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EDITOR'S MESSAGE

A legacy of leading with purpose to improve patients' health outcomes

Paula Gregory, DO, MBA, FACOFP

"Standing on the shoulders of giants allows us to see far into the future with a clearer vision" is an often-quoted phrase attributed to John of Salisbury in the twelfth century (1159). We celebrate past leaders who made our learning and profession. The giants of the past moved through the education and healthcare landscapes skillfully navigating uncertain opportunities and uncertain futures.

We are forever indebted to the giants of the past as leaders and mentors as they looked ahead to effortlessly steer us into the future—or so it seemed. Our ACOFP past presidents carried heavy responsibilities and anticipated future challenges as they steered through payment and contracting issues such as initiating diagnostic-related groups and subsequently value-based care, new and clinical challenges with emergent diseases such as HIV and hepatitis C, and even a pandemic, of all things!

Our past is full of admirable and impactful leaders: people who weathered storms and guided our entry into many of these mandated areas. Widespread uptake of the EMR, and the changes the EMR has subsequently had on practice styles, has often challenged us and made us recorders of information. We look ahead to a f uture where information is fed directly into EMRs by medical devices, changing our engagement with patients to having immediate recall of information at our fingertips rather than fragmented information that is retrospectively reviewed. Other tools available include virtual monitoring, exam and care of our patients, and the ability to help our patients remotely. Disabilities can be avoided by intervention virtually with patients and units at the patient's home. Stroke and cardiac care are now started in the home before patients are transported to hospitals or healthcare facilities. This small change in care can prevent damage to organ systems that was once so commonplace and hopeless. Monitoring that goes directly into the patient's chart and day-to-day intervention will lead to healthier patients rather than the fragmented care our patients may have experienced in the past. Morbidity and mortality can be changed for the better through monitoring and by making adjustments prior to an office visit. Patients can be treated at home and brought into the clinic if needed.

The leadership of ACOFP's past presidents has been instrumental in working with the government to advocate changes for our students, and our physicians and have shouldered the burden of an uncertain future. They've advanced our profession into an ever-growing part of the healthcare system: since 2016, unified accreditation has allowed more choices for osteopathic students. And by 2030, 30% of all medical students will be osteopathic students. But the task is not complete. Continued advocacy to show parity in our testing, curriculum, and outcomes has to be known. Using tools that improve the diagnosis and care of patients, with high touch and high treatment to ameliorate disease and improve function, osteopathic physicians have contributed to bettering our nation's health outlook. We consider the entirety of the patient, and social determinants of health have long been part of our understanding and care. Osteopathic physicians give advice on nutrition and how to achieve a healthy lifestyle. This has been the beauty of the curriculum, as we consider not only the disease but also the path to overall wellness. Osteopathic physicians are leaders and care deeply for the communities under our care.

Join me this month in appreciating our past leaders, who were instrumental to our organization working for the best of everyone.

FROM THE PRESIDENT'S DESK

Spotlighting the past to strengthen ACOFP's future

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

Over the past several months, I've had the privilege of visiting various ACOFP state society meetings and student chapter meetings at our Colleges of Osteopathic Medicine (COMs). These interactions have been inspiring, allowing me to connect with fellow osteopathic family physicians, residents, and students and to gain valuable insights into our diverse and vibrant ACOFP community. It has been a privilege to engage with them and learn about what is happening in our states, and I am deeply grateful for the warm reception I've received at every stop.

Looking ahead, I am excited about my upcoming visits to additional ACOFP state societies and COMs. These visits will give us more opportunities to foster connections, share knowledge, and strengthen our ties as a united osteopathic family. Your participation and engagement are invaluable in shaping the future of our profession. I also ask for your help by encouraging ACOFP membership to every family physician in your circle of influence. Only together can we grow our wonderful organization and achieve new heights!

My presidential theme of legacy honors the achievements and inspirations provided by past leaders and promotes the possibilities of what's to come. In this issue of Osteopathic Family Physician and in future issues, we will aim to spotlight past presidents who remain active in our organization with their service and continue to grow their legacies in the ACOFP. While our commitment to improving our patients' health is essential, so is our active involvement in leadership. There are many ways for you to involve yourself in this endeavor and one great way is to become more active in the ACOFP. Our website can provide more information on various ways to be engaged as leaders in service.

As proud members of the osteopathic family, it is vital that we continue to uphold the legacy of our profession's founder, Dr. Andrew Taylor Still, whose birthday was celebrated in August. Let's promote osteopathic medicine every chance we get and recharge our dedication to the osteopathic principles and practices that guide us as osteopathic family physicians. We can be the gold standard role models for the thousands of new osteopathic medical students and residents who recently embarked on our collective journey. Let us be inspired by the leadership of our past, with a bright outlook for the future as we continue to incorporate new technologies, innovation, and knowledge in medicine to enhance the future of health care and wellness of our patients and ourselves.

I extend my heartfelt appreciation to each of you for your commitment to the ACOFP community and wish you all the best in your practices and endeavors.

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

PAST PRESIDENTS SPOTLIGHT

THIS NEW FEATURE WILL HIGHLIGHT THE LEGACY OF ACOFP PAST PRESIDENTS.

Martin Porcelli, DO, PhD, MHPE, FAOASM, FACOFP *dist.*

TERM OF PRESIDENCY: 2003-2004

THEME OF PRESIDENCY: The Year of the Veteran (The Practicing Physician)

ACCOMPLISHMENTS:

- Publication of the history book, covering the 50+ preceding years of ACOFP from February 11, 1950, forward
- The Procedure Institute (teaching spirometry, dermatologic procedures, and joint injections at various venues)
- Setting up and attending the meeting with the Minister of Health in Calgary, Canada, to promote DO licensure

Jeffrey S. Grove, DO, FACOFP *dist.*

TERM OF PRESIDENCY: 2013-2014

THEME OF PRESIDENCY: Remembering the Past, Celebrating the Present, Anticipating the Future!

ACCOMPLISHMENTS:

- Led the creation of the ACOFP 2013-2016 Strategic Goals
- Laid the groundwork for future cooperation between DOs and MDs by attending AAFP and setting up the first historic meeting between leadership of ACOFP and AAFP (which took place during Carol Henwood's term)
- Created first ever osteopathic LGBTQ national committee (the Special Constituencies committee, as it was then known), as well as beginning the tradition of an LGBTQ+ reception at ACOFP

Robert DeLuca, DO, FACOFP *dist.*

TERM OF PRESIDENCY: 2019-2020

THEME OF PRESIDENCY: Connect and Communicate

ACCOMPLISHMENTS:

- Worked with AOBFP and the Early Entry Initial Certification program
- Increased the diversity of the board
- Coordinated and monitored the processes involved in navigating through COVID, including the transition to virtual educational formats

REVIEW ARTICLE

A SYSTEMATIC REVIEW OF TREATMENTS FOR MILD TRAUMATIC BRAIN INJURY IN ADULTS: HEADACHE-MIGRAINE, OCULOMOTOR, AND VESTIBULAR CONCUSSION SUBTYPE OUTCOMES

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KEYWORDS

Traumatic brain injury

TBI

Concussion

Headaches

Migraines

ABSTRACT

Context: Up to 15% of concussed patients experience persistent symptoms and functional impairment following injury. This is often related to headaches, dizziness, imbalance, and visual disturbances.

Objectives: To perform a systematic review of the evidence for interventions used to manage postconcussion symptoms in working-aged adults falling within the headache-migraine, ocular, and vestibular postconcussion symptom cluster subtypes.

Methods: A literature search was performed according to the PRISMA statement. PubMed, OVID, Cochrane Central, PEDro, OSTEMED, and the grey literature checklist were searched from the dates of creation of each database through December 29, 2020. The outcome measures were compared by generating the standardized mean difference (SMD) with 95% confidence intervals. GRADE (Grading of Recommendations, Assessment, Development and Evaluation) was used to rate the overall quality of the evidence.

Results: The literature search identified 496 candidate studies. After removing duplicates, 352 studies remained. The titles and abstracts of the remaining studies were screened for eligibility and 343 studies were excluded. The full text of the remaining nine studies was assessed for eligibility and risk of bias. None of these studies was excluded. This left nine studies for qualitative and quantitative analysis.

Conclusions: Moderate-quality evidence suggests 4 interventions show promise for treating adults with headache-migraine, ocular, and vestibular postconcussion subtype symptoms.

INTRODUCTION

Clinical symptoms of mild traumatic brain injury (mTBI) typically resolve spontaneously, with 80%–90% of concussed older adolescents and adults returning to preinjury levels of clinical function within 2 weeks.[1] However, up to 15% of concussed patients experience persistent symptoms and functional impairment following injury that may have severe personal costs and make it hard to resume their normal jobs and lives. Workers that sustain an mTBI have a higher risk of being out of work five years after the trauma compared to their noninjured peers. Patients in their thirties and with a higher education were found to be at higher risk of experiencing these long-term consequences.[2]

Concussions produce a heterogenous variety of symptoms, presentations, and clinical courses. The most common presenting clusters of symptoms can be used to classify probable and possible mTBI into subtypes, which can be used to develop targeted treatment strategies.[3] Headache is the most common postconcussion symptom reported by adults with a prevalence of 86%–96%.[4] Patients with headache-migraine concussion subtype (HCS) symptoms often complain of associated nausea, vomiting, and sensitivity to light and sound. Premorbid headache conditions place individuals at greater risk of postconcussion headaches.[3]

The oculomotor concussion subtype (OCS) presents with oculomotor and visual dysfunction. These dysfunctions may be detected by assessing saccades, smooth pursuit, convergence, and fixation. Oculomotor dysfunction is often found in association

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with vestibular symptoms. Patients presenting with this subtype report difficulty with visual activities (e.g., eye strain, photophobia, blurred or double vision, frontal headaches, pressure behind the eyes, vision-derived nausea, poor depth perception, difficulty tolerating visually complex environments, worsening of premorbid visual impairment).[3] Convergence insufficiency occurs in up to 65% of patients with concussion, smooth pursuit dysfunction affects approximately 60% of concussed patients, and saccadic dysfunction is present in about 30% of concussed patients.[5]

The vestibular concussion subtype (VCS) presents with at least one of the following symptoms: dizziness, fogginess, lightheadedness, nausea, vertigo, or disequilibrium. These symptoms are provoked by dynamic movement. Dysfunction may affect gait and balance. Patients with this concussion subtype often demonstrate concurrent neurocognitive defects and symptoms related to anxiety.[5] Dizziness affects about 67% of patients with concussion.[5]

There is a tremendous amount of literature reviewing concussion management; however, much of this literature includes studies of children with sports-related concussions. It is often said that children are not small adults. The converse is also true, adults are not large children. The objective of this manuscript is to perform a systematic review of the evidence for interventions used to manage headache-migraine, ocular, and vestibular postconcussion subtype symptoms in working-aged adults.

METHODS

A literature search was performed on December 29, 2020, in line with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) statement. Clinical trials studying treatment outcomes of patients diagnosed with concussion or mild to moderate traumatic brain injury (TBI) were used to generate this systematic review. There were no outside sources of funding for this research project and institutional review board approval was not required.

Studies were identified by searching PubMed, OVID, Cochrane Central, PEDro, and OSTEMED, and using the Canadian Agency for Drugs and Technologies in Health grey literature checklist. The search was limited to English language publications. The dates of coverage for each database search were from the creation of the database through December 29, 2020. The following search strategy was used to search each database: ((Concussion OR mild traumatic brain injury OR postconcussion syndrome OR postconcussion symptoms) AND (vestibular OR ocular OR cognition OR anxiety OR depression OR headache OR fatigue) AND (management OR treatment)).

Eligibility assessment was performed in an unblinded standardized manner by a single reviewer. To be eligible for this systematic review, the considered study had to be a clinical trial; the subjects had to be human; the population had to be of working age (16−70 years); the study had to be published in English; and the outcomes had to be presented numerically with 95% confidence intervals (CIs), standard deviations, or standard errors.

The outcome measures of the intervention and control groups for each study were compared by calculating standardized mean differences (SMD) with 95% CIs. Grand means would have been calculated if more than one study of the same intervention had assessed the same outcome and had lacked significant heterogeneity.[6] Statistical analysis was performed using Microsoft Excel 360.

GRADE (Grading of Recommendations, Assessment, Development, and Evaluation) was used to rate the overall quality of the evidence for risk of bias, inconsistency, indirectness, imprecision, publication bias, and magnitude of effect. The GRADE ratings of very low, low, moderate, or high-quality evidence reflect the extent to which one can be confident that the effect estimates are correct.[7] A mechanistic approach was used to minimize the risk of the single reviewer introducing bias during this process.

To ascertain the quality of eligible randomized controlled trials, the author used the National Institute for Health and Care Excellence (NICE) methodology checklist for reviewing randomized controlled trials (RCTs). The NICE methodology checklist is a tool used to qualitatively assess RCTs for risk of four types of bias: selection bias, performance bias, attrition bias, and detection bias.8 As there was only one reviewer, a quantifiable measure for determining risk of bias was incorporated into the NICE checklist. Each category of bias was initially assigned the value of one. One point was added for each "No" or "Unclear" answer. The remaining value was the quality measure for the given type of potential bias (4 = high risk, 3 = moderate-high risk, 2 = low-moderate risk, 1 = low risk). The quality measures were summed and then divided by four to generate the average risk of bias for each included RCT.

Joanna Briggs Institute (JBI) critical appraisal checklist for case series was used to assess the methodological quality of any case series meeting eligibility criteria.[9] The JBI critical appraisal tool for case series studies includes ten questions addressing the internal validity and risk of bias of case series designs, particularly confounding, selection, and information bias, in addition to the importance of clear reporting. Once again, as there was only one reviewer, a quantifiable measure for determining risk of bias was incorporated into the JBI checklist. Each case series began with an assigned value of one and a point was added for each negative response on the questionnaire. The final value was the quality measure for overall risk of potential bias (4 = high risk, 3 = moderate-high risk, 2 = low-moderate risk, 1 = low risk).

For both checklists, if the average risk of bias was between 1.0 and 2.0, the risk of within-study bias was deemed to be low. If the average risk of bias was between 2.01 and 3.0, the risk of within-study bias was considered serious. If the average risk of bias was between 3.01 and 4.0, the risk of within-study bias was determined to be very serious.

Inconsistency was considered when more than one study compared the same intervention and control using a similar outcome measure. Inconsistency was investigated using forest plots. To be consistent, each point estimate must rest within the 95% CIs of the comparable studies. If any point estimates fell outside the CIs, serious inconsistency was deemed to exist

among the considered studies. Very serious inconsistency was determined to exist when any of the CIs failed to overlap each of the other included CIs.

Indirectness exists when the study population differs from the population of interest; when the study intervention differs from the intervention of interest; when the study outcome differs from the outcome of interest; or when the interventions of interest are not tested head to head. If any one of these conditions were met, serious indirectness was noted. If two or more of these conditions were met, very serious indirectness was deemed to be present.

Serious imprecision was determined to be present when the 95% CI for the point estimate of effect of a study or group of studies crossed the null effect line. Serious imprecision was also considered to be present if a study's authors noted that the study was underpowered. Very serious imprecision existed when the CI crossed the null effect line and the contralateral clinically meaningful effect line.

Publication bias usually exists when a literature search fails to identify studies with negative outcomes. Publication bias was considered when more than one study compared the same intervention and control using a similar outcome measure and the included studies failed to present any negative findings. A funnel plot was chosen as the means of presenting this assessment.

Results of the analyses were used to generate an evidence profile table and summary of findings tables with forest plots.

RESULTS

The literature search identified 496 candidate studies. After removing duplicates, 352 studies remained. The titles and abstracts of the remaining studies were screened for eligibility and 343 studies were excluded. The full text of the remaining nine studies was assessed for eligibility and risk of bias. None of these studies was excluded. This left nine studies for qualitative and quantitative analysis (Figure 1). A summary of the interventions and controls from each of the studies is presented in Table 1 (see online version).

One study was found to be at high risk for selection bias. Two studies were at high risk for performance bias. The average risk of bias was determined to be severe for three of the studies. None of the studies was determined to have a very serious average risk of bias (Table 2) (see online version).

Studies examining headache-migraine symptom cluster subtype included four discrete interventions, four controls, and four intervention and control pairs, resulting in six measured outcomes. For oculomotor subtype outcomes, there were two unrelated interventions, two controls, two intervention and control pairs, and 11 outcomes. Under the vestibular subtype, there were two interventions, one control, and two intervention and control pairs, producing four measured outcomes. Table 2 is an evidence profile table that presents the bias assessment, quality assessment, and SMD results for each of the studies' outcome measures. No grand means were generated due to heterogeneity of injury to group allocation time, treatments, outcome measures, and timing of outcome measurement.

Synthesis of results

HEADACHE-MIGRAINE POSTCONCUSSION SYMPTOM CLUSTER SUBTYPE (FIGURE 2)

Frequency

Very low-quality evidence showed group cognitive behavioral therapy (CBT) designed to manage postconcussion headaches did not decrease the frequency of postconcussion headaches when compared to being on a waitlist [n = 71; SMD (95% CI) = 0.15 (-0.31–0.62)].[10]

Intensity (corresponding SCAT-5 symptoms: headache and "pressure in head")

Moderate-quality evidence disclosed group CBT increased outcome measures of headache intensity [n = 71; SMD (95% CI) = 0.62 (0.14−1.09)].10 Moderate-quality evidence suggested erenumab, a calcitonin gene-related peptide inhibitor, improved measures of headache intensity when measured at 12 weeks' post treatment initiation [n = 100; SMD (95% CI) = -0.67 (-0.95 to -0.38)].11 Low- and very-low–quality evidence determined hyperbaric oxygen therapy (HBOT) did not reduce measures of headache intensity at 2-year [n = 40; SMD (95% CI) = 0.49 (-0.15–1.12)] and 3-year [n = 14; SMD (95% CI) = 0.19 (-0.92–1.29)] follow-up evaluations relative to sham treatment.12 Moderate-quality evidence showed 22 weeks of multidisciplinary care (MDC) that included psychological interventions reduced measures of postconcussion headache intensity relative to usual care [n = 89; SMD (95% CI) = -0.58 (-1.01 to -0.16)].[13]

OCULAR POSTCONCUSSION SYMPTOM CLUSTER SUBTYPE (FIGURE 2)

Photosensitivity (corresponding SCAT-5 symptom: sensitivity to light)

Moderate-quality evidence suggested use of nonliquid crystal display (non-LCD) screens produced fewer photosensitivity symptoms [n = 58; SMD (95% CI) = -3.75 (-4.61 to -2.90)] and lower symptom severity [n = 58; SMD (95% CI) = -4.61 (-5.59 to -3.62)] than use of liquid crystal display (LCD) screens after a 30-minute reading task was performed by postconcussion subjects.[14]

Accommodation (corresponding SCAT-5 symptom: blurred vision)

Moderate-quality evidence showed oculomotor rehabilitation improved measures of postconcussion amplitude of accommodation [n = 24; SMD (95% CI) = 0.99 (0.14−1.84)] and accommodative facility [n = 24; SMD (95% CI) = 0.98 (0.13−1.83)] relative to sham rehabilitation.[15]

Convergence (corresponding SCAT-5 symptom: blurred vision)

Moderate-quality evidence also showed oculomotor rehabilitation improved postconcussion convergence insufficiency symptom score [n = 24; SMD (95% CI) = -2.55 (-3.62 to -1.47)], near point convergence break [n = 24; SMD (95% CI) = -1.04 (-1.89 to -0.19)], and near point convergence recovery [n = 24; SMD (95% CI) = -0.87 (-1.71 to -0.03)] relative to sham rehabilitation.[16] However, moderate-quality evidence determined

oculomotor rehabilitation did not improve postconcussion stereoacuity [n = 24; SMD (95% CI) = -0.717 (-1.54−0.11)].[16]

Oculomotor reading behaviors

Low-quality evidence revealed oculomotor rehabilitation did not improve measures of reading rate [n = 24; SMD (95% CI) = 0.59 (-0.23−1.40)], reading comprehension [n = 24; SMD (95% CI) = 0.23 (-0.57−1.03)], or grade level efficiency [n = 24; SMD (95% CI) = 0.46 (-0.35−1.27)] in postconcussion subjects.[17]

VESTIBULAR POSTCONCUSSION SYMPTOM CLUSTER SUBTYPE (FIGURE 2)

Balance (corresponding SCAT-5 symptom: poor balance/ coordination)

Low-quality evidence determined 22 weeks of MDC did not improve self-reported measures of balance in postconcussion subjects relative to usual care [n = 89; SMD (95% CI) = -0.40 (-0.82 to 0.02)].[13] Moderate-quality evidence determined 8 weeks of vestibular rehabilitation did not improve measures of postconcussion balance compared to usual care that included Epley and BBQ roll maneuvers for subjects with a positive Dix-Hallpike maneuver [n = 57; SMD (95% CI) = -0.39 (-0.92−0.13)].[18]

Vestibular symptoms (corresponding SCAT-5 symptom: dizziness)

Moderate-quality evidence suggested 22 weeks of MDC with a robust psychological component did improve measures of postconcussion vestibular symptoms compared to usual care [n = 89; SMD (95% CI) = -0.44 (-0.86 to -0.02)].13 Low-quality evidence disclosed vestibular rehabilitation did not improve measures of postconcussion vestibular symptoms relative to usual care that included Epley and BBQ roll maneuvers for subjects with a positive Dix-Hallpike maneuver [n = 63; SMD (95% CI) = -0.27 (-0.77−0.23)].[18]

FIGURE 1:

Literature search flow diagram.

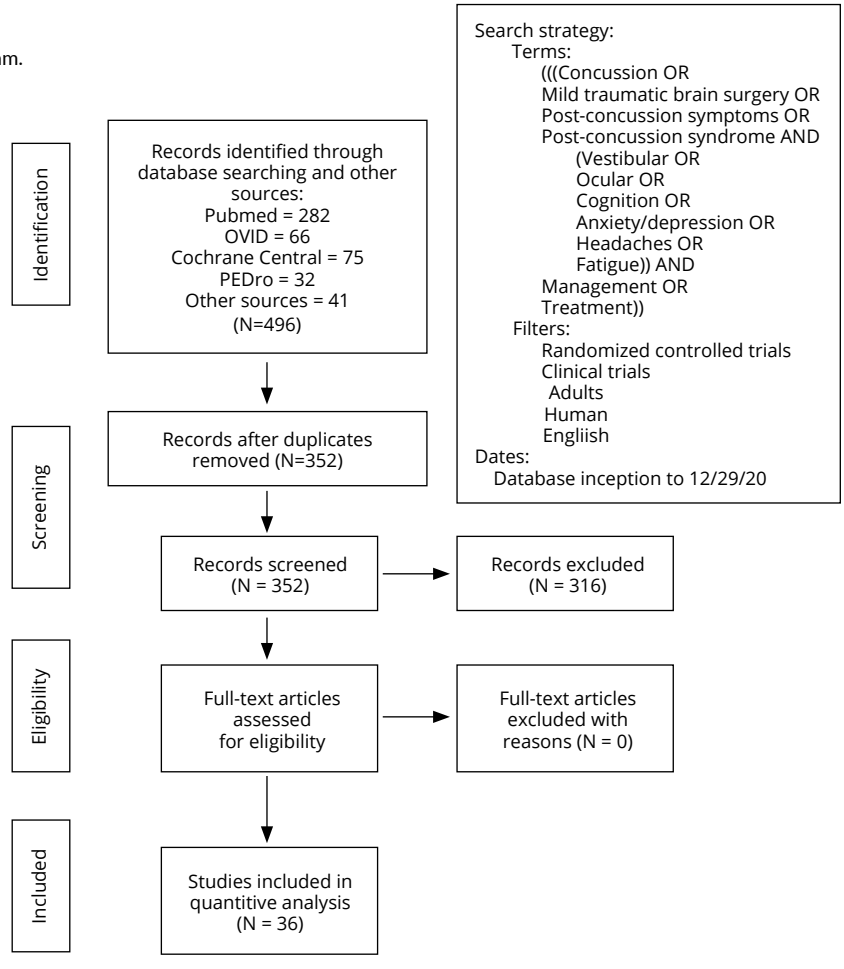

TABLE 1:
Summary of the studies meeting inclusion criteria.

AUTHOR (YEAR)	AGE	INJURY TO ALLOCATION TIME	INTERVENTION	CONTROL	COMMENTS
Ashina (2020)[11]	18 to 65 years	Mean of 59 months ± 54 months	Erenumab: 100 subjects received at least 1 dose of erenumab	Observational	Industry sponsored. Outcomes were measured at 12 weeks. 78 subjects reported at least 1 adverse event, the most common being constipation. 2 subjects experienced dizziness and worsening headache
Hart (2019)[12]	> 18 years	Not specified	HBOT: 40 chamber sessions at 1.5 atmospheres pressure with 100% oxygen over a 12-week period	Sham: 40 sessions consisting of room air at 1.2 atmospheres of pressure	This study is set apart by a 24-month and 36-month measurement of outcomes. Selection bias may have influenced the extended follow-up results, with the 24- and 36-month follow-up rates falling well below the 80% follow-up threshold that is proposed to be a threat to validity
Kjeldgaard (2014)[10]	18 to 65 years	Mean of 27 months	CBT (group): used a structured protocol for each weekly session. Week 1: introduction to the group and the diagnosis. Week 2: introduction to the cognitive model and the stress-pain connection, identification of stressors, and setting goals. Week 3: discussed memory problems and the connection to headaches. A breathing exercise was introduced as a relaxation technique. Week 4: reviewed memory and reading strategies, and management of energy. Progressive muscle relaxation was also taught. Week 5: introduction to the pain model and discussion of acceptance and behavior toward headache. A breathing exercise with body scan was presented. Week 6: reviewed acceptance of the present headache state and management of energy. Visualization of a pleasant place was taught as method of relaxation. Week 7: discussion of defining and identifying negative automatic thoughts (NAT). Week 8: introduced how to examine NAT and develop alternative more adaptive thoughts. Visualization problem solving also introduced. Week 9: discussion of integration and maintenance of new techniques and concepts		

TABLE 1 CONT.:
Summary of the studies meeting inclusion criteria.

AUTHOR (YEAR)	AGE	INJURY TO ALLOCATION TIME	INTERVENTION	CONTROL	COMMENTS
Kleffelgaard (2019)[18]	16 to 65 years	Mean of 3.5 months ± 2.1 months	Vestibular rehabilitation (group): twice weekly for 8 weeks. The intervention consisted of guidance, individually tailored exercises, a home exercise program, and an exercise diary. Exercises were Brandt-Daroff exercises for benign paroxysmal positional vertigo, habituation exercises for motion sensitivity and central posttraumatic vertigo, gaze-stabilization exercises for symptoms exhibited during eye-head coordination and reduced vestibulo-ocular reflex, and exercises for reduced balance, focusing on improving sensory integration. The home exercise program included 2 to 5 individually modified exercises and general physical activity	Usual care: did not receive any rehabilitation intervention in place of the group-based vestibular rehabilitation intervention. However, not to treat posttraumatic benign paroxysmal positional vertigo was deemed a conflict of research ethics, because of the strong existing evidence on the effect of canalith repositioning procedure. Therefore, patients with a positive Dix-Hallpike or roll test were treated with Epley and BBQ roll maneuvers	Outcomes were measured at 8 weeks. No adverse events of the intervention were registered
Mansur (2018)[14]	16 to 67 years	Not specified	Non-LCD screen	LCD screen	Randomized crossover design with outcomes measured before and after a 30-minute reading task on 2 consecutive days
Rytter (2019)[13]	18 to 65 years	> 6 months	Multidisciplinary care: psychoeducation, group therapy, psychological counselling, exercise training, and physiotherapeutic coaching. Length of the program was 22 weeks divided into 2 modules	Usual care: ranged from no treatment at all to referral to individual discipline-specific therapies	Usual care had a great degree of variability as to what treatments were offered, how much treatment was provided and at what intensity treatments were delivered. Given the complexity of the intervention, the study does not allow for one to determine whether the treatment effect is due to program intensity, interdisciplinary approach, accommodation of individual needs, or a combination of these factors. Nor does it allow one to ascertain the relative contributions of the individual treatment components

TABLE 1 CONT.:
Summary of the studies meeting inclusion criteria.

AUTHOR (YEAR)	AGE	INJURY TO ALLOCATION TIME	INTERVENTION	CONTROL	COMMENTS
Thiagarajan (2013)[16]	23 to 33 years	1 to 10 years	Oculomotor rehabilitation: twice per week, for a total of 6 weeks. At a session, each oculomotor component (version, vergence, and accommodation) was trained for 15 minutes, with 5-minute rest periods between each component. For this study only vergence training and related outcomes were presented	Sham training	Crossover, interventional, experimental design with subject blinded. Assessed convergence. Seriously underpowered
Thiagarajan (2014)[15]	23 to 33 years	1 to 10 years	Oculomotor rehabilitation: as described in Thiagarajan (2013). For this study, only the accommodative responsivity and related results were presented	Sham training	This is the second paper using data obtained from Thiagarajan (2013). Assessed accommodation. Crossover, interventional, experimental design with subject blinded. Seriously underpowered
Thiagarajan (2014)[17]	23 to 33 years	1 to 10 years	Oculomotor rehabilitation: as described in Thiagarajan (2013). For this study, only the reading-related oculomotor behavior and related results were presented	Sham training	This is the third paper using data obtained from Thiagarajan (2013). Assessed reading rate. Crossover, interventional, experimental design with subject blinded. Seriously underpowered

DISCUSSION

There is no high-quality evidence for treatment in postconcussion patients that meets inclusion criteria for this review. However, moderate-quality evidence from this systematic review has found two interventions that have shown promise for treating HCS symptoms and two interventions that show promise for treating OCS symptoms. No intervention was superior to Epley and BBQ roll maneuvers for VCS symptoms.

Rytter and colleagues compared MDC to usual treatment.[13] The injury to group allocation time for this trial was greater than 6 months. The intervention included psychoeducation, group therapy, psychological counselling, exercise training, and physiotherapeutic coaching for each subject. Usual-care treatment ranged from no treatment at all to referral to individual discipline-specific therapies.[13] Their results showed an improvement of HCS and VCS outcomes measures. The intervention in this trial was very structured, with each subject receiving each of the available interventions rather than receiving care as needed. The subjects in this trial were at least 6 months beyond their sustained head injury. This may have weeded out individuals who had spontaneous resolution of their symptoms. This suggests that early care provides little if any benefit for most patients who have sustained a concussion. This is interesting considering there is evidence suggesting psychological distress is common in the initial days following concussion. The degree of psychological distress correlates well with postconcussion syndrome symptom severity independent of injury severity and preexisting psychiatric disorders.[19]

Hyperbaric oxygen therapy (HBOT) is thought to treat TBI through generation of oxygen radicals, which facilitate production of neurotrophic growth factors and vascular endothelial growth factor, neural stem cell proliferation and mobilization, and modification of gene expression.[20] However, the findings of this review did not suggest a benefit for treating posttraumatic headache. The average risk of bias was found to be low for the included trial.

The intervention consisted of 40 chamber sessions at 1.5 atmospheres with 100% oxygen over a 12-week period. The intervention was compared to sham therapy involving 40 chamber sessions consisting of room air at 1.2 atmospheres. This result is disappointing; however, there is a great deal of debate regarding use of a sham control in HBOT research. The minimal elevated pressure a patient can sense is about 1.2 atmospheres, depending on the rate of change. This pressure can induce an elevation in tissue oxygenation of approximately 50% when the patient is breathing room air.[20] This is important to recognize because "sham" treatment under such conditions has been used as a "placebo" in experimental trials, when it may be a low-dose treatment. The results should not be generalized since the study recruited current or former military personnel who sustained head injuries in the line of duty. A larger proportion of these subjects may have experienced blast injuries, multiple TBIs, and posttraumatic stress disorder (PTSD) while also being skewed toward being younger and male without chronic medical conditions such as chronic obstructive pulmonary disease (COPD), diabetes, or coronary artery disease.

FIGURE 2:

Summary of findings for the headache-migraine postconcussion symptom cluster subtype, the oculomotor postconcussion symptom cluster subtype, and the vestibular postconcussion symptom cluster subtype. The solid vertical line is the null effect line. The dashed vertical lines are the minimal clinical effect lines. The side of the null effect line, indicative of a positive or negative effect, is dependent on the outcome measure. atm., atmospheres; SMD, standardized mean difference; CBT, cognitive behavioral therapy.

	Author (year)	Quality	Sample Size	SMD	Lower CI	Upper CI
Headache-Migraine						
CBT (group) vs Wait-list						
Headache frequency (26 weeks)	Kjeldgaard (2014)	Very Low	71	0.1528	-0.3135	0.6191
Headache intensity (26 weeks)	Kjeldgaard (2014)	Moderate	71	0.6158	0.1393	1.0923
Erenumab vs Baseline						
Headache intensity (12 weeks)	Ashina (2020)	Moderate	100	-0.6700	-0.9548	-0.3851
Hyperbaric Oxygen (1.5 atm) vs Sham						
Headache intensity (2 years)	Hart (2019)	Low	40	0.4890	-0.1470	1.1249
Headache intensity (3 years)	Hart (2019)	Very Low	14	0.1938	-0.9018	1.2893
Multidiciplinary Care vs Usual care						
Headache intensity (22 weeks)	Rytter (2019)	Moderate	89	-0.5810	-1.0052	-0.1568
Ocular	**Author (year)**	**Quality**	**Sample Size**	**SMD**	**Lower CI**	**Upper CI**
Non-LCD Screen vs LCD Screen						
Photosensitivity (Immediate)/SCAT-3 symptom severity	Mansur (2018)	Moderate	58	-4.6082	-5.5921	-3.6242
Photosensitivity (Immediate)/SCAT-3 No. of symptoms	Mansur (2018)	Moderate	58	-3.7547	-4.6101	-2.8992
Oculomotor Rehabilitation vs Sham						
Accommodation (15 weeks)/Amplitude of accommodation OU	Thiagarajan (2014)	Moderate	24	0.9932	0.1451	1.8412
Accommodation (15 weeks)/Accommodative facility OU	Thiagarajan (2014)	Moderate	24	0.9794	0.1326	1.8261
Convergence (15 weeks)/Convergence insufficiency symptom score	Thiagarajan (2014)	Moderate	24	-2.5456	-3.6221	-1.4691
Convergence (15 weeks)/Near point convergence break (cm)	Thiagarajan (2014)	Moderate	24	-1.0417	-1.8944	-0.189
Convergence (15 weeks)/Near point convergence Recovery (cm)	Thiagarajan (2014)	Moderate	24	-0.8694	-1.7066	-0.032
Convergence (15 weeks)/Stereoacuity (arc sec)	Thiagarajan (2014)	Moderate	24	-0.7173	-1.5428	0.1082
Reading (15 weeks)/Visagraph reading rate (wpm)	Thiagarajan (2014b)	Low	24	0.5872	-0.23	1.4045
Reading (15 weeks)/Visagraph comprehension (%)	Thiagarajan (2014b)	Low	24	0.2309	-0.5719	1.0337
Reading (15 weeks)/Visagraph comprehension (%)	Thiagarajan (2014b)	Low	24	0.457	-0.3535	1.2676
Vestibular	**Author (year)**	**Quality**	**Sample Size**	**SMD**	**Lower CI**	**Upper CI**
Multidisciplinary Care vs Usual Care						
Balance (22 weeks)	Rytter (2019)	Low	89	-0.396	-0.8156	0.0236
Vestibular symptoms (22weeks)	Rytter (2019)	Moderate	89	-0.4406	-0.8612	-0.0201
Vestibular Rehabilitation vs Usual Care						
Balance (8 weeks)	Kleffelgaard (2019)	Moderate	57	-0.3921	-0.9183	0.1341
Vestibular symptoms (8 weeks)	Kleffelgaard (2019)	Low	63	-0.2697	-0.7659	0.2264

TABLE 2:

GRADE evidence profile table of measured outcomes in the context of the postconcussion symptom cluster subtypes.

AUTHOR (YEAR)	COMPARISON (FOLLOW-UP)	MEASURE	DESIGN	TOTAL SUBJECTS	SELECTION BIAS	PERFORMANCE BIAS	ATTRITION	DETECTION BIAS
HEADACHE-MIGRAINE								
Headache Frequency								
Kjeldgaard (2014)[10]	CBT (group)vs waitlist (26 weeks)	Days/ 4 weeks	RCT	81	2	4	2	1
Headache Intensity								
Kjeldgaard (2014)[10]	CBT (group)vs waitlist (26 weeks	0-10 scale	RCT	81	2	4	2	1
Ashina (2020)[11]	Erenumab vs baseline (12 weeks)	HIT-6	CS	100				
Hart (2019)[12]	Hyperbaric oxygen vs sham (2 years)	MPQ-SF	RCT	40	2	2	1	1
Hart (2019)[12]	Hyperbaric oxygen vs sham (3 years)	MPQ-SF	RCT	14	2	2	1	1
Rytter (2019)[13]	Multidisciplinary care vs usual care (22 weeks)	HIT-6	RCT	89	2	3	2	2
OCULAR								
Accommodation								
Thiagara jan (2014)[15]	Oculomotor rehabilitation vs sham (15 weeks)	Amplitude	RC	24	1	2	1	3
Thiagara jan (2014)[15]	Oculomotor rehabilitation vs sham (15 weeks)	Facility OU	RC	24	1	2	1	3
Convergence								
Thiagara jan (2013)[16]	Oculomotor rehabilitation vs sham (15 weeks)	CISS	RC	24	1	2	1	3
Thiagara jan (2013)[16]	Oculomotor rehabilitation vs sham (15 weeks)	NPC Break	RC	24	1	2	1	3
Thiagara jan (2013)[16]	Oculomotor rehabilitation vs sham (15 weeks)	NPC Recovery	RC	24	1	2	1	3
Thiagara jan	Oculomotor rehabilitation	Stereo	RC	24	1	2	1	3
Photosensitivity								
Mansur (2018)[14]	Non-LCD screen vs LCD screen (Immediate)	SCAT-3 Symptom severity	RC	58	4	4	1	1
Mansur (2018)[14]	Non-LCD screen vs LCD screen (immediate)	SCAT-3 No. of symptoms	RC	58	4	4	1	1
Reading								
Thiagara jan (2014)[17]	Oculomotor rehabilitation vs sham (15 weeks)	Reading rate	RC	24	1	2	1	3
Thiagara jan (2014)[17]	Oculomotor rehabilitation vs sham (15 weeks)	Comp	RC	24	1	2	1	3
Thiagara jan (2014)[17]	Oculomotor rehabilitation vs sham (15 weeks)	GLE	RC	24	1	2	1	3

AVERAGE RISK OF BIAS	INCONSISTENCY	INDIRECTNESS	IMPRECISION	PUBLICATION BIAS	INTERVENTION MEAN	INTERVENTION SD	CONTROL MEAN	CONTROL SD	QUALITY (GRADE)
HEADACHE-MIGRAINE									
Headache Frequency									
2.25	NA	None	Very Serious	NA	26.0 0	5.13	25.10	6.51	Very Low
2.25	NA	None	None	NA	6.34	1.69	5.10	2.27	Mod
1	NA	None	None	NA	57.00	8.20	61.60	5.20	Mod[b]
1.5	NA	Serious[a]	Serious	NA	13.2	9.79	8.80	7.78	Low
1.5	NA	Serious[a]	Very Serious	NA	9.00	15.15	6.40	9.01	Very Low
2	NA	None	None	NA	57.1	8.99	61.77	6.89	Mod
OCULAR									
Accommodation									
1.75	NA	None	None	NA	8.80	1.73	6.90	2.08	Mod
1.75	NA	None	None	NA	11.0	6.93	5.00	5.20	Mod
Convergence									
1.75	NA	None	None	NA	28.00	3.00	37.00	4.00	Mod
1.75	NA	None	None	NA	9.20	3.46	15.60	7.97	Mod
1.75	NA	None	None	NA	11.9	4.50	17.9	8.66	Mod
1.75	NA	None	Serious	NA	22.90	3.81	26.20	5.20	Mod
Photosensitivity									
2.5	NA	None	None	NA	3.00	1.50	12.50	2.50	Mod
2.5	NA	None	None	NA	0.30	0.40	2.00	0.50	MOD
Reading									
1.75	NA	None	Very Serious	NA	177.00	68.59	142.00	48.99	Low
1.75	NA	None	Very Serious	NA	85.00	14.70	81.00	19.60	Low
1.75	NA	None	Very Serious	NA	6.30	5.88	4.10	3.43	Low

TABLE 2. CONT'D.

GRADE evidence profile table of measured outcomes in the context of the postconcussion symptom cluster subtypes.

AUTHOR (YEAR)	COMPARISON (FOLLOW-UP)	MEASURE	DESIGN	TOTAL SUBJECTS	SELECTION BIAS	PERFORMANCE BIAS	ATTRITION	DETECTION BIAS
VESTIBULAR								
Balance								
Rytter (2019)[13]	Multidisciplinary care vs usual care (22 weeks)	UQ	RCT	89	2	3	2	2
Kleffelgaard (2019)[18]	Vestibular rehabilitation vs usual care (8 weeks)	BESS	RCT	57	1	3	1	2
Vestibular symptoms								
Rytter (2019)[13]	Multidisciplinary care vs usual care (22 weeks)	UQ	RCT	89	2	3	2	2
Kleffelgaard (2019)[18]	Vestibular rehabilitation vs usual care (8 weeks)	VSS	RCT	63	1	3	1	2

CISS, Convergence Insufficiency Symptom Score; GLE, Grade Level Efficiency; HIT-6, Headache Impact Test-6; MPQ-SF, McGill Pain Questionnaire-Short Form; NPC, Near Point Convergence; SCAT-3, Sport Concussion Assessment Tool-3; UQ, Unvalidated Questionnaire; VSS, Vertigo Symptom Scale.

[a]Graded down for indirectness. Military sample with high incidence of blast injuries and PTSD.

[b]Graded up for large effect.

The CBT study that met inclusion criteria for this systematic review was by Kjeldgaard and associates.[10] They compared a group-based CBT intervention to being on a waitlist with the primary outcome of headache. Their trial was the largest with 70 subjects. The mean injury to group allocation time was 27 months. The range was not provided. Their intervention used a structured protocol for nine weekly group-based CBT sessions (Table 1).[10] Their results suggest their program reduced measures of headache intensity. However, there was no improvement in measures of headache frequency, anxiety, depression, or somatization.

There was one randomized, single-blind, sham-controlled crossover trial with 24 subjects that generated three papers comparing oculomotor rehabilitation to sham rehabilitation.[15-17] Injury to allocation time was 1−10 years, and age of subjects was limited to 23−33 years. The outcomes were measured 15 weeks after the start of the trial. Oculomotor rehabilitation was performed twice per week, for a total of 6 weeks. At a session, each oculomotor component (version, vergence, and accommodation) was trained for 15 minutes, with 5-minute rest periods between components.[15-17] Their findings suggested that oculomotor rehabilitation improved measures of amplitude of accommodation and accommodative facility for postconcussion subjects.[15] They also determined that oculomotor rehabilitation improved convergence insufficiency symptom score, near point convergence break, and near point convergence recovery. However, they found no improvement in measures of stereoacuity.[16] The final paper generated from this study examined reading metrics. The study showed that oculomotor rehabilitation did not improve reading rate, reading comprehension, or grade level efficiency.[17] This finding may be related to a cognitive impairment rather than an oculomotor issue.

One study of mTBI patients compared effects of reading from a non-LCD computer screen to reading from and LCD computer screen. The study used a randomized crossover design with outcomes measured before and after a 30-minute reading task on two consecutive days. There were 58 subjects and the injury to group allocation time was not specified.[14] The results showed that the number of postreading symptoms and the severity of symptoms were lower after reading from the non-LCD screen than from the LCD screen. This study can also be interpreted to demonstrate that reading from LCD flat screens appears to worsen postconcussion symptoms. This may be due to postconcussion patients being particularly sensitive to the characteristics of light emitted from LCD screens.

One RCT compared group-based vestibular rehabilitation to usual care.[10] There were 63 subjects in this study, but only 57 completed the balance assessment. The mean time from injury to group allocation was 3.5 months. The intervention group received a group-based vestibular rehabilitation intervention twice weekly for 8 weeks. The intervention consisted of guidance,

AVERAGE RISK OF BIAS	INCONSISTENCY	INDIRECTNESS	IMPRECISION	PUBLICATION BIAS	INTERVENTION MEAN	INTERVENTION SD	CONTROL MEAN	CONTROL SD	
VESTIBULAR									
Balance									
2.25	NA	None	Serious	NA	3.31	2.64	4.34	25.6	Low
1.75	NA	None	Serious	NA	19.10	10.60	23.0	9.10	Mod
Accommodation									
2.25	NA	None	None	NA	3.51	2.79	4.68	2.51	Mod
1.75	NA	None	Very Serious	NA	6.70	6.00	8.40	6.60	Low

individually tailored exercises, a home exercise program, and an exercise diary. The exercises were Brandt-Daroff exercises for benign paroxysmal positional vertigo, habituation exercises for motion sensitivity and central posttraumatic vertigo, gaze-stabilization exercises for symptoms exhibited during eye-head coordination and reduced vestibuloocular reflex, and exercises for reduced balance, focusing on improving sensory integration. The home exercise program included two to five individually modified exercises and general physical activity.[10] The control group did not receive any rehabilitation intervention in place of the group-based vestibular rehabilitation intervention. However, not to treat posttraumatic benign paroxysmal positional vertigo was deemed a conflict of research ethics because of the strong existing evidence on the effect of canalith repositioning procedures. Therefore, patients with a positive Dix-Hallpike or roll test were treated with Epley and BBQ roll maneuvers.[10] The results of this study suggested that vestibular rehabilitation did not improve measures of postconcussion anxiety, depression, balance, or vestibular symptoms relative to usual care. However, it is more accurate to state that vestibular rehabilitation provided to all VCS patients is not superior to simply performing canalith repositioning maneuvers for patients presenting with a positive Dix-Hallpike maneuver.

LIMITATIONS

Having a single reviewer and author was the most notable limitation of this systematic review. However, this limitation was mitigated by the author strictly adhering to predefined inclusion criteria and by using algorithmic application of the NICE methodology checklist for RCTs, the JBI critical appraisal checklist for case series, and the GRADE guidelines for rating the quality of evidence for systematic reviews.

Another limitation is the dearth of literature concerning the treatment of adults who sustained concussion unrelated to athletic activities. Generally, the studies meeting inclusion criteria were quite small. This led to relatively wide 95% CIs, which are reflected in the serious and very-serious concerns of imprecision for many of the included studies. An additional limitation lies in the heterogeneity of study designs, injury to group allocation times, outcome measures, and time of outcome measures. Consequently, the author was not able to calculate grand means for any of the studied interventions.

It was also disappointing that no studies examining the effectiveness of manual techniques, including osteopathic cranial manipulative medicine, met inclusion criteria for this review. However, there are case reports that provide anecdotal evidence of a possible therapeutic benefit of receiving osteopathic manipulative treatment, including osteopathic cranial manipulative medicine for adolescents[21,22] and adults[23,24] after sports-related concussions. There was also a retrospective chart review that suggested that osteopathic manipulative treatment was effective for reducing a substantial subset of sports-related postconcussion symptoms for young athletes, including those falling under the HCS, OCS, and VCS.[25]

CONCLUSIONS

To the author's knowledge, this is the most comprehensive review to date that considers the effectiveness of various treatments for HCS, OCS, and VCS symptoms in the working-aged population. Erenumab and psychologically centered MDC improved outcome measures falling under HCS. Oculomotor rehabilitation and avoiding LCD screens were shown to improve OCS outcome measures. Outcome measures within the realm of

VCS demonstrated improvement with psychologically centered MDC, and Epley and BBQ roll maneuvers for positive Dix-Hallpike assessments.

The results of this systematic review should be interpreted cautiously because of small sample sizes, serious risk of bias, imprecision, and indirectness of many of the reviewed studies. In the future, there is a need for high-quality RCTs with larger sample sizes to better demonstrate the effectiveness of the treatments for HCS, OCS, and VCS symptoms.

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REVIEW ARTICLE

LOW BACK PAIN IN ADOLESCENTS WITH AN OSTEOPATHIC COMPONENT

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ABSTRACT

Low back pain (LBP) is a common complaint in adolescents and has been increasingly reported in recent years. Affecting roughly 40% of adolescents, it leads to negative overall health, higher incidence of LBP in adulthood, and greater utilization of health care resources over one's lifetime. LBP in adolescents differs from adult populations due to variations in structural anatomy, which contribute to differing approaches in diagnosis and treatment of this condition. The differential diagnosis of LBP in this population is extremely broad and can be attributed to many underlying etiologic factors. Clinicians must conduct a thorough history and physical examination and consider the appropriate diagnostic testing to accurately diagnose adolescents early on in their conditions to provide the most effective treatment. Treatment for this condition ranges from rest and rehabilitation, to oral medications, OMT, bracing, and rarely, surgery. Physicians must also be able to recognize clear risk factors and symptoms for serious underlying pathology that can be causing LBP. This article will focus on diagnosis and treatment of the most common causes of LBP in adolescents

BACKGROUND

Low back pain (LBP) has become one of the most common chief complaints by patients but has been frequently underappreciated. Currently, more than 10% of all appointments made with primary care physicians are for complaints regarding back or neck pain, leading to roughly $86 billion in health care spending.[1–3] LBP in adolescents has been increasing in recent years, yet only 24% of adolescents who report LBP seek medical attention.[4] The prevalence of LBP generally rises with age, as an estimate of 1% of 7-year-olds experience LBP, while 6% of 10-year-olds, and 18% of 16-year-olds are found to have LBP.[5]

According to the World Health Organization, an adolescent is defined as those between the ages of 10–19 years old.4 LBP can have both short- and long-term implications for adolescents. Short-term effects can lead to restriction of daily activities, such as attending school and participating in sports.[6] In addition, studies have shown that adolescents with LBP are more likely to develop chronic LBP as adults, have impaired quality of life, and use a greater amount of health care resources throughout their lifetime.[7]

Many potential risk factors have been identified and can aid in early diagnosis and treatment of adolescent LBP. Nonmodifiable risk factors include sex, age, and ethnicity.[8] Conversely, modifiable risk factors include childhood obesity, psychosocial and socioeconomic factors, and sports specialization.[9–11] Additionally, some causes of LBP may correlate with participation in specific sports, as well as level of competition.[6,12–14]

The most common diagnosis among adolescents with LBP is muscular or nonspecific LBP. However, it is important for physicians to be able to detect other causes of LBP in adolescents.[13,15] Some of the more serious conditions are infection, masses (malignant and benign), spondylolysis, spondylolisthesis, lumbar disc herniation, degenerative disk disease, scoliosis, and ankylosing spondylitis.[13,15,16] Additionally, our institution recently began conducting a 3-year retrospective chart review regarding low back pain in adolescents seem to be suggesting similar gender breakdowns and prevalences of these conditions to the current literature (Tables 1 and 2). The pain and functional impairment these patients undergo can result from somatic dysfunction throughout the body, especially in the area of the lower back. An osteopathic structural examination assessing for TART findings (Tissue texture changes, Asymmetry, Restricted range of motion,

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Tenderness) may help physicians detect somatic dysfunction. This article will discuss these conditions to improve early detection and treatment and have provided a detailed summary of everything discussed at the end (Table 3).

RISK FACTORS

It is critical for physicians to know certain risk factors for LBP, as this will greatly aid them in their initial diagnostic interview with adolescents. During this initial encounter, the physician must note gender; past medical history; hours, type, and intensity of activity; and family history of LBP. Gender plays a large role, as females are more predisposed to have LBP than males.[11] As noted in Table 2, females are more likely to have pain that is discogenic in origin, and males are more likely to have spondylolysis when compared with females. There are many plausible explanations for this, from females starting puberty before males leading to girls reporting LBP earlier, to fat masses increasing at the end stage of pubertal development and replacing active muscle fibers, which can result in back problems.[17] Hours and intensity of activity are vital to note, as studies have shown that when both of these factors increase, adolescents are more likely to report LBP.[18] Additionally, adolescent athletes have a higher 1-year prevalence rate of LBP relative to nonathletes of the same age.[10]

Nonspecific or muscular LBP

As noted, one of the most common causes of LBP is acute or subacute muscle strain, or nonspecific LBP.[15] According to a previous study, 24% of adolescents who complained of LBP in an emergency department were experiencing muscle strain injury.[19] Table 2 shows that in our retrospective chart review, roughly 47.5% of all the adolescents who reported back pain had muscular or nonspecific LBP. Previous studies have suggested that specific sports that involve pushing and pulling heavy weights, such as football and weightlifting, can lead to a higher risk of acute muscle strains.[20] Conversely, other sports can cause chronic strains from repetitive overuse of the muscles, such as rowing or tennis.[20] Initial treatment involves a brief period of rest and oral analgesics, such as acetaminophen or nonsteroidal anti-inflammatory drugs (NSAIDs). Additional modalities, including manual therapies can also be considered. An osteopathic structural examination at the time of initial presentation can alert physicians to the presence of somatic dysfunction, especially involving the lumbar spine, pelvis, and sacrum. Osteopathic manipulation treatment (OMT) can then be incorporated to correct these somatic dysfunctions, resulting in decreased pain, decreased use of medication, and improved functional ability. If no improvement is noted in 2–4 weeks, radiographs of the lumbar spine and structured physiotherapy are reasonable considerations.

TABLE 1:

Shows slightly more females than males presented with a complaint of LBP in a 3-year retrospective chart review at our institution[70]

Male	922 (47.7%)
Female	1,010 (52.3%)
Total	**1,932**

TABLE 2:

Prevalence of specific diagnoses in adolescents presenting to our institution

[1]Includes disc herniation, disc degeneration, and radiculopathy caused by disc herniation.

[2]Includes coccydynia, sacroiliac pain, and facet cysts.[70]

	NONSPECIFIC OR MUSCULAR LBP	DISCOGENIC CAUSES OF LBP[1]	SPONDYLOLYSIS (WITH OR WITHOUT SPONDYLOLISTHESIS)	SCOLIOSIS	OTHER[2]	TOTAL
Male	423 (45.9%)	230 (24.9%)	192 (20.8%)	46 (5.0%)	31 (3.4%)	1,010 (52.3%)
Female	494	304 (30.1%)	89 (8.8%)	92 (8.8%)	31 (3.1%)	922 (47.7%)
Total	**917 (47.5%)**	**534 (27.6%)**	**281 (14.6%)**	**138 (7.1%)**	**62 (3.2%)**	**1,932**

TABLE 3:

Summary of common causes of LBP in adolescents including diagnosis and treatment options[70]

DIAGNOSIS	DEFINING CHARACTERISTICS	COMMON HISTORY AND EXAMINATION FINDINGS	DIAGNOSTIC TOOLS	TREATMENT
Nonspecific or muscular LBP	Acute reproducible muscle tenderness	Often seen in sports with pushing/pulling components	Osteopathic structural examination TART findings	Rest Oral analgesics Physical therapy OMT
Degenerative disc disease/ lumbar disc herniation	Protrusion or rupture of a disc Radiculopathy can also be present	Largely seen in obese patients and patients participating in high-impact sports	Straight leg raise test Advanced imaging to include MRI	Rest NSAIDs Extension-based physical therapy OMT for associated somatic dysfunction Epidural steroid injections (ESIs)
Spondylolysis/ spondylolisthesis	Stress fracture in the pars interarticularis of the vertebral arch Spondylolisthesis displacement or forward shift of one vertebra with respect to another	Most commonly seen in athletes with repetitive extension or twisting	Stork test Radiographs are often normal Advance imaging can include SPECT scan, MRI, and CT scans	Rest from activity Bracing Flexion-based therapy
Scoliosis	Lateral curvature of the spine Often minimal or no pain Can progress rapidly during adolescent growth spurt	Often seen in females and patients who have a strong family history of scoliosis	Adam forward bend test Radiographs to include anteroposterior and lateral standing views of the thoracic and lumbar spine and measurement of the Cobb angle	Monitoring curve progression Bracing Surgical intervention
Ankylosing spondylitis	Inflammatory arthropathy that affects the spine	More often seen in males than females. Patients will complain of night pain Pain improves with exercise and worsens with rest	Schober test Gaenslen test Posterior superior iliac spine (PSIS) distraction test	Promote exercise and activity to maintain spinal flexibility NSAIDs Rheumatologic evaluation

Degenerative disc disease/lumbar disc herniation

Both degenerative disc disease and lumbar disc herniation can cause LBP in adolescents.[21] This most commonly occurs at the L4-L5, and L5-S1 levels.[22] Some notable symptoms of the condition are radiating pain ("sciatica"), and pain worsening with flexion or the Valsalva maneuver.[22] According to our data, 27.6% of our adolescent population had discogenic causes of LBP.[23]

For early detection of degenerative disc disease or lumbar disc herniation, clinicians must monitor a patient's anthropometrics, as rapid changes in height or weight can predispose individuals to discogenic issues.[23] Obesity and participation in high-impact sports can play large roles in developing a herniation, as these place added stress on a patient's discs, causing injury.[24,25] A common physical examination done to test for the condition is a straight leg raise test, which has a high sensitivity and specificity rate.[26] Diagnostic imaging might also be necessary to confirm the condition, with magnetic resonance imaging (MRI) being a commonly used modality.[27,28]

The goal of treatment of adolescent lumbar disc herniation is to relieve symptoms and allow early return to routine life. The most common treatment plan for adolescents is a conservative approach with a mix of rest, physical therapy, and NSAIDs. If there is no improvement, then epidural steroid injections can be considered.[23] OMT to correct somatic dysfunction can be beneficial. Additionally, if conservative treatment fails, there is more aggressive surgical treatment that consists of surgical discectomy. Borgesen and Vang conducted a study that reviewed 158 adolescent patients who had all undergone surgery. According to the study, 93.7% of the patients reported good to excellent results after surgery. [29]

Spondylolysis/spondylolisthesis

Spondylolysis is a condition in which there is a bony defect within the pars interarticularis of the vertebral arch.[30] This can occur due to repetitive overuse, especially in extension; it can also be congenital. This injury most commonly occurs at the L5 level.[30] Spondylolisthesis is a displacement or forward shift of one vertebra with respect to another.[30] This typically occurs due to trauma and is categorized by different grade based on the percentage of slip of the superior body relative to the one inferior.[30] This typically occurs at the L5-S1 region of the vertebra.[30] Patients will complain of exertional LBP usually relieved by rest. Pain tends to worsen when patients extend at the lumbar spine. Adolescent athletes are at a higher risk for this condition than nonathletes.[31] Interestingly, according to our data, more adolescent males experience spondylosis than females. Of adolescents diagnosed with spondylolysis, roughly 70% were males and 30% females. While our data showed that this condition was most prevalent in adolescents who participated in gymnastics and weightlifting, it should be considered in any adolescent who engages in increased extension-based activities.

Studies have shown that there is no clear identifying physical examination maneuver that detects spondylosis; however, a positive Stork test is more often indicative of spondylolysis.[32] A positive Stork test must be paired with other distinctive indications of spondylosis to warrant further diagnostic imaging.[33] Common diagnostic imaging that can be used to detect this condition are anterior-posterior (AP) and lateral radiographs (72%−78% sensitivity), single-photon emission computed tomography (SPECT) scan (84% sensitivity), MRI (92% sensitivity), and computed tomography (CT) scans (90% sensitivity).[34]

For this condition, there are many treatment options that can be combined. Some of the options include rest from activity, bracing, and flexion-based therapy.[32,35] Rest from activity is generally recommended for anywhere from 4−12 weeks.[35] Bracing can also be incorporated for 4–12 weeks.[35] Braces used include soft lumbar corset brace and hard or soft thoracic lumbar sacral orthosis, with or without thigh extension.[35] Bracing is prescribed for many adults with LBP, but it is much more controversial for treatment of adolescents. A recent meta-analysis has found that most adolescents have a clinically successful outcome after undergoing conservative management, whether bracing was used or not.[36] Additionally, physical therapy is recommended with focus on flexion-based movement, which is prescribed for roughly 4–10 weeks.[35]

Scoliosis

Scoliosis is a condition in which the spine has a lateral curvature causing a structural alteration. Although some adolescents with the condition may not experience LBP, pain is found to be twice as common in patients who have scoliosis.[37] Strong risk factors for this condition include being a female and having a family history of scoliosis.[31] Roughly 30% of adolescents with idiopathic scoliosis also have a family history of the condition.[31]

Early diagnosis and proper management are crucial for physicians to properly treat adolescents with this condition. This is critical, as idiopathic scoliosis in adolescents is predictive of adult back pain.[38] It is common practice for physicians to perform the Adams forward bend test at yearly examinations to test for scoliosis.[38] Although the test is very accurate in confirming scoliosis, it can be skewed if the patient is overweight or obese. This can occur due to overlying soft tissue and increasing double major curve.[38] Additionally, an osteopathic structural exam can be performed to assess a patient's posture, balance, and range of motion, while also palpating for any asymmetry or tenderness.[39] In addition to the physical examination, a physician may also order additional diagnostic imaging to confirm scoliosis in a patient.

A common diagnostic test for scoliosis is AP and lateral standing radiograph of the thoracic and lumbar spine.[40] Radiographs allow for the severity of lateral spinal curvature to be assessed.[40] A Cobb angle, which is a critical measurement for diagnosing scoliosis, can also be determined by using radiography.[41,42] When measuring a Cobb angle, an angle of trunk rotation that is less than 5° is insignificant and does not require follow-up; while a measure of 5°−9° warrants reexamination in 6 months.[43] However, a measurement of 10° or greater requires further radiologic evaluation for more thorough Cobb angle measurement.[43,44] Spinal curve can change over time and must be evaluated periodically. Most notably, during an adolescent's growth spurt, spinal curvature can change dramatically.[45,46]

Treatment options for scoliosis range from monitoring, to bracing, to surgical correction. The goal of all of these treatments is to keep curves under 50° at maturity.[47] Typically, observation is recommended for skeletally immature patients with curves of less than 25°.[47] Bracing is recommended for adolescent patients with curves between 25° and 50°.[48] There are many bracing options. The Milwaukee brace, Boston brace, and Charleston bending brace are all used. Bracing does not correct the scoliotic curve but instead tries to prevent it from worsening.

Another option is surgical correction, which is considered for curvatures of greater than 45° in adolescent patients, and for curves greater than 50° in mature patients.[48] Surgical treatment is done to prevent progression and improve spinal alignment and balance. Strategies include fusion with and without instrumentation. Surgical approaches can be from the anterior, posterior, or both. Surgical treatment is dependent on curve type, age of the patient, and surgeon preference.[47]

Given these treatment options, health care providers should refer any adolescent with a curve greater than 10° to a spine specialist.[47] Primary care physicians' roles are to monitor and assess their patient's spinal curvature. However, once spinal curvature exceeds a significant degree, the primary care physician must refer the patient to a spinal specialist who can properly treat the curvature. Since scoliosis rarely progresses faster than 1° per month, referral within 3–6 months is appropriate.

Ankylosing spondylitis

Ankylosing spondylitis is an inflammatory arthropathy that affects the spine.[49] Two types of ankylosing spondylitis are juvenile spondylarthritis (patients 16 years and younger) and ankylosing spondylitis (patients 17 years and older).[49] Both types are more

common in males than females.[48] Patients will report night pain that resolves with exercise but does not resolve with rest.[50]

On physical examination, patients may have limited lumbar flexion, limited spinal side-bending, and limited chest expansion. Therefore, the posterior superior iliac spine distraction test and Gaenslen test have been used to measure this condition. Each test has a sensitivity rate of 100% and 90%, respectively, and specificity rate of 89% and less than 35%, respectively.[51] The Schober test is also a common physical exam performed on patients with ankylosing spondylitis, as it assesses the restrictions in lumbar range of motion.[52] Additionally, diagnostic testing can be performed to confirm the condition, most notably plain radiographs, but also SPECT and CT scans can be helpful.[53] Adolescents with juvenile spondylarthritis are at greater risk for developing degenerative hip disease including joint space narrowing, osteophytes, erosions, and protrusio acetabuli.[54] Therefore, it is essential for physicians to know early risk factors and diagnostic tests to detect this condition in its onset and prevent progressive damage as the adolescent ages.[54]

Treatment for ankylosing spondylitis aims at reducing symptoms and maintaining spinal flexibility, while maintaining life function. The mainstay of treatment has been NSAIDs and exercise. Slow-acting antirheumatic drugs can be used, at which point rheumatologic referral is reasonable.[55]

RED FLAGS

When diagnosing and treating LBP, it is imperative for the physician to be knowledgeable and aware of different "red flags" that can present. These "red flags" indicate the need for further diagnostics and potentially referrals to more specialized physicians. These factors include[56]:

- Morning stiffness
- Numbness
- Night pain
- Unexplained weight loss
- Motor weakness
- Fever or chills
- Loss of bowel or bladder control
- History of malignancy
- History of immunosuppression
- Prolonged use of steroids
- Neurologic compromise
- Pain that is increased or unrelieved by rest

If any of these factors are present in a patient, it warrants further evaluation by a spine specialist.

OSTEOPATHIC CONSIDERATIONS

In the modern health care climate, patients are often seeking additional or alternative means of treating their pain. As physicians, our goal is to provide safe and cost-effective care while simultaneously minimizing risk of undue harm. Particularly, with concerns over the rise in opioid prescribing, the need for safe and effective nonpharmacologic low back treatment is even more pressing. Adolescents who seek medical care for their reports of back pain receive an opioid prescription 20%–40% of the time.[57] The side effects and addiction potential of these medications are well documented. As physicians, we constantly weigh the risks and benefits of any intervention, while following best practices, current guidelines, and utilizing evidence-based medicine. There is an abundance of evidence demonstrating the utility and benefit of manual therapy in adults with back pain, with many of those conclusions being extrapolated and applied to treatment of back pain in adolescents. Studies have demonstrated not only improvements in pain, but also decreased use of pain medications.[57–59] There are studies demonstrating benefit and utility of OMT in the pediatric population for a variety of ailments, but a scarce amount of quality data exists regarding use of OMT for back pain in adolescents.[59,60]

Generally regarded as both safe and effective, OMT is a nonpharmacologic option that utilizes various manual techniques in an effort to correct somatic dysfunction and associated pain. Somatic dysfunction is defined as "impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial, and myofascial structures, and their related vascular, lymphatic, and neural elements."[58] Paramount to the ability to treat somatic dysfunction is the ability to make an accurate diagnosis, and to understand and incorporate the principles of osteopathic medicine: the body is one dynamic unit of function; the body is self-regulating and self-healing; structure and function are interrelated; a treatment regimen is designed and individualized to each patient based on the understanding and implementation of the first 3 principles.[60] Whereas other forms of spinal manipulation (as performed by chiropractors, physical therapists, massage therapists, and exercise therapists) focus primarily on axillary osteoarticular structures, OMT also addresses soft tissue structures surrounding spinal and appendicular articulations. With regard to back pain, this paradigm allows for careful assessment and treatment of the axial spine, sacrum, pelvis, extremities, rib cage, cranium, and viscera.

As individuals progress through adolescence, physical and skeletal maturity becomes less similar to that of a child, and closer to that of an adult. Additionally, movement patterns become more engrained, such that treatment may directly lead to use of new movement patterns after restoration of function versus having to unlearn certain movement patterns that have been adapted as a compensatory mechanism over time.[61] Also, as joint mechanics are influenced by maturation of primary and secondary ossification centers, care must be taken to ensure techniques are applied in a careful and gentle fashion to avoid potential harm, such as fracture, subluxation/dislocation, sprain/strain, or increased pain. Such techniques would include passive techniques with either direct or indirect force applied relative to the restrictive barrier (counterstrain, myofascial release).[60] As adolescents age, more active direct techniques, such as muscle energy, direct myofascial release, and high-velocity low-amplitude thrusts, can be incorporated as part of the treatment regimen.[58,59] Treating back pain before it becomes a more chronic issue can prevent activity avoidance, deconditioning, and poor core and lower-extremity endurance, while allowing adolescents to maintain prior levels of physical activity and sports participation.[62]

A study conducted by Selhorst and Selhorst in 2015 looked at the benefit of lumbar manipulation in adolescents with acute (<90 days) LBP, measuring efficacy, when added to a dedicated exercise program. The treatments were performed by five manual therapists with no specific lumbar segmental vertebral or somatic dysfunction diagnosis, and utilized high-velocity thrust maneuvers in a "shotgun" approach.[62] The study did not find any serious adverse events, however, the patients in this trial did not have significant improvement in pain with addition of spinal manipulation. Two recent systematic reviews of OMT and chiropractic spinal manipulation for a variety of pediatric health conditions confirmed the safety of this treatment, noting only mild exacerbation of symptoms.[62,63] Another study, conducted by Evans et al in 2018, looked at spinal manipulation in adolescents with chronic back pain, again coupled with targeted exercises versus exercise alone. A 12-week course of treatment was provided to the experimental group, with outcomes measured at 12, 26, and 52 weeks. The treatment group demonstrated improvement in pain levels at all points, with statistically significant improvement at 26 and 52 weeks.[57] Secondary outcomes demonstrated 80% reduction in medication use, decreased disability, improved quality of life, and higher patient satisfaction.[57] No patients in either trial reported any adverse effects, beyond slightly increased symptoms that ultimately abated without need for further evaluation or intervention.[57,62] Additional studies are needed to look at the benefit of OMT in adolescent patients complaining of back pain with diagnosed somatic dysfunction.

At this time, there is a paucity of high-quality randomized controlled trials regarding the utilization of OMT for back pain in the adolescent population. The limited number of studies on LBP and systematic reviews for treatment of other pediatric conditions show significant improvements in pain, decreased utilization of pain medication, and a high degree of safety with only short-lived symptom aggravation. When performed by a provider skilled in OMT, it stands to reason that this modality is a useful adjunct for treatment of back pain in the adolescent population.

TREATMENT

Treatments for individual causes of LBP can vary widely. To have an effective treatment plan, there must first be accurate and early diagnosis of the cause of LBP. The most effective treatment for nonspecific LBP has been a conservative approach, emphasizing rest from offending activities and physical therapy.[14–16,63]

Rehabilitation is a multifaceted process that focuses on preserving and promoting range of motion and strength.[63] Exercises such as hip flexibility, core stabilization, and others are prescribed to strengthen the abdominal muscles, lumbar multifidi, erector spinae, as well as other paraspinal, pelvic, and cervicothoracic musculature.[27] When prescribing rehabilitation, providers must be specific with their diagnosis, as rehabilitation protocols vary based on diagnosis. For example, spondylolysis is treated through flexion-based therapy, while conditions like disc herniations and radiculopathy are treated with extension-based therapy.[63,64]

If conservative management has failed, clinicians should consider consultation with a spine specialist. Although surgery is not an option with regard to nonspecific LBP, in rare cases it may be the only option for treatment. Some examples of patients who might require surgical treatment are adolescents with high-grade spondylolisthesis or disc herniations with persistent radicular or neurologic symptoms.[65] Typically, adolescents respond much better to spine surgery relative to adults.[66]

PREVENTION

Adolescents will always be more vulnerable to trauma, as they are skeletally immature individuals, especially during periods of rapid growth.[67] Therefore, the best method to prevent LBP is for health care providers to properly educate their patients on the vulnerability of their backs, and the need for good overall health.[53] Studies have shown that strengthening of an adolescent's quadriceps, hamstrings, and core; increasing lumbar flexibility; and weight loss are all associated with reducing one's risk for developing LBP.[27] Additionally, patients should participate in regular physical activity and maintain a body mass index (BMI) below 30 kg/m^2.[24,68,69] If adolescents play in competitive sports, studies have shown that those who participate in preseason sports conditioning programs and neuromuscular training have reduced injury rates in their upcoming season.[67] Finally, adolescents should be aggressive in seeking treatment for LBP and recognize that they may need to see their primary care physician if their symptoms persist for longer than 2–3 weeks.[14]

CONCLUSION

LBP in adolescents is common and can be caused by a range of different musculoskeletal conditions. The most common causes of LBP in adolescents were discussed. A careful history, physical examination, and osteopathic structural exam can help the provider make a specific diagnosis. An appropriate treatment plan can then be instituted in an attempt to prevent acute back pain from persisting into adulthood. Therefore, clinicians must be vigilant in identifying key risk factors for certain causes of LBP in adolescents.

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REVIEW ARTICLE

CLINICAL MANAGEMENT OF POLYPHARMACY IN THE ELDERLY POPULATION

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KEYWORDS

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ABSTRACT

Polypharmacy is defined as use of multiple medications (>5) and is common in the elderly adult population. Polypharmacy typically results from the accumulation of treatments for chronic medical conditions such as hypertension, diabetes, coronary artery disease, and psychiatric illnesses. It is associated with problems such as increased risk of falls and adverse medication events. Elderly patients take an average of two to nine medicines per day, and prevalence of polypharmacy in the elderly is 11.5%–62.5%. Elderly patients are at higher risk of adverse drug reactions due to metabolic changes and reduced drug clearance. Evaluation of polypharmacy is an important part of clinical assessment of the elderly population. This process involves performing an adequate medication reconciliation, including supplements, followed by systematic evaluation of medications looking for benefits and harms. It then involves discussing goals of care with the patient and, if necessary, creating a deprescribing plan. When prescribing new medications, prescribers should consider starting at the lower end of the dosing range and increasing only after monitoring for benefits and harms.

INTRODUCTION

There is an epidemiologic shift in the leading cause of death from infectious disease and acute illness to chronic degenerative diseases. These improvements in medical therapies have led to an elderly population with ever-increasing comorbidities. As patients age, alterations in physiologic processes lead to increased risk of medication adverse effects. It is estimated that elderly patients take an average of two to nine medicines per day, and prevalence of polypharmacy in the elderly is 11.5%–62.5%. With aging there is reduced body water and lean body mass with associated increase in fat mass leading to pharmacokinetic changes of reduced first pass metabolism, reduced renal clearance, and increased volume of distribution. Evaluation of polypharmacy defined as use of >5 medications, is an important part of clinical assessment of this population. The purpose of this review article is to address polypharmacy methods of individualization of care and deprescribing to improve care and reduce risk of medication-induced adverse events in the elderly population in the most common clinical scenarios in an outpatient setting.[1,2]

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In addition to prescription medications, elderly patients often use over-the-counter (OTC) medications such as acetaminophen, nonsteroidal anti-inflammatory drugs (NSAIDs), proton pump inhibitors (PPIs), antihistamines, and supplements. These patients are often not aware of the potential drug interactions with OTC and herbal medications and may not discuss their use. Thorough review and documentation of OTC and other herbal agents in medical record is important to optimize medication management in the elderly population.[1]

CLINICAL APPROACH

Appropriate prescribing in the elderly ensures that, on balance, the medication regimen is of benefit to the patient. When new medications are expected to aid the patient, their addition should be weighed against the risks of polypharmacy. When starting new medications, the phrase “start low, go slow” is commonly applied to the geriatric population. This phrase prompts prescribers to start new medications at the lower end of an effective range, then monitor closely for benefit or harm prior to making any dose adjustments.[2]

While many medications are started to improve symptoms or control the progression of the disease, not all medications should be used lifelong. When patients accumulate medications that are no longer beneficial or possibly even harmful, they should be deprescribed. Deprescribing is “the planned and supervised process of dose reduction or stopping of medication that may be causing harm, or no longer be of benefit.”[3] The following steps provide a clinical approach to deprescribing: (Figure 1)

1. Perform an adequate medication reconciliation, including supplements. Periodically, all the patient's medications should be reviewed in detail to create a complete and accurate list of all their prescription medications, supplements, and OTC medications. This process is known as medication reconciliation, and it forms the cornerstone of appropriate prescribing in the elderly. Optimal times to perform medication reconciliation include at annual visits, at preoperative visits, and any time there is a change in the level of care (eg, transitioning to a new primary care provider, being admitted to a hospital, or moving from home to an assisted living facility).[4]

   A complete medication reconciliation provides information regarding the drug, dose, and frequency of use. This information needs to be reviewed by the provider, but it can be obtained by allied professionals such as medical assistants, nurses, or pharmacists.

2. Systematically evaluate the medications to look for benefits and harms. For each medication, consider how the medication may be helping or harming the patient. It is important to consider not only physical or psychological harms, but also financial or social harms. Many medications are expensive or require caregivers to administer them.[5,6]

   Several methods have been developed to determine appropriateness of medications that are prescribed to the elderly population. Methods like the Medication Appropriateness Index and Prescribing Optimization Method involve questions to evaluate appropriateness of each medication. These methods are patient-tailored and allow for flexibility in assessment and individualization of the pharmacotherapy to optimize medical therapy and evaluate for appropriateness of dose, frequency, and treatment duration. These two methods can be patient-centered, though time-consuming.[1]

   Other methods like BEERS Criteria and START/STOPP screening tool are more rigid and are derived from literature review and expert consensus. BEERS criteria lists potentially inappropriate medications by drug class and disease state. [1,7] STOPP and START tools are used together to recognize medications that may be inappropriate and identify alternatives that can safely treat the condition. [1,8] However, BEERS Criteria and STOPP/START tools do not consider individual preferences, or the degree to which the patient has benefited from the medication.[1]

3. Discuss goals of care with the patient

Ask patients about their treatment goals. The benefits and harms of medications can be compared with the patient's goals of care. If a medication is being discontinued or its dose reduced, explain to the patient why this is required.

4. Create a deprescribing plan

   Once a decision has been made regarding which medications to change, consider possible consequences of the change. Some medications may create adverse drug withdrawal events, especially cardiovascular or central nervous system medications. If a medication is being used to control symptoms, a plan should be made to monitor the symptoms and ensure they continue to be adequately controlled. Discuss nonpharmacologic options with the patient.

   At the end of the visit, review the changes with the patient. They should receive a written copy of the deprescribing plan, as well as an updated medication list. Pharmacies may continue to dispense medications if they are not informed when medications are discontinued, or the dosage is changed. This can be mitigated by ensuring that pharmacies are made aware when changes occur. Many electronic health records automatically send updated information to pharmacies, making this step simple. If needed, it can be helpful to have a staff member contact the pharmacy to inform them of any changes.[1]

SPECIFIC CONSIDERATIONS IN COMMON DIAGNOSES

Hypertension

Hypertension is the leading cause of mortality worldwide contributing to up to 30% of all myocardial infarctions (Table 1).[9,10] Several recent trials have shown benefits of management of hypertension with regard to cardiac risk in the elderly population. Management of blood pressure with goal blood pressure of 130−150/70−90 mm Hg with a non−beta blocker medication such as calcium channel blockers, thiazide diuretics, or ACE/ARB [angiotensin-converting enzyme/angiotensin receptor blocker] inhibitors has been shown to reduce cardiovascular risk and improve cerebral blood flow and carotid distensibility in the elderly population without increasing the risk for orthostatic hypotension.[11-13]

TABLE 1:

Indications for treatment of hypertension and available nonpharmacologic treatments.[10]

INTERNATIONAL SOCIETY FOR HYPERTENSION CRITERIA FOR DIAGNOSIS OF HYPERTENSION
Consistent BP >140/90 mm Hg in health care setting
Consistent BP >135/85 mm Hg in home setting
Consistent BP >130/80 mm Hg on 24-hour ambulatory monitor
NONPHARMACOLOGIC MANAGEMENT OF HYPERTENSION
Reduction of salt intake
Smoking cessation
Increasing physical activity
Weight loss
Pharmacotherapy if BP >140/90 mm Hg or BP >130/80 mm Hg if individual cardiac risk is >10% with thiazide, ACE inhibitor/ ARB, or calcium channel blocker to goal BP of 130-150/70-90 mm Hg. Beta blockers are second line due to risk of orthostatic hypotension.

ACE, angiotensin-converting enzyme; ARB, angiotensin receptor blocker; BP, blood pressure

Hyperlipidemia and Vascular Disease

Statin medications are firstline therapy for management of hyperlipidemia and are frequently given to elderly patients.[14] Furthermore, statin therapy among elderly 75 years and older without atherosclerotic cardiovascular disease (ASCVD) has been associated with reduced risk of all-cause and cardiovascular disease (CVD)−related mortality.[16] Risks of statin-induced myopathy and decline in physical function remain low. Furthermore, a recent study suggests that statins may preserve function by reducing risk of vascular events and improving vascular health.[16–18] When managing cardiovascular risk in older adults it is important to incorporate concept of frailty to individualize their treatment plan as one size does not fit all. Statins should be deprescribed in setting of frailty, low functional capacity, and reduced life expectancy of less than 10 years (Table 2).[15–18] Frailty can be measured anywhere using gait speed with frailty cut off 4 m in less than 5 s.[15]

TABLE 2:

Guidelines on use of statins in the elderly population.[18]

AHA/ACC GUIDELINES ON USE OF STATINS IN THE ELDERLY POPULATION
Reasonable to initiate or continue current regimen of moderate-intensity or high-intensity statin therapy in patients 75 years and older after consideration and evaluation of: • ASCVD risk reduction • Adverse effect of medication • Medication interaction • Frailty
In nonfrail elderly patients who are on high-intensity statin therapy or maximal tolerable statin therapy with high risk for ASCVD and LDL >70 mg/dL, it would be reasonable to add ezetimibe to lower ASCVD risk.
Stop statin therapy with functional decline, increased frailty, and reduced life expectancy, as benefits of statins in this setting are limited.

ACC, American College of Cardiology; AHA, American Heart Association; ASCVD, atherosclerotic cardiovascular disease; LDL, low-density lipoprotein

Gastroesophageal Reflux Disease

Gastroesophageal reflux disease (GERD) is common in elderly patients. However, its presentation may be different from the younger population. GERD symptoms in elderly include dysphagia, vomiting, and respiratory symptoms. Additionally, severity of GERD symptoms and esophageal inflammation increases with age.[19] Pharmacologic management of GERD includes PPIs. Prolonged use of PPIs can have some risks such as small intestine bacterial overgrowth, increased risk of Clostridium difficile infection, nutrient deficiencies (vitamin B12, calcium, magnesium, iron), increased risk of pneumonia, and development of chronic kidney disease. When PPIs are appropriately prescribed their benefits potentially outweigh their risks. PPIs −should be prescribed at lowest possible dose. In the setting of uncomplicated GERD, they can be stopped after 2 months or switched to an H2 blocker.[20]

Nonpharmacologic options in management of osteoporosis include participation in a multicomponent exercise program involving balance and resistance training under supervision of a physical therapist.[21] Current pharmacologic treatments for osteoporosis include bisphosphonates, denosumab, parathyroid hormone, abaloparatide, and romosozumab.[22] National Osteoporosis Foundation guidelines recommend use of bisphosphonates as firstline therapy for management of osteoporosis with risk assessment of an individual patient after an initial period of 3–5 years of treatment. All medications reduce nonvertebral fractures except for ibandronate. Zoledronic acid, risedronate, and alendronate reduce risk of hip fractures and vertebral fractures.[22,23] Denosumab can be a safe and effective option for long-term use. Denosumab should not be stopped without continuing another antiosteoporotic medication due to increased risk in bone loss and fracture risk. Long-term use (>10 years) of bisphosphonates can increase risk of atypical femoral fractures and drug holiday should be considered after 3−5 years in most patients (Table 3).[23,24,26,27] Overall evidence on benefits of vitamin D screening and supplementation is controversial. United States Preventive Services Taskforce (USPSTF) recommends against use of vitamin D supplementation for fall risk and osteoporosis risk reduction in noninstitutionalized elderly patients.[26]

TABLE 3 :

Indications for treatment of osteoporosis and associated risk factors for atypical fractures with treatment.[24,26]

AHA/ACC GUIDELINES ON USE OF STATINS IN THE ELDERLY POPULATION
Long duration of treatment with bisphosphonate therapy of >5 years
Younger age
Asian race
Low vitamin D levels
Use of multiple antiresorptive drugs
Glucocorticoids
Diabetes
Rheumatoid arthritis
Indications for Pharmacologic Treatment of Osteoporosis
Low bone mass or osteopenia and history of fragility fracture at hip or spine
T score of less than -2.5
T score of -1 to -2.5 with FRAX score >20% for major osteoporotic fracture or >3% for hip fracture

FRAX, fracture risk assessment tool

FIGURE 1:

Clinical approach to deprescribing.

1. Perform an adequate medication reconciliation, including supplements
2. Systematically evaluate medications looking for benefits and harms
3. Discuss goals of care with the patient
4. Create a deprescribing plan

Osteoporosis

Nonpharmacologic options in management of osteoporosis include participation in a multicomponent exercise program involving balance and resistance training under supervision of a physical therapist.[21] Current pharmacologic treatments for osteoporosis include bisphosphonates, denosumab, parathyroid hormone, abaloparatide, and romosozumab.[22] National Osteoporosis Foundation guidelines recommend use of bisphosphonates as firstline therapy for management of osteoporosis with risk assessment of an individual patient after an initial period of 3–5 years of treatment. All medications reduce nonvertebral fractures except for ibandronate. Zoledronic acid, risedronate, and alendronate reduce risk of hip fractures and vertebral fractures.[22,23] Denosumab can be a safe and effective option for long-term use. Denosumab should not be stopped without continuing another antiosteoporotic medication due to increased risk in bone loss and fracture risk. Long-term use (>10 years) of bisphosphonates can increase risk of atypical femoral fractures and drug holiday should be considered after 3−5 years in most patients (Table 3).[23,24,26,27] Overall evidence on benefits of vitamin D screening and supplementation is controversial. United States Preventive Services Taskforce (USPSTF) recommends against use of vitamin D supplementation for fall risk and osteoporosis risk reduction in noninstitutionalized elderly patients.[26]

Thyroid disease

Hyperthyroidism can be treated with either I-131 iodine, thyroidectomy, or antithyroid medications, which are safe and equally efficacious. The antithyroid medications available are propylthiouracil (PTU) and methimazole. Methimazole is recommended over PTU as the antithyroid medication of choice for management of overt hyperthyroidism due to increased risk of fatal liver injury associated with PTU.[27] Subclinical hyperthyroidism generally is not treated as very few patients are symptomatic or develop hyperthyroidism and benefits of treatment remain controversial.[27,28]

Hypothyroidism can be managed by levothyroxine, which generally has a long half-life, is well tolerated, and is well absorbed. There is no evidence that treatment of subclinical hypothyroidism improves symptoms and reduces mortality and morbidity. Additionally, elevated thyroid-stimulating hormone (TSH) in the elderly is associated with increased longevity.

Anxiety

Nonpharmacologic therapies for treatment of anxiety, such as cognitive behavioral therapy (CBT), should be optimized prior to consideration of pharmacologic management of anxiety. Appropriate medication management for anxiety in the elderly includes selective serotonin reuptake inhibitors (SSRIs), serotonin–norepinephrine reuptake inhibitors (SNRIs), or buspirone.[32] Venlafaxine is effective but can raise blood pressure and is often discontinued secondary to side effects.[33] SNRIs and benzodiazepines should be avoided, particularly those that are short or intermediate acting, due to increased risk of cognitive impairment, delirium, falls, fractures, and car accidents.[34,38] Escitalopram may be more effective than citalopram in cases of panic disorder.[35] Evidence for use of mirtazapine for anxiety is limited.[36] Pregabalin has been shown to be safe and well tolerated for anxiety.[37]

Depression

Behavioral intervention and/or psychotherapy, with CBT being the most studied, should be considered in place of or in addition to pharmacotherapy. SSRIs are firstline pharmacotherapy, particularly citalopram, escitalopram, and sertraline. Bupropion and trazodone are also reasonable options.[38–41] Mirtazapine can be helpful in adults with depression and comorbid appetite and sleep disturbance. Those at risk of hyponatremia may tolerate bupropion over SSRIs, SNRIs, or mirtazapine.[38,42] Newer antidepressants such as vilazodone, vortioxetine, and levomilnacipran have limited evidence for efficacy and safety in older adults. For refractory depression, other considerations include addition of low-dose atypical antipsychotics such as aripiprazole or quetiapine, or electric convulsive therapy (ECT).[39] Tricyclic antidepressants should be avoided due to anticholinergic properties. Monoamine oxidase inhibitors pose a risk of postural hypotension and sleep disturbance and also should be avoided. Those who are taken off antidepressants should slowly be tapered and monitored for signs of relapse.[43]

Insomnia

CBT for insomnia and optimal sleep hygiene is firstline therapy for adults with chronic insomnia. There is insufficient evidence for effectiveness of melatonin, though many patients find this to be helpful and with a low side-effect profile. Ramelteon, a melatonin receptor agonist, reduces sleep onset latency with low-quality evidence but has relative lack of negative side effects.[44] Doxepin could be considered at low doses.45 Benzodiazepines should not be used for insomnia as they only have modest short-term benefit and multiple risks including increased risk of hip fractures.[46,7] As of 2019, Beers criteria added that sedative-hypnotics should be avoided in the elderly population regardless of duration as they increase risk of delirium, falls, fractures, and motor-vehicle accidents.[7,46,47]

Cognitive Decline

Treatment of mild cognitive impairment in older adults starts with early recognition, followed by implementation of aerobic activity, mental activity, and optimization of risk factors for CVD and stroke.[48] Additionally, polypharmacy should be considered in the differential for mild cognitive impairment. There is no effective medication for mild cognitive impairment and use of cholinesterase inhibitors and memantine is not recommended at this stage.[49] Pharmacologic recommendations for treatment of various types of dementia are beyond the scope of this article.

Delirium

Nonpharmacologic strategies to prevent and treat delirium in the elderly population are first line. Pharmacologic management of delirium should only be considered when the safety of the patient or those around them is at risk or to perform necessary medical interventions. Antipsychotics, such as haloperidol, and atypical antipsychotics are effective but pose a risk of extrapyramidal side

effects, QTc prolongation, and increased mortality in those with dementia. Benzodiazepines worsen the duration and severity of delirium and should not be used.[50]

Urinary Incontinence

To treat urinary incontinence, a careful review of offending medications or lifestyle factors should be done. Behavioral interventions such as timed voiding and pelvic-floor exercises should be maximized. Use of medications specifically for urinary incontinence should be used cautiously. Urinary antimuscarinics such as oxybutynin, tolterodine, and trospium pose a risk of anticholinergic side effects such as constipation, dry eye, and blurred vision.[51] Trospium may be a better-tolerated option due to less impact on the central nervous system. Long-acting formulas have better side-effect profiles than their immediate-release counterparts and are equally effective.[52] Beta-3 agonists such as mirabegron are associated with less adverse events but should not be used in those with uncontrolled or severe hypertension.[53] Procedural intervention may be considered but is beyond the scope of this article.

Constipation

When possible, eliminate or replace medications that cause constipation (eg, anticholinergics, opioids, calcium channel blockers, NSAIDs). Address contributing lifestyle factors such as poor caloric or fluid intake, low-fiber diet, and physical inactivity. Toilet training to maximize the gastro-colic reflex and minimize straining may also be helpful. Osmotic laxatives, particularly polyethylene glycol, are effective.[54] Stimulant laxatives (other than bisacodyl and sodium picosulfate) and stool softeners have a lack of supportive evidence and should not be used for chronic constipation routinely.[55,56] Lubiprostone or linaclotide can be considered as next-line agents if less-expensive treatments are not helpful. Bulking agents can be used if the patient does not have slow-transit constipation. Fecal impaction is best treated with mineral-oil enema, warm-water enema, or glycerin suppository. Note that long-term use of magnesium-based laxatives and phosphate enemas should be avoided due to potential for electrolyte disturbance.[57]

Antibiotics

Antibiotics should be selected choosing the narrowest spectrum and with the shortest treatment course possible. Fluoroquinolones should be avoided in the elderly when possible due to risk of tendon injury.[58] Nitrofurantoin is acceptable to use in those with creatinine clearance >30 for the short term, noting the uncommon but serious increased risk of pulmonary and hepatotoxicity.[7,59] Trimethoprim-sulfamethoxazole should be used with caution in combination with ACE/ARB in those with decreased renal function due to risk of hyperkalemia.[7] Long-term care facilities are common sites of development of multidrug resistant organisms such as methicillin-resistant staphylococcus aureus or vancomycin-resistant enterococci. Older patients are at particularly high risk of morbidity and mortality associated with antibiotic-induced diarrhea or Clostridium difficile colitis.[60]

CONCLUSION

As the population continues to age, addressing polypharmacy in elderly patients will become even more important. A thorough medication review should be performed for each patient. Utilizing evidence-based, risk-vs-benefit assessments, and goals-of-care conversations provides a practical yet individualized approach to reducing polypharmacy in older adults for better clinical outcomes.

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BRIEF REPORT

THE BIOMECHANICAL LINKS BETWEEN PELVIC FLOOR DYSFUNCTION AND TESTICULAR PAIN: A CLINICAL REVIEW

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KEYWORDS

chronic scrotal content pain

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OMT

pelvic floor manual therapy

male pelvic floor

ABSTRACT

Chronic scrotal content pain affects 100,000 men in the United States annually. Up to 50% of these cases do not resolve by following conventional treatment algorithms and are deemed to be idiopathic. There is little peer-reviewed literature supporting the specific cause and effect relationship between pelvic floor dysfunction and chronic scrotal content pain. Additionally, the specificity of the physical exam in these types of patients is not present in the literature. Overall, the literature is deficient in proposed treatment algorithms that address the large number of cases that are deemed to be idiopathic. Patients presenting with chronic scrotal content pain may benefit from an osteopathic diagnostic and treatment approach. In these types of patients, we recommend osteopathic manipulative therapy (OMT) or pelvic floor manual therapy prior to surgical intervention. This conservative approach may reduce the large portion of cases that are deemed to be idiopathic. The emphasis on structure and function within osteopathic medical education places osteopathic family physicians in a unique position to be able to properly diagnose and treat this type of pain. Since most cases of chronic scrotal content pain are initially addressed in the primary care setting, it is important for osteopathic primary care physicians to remain vigilant in considering musculoskeletal dysfunction when evaluating these types of patients. This clinical review is underscored by a unique case presentation of a male collegiate athlete who helps demonstrate the larger gap that is present in the literature on male pelvic floor and scrotal content pain.

INTRODUCTION

Research on male pelvic floor dysfunction is sparse when compared to that of women. Up to 5% of males presenting with symptoms associated with pelvic floor dysfunction also report chronic scrotal content pain (CSCP).[1] CSCP affects about 100,000 men per year, with up to 50% of cases presenting with an idiopathic etiology.[2-4] Unfortunately, there is very little peer reviewed literature showing a direct cause and effect relationship between pelvic floor dysfunction and CSCP.[1,5] Furthermore, there are few diagnostic algorithms proposed in the testicular pain literature; none have been validated and most exclude the pelvic floor and biomechanical dysfunction altogether.[3,6-9] It is common for patients experiencing CSCP to also present with varying degrees of hypertonic pelvic floor musculature, but it is also often unclear which issue came first.[10] As a result of this, it is too often assumed to be a symptom of pain rather than a cause of pain.

CASE REPORT

A 21-year-old Caucasian male patient, who was a Division I collegiate track runner, presented with a 12-month history of severe testicular pain. The onset of his pain was sudden following a morning distance run at practice. He presented to numerous urologists and pain specialists prior to treatment at Mayo Clinic Arizona. He received multiple magnetic resonance imaging (MRI) and computed tomography (CT) scans, which were unremarkable. Sexually transmitted infection (STI) and urine testing was negative. Complete blood count (CBC) and comprehensive metabolic panel (CMP) were also unremarkable. Throughout his 12-month history, six diagnostic scrotal ultrasounds were performed showing evidence of small bilateral hydroceles, bilateral varicoceles (grade III), left scrotal wall thickening (3–4 mm), bilateral epididymal cysts, and minor epididymal head calcification. An abdominal ultrasound was performed showing no urinary abnormalities or obstructions. He had no traumatic or surgical history to the groin

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or scrotal contents. The patient had not experienced pain during ejaculation but thought he did notice postejaculatory testicular pain. A semen analysis was performed showing normal sperm motility. Other significant medical history included severe anxiety (GAD-7 score of 18) and moderately severe depression (PHQ-9 score of 15).

The patient was prescribed a 3-week course of doxycycline, followed by an additional 2-month course of ciprofloxacin, with no pain resolution. Several rounds of amitriptyline (100 mg qd), then nortriptyline (10 mg qd), in conjunction with hydrocodone/acetaminophen (10/325 mg prn), failed to terminate the pain. Additionally, the patient took gabapentin (300 mg tid) for 3 months with no resolution of symptoms. A bilateral genitofemoral/spermatic cord nerve block was performed, using 8 mg of dexamethasone and 0.25% Marcaine, without complication. The patient presented with worse scrotal content pain 2 weeks after the procedure. He was eventually referred to pelvic floor physical therapy (PFPT) only after his case was discussed at a Mayo Clinic national conference. In the initial physical therapy assessment, the patient presented with point tenderness and hypertonicity of the right levator on digital rectal exam. He had an anterior pelvic tilt and decreased sacroiliac joint mobility. His initial pain level was 9/10. The patient was treated with PFPT for 1 hour, two to three times per week. Treatments included soft tissue release of the pelvic floor musculature, neuromuscular retraining, and sacral manipulation. Within 2 weeks of therapy, the patient subjectively felt improvement. At 6 weeks, the patient's pain level reduced to 2/10. At 8 weeks, the patient was able to return to running without pain.

DISCUSSION AND REVIEW OF THE LITERATURE

It is possible that the patient's pain was deemed to be idiopathic due to minimal research on the intersection between CSCP and pelvic floor dysfunction. Most adolescent and young men who present with CSCP have either an STI or have experienced trauma to the scrotal contents.[11] It is unusual for someone so young to be experiencing idiopathic CSCP.[9] Treating idiopathic CSCP has been a therapeutic dilemma because the published data regarding the diagnosis and treatment of reliable nonsurgical interventions are predominately derived from small studies and expert opinion.

The recommendation for men who present with idiopathic CSCP is to undergo surgical intervention when there are abnormalities within the scrotum.[6,12,13] The patient in the case above did not undergo surgery even though there were multiple scrotal abnormalities present. These decisions are inconsistent with what is recommended in the literature for a patient exhibiting such abnormalities.[2,3,6,12,13] The normal progression of treatment for a CSCP patient presenting with scrotal abnormalities, particularly varicoceles, is to undergo a varicocelectomy.[6] Our patient presented with grade III varicoceles that were tender on palpation, but he had not undergone varicocelectomy due to his young age. There are no specific examples in the literature that support treatment modalities in men of his age who do not respond to conventional methods. It is our belief that this is the reason his pain had been deemed to be idiopathic in nature.

Upon examination by the physical therapist, it was discovered that this patient had an extensive history of lower-extremity biomechanical injuries. This finding was significant because it provided reason to believe that his pain could be a result of musculoskeletal dysfunction and that it may be simply unique in its presentation. Moreover, this finding helped to validate the use of PFPT to treat his pain. Within the literature, there are few diagnostic/treatment algorithms that include PFPT. An algorithm proposed in Tatem and Kovac suggests PFPT as an optional method if pain persists.[6] In this algorithm, however, PFPT is not a requirement prior to surgery. Given the conservative and relatively inexpensive nature of PFPT, this recommendation should be a first-line treatment option, well before surgery. Another algorithm by Tan and Levine more adequately recommends PFPT as a required treatment step prior to surgery, serving as the only algorithm of its kind to consider pelvic floor dysfunction in this way.[2] Within both algorithms, however, there is no discussion on the specificity of the physical exam. It would be prudent to include a detailed structural examination of patients experiencing CSCP considering the number of cases deemed to be idiopathic.

Addressing biomechanics as a source of pain is not typically within the scope of urology, nor is it emphasized within the education of allopathically trained physicians. The relationship between structure and function is a crucial aspect to consider in CSCP patients. The contents of the pelvic floor are complex, and it is well known that musculoskeletal dysfunction can cause referral pain. While pelvic floor hypertonicity and tenderness have often been associated with CSCP, it is difficult to discern whether a dysfunctional pelvic floor is a cause of pain or a symptom of pain. This is particularly true when additional abnormalities are present, as was the case with our patient.

Review of the pelvic floor literature suggests that there is little research on men in general, as most pelvic floor complications are associated with women.[14,15] In the available literature specifically related to men, few case reports and peer-reviewed studies address the direct cause and effect relationship between pelvic floor dysfunction and CSCP.[1,5] A survey of 41 men with chronic idiopathic testicular pain showed that 93% reported a minimum of one symptom of pelvic floor dysfunction according to the Pelvic Floor Inventories Leiden (PelFIs) questionnaire. The PelFIs questionnaire is a 76-item instrument that measures the degree of pelvic floor dysfunction in men within nine different domains.[16] Within the same group, 88% had evidence of a hypertonic pelvic floor on electromyographic testing (6.7 muV, normal < 3 muV).[5] Consistent with this study, our patient presented with a hypertonic pelvic floor and CSCP with no other obvious-causing pathology present. However, the physical activity of the participants in this study was not reported. Our patient's level of physical activity could have been a significant factor in the development of his pain. Had he not been a competitive long-distance runner, his pelvic dysfunction may have never become severe enough to cause him testicular pain. Therefore, it may be worth considering the physical activity levels of CSCP patients.

A study by Farrell et al demonstrated that 50% of patients with CSCP and hypertonicity in the pelvic floor noted improvement in their symptoms after 12 sessions of PFPT.[17] The progression of our patient is consistent with this literature. However, this study

did not reach statistical significance and the sample size (30) was small, demonstrating a further need for more conclusive research on this topic.

Using current treatment algorithms, our patient likely would have undergone surgery. Instead, after completing 8 weeks of PFPT with hourly sessions up to three times per week, the patient was found to have a complete resolution of pain. It is important to note that the patient was taught how to contract and relax his pelvic floor so that he may continue to complete home exercises as a management technique for future symptoms as needed.

CONCLUSION

Patients presenting with CSCP may benefit from a more holistic osteopathic approach to diagnosis and treatment. This is especially true when considering the prevalence of CSCP that is deemed to be idiopathic in nature.[2,3] There is little peer-reviewed literature supporting the specific cause and effect relationship between the male pelvic floor and CSCP.[1,5] Additionally, biomechanics are often overlooked as a plausible source of CSCP since they are not within the scope of urology and are generally not emphasized by allopathic physicians. Coupled with this fact, there are also no details in the literature regarding the specificity of the physical exam on CSCP patients. An osteopathic structural exam could prove to be beneficial in properly diagnosing this population. It is important for osteopathic family physicians to be aware of this issue and the gaps that exist in the current literature. Since most cases of CSCP are initially addressed in the primary care setting, osteopathic primary care physicians are at a unique advantage to more adequately help these types of patients. The focus on the relationships between structure and function within osteopathic medical education allows osteopathic physicians to be more equipped to assess and treat this issue efficiently. In patients presenting with CSCP, we recommend OMT or PFPT prior to surgical intervention (Figure 1). This conservative approach may reduce the large portion of CSCP cases that are deemed to be idiopathic. Moreover, it may resolve CSCP in a more cost-effective and less invasive manner. The aforementioned patient example illustrates the importance for osteopathic physicians to remain vigilant in considering musculoskeletal dysfunction when treating patients experiencing CSCP. While additional research is necessary, a greater focus on the relationship between structure and function during the initial examination may be just as important to address this issue more adequately.

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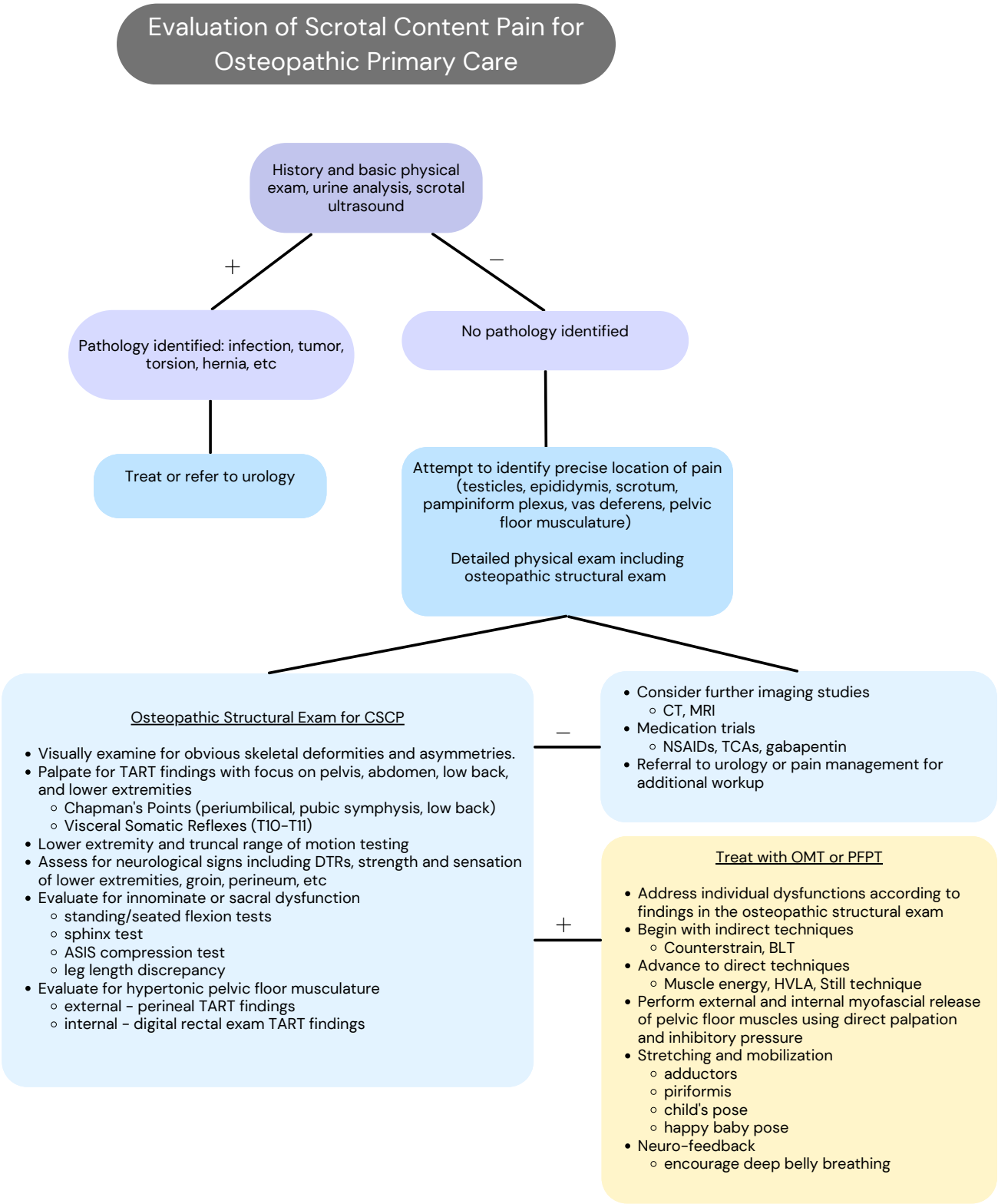
FIGURE 1:

Proposed evaluation of scrotal content pain for osteopathic primary care physicians.



CLINICAL IMAGE

NONTRAUMATIC UPPER-EXTREMITY MASS AND CONTUSION

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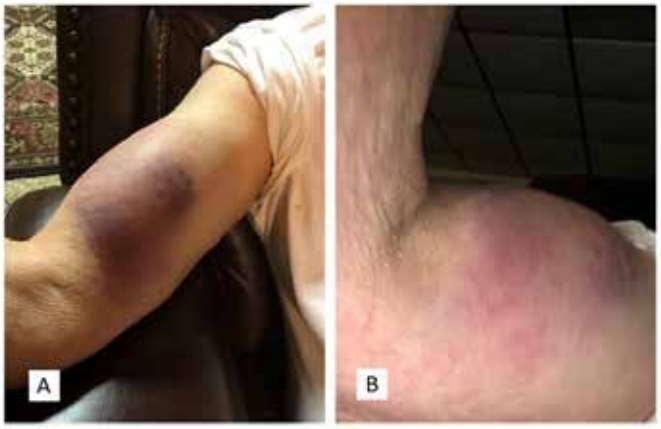
CASE PRESENTATION

A 78-year-old right-hand–dominant male presents to urgent care after experiencing sudden pain and popping in the right upper extremity while lifting an object in the garage. The patient noted weakness in his arm but denied any change in right upper-extremity range of motion after the event. No imaging was obtained, and the patient was sent home with a diagnosis of right upper-extremity contusion. Over the next couple of weeks, he noticed resolution of pain and onset of ecchymosis in the right proximal upper extremity (Figure 1). There was noticeable swelling in the right upper arm. The patient denied previous symptoms in the arm or shoulder. The patient's past medical history is positive for hypercholesterolemia, hypertension, diabetes mellitus type 2, hypothyroidism, urinary retention, and anxiety. His medications include metformin, Benicar (olmesartan), Synthroid (levothyroxine), Tragenta (linagliptin), glipizide, clonidine, Flomax (tamsulosin), Lipitor (atorvastatin), carvedilol, and Lexapro (escitalopram).

FIGURE 1:

(A) Patient's arm shown at partial extension with significant ecchymosis and bulbous Popeye deformity.
(B) Patient's arm at 90-degree flexion with ecchymosis.



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QUESTIONS

1. What is the most likely diagnosis of this patient's arm pain and swelling?

a. Biceps tendon rupture

b. Carpal tunnel syndrome

c. Humerus fracture

d. Rotator cuff tear

e. Triceps tendon rupture

Correct answer:
A. Biceps tendon rupture

Biceps tendon rupture is characterized by ecchymosis, retraction of the muscle belly, and palpable defect in the upper extremity of the patient.[1] Rupture of the long head biceps tendon may present with anterior shoulder pain that distinguishes it from a triceps tendon rupture.[1] Carpal tunnel syndrome commonly presents as pain or paresthesia following the median nerve distribution in the hand.[2] Humerus fracture is less likely due to mechanism of injury in this case. A rotator cuff tear will typically present with distinct shoulder pain and decreased range of motion due to pain.

2: What are predisposing factors that can lead to this injury?

a. Azithromycin use

b. Chronic glucocorticoid use

c. Hypertension

d. Low level of low-density lipoprotein (LDL)

e. Osteoarthritis

Correct answer:
B. Chronic glucocorticoid use

There are a variety of risk factors that increase the likelihood of biceps tendon ruptures. Chronic steroid use is the most common medication to cause this pathology.[3] Other risk factors shown to increase risk of biceps tendon rupture include smoking and hyperlipidemia.[4,5] Most pathophysiology has described an increase in tumor necrosis factor alpha (TNFα) and interleukin (IL)-6, which leads to inflammation and structural weakness in the tendon.[6] Concurrent use of fluoroquinolones with glucocorticoids is also known to increase the risk of tendon rupture by 46-fold.[7]

The incidence of distal biceps tendon rupture, also known as Popeye arm, is 2.55 out of every 100,000 patients per year in the United States.[8] Middle-aged males are most affected, particularly those with an increased body mass index and a history of smoking.[8] The biceps tendon has a long and short head that work together as supinators of the forearm and flexors of the elbow.[1] The long head of the biceps tendon is most commonly involved in ruptures.[1] The mechanism of injury for biceps tendon rupture is not fully understood; however, it is believed to be related to rotator cuff pathology.[9] The mechanism of injury most commonly seen with biceps tendon rupture is during eccentric contraction of the biceps muscle.[1] Examples of said exercise include catching a falling heavy object and forcefully extending a flexed elbow when the biceps muscle is already fully contracted.

Medications, metabolic abnormalities, and chronic disease have been shown to affect the structure and integrity of tendons.[1] Histopathology of ruptured biceps tendons shows a disorganized fiber orientation with elevated levels of proteoglycan, matrix metallopeptidases, and type III collagen.[1] Certain medications, such as fluoroquinolones and corticosteroids, are known for increasing the risk of tendinopathy and tendon rupture.[7] The rate of tendon rupture after fluoroquinolone use is approximately 2.5 cases per 10,000 patients per year.[3] The combination of fluoroquinolones and corticosteroids in patients over 60 years old increased cases to 19.6 cases per 10,000 patients.[3,10] Of tendon injuries, the Achilles tendon consists of over 95% of cases, potentially due to the weight and force on the tendon during weight-bearing movements.[7] β-hydroxy β-methylglutaryl-CoA (HMG-CoA)-reductase inhibitors and aromatase inhibitors have also been associated with weakened tendons.[4] For this patient, his use of atorvastatin for hypercholesterolemia may have increased his risk of tendon rupture.[7]

Additionally, the patient's comorbidities may have contributed to his risk of tendon tear. Increased serum cholesterol promotes lipid deposition systemically, with deposition in tendons in patients as young as 15 years old.[11] This process gives rise to elevated inflammatory cytokines, such as TNFα and IL-6, and structural alterations that may cause weakening of the tendons.[6] Elevated total cholesterol, LDL, and triglycerides have been found in patients with altered structure and increased tendon thickness.[5] Similar to the mechanism of action of tendinopathies seen with chronic corticosteroid use, there is an increased risk of rupture diabetes though less frequent.[12] Chronic glucocorticoid excess as seen in diabetes quickens collagen cross-link formation leading to thicker and stiffer tendons that are more prone to rupture.[12] Other systemic diseases that confer a greater risk of tendon rupture include gout, rheumatoid arthritis, and chronic kidney disease.[4]

Biceps tendon ruptures are commonly diagnosed clinically. They can be categorized as partial or complete tears and by location—proximal or distal. In complete tears, retraction of the biceps muscle belly results in an upper-arm mass termed "Popeye deformity" (Figure 1).[1] Other symptoms include antecubital pain and muscle weakness.[13] Partial ruptures may present with the same signs and symptoms but more subtly.[1] The most specific finding in biceps tendon injury is bicipital groove point tenderness. This can be exhibited by having the patient internally rotate the affected arm 10 degrees, which places the groove facing forward.[14] The lack of visible and palpable defects like the bulbous mass often seen in complete distal tears can lead to delayed diagnosis. The diagnosis of partial ruptures can be made with the aid of imaging when symptoms persist or the clinical picture is unclear. Though ultrasound can assist in diagnosis, magnetic resonance imaging (MRI) presents a definitive diagnosis.[15] Ultrasound overall has a sensitivity of 49% and specificity of 97%.[16] One drawback of MRI is its weakness in detecting partial biceps tendon tears.[17] However, MRI provides an excellent evaluation of the superior labral complex and biceps tendon, which are involved in full tears. Proximal biceps tendon ruptures, more often seen in elderly patients, may present with no other symptoms except pain with occasional proximal arm ecchymosis and weakness.[1] Proximal tears are often associated with rotator cuff affliction and should be monitored for shoulder girdle muscle atrophy and shoulder impingement.[1] Unlike proximal biceps tendon tears, distal tendon rupture presents with weakness of elbow flexion and forearm supination.[1]

Clinical provocative tests have been established in aiding diagnosis in biceps tendon tears. The hook test is useful in determining a complete distal biceps tendon tear by examining the lack of insertion of the biceps tendon distally at the radial tuberosity.[18] This test is conducted by positioning the patient's arm in 90-degree flexion followed by supination. A positive test is shown from the inability to hook the index finger under the distal tendon. Additionally, the Ruland biceps squeeze test is performed by positioning the patient's elbow in 60- to 80-degree flexion with pronation of the forearm.[18] A positive test results from no supination of the forearm or wrist when squeezing the distal biceps muscle belly.

Initial management of a biceps tendon rupture is typically conservative with the use of analgesia, such as nonsteroidal anti-inflammatory drugs (NSAIDs), and rest.[1] Unlike in patients with proximal biceps tendon ruptures, distal biceps tendon tears should have a surgical consultation as soon as possible.[19] Often, surgical refixation is required to regain full strength of forearm supination and function.[19] Surgical candidates should have an early repair to decrease the risk of scar-tissue formation and retraction of the tendon.[1] Complete recovery of distal biceps tendon rupture without intervention is possible with conservative treatment, but has a risk of 40% loss of supination strength and nearly 15% decrease in grip strength.[1] The two surgical techniques used for exploration and repair of distal tendon ruptures are the single-incision and dual-incision approach.[20] Adverse effects of these techniques can include lateral antebrachial cutaneous nerve and posterior interosseous nerve injury with heterotropic ossification.[20,21] Signs of abnormal bone tissue growth within soft tissue include loss of range of motion, localized inflammation, and elevated alkaline phosphatase.[22] Full postoperative recovery can take approximately 4 months with a physical therapy regimen.[20] With conservative treatment, the Popeye deformity and pain are expected to lessen in 4 to 8 weeks.[1] The recovery period for conservative treatment is less than that expected with surgery, but this option poses risk of the aforementioned loss of supination and grip strength.[23] Patients with diabetes and other metabolic syndromes have been found to have worse outcomes following treatment of tendon-related pathology.[12]

Biceps tendon rupture has the potential to be a life-altering injury with long-term effects on upper-extremity muscle strength, particularly in distal biceps tendon tears. It is imperative for the family physician to be aware of the diagnosis and possible complications to properly manage this injury. If patients have associated comorbidities or are on medications that increase their risk of tendon ruptures, they should be educated on injury prevention. The patient in this case did not undergo any treatment. His symptoms gradually improved over the following 3 weeks without any residual complications.

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CLINICAL IMAGE

PERIPHERAL WHITE NODULES

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CASE REPORT

A 92-year-old female presented with white nodules on her toes. Her nodules appeared approximately 3 weeks prior and had been slowly increasing in size. They were associated with pressure sensation but no other symptoms. She denied having skin lesions in other areas of her body. She also denied having fever, fatigue, myalgias, joint swelling, abdominal pain, and nausea. She had a past medical history of atrial fibrillation, hyperlipidemia, hypertensive chronic kidney disease, hypothyroidism, and pulmonary hypertension. Medications included amlodipine, apixaban, ferrous sulfate, levothyroxine, metoprolol tartrate, simvastatin, and vitamin D3.

Examination revealed multiple chalky-white subcutaneous nodules located on her second distal interphalangeal (DIP) joints of her bilateral toes (Figure 1). Her left second DIP toe lesion was ulcerated with chalky-white discharge (Figure 2). No other skin lesions and no acute joint findings were observed. A basic metabolic panel and uric acid level were obtained. Her glomerular filtration rate was

FIGURE 1:

The patient's toes at presentation.

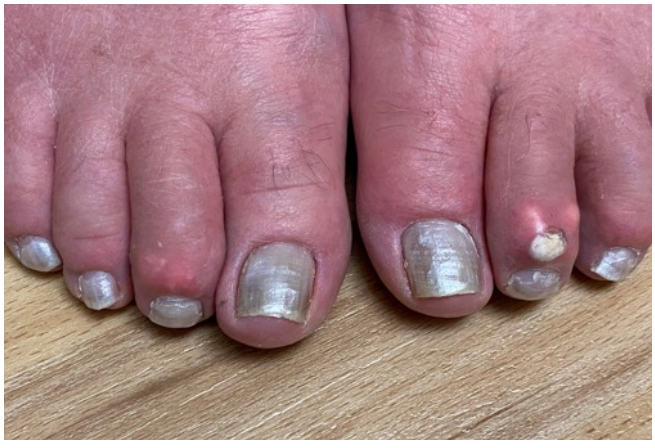

FIGURE 2:

Ulcerated lesion with chalky-white discharge.

QUESTIONS

1. Which of the following tests is required for diagnosis of gouty tophi?

a. Serum uric acid greater than 7.0 mg/dL in men or 6.0 mg/dL in women

b. Joint aspirate revealing negatively birefringent uric acid crystals

c. Characteristic imaging findings (eg, erosions on X-ray or ultrasound)

d. Tests are not required; gouty tophi can be diagnosed clinically

2. In a patient taking allopurinol for treatment of chronic tophaceous gout, what is the recommended target for serum uric acid?

a. Less than 4.0 mg/dL

b. Less than 5.0 mg/dL

c. Less than 6.0 mg/dL

d. Serum uric acid is not a useful treatment target, and treatment should be aimed at clinical resolution of symptoms

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3. Susan is a 76-year-old female with a medical history of gout, hyperlipidemia, hypertensive chronic kidney disease, hypothyroidism, and chronic low back pain. She had a recent gout flare and wonders whether any of her medications may be leading to her gout flares. Which of Susan's medications is most likely to increase serum uric acid and trigger a gout flare?

a. Amlodipine 10 mg PO daily for hypertension

b. Simvastatin 20 mg PO daily for hyperlipidemia

c. Aspirin 325 mg PO TID as needed for low back pain

d. Levothyroxine 125 mcg PO daily for hypothyroidism

ANSWERS

1. Which of the following tests is required for diagnosis of gouty tophi?

Correct Answer:

d. Tests are not required; gouty tophi can be diagnosed clinically

Gouty tophi can be diagnosed clinically based on the presence of white chalky cysts or nodules. Laboratory and imaging can also provide evidence to support the diagnosis. Elevated serum urate can be present in patients with gout, but is not considered diagnostic as patients with hyperuricemia can be asymptomatic and serum urate can be normal during an acute flare. Radiography and ultrasound can provide characteristic findings of tophaceous gout and may be helpful in supporting the diagnosis. A combination of diagnostic criteria can help estimate the likelihood of gout without aspiration. Tophi aspiration is confirmatory and should be considered if the diagnosis remains unclear.

2. In a patient taking allopurinol for treatment of chronic tophaceous gout, what is the recommended target for serum urate?

Correct Answer:

c. Less than 6.0 mg/dL

Treatment target of serum urate <6.0 mg/dL is strongly recommended as it leads to increased urate lowering therapy compliance, tophi reduction, and decreased acute flare frequency. More stringent uric acid targets have been suggested for patients with a heavier disease burden, and may facilitate resolution of tophi. However, there is a lack of study data to routinely recommend lower serum uric acid levels.

3. Susan is a 76-year-old female with a medical history of gout, hyperlipidemia, hypertensive chronic kidney disease, hypothyroidism, and chronic low back pain. She had a recent gout flare and wonders whether any of her medications may be leading to her gout flares. Which of Susan's medications is most likely to increase serum uric acid and trigger a gout flare?

Correct Answer:

c. Aspirin 325 mg PO TID as needed for low back pain

Aspirin is a hyperuricemic medication and can increase her risk of gout. Practical alternatives to aspirin include acetaminophen, naproxen, and ibuprofen. Losartan, simvastatin, and metformin are not known to cause hyperuricemia.

DISCUSSION

Gout is the most common type of inflammatory arthritis and affects up to 3.9% of adults in the United States.[1,2] Gout is closely linked to comorbid conditions including chronic kidney disease, hyperlipidemia, hypertension, insulin resistance, and obesity.[2,3] Tophi are estimated to be present in 12% to 35% of patients with gout.[4,5] A cross-sectional survey of US and EU patients with gout found that tophi have been associated with adverse effects on health care–related quality of life, employment productivity, and utilization of health care resources.[4]

Hyperuricemia is the precursor of gout, and it results from overproduction and/or underexcretion of uric acid. Urates are the ionized form of uric acid, with monosodium urate (MSU) being the most prevalent form at pH 7.4. When the serum urate concentration exceeds 6.8 mg/dL, urate crystals precipitate, which results in hyperuricemia.[6] Long-standing hyperuricemia leads to chronic gout, which classically manifests as tophi. Tophi form from the combination of proinflammatory and anti-inflammatory processes. They contain a center of MSU crystals surrounded by chronic granulomatous deposition of macrophages with overlying connective tissue.[7-10]

Tophi typically present as white chalky cysts or nodules that are firm and nontender. They can manifest either subcutaneously or intra-articularly.[3,7] Observed locations of tophi include the olecranon bursa, wrist, carpal tunnel, interphalangeal joint, metacarpophalangeal joint, spine, talus, metatarsophalangeal joint, hallux, ear, larynx, and cardiac valve.[3,11-13] Tophi can enlarge and emerge superficially, and can result in exudation of white discharge. Complications of tophi include impaired joint function, necrotic ulceration, neuropathy, radiculopathy, and bony erosion.[3,14-16] Tophi can occur even in the absence of gouty arthritis, as in the above patient. This tends to occur in patients who are older, female, take anti-inflammatory drugs or diuretics, or have kidney disease.[11,13,17]

While the diagnosis of tophi can be made clinically, laboratory and imaging evaluation can provide supportive findings.[11,18] Serum urate is typically increased in patients with gout, but it is not diagnostic. Hyperuricemia can be present in asymptomatic patients, and serum urate can be normal in the setting of an acute attack.[18,19] A combination of diagnostic criteria can help estimate the likelihood of gout without aspiration. Criteria include male gender, previous arthritic flare, onset within 1 day, joint erythema, involvement of first metatarsophalangeal joint (MTP1), presence of hypertension or other cardiovascular disease, and serum urate >5.88 mg/dL.[20] Aspiration of tophi provides confirmatory evidence by showing the presence of MSU crystals, and should be utilized when diagnosis is unclear. Aspiration helps exclude the presence of other etiologies, especially septic arthritis.[3,13,18,19]

Plain radiography has higher utility during later stages of chronic gout. Findings include bony erosion, MSU deposition in cartilaginous areas, articular or periarticular soft tissue nodularities, and joint space narrowing.[19,21] Computed tomography (CT) can provide more specific imaging of tophi as compared to ultrasound (US) and magnetic resonance imaging (MRI). CT has been effective in identifying bony erosion and tophi.[21] Dual-energy CT directly visualizes urate deposition with volume measurement, which aids diagnosis and disease monitoring.[22] MRI can help identify tophi in atypical locations, such as the axial spine,[19] and evaluate for complications, such as reduced knee mobility, in the setting of tophaceous deposition.[23] However, MRI is limited by its lower specificity and high cost.[19,20] US can diagnose tophi based on characteristic features such as MSU deposition in cartilaginous areas and identification of tophaceous material and presence of erosion.[24,25] Moreover, US has been validated in the measurement of tophi, which can be used to monitor response to therapy.[20,26]

MANAGEMENT

Starting urate-lowering therapy (ULT) is recommended when patients present with at least one subcutaneous tophus, radiographic evidence of bone erosion attributable to tophi, or have two or more gout flares in a year.[1] ULT facilitates tophi resolution by reducing serum urate concentration.[27,28] Treatment target of serum urate <6.0 mg/dL is strongly recommended as it leads to increased ULT compliance, tophi reduction, and decreased acute flare frequency. It is strongly recommended to use ULT with concurrent anti-inflammatory prophylaxis for 3 to 6 months.[1]

Allopurinol and febuxostat are xanthine oxidase inhibitors (XOIs) and are firstline ULT agents for most patients, including patients with chronic kidney disease stage ≥3. Allopurinol is recommended above febuxostat.[1] The use of febuxostat is limited by increased cost and higher risk of adverse cardiovascular events.[29] Switching to an alternative XOI may be considered if a patient is on maximum dose of the initial agent and has serum urate levels >6 mg/dL, recurrent flares, or persistent tophi.

Probenecid is a uricosuric agent that can be added to XOI therapy if a patient has limited serum urate improvement.[1] Pegloticase is a recombinant uricase that is recommended for patients who have serum urate above treatment goal and persistent tophi despite the use of XOI, uricosuric agents, and other treatments.[1,12,28] Lesinurad is a uric acid transporter 1 inhibitor that has been studied in combination with allopurinol and febuxostat. Lesinurad has mixed evidence supporting its efficacy in managing tophi, and has been withdrawn in the United States. Additional randomized controlled trials are needed for other ULTs.[1,12]

Colchicine, nonsteroidal anti-inflammatory drugs (NSAIDs), and prednisone are firstline medications in acute flare management. Initiating ULT during an acute flare may be conditionally considered. ULT can increase patient compliance and has not been shown to adversely affect duration or severity of acute flare.[1] Surgical intervention for tophi is indicated in the setting of severe complications including infection, entrapment neuropathy, and irreversible joint destruction.[16]

Hyperuricemia medications can increase the risk of gout flares. Examples include aspirin, cyclosporine, tacrolimus, and loop and thiazide diuretics. Medication changes can be considered if the benefit of reduced serum urate is greater than the risk of potential changes.[1,30] The discontinuation of low-dose aspirin, when taken for appropriate indications, is conditionally not recommended due to the lack of alternatives.[1] Thiazides at daily doses of 25 mg or greater have been associated with increased risk of gout.[31] For patients who take hyperuricemia medications, sufficient hydration and symptom and serum urate monitoring are recommended.[30] Additionally, dietary factors including alcohol intake, high-fructose corn syrup, and a high-purine diet have been linked to elevated serum urate levels and increased gout flares. Therefore, dietary recommendations along with weight loss should be advised.[1,3]

CONCLUSION

Gout, with or without tophi, is a relatively common clinical diagnosis in US adults that primary care providers often encounter. Tophi can present subcutaneously or intra-articularly in many parts of the body, and often are found as firm cysts or nodules. Hyperuricemia, either from overproduction or inadequate urate excretion, is the principal etiology for the precipitation of urate crystals that leads to gouty arthritis and tophi. ULT, often with allopurinol as firstline therapy, can help improve hyperuricemia and reduce symptoms.

The patient was started on allopurinol 100 mg daily. After 2 months, her uric acid level decreased to 5.0 mg/dL which met the goal of ULT. Her nodular lesions also improved clinically.

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PATIENT EDUCATION HANDOUT

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Melanoma and Ways to Prevent It

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Melanoma is a type of skin cancer that arises from melanocytes, the pigment-producing cells in the skin. It is the most dangerous form of skin cancer, which can spread to other parts of the body and cause serious health problems. In this handout, we explain what melanoma is, how to prevent it, and what to do if you think you may have it.

WHAT IS MELANOMA?

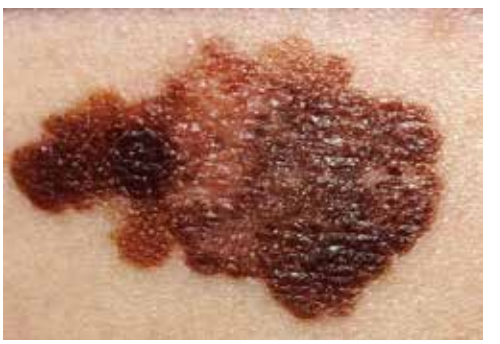
Melanoma is a type of skin cancer that develops in the cells that produce pigment in the skin.[2] It can occur anywhere on the body but is most commonly found on the face, neck, arms, and legs.[1] Melanoma happens to those exposed to ultraviolet (UV) radiation from the sun or tanning beds. It can also occur due to genetic factors, such as having a family history of the disease.

Melanoma can be a severe condition if it is not caught early. If it spreads to other body parts, it can be challenging to treat and even be life-threatening. If melanoma is caught early enough, however, it is often curable with surgery.

WHAT ARE THE SIGNS AND SYMPTOMS OF MELANOMA?

Melanoma usually appears as a new or changing mole on the skin. It may be black or brown, but it could also be pink, red, or white. Melanoma lesions can be small, but some can grow out to be as large as a pencil eraser and may be irregularly shaped or off-color. Other signs of melanoma are:[3]

- A mole that changes in size, shape, or color;
- A mole that itches or bleeds;
- A spot on the skin that looks like a bruise but does not go away; or
- A dark streak under a fingernail or toenail.



If you notice any of these symptoms, it's important to see your primary care physician (PCP) as soon as possible. Your PCP can examine the mole and determine whether it is cancerous.

SOURCE(S):

[National Institutes of Health: National Cancer Institute – 2011]

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HOW CAN MELANOMA BE PREVENTED?

Protecting your skin from the sun's harmful UV rays is the best way to prevent melanoma. Here are some tips for sun safety:[5]

- **Wear protective clothing:** When you are outside, wear clothing that covers your arms and legs. Choose clothing made from lightweight fabrics that will keep you cool and protect you from the sun. You can also wear a wide-brimmed hat to shade your face and neck.
- **Use sunscreen:** Use a broad-spectrum sunscreen with an SPF of at least 30. Apply it to all exposed skin, including your face, neck, and ears. Reapply sunscreen every 2 hours or more often if you are swimming or sweating.
- S**eek shade:** When possible, seek shade when the sun's rays are strongest, typically between 10 a.m. and 4 p.m.
- **Avoid tanning beds:** Tanning beds emit UV radiation, which can damage the skin and increase your risk of melanoma.
- **Check your skin:** Regularly examine your skin for any new or changing moles or spots. If you notice anything unusual, see your PCP right away.

WHAT SHOULD I DO IF I THINK I HAVE MELANOMA?

If you notice any new or changing moles on your skin or any other symptoms of melanoma, it is essential to see your PCP as soon as possible. Your PCP will be able to examine the mole with a dermatoscope and may perform a biopsy to determine whether it is cancerous. If it is found to be cancerous, they will work with you to develop a treatment plan. If you notice a mole, spot, or lesion you did not think was there before, schedule an exam with your PCP as soon as possible.

The most common treatment for melanoma is surgery to remove the cancerous mole and some of the surrounding tissue. If the melanoma has spread to other parts of the body, however, additional treatments such as chemotherapy or radiation therapy may be necessary.[4]

In conclusion, melanoma is a severe type of skin cancer that you can prevent through sun safety practices such as wearing protective clothing, applying sunscreen, seeking shade, avoiding tanning beds, and regularly checking your skin for unusual moles or spots. If you notice any symptoms of melanoma, it is vital to see your PCP as soon as possible for evaluation and treatment.

Melanoma is often curable with early detection and proper treatment, but prevention is always the best option. Protecting your skin from the sun's harmful UV rays can significantly reduce your risk of developing this dangerous disease.

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Migraines

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WHAT ARE MIGRAINES?

A migraine is a type of headache that affects roughly 12% of people in the United States (up to 6% of men and 17% of women).[1,2] These severe headaches can happen alone or with other sensations that involve vision problems, numbness, or tingling that are called "auras."[1] It is believed that migraines are caused by an overactivation of the trigeminal nerve (the pain-sensing nerve of the brain) due to an excess of neurotransmitters and vasodilation, but much is still unknown about them.[1,3,4]

AM I AT RISK?

Adult women are three times more likely to have migraines than men.[1,5] However, in children, boys are more commonly affected.[5] Migraines usually start between 15-24 years, but they can happen at any age.[6] If your parents had migraines, you are at an increased risk.[5] Migraine risk decreases with age, especially after 60 years.[5] Certain foods and medications may also increase your risk of having migraines.4 It is possible that your migraines are related to an unhealthy diet, lack of sleep, or inadequate exercise.[3] Some women may experience migraines related to their menstrual cycle.[1,3]

COMMON SIGNS AND SYMPTOMS

- Neck pain or stiffness
- Zigzag lines, flashes of lights, or blind spots in vision
- Sensitivity to lights or noises
- Nausea
- Vomiting
- Fatigue
- Confusion
- Throbbing headache on one side of the head
- Difficulty talking

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WHAT SHOULD I DO?

Speak with your doctor if you have migraine symptoms.[3] There is no specific test for migraine, but your doctor might recommend a blood test or imaging to rule out other causes.[3] Be prepared to describe your migraines and let your doctor know if any foods, flashing lights, or loud noises make them worse.[3] Keep a record of how often your migraines happen and how bad they are on a scale of 1-10.[1,3] Your doctor may recommend keeping track of all the foods you eat to see if they are causing your migraines.[4]

TREATMENT OPTIONS

You may also be able to stop your migraines from happening or prevent them from hurting as much if you exercise 4 to 5 times per week, work on decreasing stress, and get enough sleep. [1,4,5] It is also important that you drink enough water during the day. If these things do not help your migraines, osteopathic manipulative treatment (OMT), medications, or a combination of both may be recommended by your doctor. OMT techniques such as myofascial tissue release and suboccipital inhibition, among others, have been shown to help patients who experience migraines. Medications used for migraine treatment can either help prevent them from occurring (preventive medications) or help stop them once they start (abortive medications). Your doctor may recommend either or both types so that your migraines are better controlled. The treatment types that your doctor recommends may be tailored based on the severity and frequency of your migraines.

PROGNOSIS

The prognosis of migraine headaches can vary from complete remission to progressive worsening of symptoms and chronic migraines. Migraines tend to be most severe during early to middle adulthood and eventually become milder with aging. However, chronic migraines can last throughout life with variable frequency and severity.[1,2]

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