenerative changes associated with age. This disease can affect any of the fingers, but is most common in the ring finger, followed by the thumb, long, index, and small fingers. While the progression of TF can vary between patients, inflammation of the A1 pulley leads to hypertrophy of the retinacular sheath. As the sheath continues to hypertrophy, this eventually leads to progressive restriction of the flexor tendon.[1]

TF most commonly presents in adults at or above the sixth decade of life and has a 2% to 3% lifetime risk of development. However, there is a higher incidence in diabetic patients with a lifetime risk of development of 10%.[1-3] There also seems to be a higher risk of developing TF in patients with other conditions, such as carpal tunnel, DeQuervain's tenosynovitis, thyroid diseases, rheumatoid arthritis, amyloidosis, and renal disease.[1] Clinically, initial symptoms often include catching or clicking with manipulation of the digits in flexion or extension. The catching and clicking usually starts without pain. Still, as the disease process progresses, patients will often begin complaining of pain with digital manipulation, and occasionally palpable swelling or nodules can be seen on the palmar surface of the hand just proximal to the metacarpophalangeal (MCP) joint. Pain and restricted motion are characteristic of TF, at which point a clinical diagnosis is usually made.[2]

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Conservative first-line treatment options include resting, osteopathic manipulative techniques, splinting, and nonsteroidal anti-inflammatory drugs (NSAIDs). Osteopathic techniques have been used to treat TF. Some techniques include myofascial release, muscle energy, and articulatory techniques for the carpal bones. However, only anecdotal evidence including a case report is available.[4] Oral NSAIDs are commonly recommended for pain management for TF. However, studies have yet to be conducted on the use of oral NSAIDs and their efficacy in resolving symptoms. Splinting for 6 months has been demonstrated to be roughly 50% effective in resolving symptoms of TF. If these treatment options do not relieve the patient's symptoms, corticosteroid injections or surgical release can also be an option. Corticosteroid injection of the flexor tendon sheath is an effective treatment method for managing TF, with 88% of patients perceiving improvement in symptoms and quality of life after receiving an injection in some studies.[5,6] Often, lidocaine is injected simultaneously as an anesthetic. It has been shown that single injections can provide several months of symptomatic relief, while those who receive repeat injections can have symptomatic relief for up to a year or more. With that said, many patients still opt for surgical release after several injections for more permanent relief.[7] Studies have shown that over 90% of patients who undergo percutaneous or open TF release experience complete resolution of TF symptoms.[8] While surgical release is considered the gold standard for TF treatment, corticosteroid injection is sometimes more cost-effective and is a viable treatment option for patients who do not want surgery.[9] This review aims to investigate the current literature regarding TF injection options and techniques to identify best practices and current gaps in research that warrant further investigation.

METHODS

For this narrative-style review, a search was conducted using the keywords "trigger finger," "stenosing tenosynovitis," "injection," and "treatment." The databases searched include PubMed, Scopus, and Google Scholar. Inclusion criteria included articles discussing injectate options, injection techniques, or other therapies related to TF injection. Some articles discussing other first-line treatment modalities aside from injection, such as osteopathic manipulative therapies, NSAIDs, and splinting, were also included. Articles discussing surgical treatment or other pathologies aside from TF were excluded. Some articles outside the search parameters were included to provide context. This includes articles that discuss the etiology and epidemiology of TF and other articles that discuss the mechanism of action of various injectate options.

COMPOSITION: COMPARING VARIOUS CORTICOSTEROIDS AND THE USE OF LOCAL ANESTHETIC

Traditionally, TF injections consist of a corticosteroid in addition to lidocaine, with the steroid acting as the primary therapeutic agent while the lidocaine provides an anesthetic effect. However, not all corticosteroids are equally efficacious in the treatment of TF. The choice of corticosteroids can vary from provider to provider. Still, nonsoluble corticosteroids are typically used for intra-articular injections, while soluble corticosteroids, like methylprednisolone, are more frequently used for soft-tissue injections.[10] Several studies have shown that treatment of TF using a nonsoluble corticosteroid, triamcinolone, results in higher rates of symptomatic recurrence, and patients are more likely to undergo repeat injections when compared to soluble corticosteroids like methylprednisolone and dexamethasone.[11,12] While triamcinolone has been shown to provide more rapid relief in the first few weeks of treatment,[12] soluble corticosteroid options, such as dexamethasone and methylprednisolone, have been shown to provide longer-lasting relief and are less likely to result in the recurrence of symptoms.[11]

Studies have also investigated the inclusion of local anesthetics like lidocaine and bupivacaine and their efficacy in the treatment of TF and other tendinopathies. Currently, standard practice for TF injection typically includes a local anesthetic such as 1% lidocaine. However, studies have called into question whether these local anesthetics are necessary. When educating patients about the potential complications of injection, providers often discuss the potential chondrotoxic effects and toxic tendinopathy associated with local anesthetics such as lidocaine. Several in vitro studies conducted with human-harvested Achilles and rotator cuff tendons have found that treatment with lidocaine or bupivacaine decreased the proliferation of tendon cells and extracellular matrix production through the downregulation of cyclin A and cyclin-dependent kinase 2 (CDK2), cell cycle regulatory proteins that are important for chondrocyte proliferation. It was also found that type I and IV collagen expression were downregulated in treatment groups.[13,14] While all local anesthetics have been shown to have some degree of chondrotoxic effects, some are relatively less chondrotoxic than others. Studies that reviewed in vitro and clinical studies have found that bupivacaine is the most chondrotoxic local anesthetic commonly used in clinical practice followed by lidocaine. Ropivacaine has been demonstrated in vitro and in clinical trials to be significantly less chondrotoxic than bupivacaine and lidocaine. Other less commonly used anesthetics were also analyzed. It was found that mepivacaine is more

TABLE 1: Relative chondrotoxicity of local anesthetic agents

Most chondrotoxic	Levobupivacaine
	Bupivacaine
	Lidocaine
	Mepivacaine
Least chondrotoxic	Ropivacaine

chondrotoxic than ropivacaine, but less chondrotoxic than bupivacaine and lidocaine. Furthermore, levobupivacaine was found to be even more chondrotoxic than bupivacaine (Table 1).[15] However, this anesthetic is less commonly used in clinical practice.

Despite the chondrotoxic effects of local anesthetics, they are often still included in most injections to reduce the pain experienced by the patient. This has led clinicians to investigate whether local anesthetics are effective in reducing injection pain, and other alternative options that may reduce pain associated with injection. Two double-blind, randomized, control trials were conducted to determine the efficacy of local anesthetic when treating TF. In the first study, the J-tip system, which uses compressed CO2 to propel medication into the subcutaneous tissue without a needle, was used to anesthetize the local area before administration of a corticosteroid injection. It was found that the pain experienced by the treatment group who received lidocaine before injection had a lower mean Visual Analogue Scale (VAS) pain score compared to the control group who received normal saline. However, this difference was not statistically significant.[16] Another study also investigated the efficacy of local anesthetics by comparing VAS scores among control and treatment groups. The treatment group received 1 mL of triamcinolone 40 mg and 1 mL of lidocaine with epinephrine, and the control group received an injection consisting of 1 mL of triamcinolone 40 mg and 1 mL of normal saline. It was found that VAS scores in the treatment group were significantly higher than those in the control group.[17] This study demonstrated that those who received simultaneous injections with corticosteroids and lidocaine perceived a higher level of pain compared to those who received corticosteroids with normal saline. These studies suggest that using local anesthetic in TF injections may not be necessary, but further studies must be conducted before strong conclusions can be drawn.

INJECTION TECHNIQUES: ULTRASOUND GUIDANCE AND ALTERNATIVE NEEDLE APPROACHES

One innovation that has changed the course of medicine has been the introduction of ultrasound (US) guidance and the increasing prevalence of US-guided procedures. Traditionally, intra-articular and other soft-tissue injections were performed using landmarks. However, US-guided injections have slowly become more commonplace in many practices as they increase the accuracy of injections and allow providers to see specific structures relevant to needle placement and manipulation. This increased accuracy has been demonstrated in cadaveric studies, which found that US guidance increased the accuracy of intrasheath injections into the flexor tendon sheath near the A1 pulley.[18] Studies have demonstrated that US provides increased accuracy for TF injections. However, studies have also shown that the increased accuracy does not necessarily lead to a significant improvement in clinical outcomes in the case of TF.

One of the main advantages of using US guidance when performing TF injections is that providers can visualize the needle entering the flexor tendon sheath. It was previously hypothesized that if a greater amount of steroid could be administered within the sheath, then clinical outcomes would improve. However, several studies comparing intrasheath vs extrasheath corticosteroid injections in the treatment of TF found no significant differences in clinical outcomes and injection into either space provides comparable symptomatic relief.[19,20] However, clinical studies comparing blind vs US-guided injection techniques have mixed results. One study demonstrated that those who received US-guided TF injections did not differ significantly from the blind group when comparing pain or the need for additional injections after initial treatment. It was concluded that US guidance only created extra effort and cost without increased clinical benefits.[21]. A recent study found that patients who received US-guided injections experienced greater symptomatic relief in the first 1 to 4 weeks and could return to activities faster than the blind group. However, there was no significant long-term difference when symptoms were reassessed at weeks 6 and 12.[22] Certainly, US-guided injections depend on various factors including physician skill, the type of machine, and other factors that can all affect clinical outcomes. Due to the mixed results of these studies, further repeat studies with larger sample sizes would be necessary to determine the efficacy of US in improving clinical outcomes for TF.

In addition to introducing US guidance to improve the accuracy of injections, various techniques have also been employed to increase accuracy while decreasing pain. Currently, the traditional injection technique for TF is a blind approach in which the needle is inserted on the palmar surface of the flexor tendon over the metacarpal head. Other alternative injection approaches utilized are the proximal phalanx and midaxial techniques. The proximal phalanx technique

approaches the flexor tendon sheath from the palmar surface at the midproximal phalanx, while the midaxial technique approaches the flexor tendon sheath perpendicularly. These techniques were hypothesized to be less painful than the traditional approach, as the palmar skin contains a high density of sensory receptors. Studies comparing these techniques found that the mixaxial and proximal phalanx approaches are less painful than conventional techniques. In addition to being less painful, it was also found that recurrence rates did not differ between these alternative approaches and conventional techniques.[23,24]

ALTERNATIVE INJECTIONS: ORTHOBIOLOGICS FOR THE TREATMENT OF TF

As the treatment of musculoskeletal injuries has progressed, orthobiologics such as platelet-rich plasma (PRP), stem cells, and hyaluronic acid have become more prominent. PRP effectively treats various pathologies such as rotator cuff tendinopathy, lateral epicondylitis, and patellar tendinitis.[25] However, PRP has not been widely studied and documented in treating TF or other hand pathologies. One case report details the experience of a 63-year-old female patient who was diagnosed with TF. Over 3 weeks, the patient received three PRP injections. The triggering had entirely resolved at their 3-month follow-up and the patient no longer experienced any pain. This study concluded that PRP injection may potentially be an effective treatment for TF.[26] While this case is largely preliminary, further follow-up studies would need to be conducted to establish PRP as a viable treatment option for TF. Another case report details the experience of a 38-year-old patient who underwent PRP therapy for the treatment of wrist flexor tenosynovitis. This patient received a 3-mL injection of PRP into the carpal tunnel. At the 6- and 12-week follow-up appointments, VAS and Quick Disabilities of Arm, Shoulder and Hand (Q-DASH) scores had improved compared to baseline.[27] While the pathophysiology of TF and flexor tenosynovitis follows similar mechanisms, the results of this case cannot necessarily be applied to the treatment of TF. Further case reports and control trials regarding PRP for treating TF are necessary to establish the efficacy of this treatment modality.

Currently, there is only anecdotal evidence that discusses using PRP for treating TF. However, research in the field is growing. In 2020, a study protocol for a randomized control trial was registered and published in BMC. The protocol details a prospective, randomized, triple-blind, placebo-controlled trial that will compare the efficacy of PRP vs corticosteroid. To measure treatment outcomes, the investigators plan on using Patient Rated Wrist Evaluation (PRWE), Q-DASH, and VAS scores over 6 months. At the time of publication, the trial was in the recruitment phase and the results have yet to be published.[28] However, this protocol is the first of its kind and helps lay the groundwork for further research regarding the use of PRP for treating TF. Because there is only anecdotal evidence at this time, a randomized control trial will greatly benefit the field and help establish the efficacy of PRP compared to other injectate options. Furthermore, this protocol will benefit other investigators aiming to replicate or conduct similar randomized control trials regarding the use of PRP.

Hyaluronic acid has also been studied for use in the treatment of TF. Hyaluronic acid is an effective treatment option for musculoskeletal injuries, specifically soft-tissue injuries such as shoulder, elbow, and ankle tendinopathies.[29] One study uses hyaluronic acid injection in conjunction with methylprednisolone to treat TF. It was hypothesized that the hyaluronic acid would act as a mechanical intermediary that would aid in restoring synovial fluid viscosity, thus enlarging narrowed tendon sheaths. In conjunction with a corticosteroid, these properties would allow for optimal tendon gliding and patient recovery. Fifteen patients received a combination of hyaluronic acid and methylprednisolone, and 14 reported a complete resolution of symptoms at the 6-month follow-up.[30] This study has several limitations including a small sample size and the fact that it does not compare the hyaluronic acid group to a corticosteroid monotherapy group. Therefore, further studies would need to be conducted to establish the effect hyaluronic acid has on the treatment of TF.

Orthobiologics are becoming a high-profile option for treating musculoskeletal pathologies, but limitations exist. The cost of these treatments has led clinicians and investigators to investigate the cost-efficacy of these alternative injectate options. While many alternative injectate options can be a potentially promising avenue for the treatment of TF, the cost variability of orthobiologic treatments, such as PRP, may be a limiting factor for patient access.[31] One study conducted regarding the use of PRP for lateral epicondylitis showed that it was a more cost-effective option when compared to surgery and corticosteroid injections.[32] However, the study did not include a cost-benefit analysis of other upper-extremity disorders, so the conclusions may not apply to the treatment of TF. Currently, there are no cost-benefit analyses for using PRP or other orthobiologics for treating TF, which would be necessary to determine if this is a viable treatment recommendation for most patients.

RISKS OF OF INJECTION

Generally, traditional injections with corticosteroids and local anesthetic are considered very safe for most patients. While it is thought that these injections are less effective among those with diabetes mellitus, or a history of multiple injections, the risks are mostly identical. The most common adverse effects of TF injection include local skin hypopigmentation, dermal atrophy, fat atrophy, infection, and pain at the injection site. For patients with diabetes, there is also a risk of a transient increase in serum glucose. The most serious complication of injection, while incredibly rare, is tendon rupture.[1] One case report details the rupture of the flexor digitorum profundus (FDP) tendon following the injection of insoluble corticosteroid for the treatment of TF.[33] While this is an infrequent complication, cases have been documented and surgical intervention is required to correct the rupture.[34]

CLINICAL IMPLICATIONS AND AVENUES FOR FURTHER RESEARCH

The studies reviewed above leave much room for further study regarding the treatment of TF. Currently, standard practice for the injection of TF includes the use of corticosteroids and a local anesthetic like lidocaine. However, the chondrotoxic effects of lidocaine and other anesthetics can be concerning for both the patient and the provider. Studies have demonstrated that the use of local anesthetics in TF injections does not necessarily decrease perceived pain. The implications of this study have the potential to change the standard of practice. However, this would require more robust studies and clinical trials to be conducted so providers have a wider base of literature to draw evidence-based practice conclusions from.

Current literature also leaves much room for further studies regarding the costs and benefits of various treatment options. While patient costs can vary from practice to practice, insurance providers, and other factors, literature has shown that traditional corticosteroid injection is one of the more cost-effective options for treating TF, followed by surgical release. However, other injectate options, such as PRP and orthobiologics, have become more high-profile and readily accessible. Further studies would need to be conducted to determine the efficacy of these treatment options, and cost-benefit analyses would be necessary to determine if they are viable treatment options that can be recommended to patients.

In addition to the types of injectate agents used for the treatment of TF, further studies need to be conducted regarding various injection techniques, specifically those done under US guidance. Currently, literature has shown mixed results regarding the long-term outcomes for patients who received TF injections with and without US guidance. Some studies have found that US-guided injection provides no significant benefits, while others have found that using US guidance improves early patient outcomes. Due to these mixed results, repeat studies with larger sample sizes and stringent control measures would benefit the area of study.

While the cause of the initial inflammation that leads to TF is still unclear, the study of potential prevention methods, such as rest, or osteopathic manipulative treatment (OMT), could be beneficial additions to the pool of current literature, as studies regarding TF prevention are not widely documented. Currently, there is only anecdotal evidence in the form of a case study that discusses the successful treatment of TF using OMT in conjunction with acupuncture. However, OMT could be beneficial in preventing TF or potentially decrease the need for repeat injections. Techniques such as myofascial release and carpal articulation are effective treatment methods for other pathologies, and these principles may be applicable in treating TF. Larger studies that look into various osteopathic techniques for the treatment or prevention of TF could be beneficial for the field and osteopathic medicine as a whole.

At this point, the mainstay treatment for TF remains symptomatic management with NSAIDs, splinting, corticosteroid injection, and surgical release. For those with symptomatic TF that is not responding to conservative treatment options, corticosteroid injection, and surgical release remain options. As previously stated, further research regarding the use of local anesthetic in these injections requires further investigation. However, as the use of local anesthetic in corticosteroid injections is commonplace, it may be worthwhile to consider using a relatively less chondrotoxic anesthetic agent such as ropivacaine. Further cost-benefit analyses and studies regarding the efficacy of percutaneous vs open TF release would benefit providers making referrals for surgical release.

AN OSTEOPATHIC PERSPECTIVE

The importance of rational treatment has been a mainstay of the osteopathic practice. When considering the treatment of TF, the family physician is likely the first provider a patient may see. A wide variety of treatment options is available, and how one goes about treating this pathology can differ based on a wide variety of patient factors. As family physicians, being aware of current TF practices and the innovations yet to come will help physicians be better equipped and provide more individualized care for their patients.

REFERENCES

1. Makkouk AH, Oetgen ME, Swigart CR, Dodds SD. Trigger finger: etiology, evaluation, and treatment. *Curr Rev Musculoskelet Med*. 2008;1(2):92–96. doi: 10.1007/s12178-007-9012-1
2. Henton J, Jain A, Medhurst C, Hettiaratchy S. Adult trigger finger. *BMJ*. 2012;345:e5743–e5743. doi: 10.1136/bmj.e5743
3. Koh S, Nakamura S, Hattori T, Hirata H. Trigger digits in diabetes: their incidence and characteristics. *J Hand Surg Eur Vol*. 2010;35(4):302–305. doi: 10.1177/1753193409341103
4. Schulman R, Levchenko A, Lombardo SR, Walkoski SA, Moroz A. Trigger finger in a male with diabetes successfully treated with acupuncture and osteopathic manipulative treatment. *Med Acupunct*. 2013;25(1):74–77. doi: 10.1089/acu.2012.0896
5. Peters-Veluthamaningal C, van der Windt DA, Winters JC, Meyboom- de Jong B. Corticosteroid injection for trigger finger in adults. *Cochrane Database Syst Rev*. 2009;Jan 21(1):CD005617. doi: 10.1002/14651858.CD005617.pub2
6. Peters-Veluthamaningal C, Winters JC, Groenier KH, Jong BM d. Corticosteroid injections effective for trigger finger in adults in general practice: a double-blinded randomised placebo controlled trial. *Ann Rheum Dis*. 2007;67(9):1262–1266. doi:10.1136/ard.2007.073106
7. Dardas AZ, VandenBerg J, Shen T, Gelberman RH, Calfee RP. Long-term effectiveness of repeat corticosteroid injections for trigger finger. *J Hand Surg Am*. 2017;42(4):227–235. doi:10.1016/j.jhsa.2017.02.001
8. Lewis J, Seidel H, Shi L, Wolf J, Strelzow J. National benchmarks for the efficacy of trigger finger and the risk factors associated with failure. *J Am Acad Orthop Surg Glob Res Rev*. 2023;7(2):e22.00198. doi: 10.5435/JAAOSGlobal-D-22-00198
9. Kerrigan CL, Stanwix MG. Using evidence to minimize the cost of trigger finger care. *J Hand Surg Am*. 2009;34(6):997–1005. doi: 10.1016/j.jhsa.2009.02.029
10. Rastogi AK, Davis KW, Ross A, Rosas HG. Fundamentals of joint injection. *Am J Roentgenol*. 2016;207(3):484–494. doi: 10.2214/AJR.16.16243
11. Roberts JM, Behar BJ, Siddique LM, Brgoch MS, Taylor KF. Choice of corticosteroid solution and outcome after injection for trigger finger. *HAND*. 2021;16(3):321–325. doi: 10.1177/1558944719855686
12. Ring D, Lozano-Calderón S, Shin R, Bastian P, Mudgal C, Jupiter J. A prospective randomized controlled trial of injection of dexamethasone versus triamcinolone for idiopathic trigger finger. *J Hand Surg Am*. 2008;33(4):516–522. doi: 10.1016/j.jhsa.2008.01.001
13. Chen YC, Chang HN, Pang JHS, et al. Lidocaine inhibited tendon cell proliferation and extracellular matrix production by down regulation of cyclin A, CDK2, type I and type III collagen expression. *Int J Mol Sci*. 2022;23(15):8787. doi: 10.3390/ijms23158787
14. Sung CM, Hah YS, Kim JS, et al. Cytotoxic effects of ropivacaine, bupivacaine, and lidocaine on rotator cuff tenofibroblasts. *Am J Sports Med*. 2014;42(12):2888–2896. doi: 10.1177/0363546514550991
15. Saffarian M, Holder EK, Mattie R, et al. FactFinders for patient safety: preventing local anesthetic-related complications: local anesthetic chondrotoxicity and stellate ganglion blocks. *Interv Pain Med*. 2023;2(4):100282. doi: 10.1016/j.inpm.2023.100282
16. Patel KR, Fralinger D, MacGillis KJ, Wright-Chisem J, Mejia A. The anesthetic effectiveness of J-tip needle-free injection system prior to trigger finger injection: a double-blind randomized clinical trial. *Hand*. 2021;16(6):776–780. doi: 10.1177/1558944719890035
17. Patrinely JR, Johnson SP, Drolet BC. Trigger finger corticosteroid injection with and without local anesthetic: a randomized, double-blind controlled trial. *Hand*. 2021;16(5):619–623. doi: 10.1177/1558944719884663
18. Lee DH, Han SB, Park JW, Lee SH, Kim KW, Jeong WK. Sonographically guided tendon sheath injections are more accurate than blind injections. *J Ultrasound Med*. 2011;30(2):197–203. doi: 10.7863/jum.2011.30.2.197
19. Shinomiya R, Sunagawa T, Nakashima Y, Yoshizuka M, Adachi N. Impact of corticosteroid injection site on the treatment success rate of trigger finger: a prospective study comparing ultrasound-guided true intra-sheath and true extra-sheath injections. *Ultrasound Med Biol*. 2016;42(9):2203–2208. doi: 1 0.1016/j.ultrasmedbio.2016.05.015
20. Mardani-Kivi M, Karimi-Mobarakeh M, Babaei Jandaghi A, Keyhani S, Saheb-Ekhtiari K, Hashemi-Motlagh K. Intra-sheath versus extra-sheath ultrasound guided corticosteroid injection for trigger finger: a triple blinded randomized clinical trial. *Phys Sportsmed*. 2018;46(1):93–97. doi: 10.1080/00913847.2018.1400897
21. Cecen GS, Gulabi D, Saglam F, Tanju NU, Bekler HI. Corticosteroid injection for trigger finger: blinded or ultrasound-guided injection? *Arch Orthop Trauma Surg*. 2015;135(1):125–131. doi: 10.1007/s00402-014-2110-9
22. Tunçez M, Turan K, Aydın ÖD, Çetin Tunçez H. Ultrasound guided versus blinded injection in trigger finger treatment: a prospective controlled study. *J Orthop Surg Res*. 2023;18(1):459. doi: 10.1186/s13018-023-03950-y
23. Jianmongkol S, Kosuwon W, Thammaroj T. Intra-tendon sheath injection for trigger finger: the randomized controlled trial. *Hand Surgery*. 2007;12(02):79–82. doi: 10.1142/S0218810407003535
24. Pataradool K, Buranapuntaruk T. Proximal phalanx injection for trigger finger: randomized controlled trial. *Hand Surgery*. 2011;16(03):313–317. doi: 10.1142/S0218810411005606
25. Kia C, Baldino J, Bell R, Ramji A, Uyeki C, Mazzocca A. Platelet-rich plasma: review of current literature on its use for tendon and ligament pathology. *Curr Rev Musculoskelet Med*. 2018;11(4):566–572. doi: 10.1007/s12178-018-9515-y
26. Medina-Porqueres I. Platelet-rich plasma in the management of trigger finger: a case report. *Ağrı*. 2022;34(3):222–224. doi: 10.14744/agri.2021.03064
27. Jena D, Barman A, Sahoo J, Baral D. Platelet-rich plasma in the treatment of recurrent flexor tenosynovitis of wrist complicated with carpal tunnel syndrome in a patient with type 2 diabetes mellitus: a case report. *J Orthop Case Rep*. 2022;12(4):97–100. doi: 10.13107/jocr.2022.v12.i04.2784
28. Aspinen S, Nordback PH, Anttila T, Stjernberg-Salmela S, Ryhänen J, Kosola J. Platelet-rich plasma versus corticosteroid injection for treatment of trigger finger: study protocol for a prospective randomized triple-blind placebo-controlled trial. *Trials*. 2020;21(1):984. doi: 10.1186/s13063-020-04907-w
29. Khan M, Shanmugaraj A, Prada C, Patel A, Babins E, Bhandari M. The role of hyaluronic acid for soft tissue indications: a systematic review and meta-analysis. *Sports Health*. 2023;15(1):86–96. doi: 10.1177/19417381211073316
30. Kanchanathepsak T, Pichyangkul P, Suppaphol S, Watcharananan I, Tuntiyatorn P, Tawonsawatruk T. Efficacy comparison of hyaluronic acid and corticosteroid injection in treatment of trigger digits: a randomized controlled trial. *J Hand Surg Asian Pac Vol*. 2020;25(01):76–81. doi: 10.1142/S2424835520500101
31. Momaya AM, McGee AS, Dombrowsky AR, et al. The cost variability of orthobiologics. *Sports Health*. 2020;12(1):94–98. doi: 10.1177/1941738119880256

32. Klifto KM, Colbert SH, Richard MJ, Anakwenze OA, Ruch DS, Klifto CS. Platelet-rich plasma vs. corticosteroid injections for the treatment of recalcitrant lateral epicondylitis: a cost-effectiveness Markov decision analysis. J Shoulder Elbow Surg. 2022;31(5):991–1004. doi: 10.1016/j.jse.2021.12.010

33. amada K, Masuko T, Iwasaki N. Rupture of the flexor digitorum profundus tendon after injections of insoluble steroid for a trigger finger. *J Hand Surg Eur Vol.* 2011;36(1):77–78. doi: 10.1177/1753193410382377

34. Lu H, Yang H, Shen H, Ye G, Lin XJ. The clinical effect of tendon repair for tendon spontaneous rupture after corticosteroid injection in hands. Medicine. 2016;95(41):e5145. doi: 10.1097/MD.0000000000005145

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DISCOVER OUR REMARKABLE JOURNEY FROM 1950 TO TODAY

1950

The American Osteopathic Association (AOA) officially recognizes ACGPOMS, calling it "one of the most important to arise in our profession's history."

1953

- Eight osteopathic physicians found the American College of General Practitioners in Osteopathic Medicine and Surgery (ACGPOMS). Four "at large" members are added to the Board, for a total of 12.
- ACGPOMS is incorporated in Los Angeles County, California.

1954

- ACGPOMS is now referred to as the American College of General Practitioners (ACGP).
- Frank York Lee, DO, "MD," one of ACGP's key founders, becomes ACGP's first Asian American president.

1955

ACGP presents the first Practitioner of the Year Honor Award to Frank MacCracken, DO—now the Osteopathic Family Physician of the Year Award.

1957

The first student chapter, The Alpha Society, is founded at the Kansas City College of Osteopathic Medicine.

Kansas City University

1958

ACGP holds its first official national meeting in Des Moines, Iowa.

1960

- ACGP names Alfred Schramm, DO, as the organization's first executive secretary (executive director), the only DO to serve in this position to date.
- The ACGP Congress of Delegates is established.

1961

Amid the California Merger movement, hundreds of DOs exchange their DO degrees for an MD designation, causing significant membership losses.

1962

ACGP relocates to Chicago, Illinois, to expand its national footprint.

1965

The Conclave of Fellows inducts its first 24 Fellows, recognizing exceptional leadership and service.

1966

ACGP seeks to establish general practice as specialty within the osteopathic medical certification process and is met with intense resistance from the NBOE and the Advisory Board to Osteopathic Specialist (ABOS).

1972

- Mary M. Burnett, DO, FACGP, becomes ACGP's first female president.
- The American Osteopathic Board of General Practitioners (AOBGP) is established as the certifying board for osteopathic general practitioners. T. Robert Sharp, DO, FACGP, ACGP, is issued certificate No. 1 for being instrumental in making certification a reality.

1974

ACGP's first residency program graduates its first resident in osteopathic general practice, James B. Donahue, DO.

1986

The ACGP Education and Research Foundation is established, furthering the organization's commitment to education and philanthropy."

1987

Ray E. Piper, DO, FACOFP, becomes ACOFP's first African American president, heralding greater diversity in leadership.

1988

A groundbreaking ceremony is held for ACGP's permanent building in the northwest suburbs of Chicago, establishing a dedicated home base.

1990

The Auxiliary to the ACGP (AACGP) is launched, bringing together spouses and significant others of osteopathic physicians in support of the profession.

AACOFP Auxiliary TO THE AMERICAN COLLEGE OF OSTEOPATHIC FAMILY PHYSICIANS

1993

ACGP changes its name to the American College of Osteopathic Family Physicians (ACOFP), reflecting a focus on family medicine within the osteopathic tradition.

ACOFP
American College of Osteopathic Family Physicians

1997

To protect its members from further loss of professional privileges, ACOFP reissues all Certificates of Certification, adding the words "osteopathic manipulative treatment" to "family practice."

2000

ACOFP debuts the Osteopathic Family Medicine In-Service Training Exam, enhancing residency education and performance assessment.

AOA | ACOFP | acofp
IN-SERVICE EXAM

2004

ACOFP inducts its first class of distinguished fellows, honoring 21 recipients.

2007

The ACOFP Lifetime Achievement Award is established, celebrating members who have dedicated their careers to advancing osteopathic family medicine.

2009

- ACOFP launches its peer-reviewed medical journal, Osteopathic Family Physician (OFP).
- ACOFP holds the inaugural ACOFP Future Leaders Conference.

2012

- The Accreditation Council for Graduate Medical Education (ACGME) announces plans to exclude all AOA-certified postgraduate training. ACOFP advocates vigorously for osteopathic family physicians during this transition.
- The ACOFP Outstanding Female Leader Award is established.

acofp WOMEN'S INSTITUTE FOR LEADERSHIP

2016

Elizabeth A. Palmarozzi, DO, FACOFP, is elected as the first female speaker of the ACOFP Congress of Delegates.

2018

ACOFP launches the Clinical Osteopathic Recognition Training Examination (CORTEx) to help residency programs meet ACGME Osteopathic Recognition guidelines.

CORTEx
CLINICAL OSTEOPATHIC RECOGNITION TRAINING EXAM

2020

acofp '20

In response to the COVID-19 pandemic, ACOFP cancels its annual convention in New Orleans and launches its first virtual annual convention.

2021

- The Congress of Delegates votes to include allopathic physicians as full voting members; Brian Bixler, MD, becomes the first MD member.
- ACOFP creates the Diversity, Equity, and Inclusion (DEI) Award, honoring Tejal R. Patel, DO, as the first recipient.
- ACOFP wins the AOA Outstanding Affiliate of the Year Award.

2023

acofp FOUNDATION

- The ACOFP Foundation surpasses its $2 million fundraising goal for "Forging Our Osteopathic Future," more than a year ahead of schedule.

2024

- Ryan M. Smith, DO, FAAFP, is named the first New Physician-in-Practice Governor.
- The Foundation launches the "Family Medicine Forward" campaign, aiming to raise $750,000 to enhance programs for residents and early career physicians.

FAMILY MEDICINE FORWARD

2025

2025 Celebrating 75 YEARS

Since its founding ACOFP has grown into a diverse, influential organization dedicated to advancing osteopathic family medicine. Throughout the decades, it has navigated significant challenges—from the California Merger movement to shifts in national certification processes—and championed groundbreaking initiatives, including the establishment of robust residency programs, pivotal leadership roles for women and people of color, and the integration of allopathic physicians. These milestones reflect ACOFP's commitment to holistic, patient-centered care and underscore its vital role in shaping the future of osteopathic family medicine.

WELCOME TO THE ACOFP STORY

Founded in 1950 by eight visionary osteopathic physicians, the American College of Osteopathic Family Physicians (ACOFP) has championed comprehensive patient care, medical education, and professional growth for over seven decades. We're excited to share the highlights of our rich history, from early milestones and pioneering leaders to modern achievements and a future filled with promise.

EXPERIENCE THE

Magic of OrlanDO with ACOFP

Save the Date

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acofp '26
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acofp AMERICAN COLLEGE OF OSTEOPATHIC FAMILY PHYSICIANS

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Celebrating 75 YEARS

History, Leadership, & Innovation

A LEGACY OF EXCELLENCE IN OSTEOPATHIC FAMILY MEDICINE

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