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The Editor's Desk

Shawn M. Neff, DC, MAS, FACO
Editor-in-Chief

Welcome to the December 2017 issue of the Journal of the Academy of Chiropractic Orthopedists. We at the Journal wish to express our appreciation to our readers and contributors. We are grateful to have the opportunity to help the chiropractic orthopedic community share and stay abreast of the newest evidence in the specialty.

We are aware of the issues we are having with the archives of the Journal. We are working to correct this issue. There were files corrupted and lost in the transition of webhosting. If you have copies of any of the archived journals which are currently corrupted, please contact me at editor@dcorthoacademy.org.

The new year will bring changes as it always does. Our commitment to the doctors and patients of our profession and specialty will not change. We look forward to another year of advancing knowledge in conservative orthopedics with all of you.

I hope you all enjoy this issue.

Sincerely,

-Shawn

Original Article

An Evidence-Based Approach to the Orthopedic Physical Exam – Part 4: The Lower Extremity

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ABSTRACT

This narrative review article aims to examine the current evidence surrounding the orthopedic physical exam procedures related to various lower extremity conditions and provide an overview of these examinations. A narrative review was performed using online databases, authoritative textbooks, and a mobile orthopedic exam application. When multiple studies existed for a single orthopedic test, we reported results from the highest-quality studies and used studies with the highest Quality Assessment of Diagnostic Accuracy Studies (QUADAS) scores. Additionally, we attempted to highlight when orthopedic physical exams were described, but have yet to be evaluated in order to establish diagnostic accuracy. The purpose of this article is to provide an overview of the evidence-based orthopedic physical exams for many lower extremity conditions.

KEY WORDS (MeSH terms)

Evidence Based Practice; Injury, Leg; Injuries, Hip; Injury, Knee; Injury, Ankle; Injuries, Foot

INTRODUCTION

Clinical decision-making involves clinicians engaging in the process of probabilistic thinking, which evaluates the probability that a patient's clinical presentation is due to a given pathology.[1,2] Clinicians tend to represent this decision-making process in the context of establishing a list of differential diagnoses, but at its core the process of winnowing down a list of differential diagnoses is the same as probabilistic thinking.[2]

Framing clinical decision-making in the context of probabilistic thinking is not new. Pauker and Kassirer first described this approach in 1980.[1] We feel this work of Pauker and Kassirer clearly outlines the process of clinical decision-making. They describe the concepts of a *testing threshold* and a *diagnostic threshold* in the context of establishing a differential diagnosis. Every diagnostic challenge incorporates multitudes of information, such as the patient's chief complaint, findings from the physical examination, and even the clinician's instincts (likely from his/her education or clinical experience);[3] each of these pieces of information adds to or subtracts from the probability that any given pathology is responsible for the patient's condition (i.e. disease state).

Testing Threshold and Diagnostic Threshold

Many times the probability that a condition is responsible for a patient's condition is so unlikely that a clinician may exclude it from consideration without consciously thinking about it; when this is the case, the condition does not rise to the *testing threshold*. When conditions fail to reach the *testing threshold*, no further investigation into the presence of such an unlikely condition is warranted (see Figure 1).

Figure 1: Probabilistic Decision-Making*

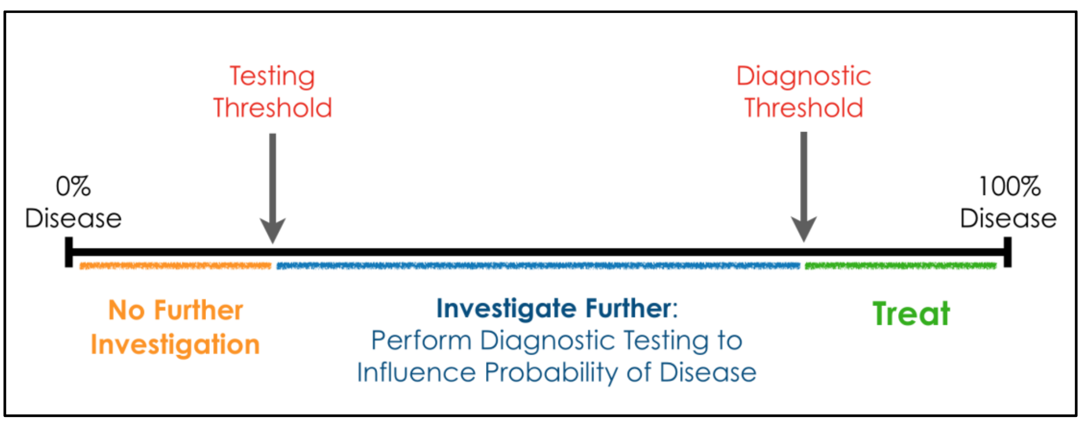


*Figure adapted from Pauker SG, Kassirer JP. The threshold approach to clinical decision making. N Engl J Med. 1980 May 15;302(20):1109-17.

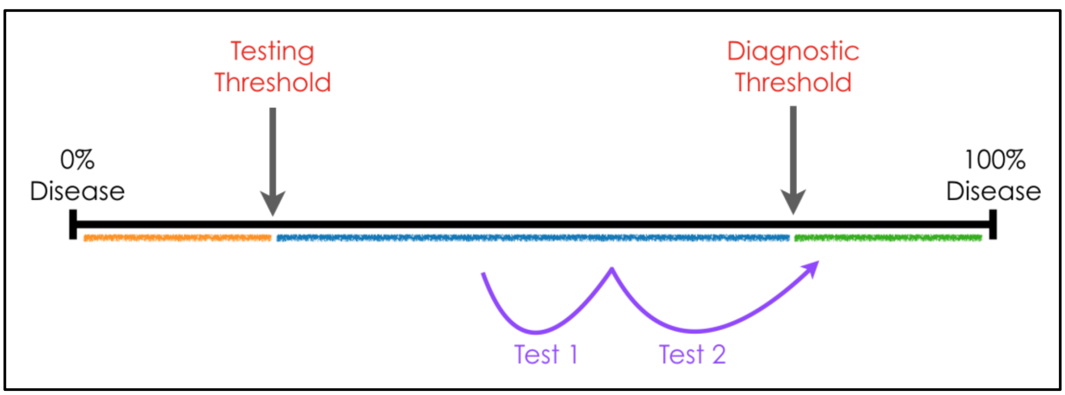
If a patient's chief complaint or past health history lead a clinician to consider a given condition, this potential diagnosis may pass the *testing threshold* and further diagnostic investigation is warranted. At this point the clinician is likely to place this condition on his/her list of differential diagnoses; additional probing questions and diagnostic tests are used to modify the probability that this differential diagnosis is responsible for the patient's presentation. Here we will use a lower extremity injury as an example of how performing a sequence of orthopedic tests may influence the probability of a condition being present, to the point of crossing a *diagnostic threshold*.

Clinical Scenario

A 22 year old female presents at your office with a sudden onset of knee pain and she points to the medial joint line of her right knee. She explains the pain came on suddenly while playing volleyball yesterday and that the pain has been intermittent ever since. She also describes the pain being associated with a locking or pinching sensation, as well as describing a sensation of her knee giving way.

This clinical scenario is likely to have elicited a short list of differential diagnoses for many of you who are clinicians or students. It's likely that you feel the patient's presentation may be due to a meniscus injury, an anterior cruciate ligament injury, or an injury to a collateral ligament of the knee. These are all examples of conditions that have passed your *testing threshold*. Are you ready to say that it is any one of these conditions? It is unlikely. It seems reasonable that you'd want to perform a few orthopedic tests to the area. These tests will serve two purposes; they will add to the probability that the correct diagnosis is identified, while also lowering the probability of other, incorrect, diagnoses. Essentially, you are investigating the clinical scenario to build a case for the correct diagnosis and pass your diagnostic threshold. Orthopedic tests serve to modify the probability that a given condition is responsible for the patient's presentation. Positive tests are likely to raise the probability of the correct diagnosis, while negative tests are likely to lower the probability of competing differential diagnoses. Figure 2 demonstrates how a series of two tests, used in tandem, serve to increase the probability of a condition to the point where it passes the *diagnostic threshold*. It is also worth noting that tests with greater diagnostic utility will have a larger impact on the probability that a condition is or is not responsible for the patient's presentation.

Figure 2: Use of Two Diagnostic Tests to Cross the Diagnostic Threshold



Likelihood Ratios

Likelihood ratios are reported to be the best measure of diagnostic accuracy and allow clinicians to quickly compare the diagnostic utility of various orthopedic tests for the same diagnosis.[4] This is why we've decided to focus on reporting likelihood ratios throughout this four part review series. While nomograms are typically used to teach the concept of likelihood ratios, these are rarely accessible and seldom used in clinical practice.[4] While likelihood ratios are continuous in nature, ranging from 0 to infinity (∞), Table 1 provides a quick way to estimate the influence of positive likelihood ratios (LR+) and negative likelihood ratios (LR-). The purpose of Table 1 is to allow clinicians to memorize the influence of a short list of likelihood ratios for practical purposes, instead of relying on a nomogram.

Table 1: Estimated Influence of Likelihood Ratios*

	Quality	Likelihood Ratio	Influence on Probability
For Positive Likelihood Ratios (LR+), **larger** is better	Best	LR+ = 10	Increased by **45%**
	OK	LR+ = 5	Increased by **30%**
	Poor	LR+ = 2	Increased by **15%**
	Useless	LR+ = 1.0	No change **(0%)**
For Negative Likelihood Ratios (LR-), **smaller** is better	Useless	LR- = 1.0	No change **(0%)**
	Poor	LR- = 0.5	Decreased by **15%**
	OK	LR- = 0.2	Decreased by **30%**
	Best	LR- = 0.1	Decreased by **45%**

*Figure adapted from McGee S. Simplifying likelihood ratios. J Gen Intern Med. 2002 Aug;17(8):646-9.

LR+ = positive likelihood ratio; LR- = negative likelihood ratio

We would like to point out that larger positive likelihood ratios have a greater influence on clinical decision-making compared to positive likelihood ratios that are smaller. As a positive likelihood ratio approaches 1.0 it becomes less useful and likelihood ratios approximating 1.0 lack any diagnostic value.[4] This relationship is reversed for negative likelihood ratios. Smaller negative likelihood ratios (e.g. LR- = 0.1) have a greater influence on clinical decision-making, while larger negative likelihood ratios that approach 1.0 are of minimal use.

The purpose of this article is to provide a succinct review of the literature related to lower extremity conditions as well as the orthopedic physical exam procedures reported to evaluate for the presence of these conditions. This article intends to focus on orthopedic exams demonstrating the most diagnostic utility, while also calling attention to orthopedic exams that have yet to establish diagnostic utility (i.e. have yet to be studied).

METHODS

This is a narrative review of the evidence-based orthopedic exams for various lower extremity conditions. Information was collected from a variety of sources (outlined in Table 2) that were combined with targeted searches of www.PubMed.gov. Original source articles were obtained when additional information was needed to verify study methods or results. Material presented in this review was selected because it originates from research articles with the highest QUADAS scores or originated from the most recent meta-analysis related to the relevant orthopedic test.

Table 2: Sources Used for this Narrative Review

Cook CE, Hegedus EJ. Orthopedic Physical Examination Tests: An Evidence-Based Approach, 2nd Ed. Indianapolis, IN. Pearson Education; 2013
Clinically Relevant Technologies, CORE - Clinical ORthopedic Exam, version 5.3.3, iOS application, last updated on October 20, 2015
Cleland JA, Koppenhaver S. Netter's Orthopedic Clinical Examination: An Evidence-Based Approach, 2nd Ed. Philadelphia, PA. Saunders Elsevier; 2011
Malanga, Gerard A., and Scott F. Nadler. Musculoskeletal Physical Examination: an Evidence-Based Approach. Elsevier Mosby, 2006.
Prentice WE. Principles of Athletic Training A Competency-Based Approach. 14th ed. New York, NY: McGraw-Hill; 2011.
Starkey, C., Brown, S. D., & Ryan, J. (2015). Examination of Orthopedic & Athletic Injuries (3rd ed.). Philadelphia: F.A. Davis Company.
Souza Thomas A. Differential Diagnosis and Management for the Chiropractor. Massachusetts: Jones & Bartlett Learning, 2016. Print.

DISCUSSION

Organization of this Article

This narrative review of the literature is focused on evidence-based orthopedic exams for lower extremity conditions. This article uses a regional approach to presenting material related to the lower extremity and begins with the hip and proceeding distally.

Hip, Groin, & Thigh Complaints

Orthopedic examination of the hip joint is very accurate in detecting the presence of a problem, with a study conducted by JW Thomas et al. showing 98% accuracy during clinical evaluation. Yet, exact diagnosis is complicated by coexisting pathologies and secondary dysfunction.[5] Hip joint dysfunction commonly coexists with lumbar spine dysfunction (i.e. disk related issues) due to biomechanical compensation.[5] In addition, overlapping symptoms lead to complications in accurate diagnosis while distinguishing between bone, musculotendinous structures, bursal structures, neurological structures, and visceral disorders, which may refer pain to the hip region.[5] Table 3 provides a list of a few clinical hypotheses, based upon the patient's presentation.

Table 3: Patient's Presentation for a Hip Complaint and the Associated Clinical Hypotheses*

Patient's Presentation or History	Initial Clinical Hypothesis
Reports of pain at the lateral thigh. Pain exacerbated when transferring from sitting to standing.	Greater trochanteric bursitis Muscle strain
Age > 60. Reports of pain and stiffness in the hip with possible radiation into the groin.	Osteoarthritis
Reports of clicking or catching in the hip joint. Pain exacerbated by full flexion or extension.	Labral tear
Reports of repetitive or overuse injury.	Muscle sprain/strain
Deep aching throb in the hip or groin. Possible history of prolonged steroid use.	Avascular necrosis
Sharp pain in groin. Often misdiagnosed by multiple providers.	Femoroacetabular (anterior) impingement
Pain in the gluteal region with occasional radiation into the posterior thigh and calf.	Piriformis syndrome, hamstring strain, or ischial bursitis

* Adapted from Cleland J, Koppenhaver S. Netter's Orthopaedic Clinical Examination: An Evidence-Based Approach. 2nd Ed., Elsevier Health Sciences; 2015.

<u>Femoral Neck Fracture (Hip Fracture)</u>

The neck is considered to be the weakest part of the femur, and when coupled with predisposing factors such as being an older, osteoporotic woman, injury is more likely to occur. Other risk factors that contribute to an increased risk of femoral neck fractures are listed below (Table 4). They are delineated into two groups, modifiable factors that can be altered to decrease risk of fracture, and nonmodifiable that cannot be changed.

Table 4: Risk Factors for Hip Fractures*

Nonmodifiable	Modifiable
Age > 65 years	Chronic medications: Levothyroxine, Loop diuretics, Proton Pump Inhibitors, Selective Serotonin Reuptake Inhibitors, Sedatives
Family history of hip fracture	Decreased bone mineral density (osteoporosis)
Female sex	Risk of falls
Low socioeconomic status	Reduced level of activity
Previous hip fracture	Vitamin D deficiency

*Adapted from LeBlanc KE, Muncie HL Jr, LeBlanc LL. Hip fracture: diagnosis, treatment, and secondary prevention. Am Fam Physician. 2014 Jun 15;89(12):945-51.

In elderly patients, the mechanism of injury is typically a fall directly onto the hip or a twisting mechanism with the patient's foot planted as the body rotates. In younger patients, femoral neck fractures can occur during significant trauma such as a vehicle collision. Patients present with an inability to bear weight and ambulate normally, pain in the groin or buttock, and possible referred pain into the distal femur and superior knee.[6] There can be significant complications post injury, such as avascular necrosis and nonunion. Hip fractures increase the risk of death in patients with advanced age.[6]

Of tests described in Table 5, the Patellar-Pubic Percussion Test was found to have the highest diagnostic accuracy, not only in one study but in two. In addition, the Fulcrum Test had a moderate diagnostic utility, whereas the Log Roll Test had only minimal utility for femoral neck fractures.

Table 5: Orthopedic Tests for Femoral Neck Fracture

Test	LR+	LR-
Patellar-Pubic Percussion Test*		
Reimab MP, et al.[7]	6.11	0.07
Borgerding LJ, et al.[8]	17.37	0.22
Fulcrum Test[7]	3.7	0.09
Log Roll Test[9]	1.5	0.10

*Multiple high-quality studies have been published; therefore multiple results are reported

Hip Osteoarthritis

The hip is the most common joint in the body to be affected by osteoarthritis (OA) which affects between 10 and 25% of the population over the age of 55.[10] Derangement of the joint can manifest due to many factors including age, repetitive trauma, acute trauma, or improper boney arrangement. An increase in body weight, especially coupled with a family history of hip OA, can increase loads transmitted through the joint, therefore increasing stress on tissues and creating dysfunctional changes in these tissues. In the initial stages of dysfunction, the primary complaint is typically pain specifically while weight bearing, with pain becoming more constant as derangement progresses. The gold standard test for the detection of hip OA is a standing anteroposterior radiograph of the pelvis.[10] Of the tests mentioned below, most have little to moderate diagnostic accuracy, yet when tests are utilized concurrently (as a cluster), accuracy increases as described below (see Cluster Testing for Hip Osteoarthritis). Abduction or adduction causing groin pain has the highest likelihood ratio showing the highest diagnostic accuracy for hip osteoarthritis. Table 6 provides a list of orthopedic tests used to evaluate for the presence of OA at the femoroacetabular (hip) joint.

Table 6: Orthopedic Tests for Hip Osteoarthritis

Test	LR+	LR-
Squat causing posterior pain[10]	6.1	0.79
Patrick's Test, <60 degrees[10]	1.9	0.61
Hip Scour, with adduction[10]	2.4	0.51
Active hip flexion causing lateral hip pain[10]	3.6	0.65
Active hip extension causing pain[10]	2.7	0.59
Abduction or adduction causing groin pain[10]	5.7	0.71
Passive Internal Rotation ≤25°[10]	1.9	0.39
Trendelenburg's Sign[7,10]	1.83	0.82

Cluster Testing for Hip Osteoarthritis

Sutlive et al. created a clinical prediction rule for identifying hip OA, which evaluated the first seven tests listed in Table 6, and the aggregate diagnostic accuracy was assessed. [10] This study revealed that having multiple positive tests for hip OA increases the specificity of the diagnosis. Additionally, this study demonstrated that having any 3 out of the 7 tests positive yielded a positive LR+ of 5.3, while at least 4 positive tests yielded a LR+ of 24.3.[10]

Femoroacetabular Impingement

Femoroacetabular impingement (FAI) is associated with the abnormal anatomical relationship between the acetabulum and femur. The classifications that cause dysfunction can be described as either a cam lesion, pincer lesion, or combination lesion where both are present. The cam deformity is characterized by an additional bony prominence in the femoral head or neck region. The pincer lesion features an additional bony protrusion on the acetabulum. The existence of FAI is a recent discovery for clinicians, yet FAI has proven important to recognize due to its contribution to a multitude of hip related morbidities such as labral injuries and early OA in the active population.[11] Imaging techniques such as radiograph and MRI can be utilized for diagnosis. In addition, relief post anesthetic injection can be utilized as a confirmatory test.[11]

As established in the table below, there is only minimal evidence to support the diagnostic accuracy of test that are typically utilized to identify FAI pathology. Patient history and physical examination are crucial to early detection and treatment. Table 7 provides a list of orthopedic tests used to evaluate for the presence of FAI.

Table 7: Orthopedic Tests for Femoroacetabular Impingement

Test	LR+	LR-
FABER (Patrick's Test)[11]	1.09	0.72
Scour Test[11]	0.82	1.72
Stinchfield Test (Resisted Straight Leg Raise)[11]	0.87	1.28
Maximal Squat Test[12]	1.27	0.61

FABER: abbreviation for flexion, abduction, and external rotation

Acetabular Labral Injuries

Acetabular labral injuries occur due to mechanical stresses placed on the fibrous rim of cartilage and tissue associated with the acetabulum. Patients typically present with anterior hip or groin pain, as well as an associated catching or clicking sensation within the joint. MRI, which is considered the gold standard, is commonly utilized in diagnosing acetabular labral tears. The Thomas and Modified Thomas tests have the greatest diagnostic utility in comparison to other orthopedic examinations that are commonly associated with acetabular labral injuries. Table 8 provides a list of orthopedic tests used to evaluate for the presence of acetabular labral injuries.

Table 8: Orthopedic Tests for Acetabular Labral Injuries

Test	LR+	LR-
Hip Scour Test (Quadrant Test)[13]	1.32	0.58
Impingement Test (FADIR Test)[7]	1.02	0.48
FABER/Patrick Test		
Martin et al. 2006[14]	0.73	2.2
Sutlive et al. 2008[10]	1.9	0.61
Resisted Straight Leg Raise[10,15]	0.87	1.28
Fitzgerald Test[10]	2.4	not reported
Thomas Test[7,10]	11.1	0.12
Modified Thomas Test[13]	11.13	0.12

Trochanteric Bursitis/Greater Trochanteric Pain Syndrome

Trochanteric Bursitis typically occurs due to friction from the iliotibial band crossing the bursa during hip flexion, extension, internal rotation, and external rotation. The bursa lies between the insertion of the gluteus medius and minimus muscles as they attach onto the greater trochanter and shaft of the femur. Symptoms include pain and tenderness over the lateral hip, pain with walking, inability to comfortably lie on the affected side, and paresthesia.[16] The incidence of trochanteric bursitis is approximately 1.8 per 1000 per year with women being most affected,[17] who are possibly predisposed due to increased Q angle. Clinicians should ensure that the patient does not have a femoral neck injury, as trochanteric bursitis can mask its signs and symptoms. Ober's Test was described for the identification of trochanteric bursitis, with a positive test causing pain over the greater trochanter.[18] Although it was described in literature, it was not evaluated for diagnostic accuracy. Trendelenburg test was evaluated for diagnostic accuracy for trochanteric bursitis and proved to be a good measure for identification of this pathology. Table 9 outlines two orthopedic tests used to evaluate for the presence of trochanteric bursitis.

Table 9: Orthopedic Tests for Trochanteric Bursitis

Test	LR+	LR-
Trendelenburg Test[19]	3.64	0.82
Ober's[18]	described, but not evaluated	

Coxa Saltans/Snapping Hip Syndrome

Snapping Hip Syndrome, also known as coxa saltans or dancer's hip, is characterized by a snapping sensation felt when the hip is flexed and extended. There may be an audible pop or snap associated with this sensation, as well as pain or discomfort. This sensation is caused by a taut iliopsoas tendon or iliotibial band (IT band) passing over the greater trochanter of the femur. If the cause stems from the iliopsoas it is termed internal coxa saltans, whereas if the IT band is involved it is considered external coxa saltans which presents more laterally. The FABER and Ober's tests are utilized to distinguish between internal and external snapping hip, respectively. A palpable or audible snap is considered a positive FABER's test for internal snapping hip. It is important to distinguish iliopsoas tendon or IT band snapping from intra-articular disorders that also cause popping or snapping sensations. The snapping sensation of coxa saltans may be solicited with more ease during dynamic movements performed by the patient.[20] Other methods of evaluating snapping hip syndrome include ultrasonography. Patient history, primarily self-reported symptoms, is crucial to consider when evaluating snapping hip. Although described in literature, diagnostic accuracy for snapping hip syndrome has not been evaluated. Diagnosis of these conditions relies heavily on history and inspection.

Athletic Pubalgia/Sports Hernia

Athletic pubalgia, also known as a sports hernia, is typically the result of increased muscular loads, due to high velocity twisting motions. Overuse or muscular imbalance of the abdominal, hip and pelvic musculature places increased stress on the pubic symphysis or pubic bone. Pain related to athletic pubalgia is associated with stresses placed on the transversalis fascia, tendons of the adductor group, insertion of the rectus abdominis, insertion of the internal oblique and aponeurosis of the external oblique, as well as the genital branches of the ilioinguinal or genitofemoral nerves. Repeated stress across the pubic symphysis can lead to weakening or tearing of the pelvic floor musculature. The tests listed below are pain provocative test which place a shearing force across the pubic symphysis. The tests listed below in Table 10 have moderate clinical diagnostic accuracy for the presence of athletic pubalgia, with the Bilateral Adductor Test having the most clinical accuracy.

Table 10: Orthopedic Tests for Athletic Pubalgia

Test	LR+	LR-
Adductor Squeeze Test[21]	4.78	0.63
Single adductor[21]	3.33	0.77
Bilateral Adductor Test[21]	7.71	0.49

Piriformis Syndrome

Pain in the gluteal region is becoming a more commonly recognized complaint.[22] The pain associated with piriformis syndrome stems from the compression of the sciatic nerve by the overlying piriformis musculature.[23] The diagnosis of piriformis syndrome is considered one of exclusion, and examined only after lumbar disease (i.e. disk related issues) were thoroughly investigated.[23] Early consideration and diagnosis of piriformis syndrome can aid in avoiding unsuccessful treatment and prolonged disability.[24] The examinations listed below have a mild to moderate level of diagnostic accuracy for identifying piriformis syndrome. There is no recognized gold standard for evaluating piriformis syndrome.[25] Table 11 provides a list of orthopedic tests used to evaluate for the presence of piriformis syndrome.

Table 11: Orthopedic Tests for Piriformis Syndrome

Test	LR+	LR-
Piriformis/FAIR test[26]	5.18	0.14
Straight Leg Raise Test[22]	3.20	0.90
Active Piriformis Test[22]	3.90	0.27
Seated Piriformis Stretch Test[22]	5.22	0.53
Combined Active Piriformis Test and Seated Piriformis Stretch Test[22]	4.57	0.11

FAIR: abbreviation for flexion, adduction and internal rotation as abbreviated by the cited source

Hip Flexor Pathology

The commonly referred to hip flexor muscular group, is comprised of the psoas major and iliacus muscles. The rectus femoris of the quadriceps group performs hip flexion, and when injured can lead to hip flexion dysfunction.[27] If the range of motion of the hip flexor muscle group (including the iliopsoas and rectus femoris) is decreased or restricted, it can predispose patients to musculoskeletal injuries of the lower extremity. In fact, it has been theorized that pathology of the hip flexors can act as a reciprocal inhibitor to the gluteus maximus.[27,28] The inhibition of the gluteus maximus then causes other hip extensors to be overworked, causing greater tissue stress and dysfunction.[27,28] According to a study conducted by Eckard et al. assessing the epidemiology of hip flexor injury in sport, men's soccer and men's hockey have the highest number of hip flexor strains than any other sport; Furthermore, men's soccer athletes injured their hip flexors more often than women's soccer athletes.[27] Of the orthopedic examinations listed below, Ely's test has the highest diagnostic usefulness, whereas the Modified Thomas test has very limited usefulness during an assessment. The Thomas Test, which is commonly described in assessing hip flexor pathologies, has not been evaluated for diagnostic accuracy. Table 12 provides a list of orthopedic tests used to evaluate for the presence of hip flexor pathology.

Table 12: Orthopedic Tests for Hip Flexor Pathology

Test	LR+	LR-
Thomas Test[29]	described, but not evaluated	
Ely's Test (Duncan-Ely's Test)[30]	2.25	0.57
Modified Thomas[31]	0.74	1.19

Iliotibial Band Friction Syndrome

IT band friction syndrome is a frequent overuse injury caused by excess friction between the IT band and the lateral femoral epicondyle. IT band issues account for the sixth most common overuse injury in runners.[32] The biomechanics associated with the weight bearing movements expose the tissue to an increase in friction and inflammation. Early in the swing phase of gait, the IT band is anterior to the greater trochanter to aid in hip flexion. Yet, as the leg transitions into hip extension, the tissue is pulled across the greater trochanter proximally and the lateral femoral condyle distally, possibly causing friction.[32] Excess friction is caused by improper mechanics including genu valgum, excessive foot pronation, and leg length discrepancy, as well as training behaviors such as running excessive distances and changing running surfaces too quickly. Along with proper mechanics and training habits, orthotics and a consistent stretching routine can assist with prevention and treatment of IT band friction syndrome.[32] Several orthopedic examinations have been indicated in assessing the IT band including Noble's Compression Test, Ober's Test, and Rennes Test. Unfortunately, there is a lack of evidence that evaluates the reliability of these examinations. Therefore, further studies need to be conducted to evaluate their diagnostic accuracy.[32-34]

Knee Complaints

Knee pain is a common reason for patients to seek care from health care providers, and knee injuries are among the most common sports-related injuries.[35] A wide variety of pathologic conditions may cause knee pain, which include acute trauma to the knee, overuse injuries, or inflammatory arthritides. Soft tissue injuries to the knee commonly present with a sudden onset of pain and are characteristically associated with specific mechanisms of onset. Clinicians are presented with a diagnostic challenge each time a patient presents with knee pain and are challenged with formulating a working list of diagnoses during the history and physical exam process. Table 13 outlines characteristic patient presentations related to knee pain.

Table 13: Patient's Presentation for a Knee Complaint and the Associated Clinical Hypotheses*

Patient's Presentation or History	Initial Clinical Hypothesis
Locking or clicking within the knee, joint line tenderness, and edema	Meniscal tear Loose bodies within the joint (joint mice)
Traumatic onset of knee pain after jumping, twisting, or pivoting on a planted foot	Ligamentous injury (e.g. torn ACL) Meniscal tear Patellar dislocation Quadriceps muscle strain or rupture
Traumatic onset of knee pain and/or deformity following posteriorly-directed force to the tibia while the knee is in a flexed position	Posterior cruciate ligament (PCL) injury
Traumatic onset of knee pain following a varus or valgus force to the knee	Collateral ligament injury ● Valgus force = MCL injury ● Varus force = LCL injury
Anterior knee pain following jumping activities and/or deep flexion of the knee, while ascending/descending stairs, or while performing squats	Patellar tendonitis Patellofemoral pain syndrome Osgood-Schlatter disease, with pain at the tibial tubercle
Morning stiffness that diminishes following 10-60 minutes of becoming active	Osteoarthritis of the knee joint

* Adapted from Cleland J, Koppenhaver S. Netter's Orthopaedic Clinical Examination: An Evidence-Based Approach.
2nd Ed., Elsevier Health Sciences; 2015.

ACL, anterior cruciate ligament; PCL, posterior cruciate ligament; MCL, medial collateral ligament; LCL, lateral collateral ligament; OA, osteoarthritis

Evaluating for Fracture around the Knee

Acute injury to the knee may require advanced imaging. While ultrasound or MRI are frequently used to assist in the evaluation of soft tissue injuries of the knee, plain film radiography may be required to evaluate for the presence of an osseous fracture. The Ottawa Knee Rules are a set of clinical features that are strongly associated with fracture in the area of the knee (see Table 14). The presence of at least one of the five clinical features indicates that radiography should be performed. The Ottawa Knee Rules are extremely sensitive (100% sensitive) for acute fracture;[36] therefore, use of this decision making aid is ideal for screening patients for a knee fracture. The absence of each of the five clinical features dramatically reduces the need for x-ray and has been shown to reduce the use of unnecessary x-rays following acute knee injury.[36,37]

Table 14: Ottawa Knee Rules for Assessing Fracture of the Knee[*]

≥55-years-old
Tenderness at the fibular head
Patellar Tenderness
Inability to flex the knee more than 90°
Inability to bear weight (≥4 steps) on the involved knee ● Immediately following trauma ● In emergency room (clinical setting)

* Adapted from Stiell IG, Greenberg GH, Wells GA. Derivation of a decision rule for the use of radiography in acute knee injuries. Ann Emerg Med. 1995 Oct;26(4):405-13.

Meniscal Injury at the Knee

A meniscus injury of the knee frequently occurs during an athletic activity as the knee twists while the foot is planted on the ground or a shear force is applied to the knee.[38] Each knee joint contains a medial meniscus and a lateral meniscus; injury to the medial meniscus is about five times more common, and meniscus injuries are commonly associated with injuries to other ligaments of the knee.[39] Patients suspected of having suffered a meniscus injury will frequently have an acute onset of joint line tenderness, edema/effusion, limited range of motion, or report a “catching” or locking” sensation.[40,41]. Table 15 provides an overview of the orthopedic exams associated with evaluating for the presence of a meniscus injury of the knee.

Table 15: Orthopedic Tests for a Meniscus Injury of the Knee*

Orthopedic Test	LR+	LR-
McMurray's Test**		
Miao Y, et al. 2011[42]	6.25	0.78
Jaddue, et al. 2010[43]	Medial = 2.27 (no lateral reported)	Medial = 0.64 (no lateral reported)
Pookarnjanamorakot C, et al. 2004[44]	3.5	0.78
Akseki D, et al. 2004[40]	Medial = 2.2 Lateral = 4.4	Medial = 0.48 Lateral = 0.53
Apley's Compression Test[45]	1.0	0.91
Apley's Distraction Test[46]	0.13	1.87
Thessaly Test*		
Pookarnjanamorakot C, et al. 2004[44]	6.8	0.76
Mirzatolooei F, et al. 2010[47]	1.3	0.51
Harrison BK, et al. 2009[48]	39.3	0.10
Ege's Test (Akseki Test)[40]	Medial = 3.5 Lateral = 6.4	Medial = 0.41 Lateral = 0.40
Axial Pivot-shift Test[49]	4.2	0.35
Steinmann I Sign[43]	Medial = 3.88 (no lateral reported)	Medial = 0.41 (no lateral reported)
Dynamic Test (for lateral meniscus injury)[50]	Lateral = 3.88 (no medial reported)	Lateral = 0.41 (no medial reported)

Bounce Home Test (Forced Extension Test)[49]	1.4	0.70
Childress Test (Squat Test or Duck Waddle Test)[44]	1.7	0.53
Payr Test/Sign[44,45]	0.96	1.05

* Results are reported for combined meniscal injury (medial or lateral), unless medial or lateral are directly listed

** Multiple high-quality studies have been reported; therefore, we've included the results from a few of the most rigorous studies.

Cluster Testing for a Meniscus Injury

The use of multiple orthopedic tests may be useful for clinicians attempting to diagnose a suspected meniscal injury of the knee. Miao, et al. evaluated the combination of 4 clinical features (edema, joint line tenderness, a sensation of "locking," and a positive McMurray's test).[42] This evaluation of the cluster combination discovered that the presence of any 2 of the 4 clinical features is highly useful for diagnosing a meniscal injury with a positive likelihood ratio of 14.5 and a negative likelihood ratio of 0.44.

Anterior Cruciate Ligament Injury

Injury to the anterior cruciate ligament (ACL) is a common knee injury among adolescent athletes. The sports which place individuals at the greatest risk for suffering an ACL injury are soccer, football, basketball, skiing, and lacrosse.[51] Additionally, females participating in sport are about 1.5-to-10 times more likely than males to injure their ACL, depending on the specific sport.[52,53] Table 16 provides a list of orthopedic tests used to evaluate for the presence of an ACL injury.

Table 16: Orthopedic Tests for an Anterior Cruciate Ligament Injury

Orthopedic Test	LR+	LR-
Lachman's Test[54]	10.2	0.2
Prone Lachman's Test[55]	20.17	0.32
Active Lachman's Test[56]	described, but never evaluated	
Anterior Drawer Test[57]	1.67	0.91
Anterior Drawer Test in External Rotation[58]	described, but never evaluated	
Anterior Drawer Test in Internal Rotation[58]	described, but never evaluated	
Lever Sign Test (Lelli's test)[59]	6.3	0.41
Pivot-Shift Test[54]	8.5	0.90
Fibular Head Sign[60]	described, but never evaluated	

Cluster Testing for ACL Injury

Wagemakers et al. established how accurately a clinician could identify ACL injury by evaluating for a few clinical features (effusion around the knee, a "popping sensation," and a sensation of "giving way") and assessing the ACL utilizing the anterior drawer test.[61] When they evaluated patients with knee pain for this combination of findings, their study yielded a positive likelihood ratio (LR+) of 19.9 and a negative likelihood ratio (LR-) of 0.80. These findings suggest that patients presenting with swelling around the knee, combined with popping, and a sensation of giving way may only require an additional positive anterior drawer test before clinicians can be fairly confident that the patient has a torn ACL.

Posterior Cruciate Ligament Injury

The posterior cruciate ligament (PCL) is broader and stronger than the ACL and is also injured less frequently than the ACL.[62] The PCL prevents excessive posterior translation of the tibia, relative to the femur, and PCL injuries characteristically involve a history of knee hyperextension or posteriorly-directed force to the patient's flexed knee (e.g. during an automobile accident).[63] Also, when compared to ACL injuries, PCL injuries are less likely to be associated with a "popping" sound at the time of onset.[64] Table 17 provides a list of orthopedic tests used to evaluate for the presence a PCL injury.

Table 17: Orthopedic Tests for a Posterior Cruciate Ligament (PCL) Injury

Orthopedic Test	LR+	LR-
Posterior Drawer Test[65]	90	0.10
Posterior Functional Drawer Test[66]	described, but never evaluated	
Posterolateral Drawer Test[67]	described, but never evaluated	
Modified Posterolateral Drawer Test (Loomer's Test)[68]	described, but never evaluated	
Posterior Sag Sign (Godfrey's Test, Gravity Drawer Test)[69]	∞	0.21
Reverse Lachman's Test (Trillat's Test)[65]	5.64	0.43
Quadriceps Active Test[65]	18.0	0.47
Reverse Pivot-Shift Test[65]	5.2	0.78
Varus/Valgus Stress at 0° Flexion[70]	described, but conclusions about diagnostic utility are possible, due to methodological issues	
External Rotation Recurvatum Test[65]	3.0	0.98
Anterior Abrasion Sign[71]	described, but never evaluated	
Proximal Tibial Percussion Test[66]	described, but never evaluated	
Dial Test (Posterolateral Rotation Test)[72]	described, but never evaluated	
Standing Apprehension Test[73]	described, but never evaluated	

∞ = infinity, which occurs when the sensitivity or specificity of an exam is 100%, thus preventing the ability to calculate a likelihood ratio. In clinical practice, this should be thought of as a test that is of very high quality.

Collateral Ligament Injuries of the Knee

Medial collateral ligament (MCL) injury occurs when a valgus force is applied to the knee, and injuries to the MCL are much more common than injuries to the lateral collateral ligament

(LCL).[74] Many MCL injuries occur when a traumatic force is applied to a partially flexed knee and patients may be able to ambulate following the injury.

The Valgus Stress Test is performed at 0° and 30° of flexion and is the hallmark orthopedic test for evaluating for an MCL injury. Pain on the Valgus Stress Test equals a LR+ of 2.3 and the absence of pain equals a LR- of 0.30, while *laxity* on the Valgus Stress Tests equals a LR+ of 1.8 and the absence of laxity equals a LR- of 0.20.[75]

A test cluster for evaluating an MCL injury has also been developed.[75] Kastelein et al. discovered that patients who have experienced either trauma by external force to the leg or rotational trauma to the leg along with laxity and pain on the Valgus Stress Test have a LR+ of 6.4 and a LR- of 0.5.[75] This shows that combining information from the history and orthopedic exam may provide the most useful information when evaluating potential MCL injuries.

The LCL is one component of a complex of ligaments known as the posterolateral corner of the knee. Injury to the LCL occurs when a varus force is applied to the knee, which results in localized pain, edema, and a sensation of instability in more severe cases.

The Varus Stress Test is also performed at 0° and 30° of flexion and is the hallmark orthopedic test for evaluating for an LCL injury. Unfortunately, the studies that have attempted to evaluate the clinical utility of the Varus Stress Test have methodological issues, which limits the ability to draw conclusions from these studies.[7677]

<u>Anterior Knee Pain</u>

Anterior knee pain, sometimes called patellofemoral pain is a symptom that may originate from various pathologies.[78] Anterior knee pain is most likely to affect adolescents and young adults and is more commonly reported among females.[79] While the features of anterior knee pain vary by individual diagnosis, common symptoms include pain with activity, such as those that involving walking down stairs, squatting, prolonged sitting, or when wearing high-heels.[7880] We have provided a brief overview of the common causes of anterior knee pain in Table 18, along with the relevant orthopedic tests for each diagnosis.

Table 18: Causes and Orthopedic Tests for Anterior Knee Pain

Condition	Orthopedic Test	LR+	LR-
Patellofemoral Pain Syndrome (chondromalacia patella)	Waldron Test, Phase I[81]	1.7	0.81
	Waldron Test, Phase II[81]	1.05	0.99
	Pain During Resisted Knee Extension[80]	2.2	0.75
	Medial & Lateral Movement of the Patella[80]	1.5	0.80
	Patellar Compression[80]	1.5	0.60
	Pain During Functional Activity (squatting)[80]	1.8	0.20
	History of "pain with squatting"[82]	5.4	0.66
	History of peripatellar pain[82]	19.0	0.43
	History of pain while navigating stairs or sitting[82]	11.6	0.44
Patellar Dislocation	Patellar Apprehension (Fairbank's Test)[81]	2.3	0.79
Patellar Instability	Passive Patellar Tilt Test[83]	5.4	0.62
	Lateral Pull Test (Active Instability Test)[83]	∞	0.75
	Moving Patellar Apprehension Test[84]	8.62	0.0
	Medial and Lateral Patellar Glide Tests[85]	not evaluated	not evaluated
Abnormal Patellofemoral Tracking	Vastus Medialis Coordination Test[81]	2.26	0.90
Patellofemoral Joint Pathology	Clarke's Sign (Patellar Grind Test)[81]	1.94	0.69
Patellofemoral Joint Dysfunction	Eccentric Step Test[81]	2.34	0.71
Plica Syndrome*	Medial Patellar Plica Test (MPP Test)[81,86]	8.18	0.11
	Plica Stutter Test[87]	not evaluated	not evaluated
Patella Alta	Patella Alta Test[83]	1.75	0.71
Patellar Tendinopathy (Jumper's Knee)	Palpation of the inferior pole of the patella for moderate or severe pain[88]	2.18	0.76

Chondral Fracture (osteochondritis dissecans)	Wilson's Test[89]	described, but not evaluated
Osgood-Schlatter's	Physical examination and radiography[90]	described, but not evaluated
Patellofemoral Bursitis (Housemaid's Knee)	Physical examination combined with a history of frequent kneeling[91]	described, but not evaluated
Infrapatellar Fat Pad Injury (Hoffa's disease)	Physical examination and MRI[92,93]	described, but not evaluated

∞ = infinity, which occurs when the sensitivity or specificity of an exam is 100%, thus preventing the ability to calculate a likelihood ratio

* Many tests have been described with the intention to diagnoses plica syndrome, but nearly all have not been evaluated for diagnostic accuracy

Leg Complaints

Complaints of the leg can present as a challenge for clinicians to properly diagnose as many of them have overlapping symptoms. It is important for the clinician to recognize subtle differences in the presentation and formulate a correct diagnosis, as management strategies vary greatly with each condition. Although not an exhaustive list, the three leg conditions focused on in this article are medial tibial stress syndrome, chronic exertional compartment syndrome, and stress fracture. Table 19 provides a list of a few clinical hypotheses for patients presenting with leg pain, based upon the patient's presentation.

Table 19: Patient's Presentation for a Leg Complaint and the Associated Clinical Hypotheses*

History/Presentation	Initial Hypothesis
Runner, pain at distal third of posteromedial tibia, worse at the beginning and conclusion of activity/sport	Medial tibial stress syndrome (shin splints)
Pain in the lower leg, oftentimes described as burning or cramping, experienced with exercise. Symptoms resolve with rest	Chronic Exertional Compartment Syndrome
An insidious onset of pain with a concurrent reported change in activity, localized bony pain	Tibial Stress Fracture

* Adapted from Cleland J, Koppenhaver S. Netter's Orthopaedic Clinical Examination: An Evidence-Based Approach. 2nd Ed., Elsevier Health Sciences; 2015.

Medial Tibial Stress Syndrome

Medial tibial stress syndrome (MTSS), commonly referred to as shin splints, affects many runners and persons involved in other activities that involve running and jumping on hard surfaces. Although the exact etiology of MTSS is unknown, it is believed that bony overload and periosteal inflammation or traction are involved.[94,95] There are no orthopedic tests designed to evaluate for MTSS. Research has shown that it can be reliably clinically diagnosed using history and physical examination findings.[96] Common features of MTSS include;

- Pain provoked upon palpation of the posteromedial tibia over an area of at least 5 cm
- Pain located within the distal third of the tibia
- Pain improves with relative rest
- Pain exacerbated with physical activity, especially at the beginning and end

Chronic Exertional Compartment Syndrome

Chronic exertional compartment syndrome (CECS) is a rare condition that typically affects young adult distance runners and other running athletes. There is an increase in pressure within the confinement of a closed fascial compartment during exercise. There are currently no orthopedic tests used to evaluate CECS. A good history is paramount, as the physical exam is often unrevealing.[97] It is important to note for the chiropractor that CECS can mimic other conditions, such as MTSS, and that there is an average 2 year delay in diagnosis, making it important to rule out other causes.[98] The gold standard in diagnosis is intracompartmental pressure testing, which is outside the scope of a chiropractor. Common history findings for CECS include;

- Bilateral symptoms 70-80% of the time
- Pain, swelling, sensation of burning, cramping, tightness develop during exercise
- Development of pain in a certain area of the leg develops at the same time, distance, or intensity of the exercise
- Pain is relieved with rest

Stress Fracture

Stress fractures of the leg are associated with repetitive activities of impact, such as running and marching. They most commonly occur in the tibia, although they can occur in the fibula. Common physical exam findings associated with a stress fracture include;[99,100]

- Recent increase in physical activity
- Gradual onset
- Pain with weight bearing
- Localized bony pain
- Begins as pain with stress, eventually progressing to pain at rest and at night

Although radiographs are commonly the first image ordered, they have a poor sensitivity in the diagnosis of stress fractures, as they are not visible on radiographs for 2-6 weeks post injury. Scintigraphy and MRI are considered the gold standard in diagnosing stress fractures. Although there are no traditional orthopedic tests used in helping diagnose stress fractures, the use of a tuning fork and therapeutic ultrasound have been studied; reproduction of pain following the application of the tuning fork or ultrasound to the bone is a positive finding for a stress fracture. Table 20 provides an overview of the exams associated with evaluating for the presence of a stress fracture in the lower leg.

Table 20: Orthopedic Tests for a Stress Fracture

Exam	LR+	LR-
Tuning Fork (128-Hz)[101]	2.3	0.3
Ultrasound[102]	2.1	0.3

Ankle Complaints

Ankle sprains are the most common musculoskeletal injuries seen by primary care providers.[103] Although sprains are the most common injury to the ankle, other conditions (ex. tendinopathy, fracture, nerve compression, arthritis, etc.) can cause pain and dysfunction in the ankle. Table 21 outlines characteristic patient presentations associated with ankle pain.

Table 21: Patient's Presentation for an Ankle Complaint and the Associated Clinical Hypotheses*

Patient's Presentation or History	Initial Clinical Hypothesis
Patient reports a traumatic incident in either forced inversion or eversion	Possible ankle sprain Possible fracture Possible peroneal nerve involvement (with inversion)
Patient reports trauma to ankle that included tibial rotation on a planted foot	Possible syndesmotic sprain
Patient reports traumatic event resulting in inability to plantarflex the ankle	Possible Achilles tendon rupture
Patient reports pain with stretch of calf muscles and during gait (toe push off)	Possible Achilles tendonitis Possible Sever's disease

* Adapted from Cleland J, Koppenhaver S. Netter's Orthopaedic Clinical Examination: An Evidence-Based Approach.
2nd Ed., Elsevier Health Sciences; 2015.

Ankle Sprain

An ankle sprain is the most frequent injury to the ankle, with inversion sprain being the most prevalent. An inversion ankle sprain damages the anterior talofibular ligament most commonly, but can also affect the calcaneofibular and posterior talofibular ligaments.[104] Eversion ankle sprains can damage the deltoid ligament, and are commonly associated with fractures of the medial malleolus and syndesmotic injuries.[105] Common exam findings include tenderness, swelling, and bruising around the ankle with an inability or difficulty bearing weight on the affected side. Acute injury to the ankle may necessitate advanced imaging to screen for fracture. The Ottawa Ankle Rules (OAR) (Table 22) were developed to guide clinicians as to when advanced imaging of the ankle is appropriate after injury. The absence of each of the five clinical features dramatically reduces the need for an x-ray and has been shown to reduce the use of unnecessary x-rays following acute ankle injury.[106] If any of the below features are present, then an x-ray should be obtained. The OAR were found to have a LR+ of 1.52 and a LR- of 0.03, which means that they are best used to rule out a fracture. The extremely low LR- provides the clinician with a lot of confidence that if none of the below findings are present, then the patient is very unlikely to have a fracture.[107] Table 23 outlines the orthopedic tests for evaluating patients who may have a sprained ankle.

Table 22: Ottawa Ankle Rules to Evaluate for Fracture Following an Ankle Injury*

Bony tenderness along the distal 6 cm of posterior edge of fibula or tip of lateral malleolus
Bony tenderness along distal 6 cm of posterior edge of tibia/tip of medial malleolus
Bony tenderness at the base of the 5th Metatarsal
Bony tenderness at the navicular
Inability to bear weight both immediately after injury and for 4 steps during initial evaluation

*Adapted from Stiell IG, McKnight RD, Greenberg GH. Implementation of the Ottawa ankle rules. JAMA. 1994 Mar 16;271(11):827-32.

Syndesmotic Injuries

Syndesmotic injuries, also known as "high ankle sprains" are a relatively rare ankle injury, with the incidence estimated from 1-11% of all ankle sprains. These injuries are usually a result of an external rotation force combined with dorsiflexion and/or eversion, which can sprain or rupture the syndesmosis between the tibia and fibula.[108] Table 23 outlines the orthopedic tests for evaluating patients who may have a syndesmotic injury.

Ankle Impingement

Ankle impingement is divided into anterior and posterior ankle impingement. Anterior impingement is a condition in which pain is experienced at the front of the ankle due to compression of bony or soft tissue structures in the ankle mortise joint during maximal dorsiflexion. Posterior impingement refers to pain felt at the back of the ankle due to compression of structures in the ankle mortise during maximal plantar flexion. There are no orthopedic tests in the literature that have been studied for posterior ankle impingement. There has been one orthopedic test studied for anterior impingement, the forced dorsiflexion test. It should be noted that this study had a high risk of bias.[109] Table 23 outlines the orthopedic test for evaluating patients who are suspected of ankle impingement.

Achilles Rupture and Achilles Tendinopathy

The Achilles tendon is the most commonly ruptured tendon. The main causes are a forceful contraction of the calf muscles, overstretching of the tendon, and a fall from a height. As opposed to a complete rupture, some patients' tendon will still be intact and will have Achilles tendinopathy, which is usually associated with overuse. Table 23 outlines the orthopedic tests for evaluating patients who may have Achilles tendon rupture or tendinopathy.

Table 23: Causes and Orthopedic Tests for Ankle Pain

Condition	Orthopedic Test	LR+	LR-
Inversion Ankle Sprain	Anterior Drawer Test		
	Croy T, et al.[104]	1.4	0.41
	Schwieterman B, et al.[110]	∞	0.42
	Inversion Talar Tilt Test[110]	4.00	0.57
	Posterior Drawer Test	described, but never evaluated	
Eversion Ankle Sprain	Eversion Talar Tilt Test	described, but never evaluated	
Syndesmotic Injury	External Rotation Stress Test (Kleiger's Test)[110]	only a specificity of 99% was reported	
	Squeeze Test[110]	4.60	0.75
	Fibular Translation[110]	6.30	0.28

Anterior Ankle Impingement	Forced Dorsiflexion [109]	8.06	0.06
Achilles Rupture)	Thompson Test[110]	13.47	0.04
	Matles Test[110]	6.18	0.14
	Palpable Gap Test[110]	6.81	0.30
	Copeland Test[110]	only a sensitivity of 78% was reported	
Achilles Tendinopathy	Palpation[111]	3.15	0.48
	Arc Sign[111]	3.24	0.68
	Royal London Hospital Test[111]	3.84	0.54

Foot

The foot is a complex structure of the body with all of its associated weight-bearing bones, joints, ligaments, and tendons. Due to the complexity and frequent use of the foot, there are a multitude of injuries that can occur, many of which do not have any associated orthopedic test. This article will attempt to cover the most common foot injuries that have orthopedic tests. Table 24 outlines a characteristic patient presentation related to foot pain.

Table 24: Patient's Presentation for an Ankle Complaint and the Associated Clinical Hypotheses*

Patient's Presentation or History	Initial Clinical Hypothesis
Patient reports pain at heel with first few steps out of bed after prolonged periods of walking	Possible plantar fasciitis
Patient reports pain or paresthesias in plantar surface of foot	Possible tarsal tunnel syndrome Possible sciatica Possible lumbar radiculopathy
Patient reports pain on plantar surface of foot between 3rd and 4th metatarsals. Might also state that pain is worse when walking with shoes compared with barefoot	Possible Morton's neuroma Possible metatarsalgia

* Adapted from Cleland J, Koppenhaver S. Netter's Orthopaedic Clinical Examination: An Evidence-Based Approach. 2nd Ed., Elsevier Health Sciences; 2015.

Turf Toe

Turf toe is a hyperextension injury of the first metatarsophalangeal joint, usually combined with axial compression. It can damage a variety of structures of the capsular ligamentous complex that supports the joint.[112] It is suggested in the literature to assess the joint with valgus and varus stress tests along with a Dorsoplantar Drawer test, although these tests have not been studied to determine diagnostic accuracy. Table 25 outlines the orthopedic tests for evaluating patients who may have turf toe.

Morton's Neuroma

Morton's Neuroma is a condition associated with the common plantar digital nerves and is thought to occur from repetitive trauma, which leads to a disorganized overgrowth of neuronal and fibrous tissues. Morton's neuroma may produce pain or numbness in the foot, and the most common locations of a Morton's neuroma are the second and third intermetatarsal spaces.[113] Table 25 outlines the orthopedic tests for evaluating patients suspected of having a Morton's neuroma.

Plantar Fasciitis

Plantar fasciitis can be a cause of plantar heel pain that usually localizes to the anterior, medial heel. It is commonly seen in runners, as it is one of the top three most common running injuries, but is also seen in people who have recently increased their amount of physical activity.[114] The pain is commonly at its worse the first thing in the morning, or after a period of non-weight bearing, but usually improves with light activity.[115] Table 25 outlines the orthopedic tests for evaluating patients who may have plantar fasciitis.

Sever's Disease

Sever's disease, also called calcaneal apophysitis, is a traction apophysitis that occurs where the Achilles tendon attaches to the calcaneus. It causes inferior heel pain in children and adolescents. The pain is usually absent in the mornings and is aggravated by physical activity, particularly running and jumping.[116] Table 25 outlines the orthopedic tests that have been reported for evaluating Sever's disease.

Tarsal Tunnel Syndrome

Tarsal tunnel syndrome is a compressive neuropathy of the posterior tibial nerve as it passes under the flexor retinaculum of the tarsal tunnel. Common symptoms include paresthesia and pain on the plantar surface of the foot and the medial ankle.[117] Table 25 outlines the orthopedic tests for evaluating patients suspected of having tarsal tunnel syndrome.

Hallux Valgus

Hallux valgus is a progressive foot deformity in which the first metatarsal deviates medially while the first phalange deviates laterally, creating a characteristic foot deformity (see Figure 3). Hallux valgus can lead to bony and soft tissue changes that result in the formation of a bunion, along with pain and functional deficits. Although no orthopedic tests have been reported to diagnose hallux valgus, the Manchester scale was developed to grade the level of severity of hallux valgus. The Manchester scale is pragmatic in that no advanced imaging is required and can be applied by both clinician and patient. It has been shown to be reliable in terms of retest and inter-rater reliability.[118-120]

Figure 3: Gross and Radiological Appearance of Hallux Valgus*

* Image "Best Shoes for Bunions," licensed under Creative Commons (CC) BY 2.0, accesses on Nov. 11, 2017 at: https://c2.staticflickr.com/6/5599/30475577936_94dbfba526_b.jpg

Table 25: Causes and Orthopedic Tests for Foot Pain

Condition	Orthopedic Test	LR+	LR-
Turf Toe	Valgus/Varus Stress Test	described, but never evaluated	
	Dorsoplantar drawer test	described, but never evaluated	
Morton's Neuroma	Thumb Index Finger Squeeze[121]	∞	0.04
	Mulder's Click[121]	∞	0.38
	Foot Squeeze[121]	0.41	∞
	Plantar Percussion[121]	∞	0.64
	Dorsal Percussion[121]	∞	0.74
	Abnormal Light Touch/Pin Prick[121]	∞	0.75
	Digital Nerve Stretch Test[122]	only reported a sensitivity of 100%	
Plantar Fasciitis	Windlass Test[123]	∞	0.68
Sever's Disease	One-Leg Heel Standing[124]	∞	0
	Calcaneal Squeeze Test[124]	∞	0.03
	Palpation Test[124]	∞	0.2
Tarsal Tunnel Syndrome	Tinel's Sign[110]	only reported a sensitivity of 58%	
	Triple Compression Test[110]	∞	0.14
	Dorsiflexion Eversion Test[110] • when a + sign was increased palpatory tenderness at posterior tibial nerve in tarsal tunnel	∞	0.02
	• when a + sign was increase in pain in foot/ankle	∞	0.43
	• when a + sign was increased numbness in foot/ankle	∞	0.75

LIMITATIONS

Due to the nature of this being a narrative review, selection bias may have influenced our selection of relevant reference articles or source materials. Also, the lack of search criteria, which would have been involved with a systematic review, make it so our results are less reproducible. While we attempted to select the highest quality reference materials, using the QUADAS grading scale, we did not systematically grade every source article that was used in this report.

CONCLUSION

The purpose of this article is to provide clinicians with an evidence-based overview of the available orthopedic tests for a variety of lower extremity conditions. Additionally, we have attempted to emphasize when orthopedic tests do not exist or when they have only been described, but have never been evaluated for accuracy. Many orthopedic tests for lower extremity conditions have limited utility. When no test exists or when the available tests are not of a high quality, we would like to emphasize that this is when clinical decision-making should rely more heavily on the other aspects of an evaluation such as the history, patient's presentation, doctor's experience, and even the patient's preferences.

Throughout this 4-part orthopedic review series the authors have attempted to encourage clinicians to adopt an evidence-based approach to clinical decision-making. We have suggested that clinicians adapt the use of likelihood ratios as one of the best ways to quickly judge the usefulness of orthopedic exams. Our hope is that this review series will assist clinicians in their ability to provide a concise orthopedic exam. Using the fewest number of exams that are of the highest utility is intended to yield the most accurate results. Additionally, the use of high-quality exams is intended to reduce the rate of false positive and false negative exam findings, which may mislead a clinician in achieving an accurate diagnosis. Orthopedic tests are tools to assist clinicians, and the usefulness of these tests is highly variable. While clinical decision-making must be made in the face of uncertainty, we hope this 4-part series will help to provide a framework for clinicians to successfully navigate this process.

List of Abbreviations

ACL = anterior cruciate ligament of the knee

CECS = Chronic Exertional Compartment Syndrome

FABER = abbreviation for flexion, abduction, and external rotation

FAI = Femoroacetabular impingement

IT band = Iliotibial band

LCL = lateral collateral ligament of the knee

LR+ = Positive Likelihood Ratio

LR- = Negative Likelihood Ratio

MCL = medial collateral ligament of the knee

MRI = Magnetic Resonance Imaging

MTSS = Medial Tibial Stress Syndrome

OAR= Ottawa Ankle Rules

OA = Osteoarthritis

PCL = posterior cruciate ligament of the knee

∞ = infinity

Competing Interests

Each of the three authors declare that they have no competing interests related to this work.

Author's Contributions

CBR conceived this project and CBR, EFL, and ZJ each contributed to the literature review and participated in the drafting and revisions of this work. Each of the three authors of this article have met the criteria for authorship.

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List of co-authors involved with this this 4-part series:

- Dr. Rebecca Warnecke: Palmer College of Chiropractic DC student at the time of writing
- Dr. Milad Asefi: practicing doctor of chiropractic
- Dr. Casey Okamoto: practicing doctor of chiropractic
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REFERENCES

1. Pauker SG, Kassirer JP. The threshold approach to clinical decision making. N Engl J Med. 1980 May 15;302(20):1109–17.

2. Cahan A, Gilon D, Manor O, Paltiel O. Probabilistic reasoning and clinical decision-making: do doctors overestimate diagnostic probabilities? QJM. 2003 Oct;96(10):763–9.

3. Mukherjee S. The Laws of Medicine: Field Notes from an Uncertain Science. Simon and Schuster; 2015.

4. McGee S. Simplifying likelihood ratios. J Gen Intern Med. 2002 Aug;17(8):646–9.

5. Byrd JWT, Jones KS. Diagnostic accuracy of clinical assessment, magnetic resonance imaging, magnetic resonance arthrography, and intra-articular injection in hip arthroscopy patients. Am J Sports Med. 2004 Oct;32(7):1668–74.

6. LeBlanc KE, Muncie HL Jr, LeBlanc LL. Hip fracture: diagnosis, treatment, and secondary prevention. Am Fam Physician. 2014 Jun 15;89(12):945–51.

7. Reiman MP, Mather RC 3rd, Cook CE. Physical examination tests for hip dysfunction and injury. Br J Sports Med. 2015 Mar;49(6):357–61.

8. Borgerding LJ, Kikillus PJ, Boissonnault WG. Use of the patellar-pubic percussion test in the diagnosis and management of a patient with a non-displaced hip fracture. J Man Manip Ther. 2007;15(4):E78–84.

9. Shin AY, Morin WD, Gorman JD, Jones SB, Lapinsky AS. The superiority of magnetic resonance imaging in differentiating the cause of hip pain in endurance athletes. Am J Sports Med. 1996 Mar;24(2):168–76.

10. Sutlive TG, Lopez HP, Schnitker DE, Yawn SE, Halle RJ, Mansfield LT, et al. Development of a clinical prediction rule for diagnosing hip osteoarthritis in individuals with unilateral hip pain. J Orthop Sports Phys Ther. 2008 Sep;38(9):542–50.

11. Pacheco-Carrillo A, Medina-Porqueres I. Physical examination tests for the diagnosis of femoroacetabular impingement. A systematic review. Phys Ther Sport. 2016 Sep;21:87–93.

12. Ayeni O, Chu R, Hetaimish B, Nur L, Simunovic N, Farrokhyar F, et al. A painful squat test provides limited diagnostic utility in CAM-type femoroacetabular impingement. Knee Surg Sports Traumatol Arthrosc. 2014 Apr;22(4):806–11.

13. Burgess RM, Rushton A, Wright C, Daborn C. The validity and accuracy of clinical diagnostic tests used to detect labral pathology of the hip: a systematic review. Man Ther. 2011 Aug;16(4):318–26.

14. Martin RL, Enseki KR, Draovitch P, Trapuzzano T, Philippon MJ. Acetabular labral tears of the hip: examination and diagnostic challenges. J Orthop Sports Phys Ther. 2006 Jul;36(7):503–15.

15. Maslowski E, Sullivan W, Forster Harwood J, Gonzalez P, Kaufman M, Vidal A, et al. The diagnostic validity of hip provocation maneuvers to detect intra-articular hip pathology. PM R. 2010 Mar;2(3):174–81.

16. Lustenberger DP, Ng VY, Best TM, Ellis TJ. Efficacy of treatment of trochanteric bursitis: a systematic review. Clin J Sport Med. 2011 Sep;21(5):447–53.

17. Williams BS, Cohen SP. Greater trochanteric pain syndrome: a review of anatomy, diagnosis and treatment. Anesth Analg. 2009 May;108(5):1662–70.

18. Herrington L, Rivett N, Munro S. The relationship between patella position and length of the iliotibial band as assessed using Ober's test. Man Ther. 2006 Aug;11(3):182–6.

19. Woodley SJ, Nicholson HD, Livingstone V, Doyle TC, Meikle GR, Macintosh JE, et al. Lateral hip pain: findings from magnetic resonance imaging and clinical examination. J Orthop Sports Phys Ther. 2008 Jun;38(6):313–28.

20. Byrd JWT. Evaluation of the hip: history and physical examination. N Am J Sports Phys Ther. 2007 Nov;2(4):231–40.

21. Verrall GM, Slavotinek JP, Barnes PG, Fon GT. Description of pain provocation tests used for the diagnosis of sports-related chronic groin pain: relationship of tests to defined clinical (pain and tenderness) and MRI (pubic bone marrow oedema) criteria. Scand J Med Sci Sports. 2005 Feb;15(1):36–42.

22. Martin HD, Kivlan BR, Palmer IJ, Martin RL. Diagnostic accuracy of clinical tests for sciatic nerve entrapment in the gluteal region. Knee Surg Sports Traumatol Arthrosc. 2014 Apr;22(4):882–8.

23. Rodrigue T, Hardy RW. Diagnosis and treatment of piriformis syndrome. Neurosurg Clin N Am. 2001 Apr;12(2):311–9.

24. Foster MR. Piriformis syndrome. Orthopedics. 2002 Aug;25(8):821–5.

25. Filler AG, Haynes J, Jordan SE, Prager J, Villablanca JP, Farahani K, et al. Sciatica of nondisc origin and piriformis syndrome: diagnosis by magnetic resonance neurography and interventional magnetic resonance imaging with outcome study of resulting treatment. J Neurosurg Spine. 2005 Feb;2(2):99–115.

26. Fishman LM, Dombi GW, Michaelsen C, Ringel S, Rozbruch J, Rosner B, et al. Piriformis syndrome: diagnosis, treatment, and outcome--a 10-year study. Arch Phys Med Rehabil. 2002 Mar;83(3):295–301.

27. Eckard TG, Padua DA, Dompier TP, Dalton SL, Thorborg K, Kerr ZY. Epidemiology of Hip Flexor and Hip Adductor Strains in National Collegiate Athletic Association Athletes, 2009/2010-2014/2015. Am J Sports Med. 2017 Oct;45(12):2713–22.

28. Mills M, Frank B, Goto S, Blackburn T, Cates S, Clark M, et al. Effect of restricted hip flexor muscle length on hip extensor muscle activity and lower extremity biomechanics in college-aged female soccer players. Int J Sports Phys Ther. 2015 Dec;10(7):946–54.

29. Cook C, Hegedus E. Orthopedic Physical Examination Tests: Pearson New International Edition: An Evidence-Based Approach. Pearson Higher Ed; 2013. 552 p.

30. Marks MC, Alexander J, Sutherland DH, Chambers HG. Clinical utility of the Duncan-Ely test for rectus femoris dysfunction during the swing phase of gait. Dev Med Child Neurol. 2003 Nov;45(11):763–8.

31. Vigotsky AD, Lehman GJ, Beardsley C, Contreras B, Chung B, Feser EH. The modified Thomas test is not a valid measure of hip extension unless pelvic tilt is controlled. PeerJ. 2016 Aug 11;4:e2325.

32. Lucas CA. Iliotibial band friction syndrome as exhibited in athletes. J Athl Train. 1992;27(3):250–2.

33. Rosenthal MD. Clinical testing for extra-articular lateral knee pain. A modification and combination of traditional tests. N Am J Sports Phys Ther. 2008 May;3(2):107–9.

34. Khaund R, Flynn SH. Iliotibial band syndrome: a common source of knee pain. Am Fam Physician. 2005 Apr 15;71(8):1545–50.

35. Hyde TE, Gengenbach MS. Conservative Management of Sports Injuries. Jones & Bartlett Learning; 2007. 1173 p.

36. Jackson JL, O'Malley PG, Kroenke K. Evaluation of acute knee pain in primary care. Ann Intern Med. 2003 Oct 7;139(7):575–88.

37. Bachmann LM, Haberzeth S, Steurer J, ter Riet G. The accuracy of the Ottawa knee rule to rule out knee fractures: a systematic review. Ann Intern Med. 2004 Jan 20;140(2):121–4.

38. Stanitski CL, Harvell JC, Fu F. Observations on acute knee hemarthrosis in children and adolescents. J Pediatr Orthop. 1993 Jul;13(4):506–10.

39. Hagino T, Ochiai S, Senga S, Yamashita T, Wako M, Ando T, et al. Meniscal tears associated with anterior cruciate ligament injury. Arch Orthop Trauma Surg. 2015 Dec;135(12):1701–6.

40. Akseki D, Ozcan O, Boya H, Pinar H. A new weight-bearing meniscal test and a comparison with McMurray's test and joint line tenderness. Arthroscopy. 2004 Nov;20(9):951–8.

41. Lowery DJ, Farley TD, Wing DW, Sterett WI, Steadman JR. A clinical composite score accurately detects meniscal pathology. Arthroscopy. 2006 Nov;22(11):1174–9.

42. Miao Y, Yu J-K, Ao Y-F, Zheng Z-Z, Gong X, Leung KKM. Diagnostic values of 3 methods for evaluating meniscal healing status after meniscal repair: comparison among second-look arthroscopy, clinical assessment, and magnetic resonance imaging. Am J Sports Med. 2011 Apr;39(4):735–42.

43. Jaddue DAK, Tawfiq FH, Sayed-Noor AS. The utility of clinical examination in the diagnosis of medial meniscus injury in comparison with arthroscopic findings. Eur J Orthop Surg Traumatol. 2010;20(5):389–92.

44. Pookarnjanamorakot C, Korsantirat T, Woratanarat P. Meniscal lesions in the anterior cruciate insufficient knee: the accuracy of clinical evaluation. J Med Assoc Thai. 2004 Jun;87(6):618–23.

45. Jerosch J, Riemer S. [How good are clinical investigative procedures for diagnosing meniscus lesions?]. Sportverletz Sportschaden. 2004 Jun;18(2):59–67.

46. Fowler PJ, Lubliner JA. The predictive value of five clinical signs in the evaluation of meniscal pathology. Arthroscopy. 1989;5(3):184–6.

47. Mirzatolooei F, Yekta Z, Bayazidchi M, Ershadi S, Afshar A. Validation of the Thessaly test for detecting meniscal tears in anterior cruciate deficient knees. Knee. 2010 Jun;17(3):221–3.

48. Harrison BK, Abell BE, Gibson TW. The Thessaly test for detection of meniscal tears: validation of a new physical examination technique for primary care medicine. Clin J Sport Med. 2009 Jan;19(1):9–12.

49. Kurosaka M, Yagi M, Yoshiya S, Muratsu H, Mizuno K. Efficacy of the axially loaded pivot shift test for the diagnosis of a meniscal tear. Int Orthop. 1999;23(5):271–4.

50. Mariani PP, Adriani E, Maresca G, Mazzola CG. A prospective evaluation of a test for lateral meniscus tears. Knee Surg Sports Traumatol Arthrosc. 1996;4(1):22–6.

51. Gornitzky AL, Lott A, Yellin JL, Fabricant PD, Lawrence JT, Ganley TJ. Sport-Specific Yearly Risk and Incidence of Anterior Cruciate Ligament Tears in High School Athletes: A Systematic Review and Meta-analysis. Am J Sports Med. 2016 Oct;44(10):2716–23.

52. Agel J, Arendt EA, Bershadsky B. Anterior cruciate ligament injury in national collegiate athletic association basketball and soccer: a 13-year review. Am J Sports Med. 2005 Apr;33(4):524–30.

53. Arendt EA, Agel J, Dick R. Anterior cruciate ligament injury patterns among collegiate men and women. J Athl Train. 1999 Apr;34(2):86–92.

54. Benjaminse A, Gokeler A, van der Schans CP. Clinical diagnosis of an anterior cruciate ligament rupture: a meta-analysis. J Orthop Sports Phys Ther. 2006 May;36(5):267–88.

55. Mulligan EP, Harwell JL, Robertson WJ. Reliability and diagnostic accuracy of the Lachman test performed in a prone position. J Orthop Sports Phys Ther. 2011 Oct;41(10):749–57.

56. Cross MJ, Schmidt DR, Mackie IG. A no-touch test for the anterior cruciate ligament. J Bone Joint Surg Br. 1987 Mar;69(2):300.

57. Ostrowski JA. Accuracy of 3 diagnostic tests for anterior cruciate ligament tears. J Athl Train. 2006 Jan;41(1):120–1.

58. Larson RL. Physical examination in the diagnosis of rotatory instability. Clin Orthop Relat Res. 1983 Jan;(172):38–44.

59. Jarbo KA, Hartigan DE, Scott KL, Patel KA, Chhabra A. Accuracy of the Lever Sign Test in the Diagnosis of Anterior Cruciate Ligament Injuries. Orthop J Sports Med. 2017 Oct;5(10):2325967117729809.

60. al-Duri Z. Relation of the fibular head sign to other signs of anterior cruciate ligament insufficiency. A follow-up letter to the editor. Clin Orthop Relat Res. 1992 Feb;(275):220–5.

61. Wagemakers HP, Luijsterburg PA, Boks SS, Heintjes EM, Berger MY, Verhaar JA, et al. Diagnostic accuracy of history taking and physical examination for assessing anterior cruciate ligament lesions of the knee in primary care. Arch Phys Med Rehabil. 2010 Sep;91(9):1452–9.

62. Amis AA, Gupte CM, Bull AMJ, Edwards A. Anatomy of the posterior cruciate ligament and the meniscofemoral ligaments. Knee Surg Sports Traumatol Arthrosc. 2006 Mar;14(3):257–63.

63. Wang C-J. Injuries to the posterior cruciate ligament and posterolateral instabilities of the knee. Chang Gung Med J. 2002 May;25(5):288–97.

64. Roberts DM, Stallard TC. Emergency department evaluation and treatment of knee and leg injuries.

Emerg Med Clin North Am. 2000 Feb;18(1):67–84, v – vi.

65. Rubinstein RA Jr, Shelbourne KD, McCarroll JR, VanMeter CD, Rettig AC. The accuracy of the clinical examination in the setting of posterior cruciate ligament injuries. Am J Sports Med. 1994 Jul;22(4):550–7.

66. Feltham GT, Albright JP. The diagnosis of PCL injury: literature review and introduction of two novel tests. Iowa Orthop J. 2001;21:36–42.

67. Hughston JC, Norwood LA Jr. The posterolateral drawer test and external rotational recurvatum test for posterolateral rotatory instability of the knee. Clin Orthop Relat Res. 1980 Mar;(147):82–7.

68. Loomer RL. A test for knee posterolateral rotatory instability. Clin Orthop Relat Res. 1991 Mar;(264):235–8.

69. Stäubli HU, Jakob RP. Posterior instability of the knee near extension. A clinical and stress radiographic analysis of acute injuries of the posterior cruciate ligament. J Bone Joint Surg Br. 1990 Mar;72(2):225–30.

70. Loos WC, Fox JM, Blazina ME, Del Pizzo W, Friedman MJ. Acute posterior cruciate ligament injuries. Am J Sports Med. 1981 Mar;9(2):86–92.

71. Fowler PJ, Messieh SS. Isolated posterior cruciate ligament injuries in athletes. Am J Sports Med. 1987 Nov;15(6):553–7.

72. Bae JH, Choi IC, Suh SW, Lim HC, Bae TS, Nha KW, et al. Evaluation of the reliability of the dial test for posterolateral rotatory instability: a cadaveric study using an isotonic rotation machine. Arthroscopy. 2008 May;24(5):593–8.

73. Ferrari DA, Ferrari JD, Coumas J. Posterolateral instability of the knee. J Bone Joint Surg Br. 1994 Mar;76(2):187–92.

74. Souza TA. Differential Diagnosis and Management for Chiropractors. Jones & Bartlett Publishers; 2016.

75. Kastelein M, Wagemakers HPA, Luijsterburg PAJ, Verhaar JAN, Koes BW, Bierma-Zeinstra SMA. Assessing medial collateral ligament knee lesions in general practice. Am J Med. 2008 Nov;121(11):982–8.e2.

76. Harilainen A. Evaluation of knee instability in acute ligamentous injuries. Ann Chir Gynaecol. 1987;76(5):269–73.

77. Harilainen A, Myllynen P, Rauste J, Silvennoinen E. Diagnosis of acute knee ligament injuries: the value of stress radiography compared with clinical examination, stability under anaesthesia and arthroscopic or operative findings. Ann Chir Gynaecol. 1986;75(1):37–43.

78. Sanchis-Alfonso V. Holistic approach to understanding anterior knee pain. Clinical implications. Knee Surg Sports Traumatol Arthrosc. 2014 Oct;22(10):2275–85.

79. Powers CM, Bolgla LA, Callaghan MJ, Collins N, Sheehan FT. Patellofemoral pain: proximal, distal, and local factors, 2nd International Research Retreat. J Orthop Sports Phys Ther. 2012 Jun;42(6):A1–

54.

80. Cook C, Hegedus E, Hawkins R, Scovell F, Wyland D. Diagnostic accuracy and association to disability of clinical test findings associated with patellofemoral pain syndrome. Physiother Can. 2010 Feb 22;62(1):17–24.

81. Nijs J, Van Geel C, Van der auwera C, Van de Velde B. Diagnostic value of five clinical tests in patellofemoral pain syndrome. Man Ther. 2006 Feb;11(1):69–77.

82. Elton K, McDonough K, Savinar-Nogue E, Jensen GM. A Preliminary Investigation: History, Physical, and lsokinetic Exam Results versus Arthroscopic Diagnosis of Chondromalacia Patella *. J Orthop Sports Phys Ther. 1985;7(3):115–23.

83. Haim A, Yaniv M, Dekel S, Amir H. Patellofemoral pain syndrome: validity of clinical and radiological features. Clin Orthop Relat Res. 2006 Oct;451:223–8.

84. Ahmad CS, McCarthy M, Gomez JA, Shubin Stein BE. The moving patellar apprehension test for lateral patellar instability. Am J Sports Med. 2009 Apr;37(4):791–6.

85. Nissen CW, Cullen MC, Hewett TE, Noyes FR. Physical and arthroscopic examination techniques of the patellofemoral joint. J Orthop Sports Phys Ther. 1998 Nov;28(5):277–85.

86. Kim S-J, Lee D-H, Kim T-E. The relationship between the MPP test and arthroscopically found medial patellar plica pathology. Arthroscopy. 2007 Dec;23(12):1303–8.

87. Magee DJ. Orthopedic Physical Assessment. Elsevier Health Sciences; 2008. 1138 p.

88. Cook JL, Khan KM, Kiss ZS, Purdam CR, Griffiths L. Reproducibility and clinical utility of tendon palpation to detect patellar tendinopathy in young basketball players. Victorian Institute of Sport tendon study group. Br J Sports Med. 2001 Feb;35(1):65–9.

89. Conrad JM, Stanitski CL. Osteochondritis dissecans: Wilson's sign revisited. Am J Sports Med. 2003 Sep;31(5):777–8.

90. Reid DC. Sports Injury, Assessment and Rehabilitation. Med Sci Sports Exercise. 1993;25(10):i.

91. McAfee JH, Smith DL. Olecranon and prepatellar bursitis. Diagnosis and treatment. West J Med. 1988 Nov;149(5):607–10.

92. Kumar D, Alvand A, Beacon JP. Impingement of infrapatellar fat pad (Hoffa's disease): results of high-portal arthroscopic resection. Arthroscopy. 2007 Nov;23(11):1180–6.e1.

93. Morini G, Chiodi E, Centanni F, Gattazzo D. [Hoffa's disease of the adipose pad: magnetic resonance versus surgical findings]. Radiol Med. 1998 Apr;95(4):278–85.

94. Reshef N, Guelich DR. Medial tibial stress syndrome. Clin Sports Med. 2012 Apr;31(2):273–90.

95. Edwards PH Jr, Wright ML, Hartman JF. A practical approach for the differential diagnosis of chronic leg pain in the athlete. Am J Sports Med. 2005 Aug;33(8):1241–9.

96. Winters M, Bakker EWP, Moen MH, Barten CC, Teeuwen R, Weir A. Medial tibial stress syndrome can be diagnosed reliably using history and physical examination. Br J Sports Med [Internet]. 2017 Feb 8; Available from: http://dx.doi.org/10.1136/bjsports-2016-097037

97. Vajapey S, Miller TL. Evaluation, diagnosis, and treatment of chronic exertional compartment syndrome: a review of current literature. Phys Sportsmed. 2017 Nov;45(4):391–8.

98. Tucker AK. Chronic exertional compartment syndrome of the leg. Curr Rev Musculoskelet Med. 2010 Sep 2;3(1-4):32–7.

99. Behrens SB, Deren ME, Matson A, Fadale PD, Monchik KO. Stress fractures of the pelvis and legs in athletes: a review. Sports Health. 2013 Mar;5(2):165–74.

100. van der Velde GM, Hsu WS. Posterior tibial stress fracture: a report of three cases. J Manipulative Physiol Ther. 1999 Jun;22(5):341–6.

101. Lesho EP. Can tuning forks replace bone scans for identification of tibial stress fractures? Mil Med. 1997 Dec;162(12):802–3.

102. Schneiders AG, Sullivan SJ, Hendrick PA, Hones BDGM, McMaster AR, Sugden BA, et al. The ability of clinical tests to diagnose stress fractures: a systematic review and meta-analysis. J Orthop Sports Phys Ther. 2012 Sep;42(9):760–71.

103. Levine AM, Kolker D. Ankle and Foot Injuries. In: Essential Sports Medicine. 2008. p. 146–68.

104. Croy T, Koppenhaver S, Saliba S, Hertel J. Anterior talocrural joint laxity: diagnostic accuracy of the anterior drawer test of the ankle. J Orthop Sports Phys Ther. 2013 Dec;43(12):911–9.

105. Fong DT, Chan Y-Y, Mok K-M, Yung PS, Chan K-M. Understanding acute ankle ligamentous sprain injury in sports. Sports Med Arthrosc Rehabil Ther Technol. 2009 Jul 30;1:14.

106. Bachmann LM, Kolb E, Koller MT, Steurer J, ter Riet G. Accuracy of Ottawa ankle rules to exclude fractures of the ankle and mid-foot: systematic review. BMJ. 2003 Feb 22;326(7386):417.

107. Stiell IG, McKnight RD, Greenberg GH, McDowell I, Nair RC, Wells GA, et al. Implementation of the Ottawa ankle rules. JAMA. 1994 Mar 16;271(11):827–32.

108. Molinari A, Stolley M, Amendola A. High ankle sprains (syndesmotic) in athletes: diagnostic challenges and review of the literature. Iowa Orthop J. 2009;29:130–8.

109. Molloy S, Solan MC, Bendall SP. Synovial impingement in the ankle. A new physical sign. J Bone Joint Surg Br. 2003 Apr;85(3):330–3.

110. Schwieterman B, Haas D, Columber K, Knupp D, Cook C. Diagnostic accuracy of physical examination tests of the ankle/foot complex: a systematic review. Int J Sports Phys Ther. 2013 Aug;8(4):416–26.

111. Reiman M, Burgi C, Strube E, Prue K, Ray K, Elliott A, et al. The utility of clinical measures for the diagnosis of achilles tendon injuries: a systematic review with meta-analysis. J Athl Train. 2014 Nov;49(6):820–9.

112. McCormick JJ, Anderson RB. Turf toe: anatomy, diagnosis, and treatment. Sports Health. 2010 Nov;2(6):487–94.

113. Sault JD, Morris MV, Jayaseelan DJ, Emerson-Kavchak AJ. Manual therapy in the management of a patient with a symptomatic Morton's Neuroma: A case report. Man Ther. 2016;21:307–10.

114. Lopes AD, Hespanhol Júnior LC, Yeung SS, Costa LOP. What are the main running-related musculoskeletal injuries? A Systematic Review. Sports Med. 2012 Oct 1;42(10):891–905.

115. Muth CC. Plantar Fasciitis. JAMA. 2017 Jul 25;318(4):400.

116. Cassas KJ, Cassettari-Wayhs A. Childhood and adolescent sports-related overuse injuries. Am Fam Physician. 2006 Mar 15;73(6):1014–22.

117. Kinoshita M, Okuda R, Morikawa J, Jotoku T, Abe M. The dorsiflexion-eversion test for diagnosis of tarsal tunnel syndrome. J Bone Joint Surg Am. 2001 Dec;83-A(12):1835–9.

118. Roddy E, Zhang W, Doherty M. Validation of a self-report instrument for assessment of hallux valgus. Osteoarthritis Cartilage. 2007 Sep;15(9):1008–12.

119. Menz HB, Fotoohabadi MR, Wee E, Spink MJ. Validity of self-assessment of hallux valgus using the Manchester scale. BMC Musculoskelet Disord. 2010 Sep 20;11:215.

120. Menz HB, Munteanu SE. Radiographic validation of the Manchester scale for the classification of hallux valgus deformity. Rheumatology . 2005 Aug;44(8):1061–6.

121. Mahadevan D, Venkatesan M, Bhatt R, Bhatia M. Diagnostic Accuracy of Clinical Tests for Morton's Neuroma Compared With Ultrasonography. J Foot Ankle Surg. 2015 Jul;54(4):549–53.

122. Cloke DJ, Greiss ME. The digital nerve stretch test: A sensitive indicator of Morton's neuroma and neuritis. Foot Ankle Surg. 2006;12(4):201–3.

123. De Garceau D, Dean D, Requejo SM, Thordarson DB. The association between diagnosis of plantar fasciitis and Windlass test results. Foot Ankle Int. 2003 Mar;24(3):251–5.

124. Perhamre S, Lazowska D, Papageorgiou S, Lundin F, Klässbo M, Norlin R. Sever's injury: a clinical diagnosis. J Am Podiatr Med Assoc. 2013 Sep;103(5):361–8.

Original Article

Resolution of Post-Surgical Low Back Pain in a Patient with Chronic Cauda Equina Syndrome: A Case Study

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ABSTRACT

Objective: The purpose of this report is to describe the management of a patient experiencing post-surgical low back pain, secondary to a corrective thoracolumbar surgical fusion, as a consequence of traumatically-induced chronic cauda equina syndrome.

Clinical Features: A 59-year-old male with low back pain, buttock pain, anal hyperesthesia constipation, and urinary retention presented on consult for consideration of chiropractic care for low back pain. This patient was involved in a motorcycle collision 18-months prior, which resulted in a burst fracture of the first lumbar vertebral body, requiring corpectomy of L1 and spinal fusion T12-L2. This patient continued to have low back pain, chronic cauda equina syndrome with neurogenic bladder and perianal hyperesthesia following the spinal fusion. His residual neurologic defects were considered stable by his neurologist and primary care providers.

Intervention and Outcome: Care consisted of two separate trials of chiropractic care, totaling 13 visits over a 4-month period. The care provided to this patient included graded lumbosacral mobilization, thoracic, lumbar and sacral spinal manipulation, moist heat, and rehabilitation exercises. Following this course of care, the patient reported improvement of his low back pain.

Conclusion: In this case, management of post-surgical low back pain with concomitant chronic cauda equina syndrome responded favorably to a course of chiropractic care, over two separate trials, including spinal manipulation therapy.

INTRODUCTION

The use of lumbar spinal manipulation and spinal rehabilitation for the management of low back pain is not well documented for patients with concomitant chronic cauda equina syndrome (CES). The authors of this report were able to identify only a single case report involving spinal manipulation for the management of low back pain (LBP) in a patient with chronic CES (1).

In patients experiencing acute CES symptomatology, manipulation is an absolute contraindication (2,3). Post-surgically, chronic CES is known to be associated with back pain and neurologic deficits (4). Cauda equina syndrome may involve LBP, radicular pain in the lower extremities, saddle anesthesia, urinary incontinence, or lower extremity motor and/or sensory deficits (4,5). Acute CES is a medical emergency, however there is controversy regarding the risk of immediate (within 48 hours) versus delayed surgical intervention, as surgery in an emergency setting may involve suboptimal results and prolonged morbidity (6).

CASE STUDY

A 59-year-old male with low back pain of 18-months duration referred on consult by his primary care provider (PCP) for chiropractic care. This patient experienced a motorcycle accident 18-months prior which resulted in a burst fracture of the first lumbar vertebral body (L1), necessitating emergency L1 corpectomy and T12-L2 fusion.

Following surgery, the patient was diagnosed with residual chronic CES and neurogenic bladder, which necessitated the need for intermittent self-catheterization to void his bladder. The patient also reported perianal hyperesthesia, which remained unchanged since the time of the surgery and was primarily noticeable while wiping after passing a bowel movement. This patient was able to defecate on his own via Valsalva maneuver on a 1-hour postprandial schedule. He also reported transient radicular pain, extending to his feet, while performing the Valsalva maneuver while voiding his bowels. No other lower extremity symptoms were noted by the patient.

This patient's low back pain was described as a constant, "sharp ache" over the lumbosacral region and was worse with all end-range lumbosacral ranges of motion. The patient rated his average pain as a 4/10 on a Numeric Pain Rating Scale (NPRS) with range from 2/10 to 6/10, depending on provocative and palliative factors. The patient completed the NPRS, but refused to complete any other outcome measures. Patient described that his pain was relieved while laying down supine, with moist heat (e.g. with a hot tub), but the pain was worsened during any activity that involved prolonged lumbar flexion (e.g. sitting).

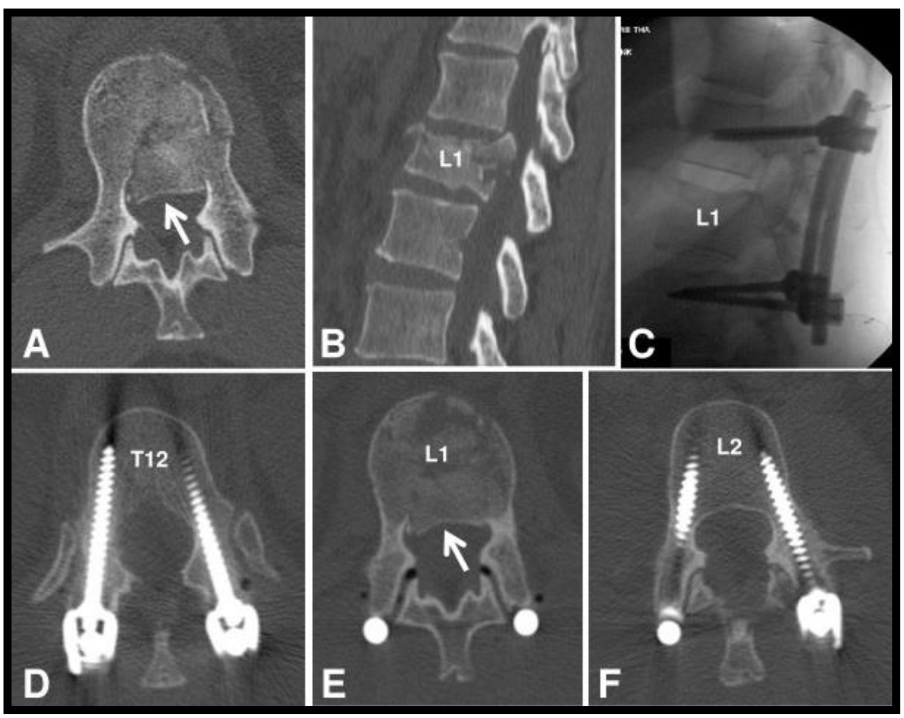
The patient had received chiropractic care for LBP previous to this injury, but denied any chiropractic care since his accident and surgery 18 months ago. The patient also underwent physical therapy for the management of his low back pain, previous to this injury, for approximately 2-3 months. He noted this trial of physical therapy failed to provide significant relief.

The patient's past medical history and surgical history, prior to the accident in question and subsequent surgery, are non-contributory with no noted prior injuries, surgeries, injections, or procedures related to the lumbar spine other than the motorcycle injury noted and subsequent surgery.

The patient is married with children and he enjoys regularly performing light home construction. He drinks socially and used recreational marijuana to improve his mood. Functionally, the patient reported an ability to walk a mile, and denied any difficulty climbing the stairs of his 3-story home. He utilized a cane while ambulating to assist with balance, which he notes is difficult at times due to loss of strength and coordination.

The patient underwent standard post-surgical lumbar spine x-ray examination, which revealed successful orthopedic fusion of T12-L2 along with generalized lumbar spinal degenerative joint disease (see Figure 1).

Figure 1: Computed tomography images of patient's surgical repair



The physical examination revealed a 12-inch surgical scar over the patient's left thoracolumbar paravertebral gutter. Lumbosacral active range of motion were decreased by approximately 20%, in all directions with no description of "pain" noted and only "soreness" if continued movement pushing end-ranges. The patient reported pain following palpation of his lumbosacral paravertebral muscles and moderate hypertonicity was noted, bilaterally. Bilateral lower extremity manual muscle testing bilaterally graded as 4-4+/5, including extensor hallucis longus with only exception plantar flexion graded 4/5 bilaterally. Deep tendon reflexes were rated as 3+ on the right quadriceps tendon, 2+ on the left quadriceps tendon, 0 on Achilles tendon bilaterally with reinforcement. Babinski's test was negative, bilaterally. Bilateral hypoesthesia to pinwheel and light touch was noted along the posterior calves (S1/S2 dermatomes). No other sensory deficits were detected in the regions of the L1-L5 dermatomes. Perianal hyperesthesia was reported by the patient and was not examined, per patient's request. Seated and supine Straight-Leg Raise tests were negative, bilaterally, with note of localized low back pain when the legs were raised above 45 degrees. When the Valsalva maneuver was performed that patient denied lower extremity symptoms (i.e. pain or paresthesia). The patient was hesitant to perform an aggressive Valsalva maneuver, due to a fear of fecal incontinence during the exam. On evaluation patient also continues to report a history of low back pain and diffuse leg paraesthesias while straining to void his bowels since accident (i.e. positive Dejerine's triad).

The patient was diagnosed as follows: 1) status-post corpectomy and thoracolumbar fusion T12-L2 with chronic CES and neurogenic bladder, 2) chronic low-back pain with myofascial contribution as sequelae of injury, subsequent surgical intervention and deconditioning, and 3) lumbar degenerative joint disease, per the radiology report.

INTERVENTION

Care consisted of two separate trials of care totaling 13 chiropractic visits over a 4-month period. The first trial of care involved 6-visits and care was extended past the first trial of care, based on improved LBP. Therapies included graded lumbosacral mobilization, thoracic, lumbar and sacral spinal manipulation, moist heat, and rehabilitation exercises. Treatment began with graded mobilization over the lumbosacral area (sacral extension) while prone, high velocity-low amplitude (HVLA) chiropractic spinal manipulation (diversified technique) was applied to the mid-thoracic spine along with hydrocollator therapy for 10-minutes, during each visit. As a safely measure, pre-manipulative positional stress was utilized and symptoms were assessed prior to each treatment. Rehabilitation exercises began with supine single-leg, knee to chest stretching for 30 seconds in 3 separate positions (same shoulder, opposite shoulder and across waist).

Improvement in low back pain was noted immediately post-treatment at each visit. After each of the first 3 visits, the patient reported lasting improvement from his LBP. Drop table chiropractic manipulation therapy was applied to the patient's lumbosacral area midway into initial trial of care (3rd visit), as was the introduction of Quadruped Reach (also known as "Bird Dog"), Side Bridge and Curl-up exercises (also known as McGill's Big 3). These were chosen as they are considered to be safe exercises for low back pain patients (8,9). The patient continued to respond positively and had no difficulties understanding or performing his home-based exercises. Handouts were dispensed detailing his home exercises instructions with progressions. He was instructed to progress when he was able to perform 3 sets of 12-15 repetitions with good control of movement, performed and demonstrated on follow-ups visits at clinic, without worsening of pain following performance of the exercises. The patient was instructed that mild post-exercise soreness may be experiences and is considered to be a normal side-effect of this activity. Side bridge was instructed initially at 8-10 slow repetitions with 2 breath holds in the elevated position done 1-2 times daily. Quadruped Reach was instructed initially with 3-4 repetitions, which were easily performed and demonstrated in-office. These exercises were progressed, on initial visit, to 10-12 repetitions with 5-6 second hold, 1-2x daily. The patient was instructed to perform Curl-Up exercise for 12 repetitions with a 2-breath hold, 1-2 times daily. All exercise progressions were based on the patient's ability to achieve a minimum of 2 times daily with adequate comfort and good control as assessed by clinician. On the patient's 6th visit, he was shown an eyes-open, one-legged standing balance exercise. Initially he could not do this for more than 2-3 seconds on either leg without a major correction or having to touch other foot to ground due to poor balance. No pain, weakness or fear-avoidance was noted on performance of this exercise. Of note, improving balance was a patient-specific goal of care as he noted he would like to sail comfortably on his sail boat and he was avoiding this, due to his imbalance issues.

Lumbosacral mobilization progressed to HVLA side-posture spinal manipulation (diversified technique) during final two visits in initial trial. Manipulation targeted the L4-S1 segments, with no adverse events reported. The patient noted a preference and greater pain relief from the drop-assisted spinal manipulation, subsequent visits continued utilization of drop table technique to the lumbosacral spine. Due to success with pain control and home exercise intervention, the patient was offered, and declined, a physical therapy consultation for a more focused progression and monitoring of home exercise plan.

The patient did not attend his follow-up re-examination and lost to follow-up, until he presented as a walk-in 6-months later requesting care as need for exacerbations. On this visit he rated his LBP as a 3/10, following recent snow shoveling and flare of LBP. He reported that he had intentionally missed his post-trial follow-up appointment as his LBP had resolved and he "felt great and didn't feel he needed care."

On his return following a 6-month absence, the patient presented without the need of a cane for ambulatory assistance and reported rarely using his cane. He denied any LBP for the last 6-months, rated as 0/10 NPRS, until this recent exacerbation as noted above. He further stated that he was able to continue to build his house remaining very active, “more active than ever”, also that he had taken up sailing again. He reported that his chronic CES symptoms were unchanged and he continued to experience perianal hyperesthesia and continued to require urinary catheterization and complies with follow-up evaluations from his neurologist to monitor his chronic CES.

Currently, patient continues to follow-up on an as-needed basis for the management of his LBP exacerbations. He has been advised to continue his activity level and other activities of daily living and was instructed to continue his home exercises, as instructed. He declined any other referrals or intervention and was happy to continue his home-based exercises.

DISCUSSION

This case is notable as cauda-equina syndrome (CES) represents a relative contraindication and when acute a medical emergency. There are few case reports noting the multimodal care chiropractors generally deliver in practice, along with spinal manipulative therapy involving these conditions. This case describes the successful chiropractic treatment of 18-month chronic low-back pain in a post-surgical patient with chronic cauda-equina syndrome. Multimodal care consisted of graded lumbar mobilization, spinal manipulation, moist heat, and home-based lumbar stability exercises.

There were no adverse events reported throughout this course of care and spinal manipulation appeared to be safe and efficacious in the long-term management strategy for this patient’s LBP with concomitant chronic CES. In this specific case, multimodal conservative chiropractic treatment including spinal manipulation, resolved this patient’s low back pain.

Clinicians should be aware of the absolute contraindication of spinal manipulative therapy in patients with acute CES. However, awareness of only acute CES guidelines and recommendations may prevent referral of patients with chronic CES and spinal pain complaints for conservative management, such as spinal manipulative therapy, spinal rehabilitation exercises and other conservative measures.

LIMITATIONS

As a retrospective study, this case involves some limitations. First, only a NPRS and the patient’s subjective interpretation of increased function were assessed as outcomes. The patient’s response to care would have be better evaluated using additional outcomes assessments, particularly an outcome measure involving the patient’s functional status. Secondly, although mobilization and two chiropractic techniques were used (drop table and diversified side-posture HVLA) there can be no statement on efficacy of those other than the stated individual patient preference. No generalized effectiveness can be gained from this type of individual case-report.

CONCLUSION

Select patients with chronic CES may benefit from spinal manipulative therapy for the management of LBP. However, further research in the form more rigorous testing, beyond case reports, is needed to establish the safety and efficacy of this approach to care.

While the effectiveness and safety of this management strategy is not able to be applied to the general population from this report, it does provide some insight into the clinical management of patients with chronic CES via conservative care. It appears spinal manipulation therapy and spinal rehabilitation exercises may be considered as a viable consideration in the treatment of spinal pain complaints among select patients with chronic CES.

REFERENCES

1. Lisi A, Bhardwaj M. Chiropractic High-Velocity Low-Amplitude Spinal Manipulation in the Treatment of A Case of Postsurgical Chronic Cauda Equina Syndrome. J Manipulative Physiol Ther 2004; 27:574-578.
2. Haldeman S, Chapman-Smith D, Peterson D, editors. Guidelines for chiropractic quality assurance and practice parameters. Gaithersburg, MD: Aspen Publishers; 1993. p. 175.
3. Bigos S, Bowyer O, Braen G, et al. Acute low back problems in adults. Clinical practice guideline #14. AHCPR Publication #95-0642. Rockville, MD: Agency for Health Care Policy and Research, Public Health Service, U.S. Department of Health and Human Services; 1994.
4. Memmo PA, Nadler S, Malanga S. Lumbar disc herniations: a review of surgical and non-surgical indications and outcomes. J Back Musculoskel Rehab 2000;14:79-88.
5. Shapiro S, Kostuik JP. Medical realities of cauda equina syndrome secondary to lumbar disc herniation. Spine 2000;25:348-352.
6. Gleave JR, MacFarlane R. Cauda equina syndrome: what is the relationship between timing of surgery and outcome? Br J Neurosurg 2002;16:325-8.
7. Ahn UM, Ahn NU, Buchowski JM, Garrett ES, Sieber AN, Kotsuik JP. Cauda equina syndrome secondary to lumbar disc herniation: a meta-analysis of surgical outcomes. Spine 2000;25:1515-22
8. Axler, C. and McGill, S.M. (1997). Low back loads over a variety of abdominal exercises: Searching for the safest abdominal challenge, Med.Sci.Sports.Ex. 29(6): 804-811.
9. Kavcic, N., Grenier, S.G., and McGill, S.M. (2004a) Determining tissue loads and spine stability while performing commonly prescribed stabilization exercises. Spine, 29(11): 1254-1265.

Editorial Review

Biological Treatment Approaches for Degenerative Disk Disease: A Literature Review of In Vivo Animal and Clinical Data

Yu Moriguchi, Marjan Alimi, Thamina Khair, George Manolarakis, Connor Berlin, Lawrence J. Bonassar, Roger Härtl

Global Spine J 2016;6:497–518

JACO Editorial Reviewer: Gregory C. Priest, DC, FACO

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AUTHORS' ABSTRACT

Study Design: Literature review

Objective: Degenerative disk disease (DDD) has a negative impact on quality of life and is a major cause of morbidity worldwide. There has been a growing interest in the biological repair of DDD by both researchers and clinicians alike. To generate an overview of the recent progress in reparative strategies for the treatment of DDD highlighting their promises and limitations, a comprehensive review of the current literature was performed elucidating data from in vivo animal and clinical studies.

Methods: Articles and abstracts available in electronic databases of PubMed, Web of Science, and Google Scholar as of December 2014 were reviewed. Additionally, data from unpublished, ongoing clinical trials was retrieved from clinicaltrials.gov and available abstracts from research forums. Data was extracted from the most recent in vivo animal or clinical studies involving any of the following: (1) treatment with biomolecules, cells, or tissue-engineered constructs and (2) annulus fibrosus repair.

Results: Seventy-five articles met the inclusion criteria for review. Among these, 17 studies involved humans; 37, small quadrupeds; and 21, large quadrupeds. Findings from all treatments employed demonstrated improvement either in regenerative capacity or in pain attenuation, with the exception of one clinical study.

Conclusion: Published clinical studies on cell therapy have reported encouraging results in the treatment of DDD and resultant back pain. We expect new data to emerge in the near future as treatments for DDD continue to evolve in parallel to our greater understanding of disk health and pathology.

Keywords: Intervertebral disk, disk regeneration, back pain, growth factor, cell therapy, platelet-rich plasma, tissue engineering, annular repair

Clinical Relevance

The prevalence of degenerative disk disease is ubiquitous, reportedly occurring in more than 90% of people over 50 years of age. Degenerative disk disease can result in chronic back pain and frequently results in surgical intervention in an attempt to alleviate the related pain and disability. Unfortunately, the surgery itself may result in altered spinal biomechanics with secondary adjacent segment degeneration. Current treatment options do not adequately address the actual diseased tissue, the degenerated disk. This robustly-referenced literature review explores biologic treatment options that may hold promise for direct treatment of the degenerated disk.

JACO Editorial Summary:

- The intervertebral disk (IVD) is a complex of three tissues: the nucleus pulposus (NP), the annulus fibrosus (AF), and the cartilaginous end plates.
- Degenerative disk disease is a multifactorial process that involves one or more of the three tissues comprising the IVD complex.
- Intervertebral disk degeneration is said to occur in 40% of persons younger than 30 years of age, and in more than 90% of those older than 50.
- Degenerative disk disease (DDD) can lead to chronic lower back pain and disability.
- Surgical intervention for DDD is reported to be performed in nearly 4 million patients worldwide.
- Spinal fusion surgery presents risks for pseudarthrosis and adjacent segment degeneration, resulting in higher rates of reoperation.
- This literature review evaluates in vivo animal and clinical data, with strong inclusion/exclusion criteria, and gives consideration to published data as well as data from ongoing, unpublished clinical studies.
- Biological repair of the degenerated IVD are classified into three categories: biomolecular therapy, cell therapy, and tissue-engineered IVD construction. These treatment strategies are further stratified into categories specific to the stage of degeneration.
- The goal of biologic treatment of the degenerated IVD is to repair/reconstruct the actual diseased tissue.
- Biomolecular treatment may include protein injection, gene therapy and platelet-rich plasma.
- Cell therapy may include differentiated cells such as disk-relevant cells and articular chondrocytes, and stem cells.

- Tissue-engineering strategies may include scaffold development and whole disk transplantation with tissue-engineered construct.
- The authors acknowledge the difficulties associated with extrapolation of data from animal studies to human applications, and are aware of model-based limitations such as subjective assessment of pain and physical function.
- The authors feel that significant progress within the field of biologic therapies for DDD has been made in the past decade, although there are a paucity of clinical studies. They have attempted to consolidate and analyze a plethora of data from published and ongoing research.

Pain sensitization and osteoarthritis

Lars Arendt-Nielsen, Prof., DMSc, PhD

Clinical Experimental Rheumatology 2017; 35 (Suppl. 107): S68-S74

JACO Editorial Reviewer: Scott E Kilmer, DC, FACO, FACC

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AUTHOR'S ABSTRACT:

Treating chronic musculoskeletal pain, and chronic joint pain (osteoarthritis (OA) in particular), is challenging as the peripheral and central pain mechanisms are not fully discovered, and safe and as effective analgesic drugs are not available. In general, the preclinical models of OA are limited to provide fundamental understanding of the pain mechanisms involved in patients with chronic joint pain (1). The pain associated with joint discomfort is highly variable, often underestimated by clinicians, and shows only modest association with crude radiological scorings. One reason for the disconnect between the extent of structural damage and pain is neural plastic changes occurring in the peripheral and central nervous system resulting in pain sensitization impacting the patient's experience of pain. In recent years, a variety of human quantitative and mechanistic pain assessment tools (Quantitative Sensory Testing, QST) have been developed, providing new opportunities for diagnostic phenotyping of OA patients and the associated degree of sensitization. Mechanistic phenotyping has revealed specific subgroups of specifically sensitized OA patients, and been used as a predictive guideline to evaluate which patients are most likely to experience continued chronic pain after an otherwise technically successful knee replacement (chronic postoperative pain). Furthermore, such techniques may be used to profile new or existing drugs together with other e.g. cognitive or behavioral therapies with the potential to manage joint pain.

JACO Editorial Summary:

- This article was written by Lars Arendt-Nielsen, Prof.,DMSc.,PhD. Dr. Arendt-Nielsen is on the Faculty of Medicine at Aalborg University, School of Medicine in Denmark.

- The purpose of this paper is to explain pain sensitization in osteoarthritis and show how selected biomarkers are used to help predict those patients that may develop increased pain after total joint arthroplasty (TJA).
- It is estimated that by 2030 total hip arthroplasty in the US will increase by 200% and total knee arthroplasty will increase by 700%. Additionally, around 20% of total knee arthroplasty patients will have continued pain after surgery and approximately 10% of total hip arthroplasty patients will have continued pain after the surgery.
- Pain sensitization occurs as a result of "continuous and intense nociceptive input from joints". Studies have shown this type of nociceptive input occurs with osteoarthritis and is a main driver of peripheral and central sensitization.
- There are two basic ways to clinically evaluate for pain sensitization. The first is assessment using scores/questionnaires such as VAS, McGill Pain Questionnaire, WOMAC and others. The second method is the use of quantitative mechanistic pain biomarkers. There are two quantitative mechanistic biomarkers used in the study. 1. Temporal Summation. This occurs when neurons in the dorsal horn become more excitable due to repeated nociceptive input. Bedside testing using this method is accomplished by using computer controlled pressure algometry. After repeated stimulation, the patient reports an increase in severity and duration of pain which is not supported by the radiographic findings. 2. Descending Pain Modulation. This type of pain modulation occurs when nociceptive input is received in the brain from a segment remote from the site of pain caused by the osteoarthritis. Clinical testing of descending pain modulation is accomplished with the use of cuff algometry when one cuff delivers a conditioning stimulus and the other delivers a test stimulus. This triggers a "pain inhibits pain phenomenon". This phenomenon can be diminished in patients with OA and can be used to help identify those patients that may have continued pain after total joint arthroplasty.
- Using the methods mentioned above to measure for pain sensitization, there is evidence to show that there is a correlation of preoperative hyperalgesia and chronic postoperative pain. Further, joint revision surgery based only on the patient's report of pain can result in 50% of those patients experiencing chronic/increased pain after the revision.
- This paper demonstrates how mechanistic phenotyping can be used to aid in predicting which patients would likely develop chronic or increased pain after total joint arthroplasty. Pain sensitization should be evaluated prior to surgery and pain alone should not be used as the sole indicator for total joint arthroplasty.

Structural Correlates of Pain in Osteoarthritis

T. Neogi

Clinical and Experimental Rheumatology 2017;35 (Suppl. 107):S75-S78

JACO Editorial Reviewer: Daniel P. Dock, DC, FACO

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AUTHOR'S ABSTRACT:

Osteoarthritis (OA) is the most common form of arthritis, with knee OA itself being among the most common conditions and a leading cause of disability among older adults worldwide. Pain is a key symptom in the decision to seek medical attention, yet available therapies for managing OA are limited with only minimal or moderate efficacy. Current approaches to pain management in OA have been rather non-specific, limited to acetaminophen or NSAIDs primarily, without targeting underlying structural lesions that may be contributing to pain in OA. With the advent of MRI, a number of studies have noted the importance of bone marrow lesions and synovitis/effusion to the pain experience in OA. These pathologic features are therefore attractive treatment targets, with some proof-of-concept studies demonstrating the potential efficacy of targeting these lesions. Another increasingly recognised important contribution to pain in OA is sensitisation, which is associated with pain severity. Synovitis/effusion have been identified as potentially leading to development and worsening of sensitisation. Much work remains to be done in understanding the mechanisms by which structural pathology causes pain; such insights are urgently needed to develop new treatment approaches to help millions of people worldwide who are burdened by pain from OA.

Editorial Review:

Knee Osteoarthritis (OA) is among the most common conditions and leading cause of disability among older adults. With the advent of MRI, a number of studies have noted the importance of bone marrow lesions and synovitis effusion to the pain experience in OA. Another increasing recognized important contribution to pain in OA is sensitisation. [*Editor's note: The original article author's spelling of sensitisation, rather than sensitization, is maintained in this Editorial Review*]

Results: Approximately 10-12% of the adult population have symptomatic OA of any joint. Knee OA is a leading cause of disability among older adults, accounting for a greater risk of mobility disability.

One barrier to understanding the genesis of pain in OA is the so-called "structure-symptom discordance", some individuals have radiographic changes with minimal symptoms, while others have more significant pain with only minimal (if any) structural pathology noted on radiograph. Findings of osteophyte with joint space narrowing characterized as *radiographic OA,* while its combination of symptoms (i.e. pain, aching, stiffness) in the same joint attributable to OA is considered to be *symptomatic OA*. The discordance diminishes with more severe stages of disease. It has been recognized that radiographs are relatively insensitive to numerous pathologic changes, and are better visualized on MRI.

Something within the knee must be contributing to symptoms. Joint replacement, intra-articular lidocaine injection, and intra-articular corticosteroid injection give relief of pain.

As OA progresses, neurovascular invasion may disrupt the osteochondral junction, accompanied by growth of sensory nerves and increased expression of nerve growth factor (NGF), which can facilitate *sensitisation*. Neurobiologic mechanisms have been recognized, particularly of sensitisation.

MRI studies give further insight into structural pathology contributing to OA pain. MRI studies have shown subchondral bone changes (bone marrow lesions), and synovitis/effusion. The bone marrow lesions and synovitis/effusion have been associated with pain and with pain fluctuation.

Use of a patellofemoral knee brace caused reduction in pain and reduction of bone marrow lesions.

Meniscal lesions are common in OA. Meniscal tears were equally occurring among those with knee pain as those without knee pain. MRI findings in knees of older adults can be challenging as 89% of people without any radiographic evidence of knee OA have at least one MRI feature, seen in both painful and pain-free knees.

The contribution of structural joint pathology to the pain experience in OA remains incompletely understood.

MRI sheds light onto specific pathologic features that may play an important role in causing pain.

Factors beyond structural pathology also contribute to the pain experience factors, including psychologic factors.

Pain sensitisation and the overall efficiency of CNS pain modulation mechanisms, merit further studies.

JACO Editorial Summary: Two points that jumped out at me when reading this article was the reduction in pain and the reduction of bone marrow lesions when the patient used a patellofemoral knee brace, and the strengthening of the lower extremity muscles helping with proper patellar glide. This outlines the possibility of using arch supports to cause relief of pain and relief of mobility disability (remember, the "foot goes flat, and the leg twists in" causing twisting of the knee structures).

- Knee OA was pointed as a leading cause of disability in older adults, accounting for a greater risk of mobility disability. This is important considering deep venous thrombosis, pneumonia, cardiovascular conditions, and cerebrovascular conditions.
- Patients with knee pain may have normal appearing radiographs; osteoarthrosis may be revealed on the radiographs of patients without knee pain. The article points out that there can be knee pain with normal appearing x-rays.
- Sensitisation can explain why the patient can have more pain than is justified by the exam.
- The future: Stem Cell Injections and Platelet-Rich Plasma (PRP) are now having success in some cases for pain relief and even reduction of MRI visible bone marrow edema. This is important as it may help take away some of the mobility disability (and the adverse conditions that may occur with mobility disability).

Editorial Review

Strawberries Improve Pain and Inflammation in Obese Adults with Radiographic Evidence of Knee Osteoarthritis

Jace Schell, R. Hal Scofield, James R. Barrett, Biji T. Kurien, Nancy Betts, Timothy J. Lyons, Yan Daniel Zhao, and Arpita Basu

Nutrients 2017, 9, 949; doi:10.3390/nu9090949.

JACO Editorial Reviewer: Scott D. Banks, DC, MS

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AUTHORS' ABSTRACT:

Osteoarthritis (OA), the most common form of arthritis, is a significant public health burden in U.S. adults. Among its many risk factors, obesity is a key player, causing inflammation, pain, impaired joint function, and reduced quality of life. Dietary polyphenols and other bioactive compounds in berries, curcumin, and tea have shown effects in ameliorating pain and inflammation in OA, but few clinical studies have been reported. The purpose of the present study was to examine the effects of dietary strawberries on pain, markers of inflammation, and quality of life indicators in obese adults with OA of the knee. In a randomized, double-blind cross-over trial, adults with radiographic evidence of knee OA (n = 17; body mass index (BMI): (mean SD) 39.1 $\pm$ 1.5; age (years): 57 $\pm$ 7) were randomized to a reconstituted freeze-dried strawberry beverage (50 g/day) or control beverage daily, each for 12 weeks, separated by a 2-week washout phase (total duration, 26 weeks). Blood draws and assessments of pain and quality of life indicators were conducted using the Visual Analog Scale for Pain (VAS Pain), Measures of Intermittent and Constant Osteoarthritis Pain (ICOAP), and Health Assessment Questionnaire-Disability Index (HAQ-DI) questionnaires, which were completed at baseline and at weeks 12, 14, and 26 of the study. Among the serum biomarkers of inflammation and cartilage degradation, interleukin (IL)-6, IL-1, and matrix metalloproteinase (MMP)-3 were significantly decreased after strawberry vs. control treatment (all $p < 0.05$). Strawberry supplementation also significantly reduced constant, intermittent, and total pain as evaluated by the ICOAP questionnaire as well as the HAQ-DI scores (all $p < 0.05$). No effects of treatment were noted on serum C-reactive protein (CRP), nitrite, glucose, and lipid profiles. Dietary strawberries may

have significant analgesic and anti-inflammatory effects in obese adults with established knee OA.

JACO Editorial Summary:

- The morbidity rates with drug treatments for chronic osteoarthritic pain have driven interest in natural therapies noted for the far superior safety profile. Some of the more studied include plant phenols such as derived from turmeric, ginger and other food items.
- Clinical benefit has been previously been demonstrated in metabolic syndrome with phenolics from berries such as strawberries. As metabolic syndrome and osteoarthritis share a major driving mechanism, inflammation, it was sought to measure their impact on both inflammatory and clinical features in osteoarthritic patients.
- Inflammatory biomarkers (IL-6, IL1b), tissue degeneration markers (matrix metalloproteinase (MMP)-3) and clinical outcome markers (ICOAP questionnaire and HAQ-DI scores) significantly improved after 12 weeks of active supplementation confirming the original hypothesis.
- No adverse effects were seen although the study population was only 17 subjects. Some adverse effects would be anticipated over the 12 weeks of observation in this study with NSAIDs.
- The applicability of the data to broader populations is difficult as the study population was significantly obese. As obesity is highly associated with a background pro-inflammatory state could be argued that the clinical effect may be greater in a non-obese population.
- Although similar outcomes have been shown with many phenolic food sources, very little is known about their comparative efficacy to guide current clinical application beyond clinician experience. Some study has found synergistic combinations of phenolic food sources to produce better outcomes that monotherapies and this should also be considered.

Summary:

The study supports the idea of the use of plant phenolics such as strawberries are a beneficial option perhaps better suited to long-term use as is needed in osteoarthritis. They may be particularly helpful in the management of patients who have already had adverse effects with drug therapies.

Radiology Corner

Retrosomatic Cleft: A Radiology Review

Jacinda D. Woods, DC[1]
[1]Radiology Resident, Palmer College of Chiropractic, Davenport, Iowa

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December 2017, Volume 14, Issue 4

History

A 25 year old male presented with right sided low back pain and left hip pain for the past five months. The patient had also reported history of left iliac fracture.

Plain Films

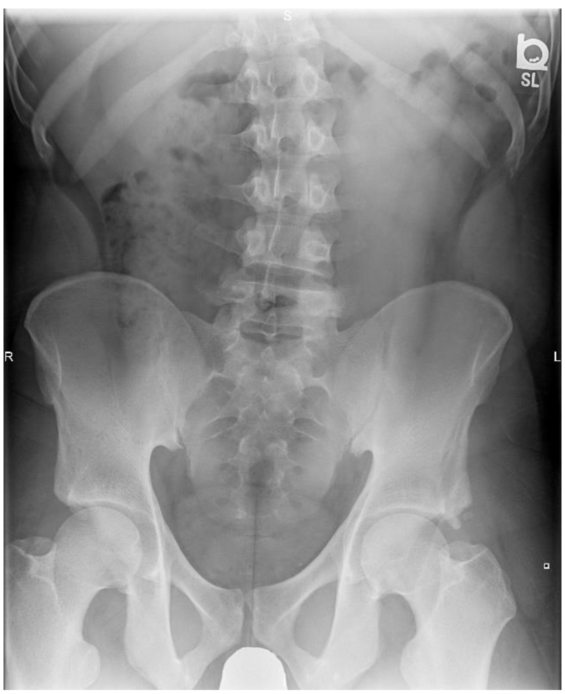

Figure 1: AP Lumbopelvic

Figure 2: Lateral Lumbar

Report

The AP and lateral lumbopelvic radiographic examination revealed L5 left pedicle region sclerosis with a suspected right-sided pars defect. A well-corticated osseous irregularity was detected at the lateral aspect of the left ilium in the region of the anterior inferior iliac spine with adjacent small corticated dystrophic calcification. This was most likely due to a prior avulsion fracture of the rectus femoris. Well-corticated ossicle was noted incidentally at the inferior aspect of the L4 spinous. Mild left lumbar lateral curvature was noted with pelvic unleveling, low on the left. There was no evidence of abdominal mass or calcification and the bowel gas distribution is normal.

CT Images

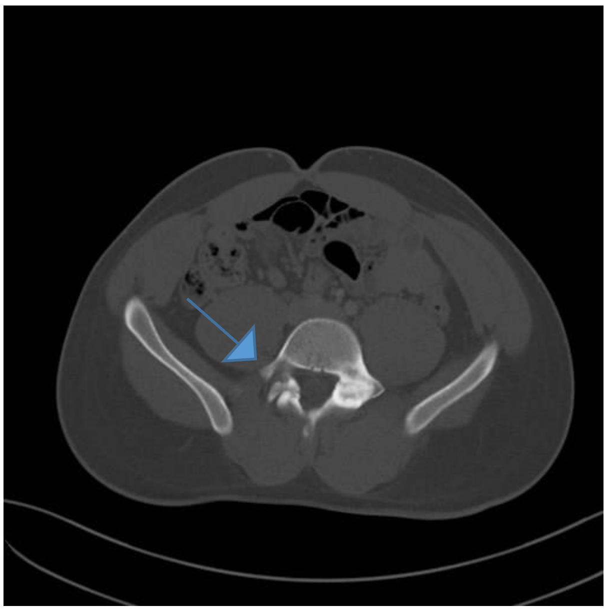

Figure 3: CT Axial slice

Pars defect (right)

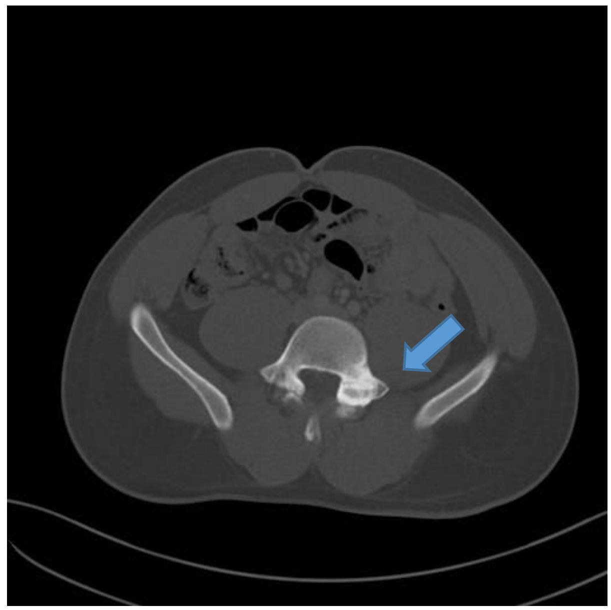

Figure 4: CT Axial slice

Retrosomatic cleft (left)

Figure 5: CT Sagittal slice

Retrosomatic cleft (left)

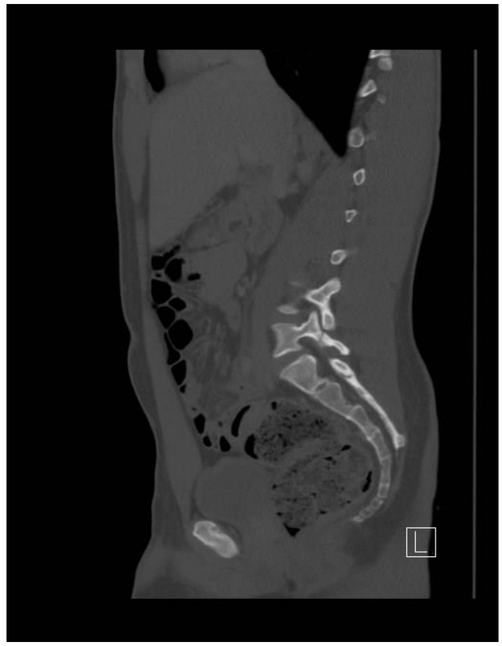

Figure 6: CT Sagittal slice

Pars defect (right)

A previous abdominal computed tomography (CT) was located at a local hospital. The examination revealed a right-sided pars defect at L5 with contralateral sclerosis involving the left pedicle region. A linear defect was also observed involving the posterior aspect of the left pedicle (retrosomatic cleft). These findings are consistent with a grade I spondylolisthesis of L5 with right pars and left retrosomatic defects.

Discussion

A retrosomatic cleft is most commonly defined as linear, vertical defects of the vertebral pedicle.[1] This can often be confused with traumatic pedicle fractures. A retrosomatic cleft is commonly seen with contralateral spondylolisthesis.[2]

Other clefts, in addition to retrosomatic cleft, that are associated with the posterior elements of vertebral bodies are retroisthmic cleft, isthmic spondylolisthesis, spondylolysis, and spina bifida. Retroisthmic cleft is a defect that is posterior to the pars interarticularis and is rare. Isthmic spondylolisthesis is a defect at the pars interarticularis with anterior displacement of the vertebral body. If there is no anterior translation of the vertebral body, this is termed a spondylolysis. Both spondylolysis and isthmic spondylolisthesis can be either developmental or acquired in origin.[3]

References

1. MDCT of variations and anomalies of the neural arch and its processes: part 1--pedicles, pars interarticularis, laminae, and spinous process. AJR Am J Roentgenol. 2011 Jul;197(1):W104-13. doi: 10.2214/AJR.10.5803.
2. M Soleimanpour, M L Gregg and R Paraliticci. Bilateral retrosomatic clefts at multiple lumbar levels. American Journal of Neuroradiology. September 1995, 16 (8) 1616-1617.
3. Jarrah Ali Al-Tubaikh, Maximiliun F. Reiser. *Congenital Diseases and Syndromes An Illustrated Radiological Guide*. 2009, 145-146.

Ortho Quiz

by Steven L. Kleinfield D.C., F.A.C.O.

1. Which of the following is not considered to be causational for olecranon bursitis?
    a. Trauma
    b. Infection
    c. Gout
    d. Prolonged Pressure
    e. All of the above are considered causational
    f. None of the above are considered causational

2. The Orthopedic Test “Dugas” is considered a classic test for evaluation for the presence of which condition:
    a. Carpal Tunnel Syndrome
    b. Postural Compression Syndrome
    c. Shoulder Dislocation
    d. Rib Fracture

3. Which of the following orthopedic tests should be performed first when suspecting Thoracic Outlet Syndrome:
    a. Adson’s Test
    b. Allen’s Test
    c. Wright’s Test
    d. Halstead Maneuver

4. Your patient is a 60 year old male. He is of Scottish heritage. His main complaints are as follows: He has recently been noticing that he is developing bone pain, generally stiff all over and that he tires easily. At present he seems to be getting headaches quite frequently and he notices that he feels like his hat size is increased since he can't wear his old Stetson any longer.
    a. Cervicogenic Encephalgia
    b. Osteoarthrosis
    c. Paget’s Disease
    d. Osteogenesis Imperfecta

5. The most common type of headache is:
    a. Cluster Headache
    b. Classic Migraine Headache
    c. Common Migraine Headache
    d. Tension Headache

Current Events

- The dates for the 2018 Academy of Chiropractic Orthopedists online examination are as follows:
    - Friday, May 18, 2018
    - Friday, July 20, 2018

  Please contact the Academy as soon as you can with your notice of intent to sit the Academy Board examination.

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- Apply for the Lipe Scholarship

  Details at http://www.accoweb.org/lipescholarship.html

- The full hours of the following conventions have been accepted by the Academy as qualifying for re-credentialing.

    - American College of Chiropractic Orthopedists 2018 Annual Convention
      19 Apr to 21 Apr 2018
      Hilton Garden Inn Carlsbad Beach, Carlsbad CA
      https://acoco.wildapricot.org/event-2436518

    - CFS 2017 Annual Fall Convention
      October 5-7, 2017
      Chicago Marriott Oak Brook
      1401 W 22nd Street | Oak Brook, IL 60523
      630.573.8555 www.marriott.com/chiob
      http://www.forensic-sciences.org/convention/

- ACC-RAC 2018 Association of Chiropractic Colleges 25th Educational Conference and Research Agenda Conference

  March 8-10, 2018

  InterContinental Dallas

  15201 Dallas Parkway, Addison, TX 75001

  http://chirocolleges.org/acc-rac-conference/

Answers to Ortho Quiz

1. e. *All of the above can be considered causational*

https://orthoinfo.aaos.org/en/diseases--conditions/elbow-olecranon-bursitis

2. *c. Shoulder Dislocation*

https://medisavvy.com/shoulder-dugas-test/

3. *b. Allen's Test*

https://www.thestudentphysicaltherapist.com/allens-test.html

4. *c. Paget Disease*

https://www.mayoclinic.org/diseases-conditions/pagets-disease-of-bone/symptoms-causes/syc-20350811

5. *d. Tension Headache*

https://www.healthxchange.sg/head-neck/brain-nervous-system/three-types-primary-headaches