Plica Syndrome and its Embryological Origins

Article · April 2017

DOI: 10.5348/O03-2017-5-RA-1

1 author:

Christopher L Hoehmann
Nassau University Medical Center
22 PUBLICATIONS **83** CITATIONS

SEE PROFILE

REVIEW ARTICLE PEER REVIEWED | OPEN ACCESS

Plica syndrome and its embryological origins

Christopher L. Hoehmann

ABSTRACT

Plica syndrome is a pathologic process due to inflammation of synovial plicae. Synovial plicae are frequently asymptomatic, but can become diseased with trauma and overuse. Synovial plicae are embryologic remnants of the synovial lining of the knee. The mechanism of their existence is controversial. Many agree that plicae are vestigial septums that once separated the knee joint into three cavities during embryogenesis. A second theory details a failure to resorb the mesenchyme that once occupied the knee joint during cavitation. The variable morphology of plicae can present as suprapatellar, infrapatellar, lateral and mediopatellar, with the latter most commonly progressing to plica syndrome. Synovial plicae remain an interesting challenge to clinicians, as plica syndrome is a common condition with a wide differential diagnosis. Many physical examination techniques can be used in conjunction with a thorough history to elucidate the presence of plica syndrome. The diagnosis is made by exclusion, therefore a multitude of imaging modalities are necessary to rule out other causes of knee pain. Conservative treatment is recommended initially, however, its effectiveness is variable. Surgical intervention is a robust method for treating plica syndrome when conservative treatment fails. This brief review provides an overview of knee embryogenesis, pertinent information regarding the etiology and pathology of plica syndrome, as well as insight into the diagnosis and management of this condition.

Keywords: Arthroscopy, Knee embryogenesis, Knee pain, Orthopedic surgery, Plica syndrome, Synovial plica

Christopher L. Hoehmann

Affiliations: Third year medical student, New York Institute of Technology College of Osteopathic Medicine, Old Westbury, New York, USA.

Corresponding Author: Christopher L. Hoehmann, BS, Northern Boulevard, Old Westbury, New York 11568 USA, New York Institute of Technology College of Osteopathic Medicine; Email: choehman@nyit.edu

Received: 26 January 2017
Accepted: 16 March 2017
Published: 13 April 2017

How to cite this article

Hoehmann CL. Plica syndrome and its embryological origins. Edorium J Orthop 2017;3:1–12.

Article ID: 100005O03HS2017

****

doi:10.5348/O03-2017-5-RA-1

INTRODUCTION

Synovial plicae are embryological remnants of the synovial lining and are found in many knees [1–3]. They are commonly asymptomatic but may become diseased when subject to an inflammatory process, commonly due to overuse or trauma [1–9]. A plica that is symptomatic will result in a constellation of symptoms highlighted by intermittent knee pain termed plica syndrome.

Understanding the mechanism of plicae development can provide great insight into how the knee joint itself forms. As plica syndrome is a common condition, it is relevant for clinicians to have a general understanding of

its origins and pathogenesis. This brief review provides an overview of the origins of the synovial plica of knee joint as well as the etiology, diagnosis, and treatment for its pathologic sequlea, plica syndrome.

METHODS

Sources were retrieved via a PubMed search. Search terms such as plica syndrome, synovial plicae, and synovial joint morphogenesis were utilized to identify relevant sources. These searches yielded 142, 112, and 446 results, respectively. Sources were included if they were considered relevant to the current review article, which was determined by analyzing the title or abstract of the source. Additionally, sources that were cited in other review articles were retrieved if they were relevant to the current review article and they were not retrieved in the original PubMed search.

EMBRYOLOGY

Bone development

Ossification occurs during fetal development by two mechanisms: intramembranous ossification and endochondral ossification. Intramembranous ossification is the process of bone forming directly from mesenchymal tissue. It occurs during the construction of the flat bones of the skull, clavicles, maxilla and mandible. Endochondral ossification differs in that it requires an intermediate cartilage model, termed the skeletal blastema, which is replaced by bone tissue. The intermediate cartilaginous phase is highlighted by avascular, densely packed mesenchymal cells and chondrocytes. This process is regulated by growth factors and cellular interactions with the surrounding extracellular matrix that work to modulate cellular signaling pathways and the transcription of distinct genes. Endochondral ossification derives long bones and most bones in the body, including the femur, tibia, and fibula that comprise the knee joint [10–13].

Synovial joint development: Cavitation

Cavitation occurs alongside endochondral ossification. The hyaline cartilage matrix is ossified to bone tissue, except at the interzone, which is the interface between two neighboring bones (Figure 1). This leaves behind an area of non-ossified cartilaginous tissue between two bones, which will form a joint. The interzone is an avascular and highly cellular region, which contains mesenchymal cell precursors that give rise to the joint

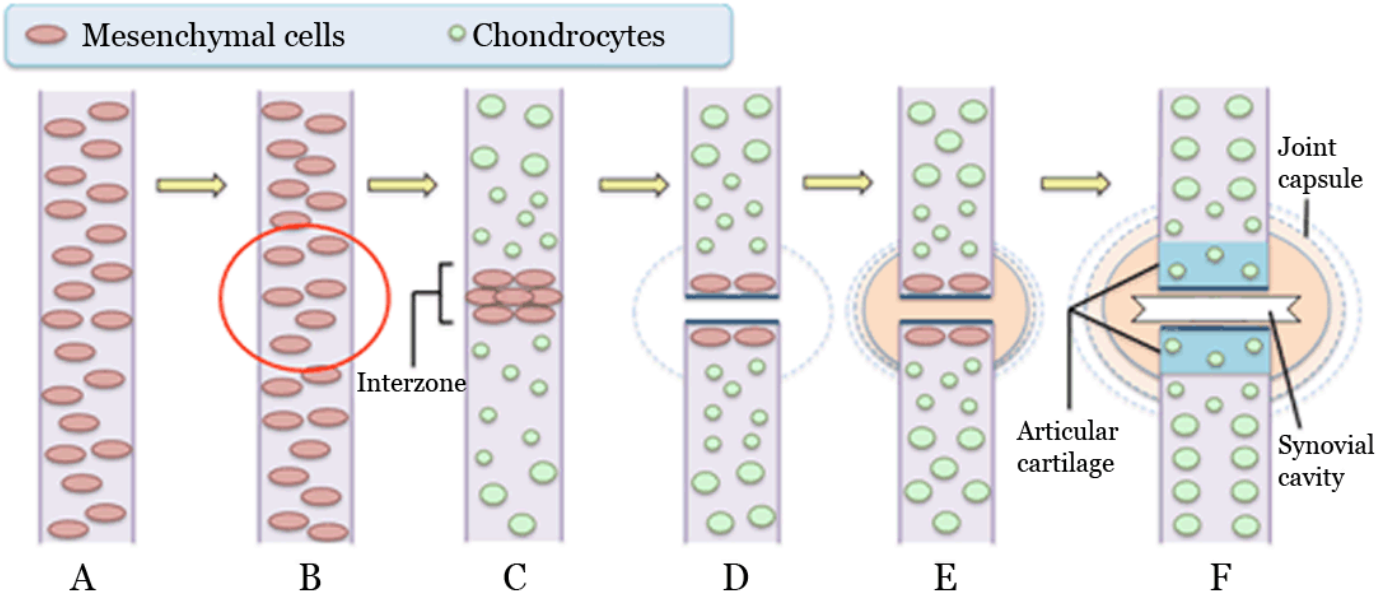

Figure 1: Synovial Joint Morphogenesis
The arrows depict the progression of condensed mesenchymal tissue to a matured knee joint. (A) Condensed mesenchymal cells are present as the process of endochondral ossification is occurring. At this point in time, the location of joint development has not been specified, as high levels of type II collagen will be expressed. (B) The location of joint development has been determined. Expression of COL2A1 will sharply decrease while expression of growth differentiation factor 5 (GDF5) will increase. (C) Condensation of mesenchymal cells occurs to form the highly cellular and avascular interzone, which will express wingless-related integration site 14 (Wnt14). Neighboring chondrocytes increase expression of type II collagen and the mutant fibroblast growth factor (FGF), which will further regulate joint morphogenesis. (D) Cavitation will occur as the opposite ends of the interzone begin to differentiate into articular cartilage surfaces. (E) Synovial mesenchyme is formed from the periphery of the interzone as it is invaded by blood vessels. The interzone will continue to expand as it accumulates hyaluronan. (F) Cavities derive in the peripheral and central interzone and coalesce to form a confluent joint cavity. The joint capsule itself will derive from a condensation of the surrounding mesoderm.

capsule, intracapsular ligaments, menisci, tendons, and synovial lining. Plicae are specifically derived from the synovial mesenchyme, which is the result of vascular proliferation into the periphery of the interzone [11–16].

Regions designated to become joints are initially specified by an accumulation of collagen type II alpha 1 (COL2A1) [12–14, 17]. Once the location of the joint has been specified, bone maturation will continue as expression of COL2A1 sharply decreases and expression of growth differentiation factor 5 (GDF5) increases [13, 15, 17]. The process of cavitation is further facilitated by expression of wingless-related integration site 14 (Wnt14), which initiates synovial joint formation, and GDF5, which induces chondrogenesis [12, 13, 15, 18, 19].

Synovial-layer formation is regulated by a number of cells and molecular markers. Increased proliferation of fibroblast-like cells and recruitment of macrophage-like (type A) cells from the bloodstream contribute to the synovial-lining cell layers. The cells on the intimal surface of the synovial-lining express uridine-diphosphate glucose dehydrogenase (UDPGD) and hyaluronan receptor CD44, which contribute to a high level of glycosaminoglycans in the joint [11, 20]. Additionally, these cells express Cadherin 11, which is largely responsible for the architecture of the synovial lining as it regulates tissue outgrowth and cell migration [11].

Formation of plicae

The synovial plicae are embryological remnants of the synovial lining of the knee joint capsule [1–4, 5, 14]. They manifest as inward folds of the membrane that lines the synovium of the knee joint capsule. The formation of these remnants is rather controversial as there are two proposed mechanisms for the development of the knee joint [1, 2].

Compartment theory

The first and more widely accepted theory proposes the knee joint to form three separate compartments during development. They are separated by mesenchymal tissue into medial, lateral, and suprapatellar compartments. This tissue fuses during the 11th or 12th week of development and resorbs during the 16th week of development. As the membranes resorb, the three compartments coalesce to form one confluent joint cavity. If these membranes fail to resorb completely, they remain as synovial plicae [1, 2, 8, 14].

Condensation and cavitation theory

The second theory advocates that during the eighth week of development the space between the distal femur and proximal tibial epiphysis becomes filled with mesenchymal tissue. Thereafter, specific territories of this tissue will either condense to form solid structures, such as ligaments and the menisci, or the tissue will resorb to form the meniscotibial, femoromeniscal, patellofemoral cavitations. By the 10th week of development, these cavitations fuse to form a confluent joint cavity with a synovial lining. If cavitation is incomplete, or mesenchymal tissue fails to resorb completely, synovial plicae will form.

Morphogenetic time table of the knee joint

Understanding the morphogenic timeline of knee joint development allows greater insight to the embryologic origin of the knee. In light of this timeline, one may better understand the context in which development and reabsorption of the synovial plicae takes place.

Developmental timeline of the bones comprising the knee joint

During the 7th week of development (Carnegie stage 18 of embryo development) the cartilaginous template of the femur and tibia is formed, and their ossification begins during the 13th week of development. The patella first appears during Carnegie stage 19 as a dense blastema. It further becomes condryfied during Carnegie stage 22, and begins ossification during the 14th week of development. During Carnegie stage 22, the knee cavity first appears as the femoropatellar joint, which derives from the periphery of the interzone. The lateral meniscotibial joint connects with the superior tibiofibular joint by the 11th week of development and separates entirely after the 13th week of development. The menisci, on the other hand, derive from the eccentric part of the interzone during Carnegie stage 22 [4, 14, 20, 21].

Developmental timeline of the ligaments comprising the knee joint

During Carnegie stage 20 the patellar ligament begins to form. Soon thereafter during Carnegie stage 21, the cruciate ligament system begins to form from the interzone. This begins with the posterior cruciate ligament and ends by the 10th week of development with the formation of Wrisberg's meniscofemoral ligament. Formation of the lateral collateral ligament occurs separately from the knee joint capsule and begins during Carnegie stage 23. The tendon of the popliteus muscle forms simultaneously with the lateral collateral ligament. Conversely, the medial collateral ligament develops during the 9th week of development as an aggregation of the joint capsule. During the 11th week of development, mesenchymal tissue inferior to the patella and between the many ligaments gives rise to the intra-articular fat pad. Finally, during the 14th week of development the suprapatellar bursa forms, which completes the embryonic morphogenesis of the knee joint [17, 20].

ANATOMY AND CLASSIFICATION

Plicae exist as four distinct anatomical morphologies: mediopatellar, suprapatellar, infrapatellar, and lateral plicae (Figure 2) [1, 2, 4, 16]. Each morphology exists with its own prevalence, anatomy, and clinical significance.

Mediopatellar plica

The mediopatellar plica most classically produces plica syndrome, and has been referred to in literature as plica synovialis mediopatellaris, meniscus of the patella, medial shelf, Aoki's ledge, Iino's band, and plica alaris elongate. [1–3, 5, 9]. The medial plica is a large structure with free borders that originates either from under the medial retinaculum or the medial wall of the pouch, and it does this at the level of the vastus medialis oblique muscle in the suprapatellar region [1, 4, 5, 22, 23]. It courses inferiorly and parallel to the medial edge of the patella in the coronal plane and ends when it becomes continuous with the synovium, termed plicae alaris, that covers the infrapatellar (Hoffa) fat pad [1, 3–5, 22, 24]. In some cases, the mediopatellar plica may begin superiorly as an extension of the suprapatellar plica, while in other cases it may continue inferiorly as the infrapatellar plica [5].

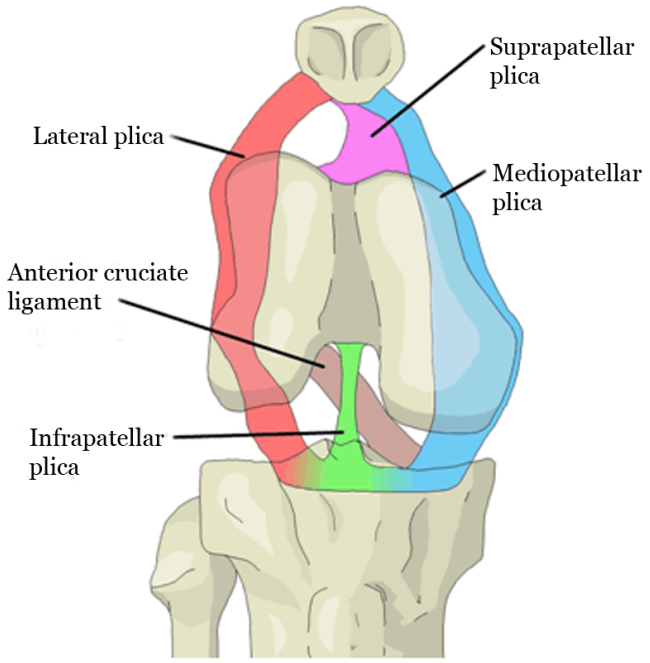

Figure 2: An anatomical depiction of the various manifestations of plicae in the knee joint.
An anterior view of a bent knee joint is depicted. The bottoms of the femoral condyles are visible, as would be in a bent knee. The patella is rolled away upwards, exposing the sesamoid bone's underside. Four plicae are depicted here, however, any variation of these plicae may exist in a live knee. This image depicts the approximate locations that each of the plicae may exist in a live knee. Each plicae is represented here with a unique color; the mediopatellar plicae is depicted in blue, the suprapatellar plica is depicted in magenta, the lateral plica is depicted in red, and the infrapatellar plica is depicted in green. The anterior cruciate ligament is also visible.

The anatomy of the medial plica itself is variable and thus can be classified into four types, A–D, according to a widely accepted and clinically significant scheme authored by Sakakibara [25, 26]. Type A and type B are largely asymptomatic because of their small size. Type C and type D often become symptomatic due to their larger size and propensity to become trapped between the medial condyle of the femur and the patella [1, 4, 5, 22, 25]. Type A can be found as a cord-like, thin elevation of the synovial wall. Type B presents as synovium with a shelf-like appearance, which is not wide enough to cover the anterior surface of the medial femoral condyle. Type C has a similar shelf-like appearance, but it is larger and partially covers the anterior surface of the medial fermoral condyle. Furthermore, a type D medial plica exists similarly to a type C medial plica, except that it is additionally fenestrated and has pedunculated tags that may impinge upon the patella-femoral joint [1, 5, 25].

There are four synovial structures located in the medial gutter that may be mistaken as the medial plica: the anteriomedial fringe of the synovium, the superomedial plica, the plica alaris elongate, and the transverse arcuate folds. The anteromedial fringe of synovium is a structure that covers the anterior horn of the medial meniscus that can cause painful symptoms, similar to the mediopatellar plica, when it is impinged. The superomedial plica may be mistaken as the medial plica but it is actually part of the superior plica; noticing its location superior to the patella itself can differentiate this as a distinct entity. The plica alaris elongate can be found as a fold of synovium adjacent to the patella that can be distinguished from the medial plica with a skyline view of arthrography. The transverse arcuate folds lie at the base of the medial gutter and may be confused as the medial plica as well [1, 22].

Suprapatellar plica

The morphology known as suprapatellar plica, also known as plica synovalis supraptellaris, is the most common plica morphology overall [1, 4]. This structure usually exists as a crescent-shaped fold that originates from either the anterior femoral metaphysis or the posterior quadriceps tendon and extends to the medial portion of the knee [2, 5, 23]. Approximately half of the time, it will blend into the mediopatellar plica. This structure divides the suprapatellar pouch from the rest of the knee joint. It can be classified as type A if it is a complete septum, type B if it is an incomplete septum or contains a central portal, and type C if it is restricted to only the superomedial side. The suprapatellar plica may become impinged between femoral trochlea and quadriceps tendon at 70–100 degrees of flexion. This plica may additionally contribute to the pathogenesis of chondromalacia or suprapatellar bursitis [27–29].

Infrapatellar plica

The infrapatellar plica morphology is also known as the ligamentum mucosum This structure begins from the intercondylar notch near the anterior cruciate ligament and broadens anteriorly to the synovial lining of the infrapatellar fat pad. Furthermore, this structure can be classified as, split, separate, fenestrated, vertical septum, or not meeting any of these criteria. This morphology is different from the other morphologies in that its clinical significance is not related to pain or presenting symptoms, but rather that it blocks a full view of the joint during knee arthroscopy [2, 30].

Lateral synovial plica

The morphology known as the lateral synovial plica can be found as an irregular band-like mass arising from the lateral parapatellar synovium and extending to the lateral patellar facet. It is usually larger and thicker than the mediopatellar plica. The origin of this structure is somewhat controversial, as many believe that it is not a vestigial septum, but rather is derived from the lateral parapatellar adipososynovial fringe. There have been rare cases of lateral plica syndrome occurring bilaterally and symmetrically. Therefore, the condition may be due to a congenital hypertrophy of the lateral parapatellar structures [2, 31].

EPIDEMIOLOGY

Prevalence and incidence rates of synovial plicae have been difficult to establish as the existing literature exhibits widely varying results. A landmark study by Kim et al., used arthroscopy to investigate 400 knees and found a prevalence of 72% for mediopatellar plica, 87% for suprapatellar plica, 86% for infrapatellar plica, and 1.3% for lateral patellar plica [32]. These numbers vary per study, however most studies have been able to validate the presence of synovial plicae as a relatively common occurrence [5, 14, 23, 30, 33, 34].

Rather than knowing the prevalence of asymptomatic plicae, it is more important for clinicians to know the rate at which plicae become pathologic. However, literature has not clearly established the true prevalence of pathologic plicae; rather, it suggests an approximate prevalence of 3.8–5.5% for plica syndrome, with some outliers suggesting a prevalence as high as 25% [1, 5]. Although the prevalence of mediopatellar plica is lower than some of the other morphologies, it most commonly causes plica syndrome and is therefore the most reported and clinically relevant plica [2, 4]. The prevalence of lateral patella plica is the least explored and most controversial plica as some question its existence and clinical relevance [1, 2, 5, 22, 35]. Furthermore, synovial plicae are more likely to become pathologic in active individuals, females, and those in the second or third decade of life [2, 5, 23, 34].

ETIOLOGY

It is possible to have asymptomatic plicae that do not result in plica syndrome. The pathologic plica syndrome occurs in a predisposed person who suffers an environmental harm, resulting in irritation and inflammation of the plicae [1]. Possible mechanisms include: direct or blunt trauma to the plica, twisting injuries, repetitive flexion and extension of the knee (overuse), increased activity levels, or any process causing intra-articular bleeding or synovitis, such as osteochondritis dessicans, a loose body, a subluxing patella, a torn meniscus, or after knee arthroscopy [1, 4–9].

PATHOLOGY

A normal plica will appear as a thin, soft, and flexible structure that is well vascularized [36, 37]. A healthy plica will freely alter its orientation with knee movement [5]. A pathological plica will present as swollen and thickened with fibrosis, hyalinization, and calcification substituting its normally elastic components [4, 5, 7, 36, 38–40]. A histological specimen of a diseased plica will demonstrate type A macrophage like cells and type B fibroblast like cells surrounded by an inflammatory reaction, dense fibrosis, vascular proliferation, and small nerves with deceased myelin [41, 42]. In some cases that involve direct trauma or twisting, the plica can be torn [1]. The degree of tear varies and can be visualized during knee arthroscopy [2]. It is most commonly the mediopatellar plica that becomes pathologic [1, 2, 5].

As the pathologic plica becomes scarred and inelastic it loses the ability to move fluidly with the knee [2, 5]. As a result, the pathologic plica can form a bowstring over the trochlea and the medial femoral condyle (Figure 3) [4, 43]. This predisposes to impingement of the mediopatellar plica between the patella and femoral condyle, causing pathological abrasion on the femoral condyle during knee flexion (Figure 4) [1, 2, 4]. This can further lead to erosion of the medial aspect of the femoral condyle, and also to erosion of the medial patellar cartilage [1, 2]. As a result, the mechanism of articulation between the patellofemoral joint will be disturbed, causing further inflammation and edema of the knee joint [1]. This destructive process can promote softening and degeneration of the articular cartilage in the form of chondromalacia, or it can lead to secondary synovitis due to inflammation and progressive fibrosis [8, 24, 28, 44].

CLINICAL SIGNS

Symptomatology

Plica syndrome usually presents as a dull, achy, and intermittent pain over the anteromedial aspect of the knee

[1, 2, 5, 22]. The mediopatellar plica is the most commonly implicated culprit of plica syndrome, however, additional referred pain to the superior border of the patella may indicate the presence of a concomitant pathological suprapatellar plica [5]. The pain of plica syndrome increases with activity, especially when the knee is flexed in the 45–90 degree range [1]. Patients may experience locking, pseudo-locking, tightness, popping, clicking, high-pitched snapping, aggravation with use, and giving way of the knee joint [5, 37]. Effusions and swelling are not typically associated with plica syndrome, but if found may indicate concomitant intra-articular pathology [2, 5, 36]. Approximately half of cases will occur in the setting of knee trauma. However, it is not uncommon for there to be a delayed onset where symptoms may not occur weeks to months after the initial injury [5]. Furthermore, symptoms are typically more severe in those with torn plicae [2]. Unfortunately, many of these findings are nonspecific as they are consistent with other types of patella femoral pain (Figure 5) [4, 24].

Physical Examination

The physical examination is a critical component of the investigation for plica syndrome. Localized tenderness on palpation may be appreciated at the inferomedial quadrant of the knee, relative to the patella [1, 2, 9]. Seldom a cord-like structure or taut articular band, that is the plica, may be found as it borders the medial patella [1, 2]. This may produce a pop or clicking noise if the knee is flexed between 30 and 60 degrees [5]. Extending, internally rotating, and gliding the patella medially from a knee flexed at 90 degrees may also produce a distinctive popping sound [1, 37, 45]. Sometimes crepitus can be appreciated while flexing and extending the knee joint [1]. Tightness and shortening of the gastrocnemius and hamstring muscles and mild atrophy of the quadriceps muscles (1 to 2 cm) may be appreciated in those with plica syndrome [2, 5, 37]. These signs however are non-specific, making the diagnosis of plica syndrome especially difficult; therefore a diagnosis must be made using exclusion of clinical and radiological findings [1, 2, 43, 46]. Additionally, a number of special tests may be utilized to aid in making the diagnosis.

Active extension test

This special test begins by having the patient lie supine with the knee held in 90 degrees of flexion and ends with having the patient perform a swift kick. The test is positive if pain is produced due to a concentric pull of the quadriceps tendon on the pathologic plica [1, 6].

Flexion test

This special test begins by having the patient lie supine with the lower leg extending off the examining table. The patient is then instructed to swiftly move the leg into flexion, but stopping at 30–60 degrees of flexion "blocking the swing". The test is positive if pain is produced due to eccentric contraction of the quadriceps muscles stretching the plica [1, 6].

Hughston plica test

This special test has the patient lie supine as the examiner flexes the knee, medially rotates the lower leg, glides the patella medially, and palpates the medial femoral condyle. The test is positive if a pop is appreciated under the examiners fingers [47].

Patellar apprehension test

This special test has the patient lie supine with the knee in full extension or slight flexion. The examiner applies force to the medial aspect of the patella, gliding it laterally, while the patient tightens the quadriceps muscle. The test is positive if pain is elicited and indicates that the patella is unstable. In plica syndrome, the patella is not typically unstable, however, the test may be falsely

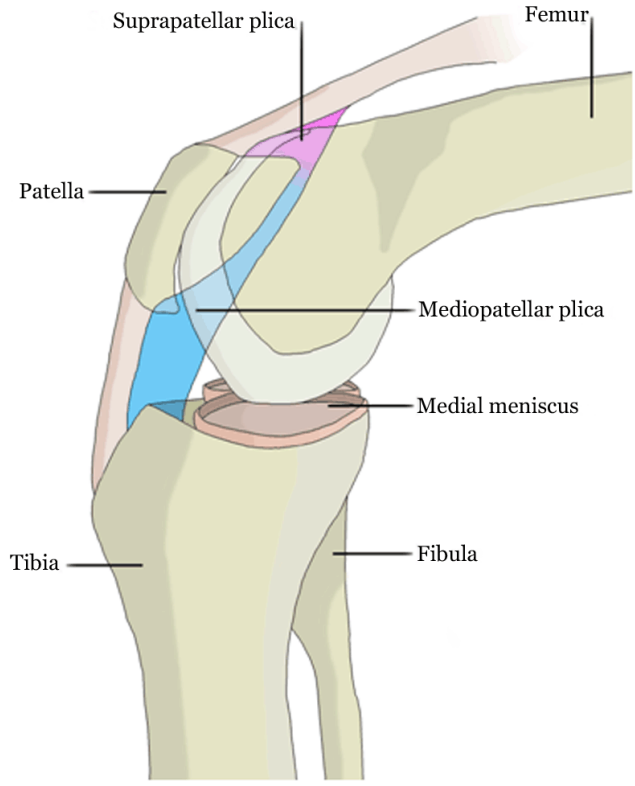

Figure 3: Medial view of a knee joint with mediopatellar and suprapatellar plicae.
The anatomical proximity of the mediopatellar plica and the medial epicondyle of the femur can be appreciated here. The plica forms a taut "bowstring" as the knee moves into flexion, creating friction between the plica and femoral condyle. This contributes to erosion of the bone as well as secondary pathologies, such as chondromalacia and synovitis. The anatomic location of the suprapatellar plica, the most common morphology of plica overall, can be observed here as well, and may additionally contribute to chondromalacia.

positive due to localized pain [2].

Mediopatellar plica test

This special test has the examiner force the knee into 90 degrees of flexion while applying a manual force to the inferomedial portion of the patellofemoral joint. The test is positive if pain can be produced in extension, but is relieved by 90 degrees of flexion [2, 48].

IMAGING

Plica syndrome is diagnosed by exclusion, therefore other potential causes of knee pain must be ruled out [49]. Standard radiographs of the knee are not diagnostic of plica syndrome as they cannot visualize synovial plicae, but they are indispensible in the workup of plica syndrome as they help rule out other pathologies [1, 2, 4, 50]. This modality can exclude bony pathologies that contribute to irritation of the medial plica, such as osteochondritis dessicans, patellar maltracking, loose bodies, osteophyte formation, fractures, and arthritis

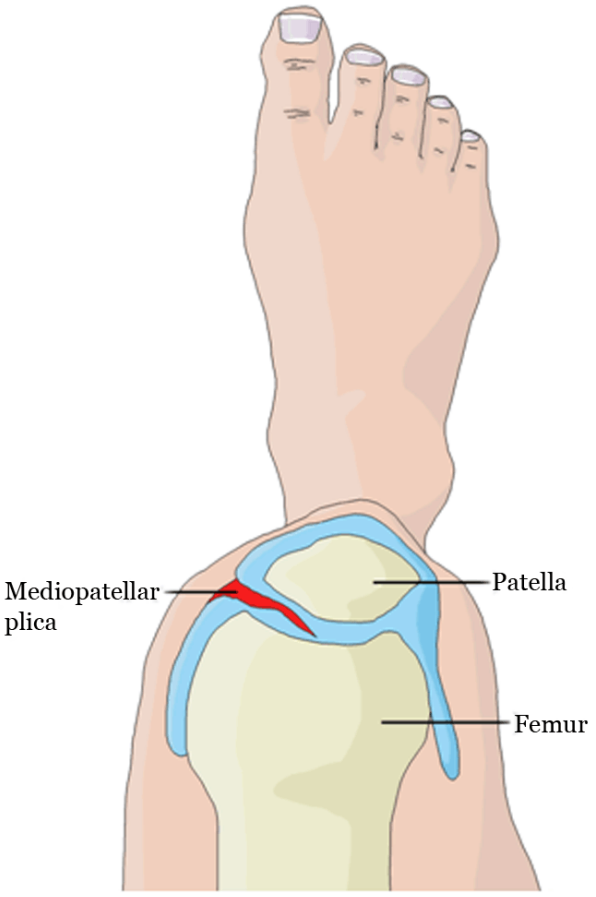

Figure 4: Superior view of the knee joint with pathologic mediopatellar plica.
As the knee moves into flexion, a pathologic mediopatellar plica can impinge between the femur and patella. The impingement more commonly occurs when the knee is flexed 30 to 45 degrees [1]. This contributes to the knee pain associated with this disorder.

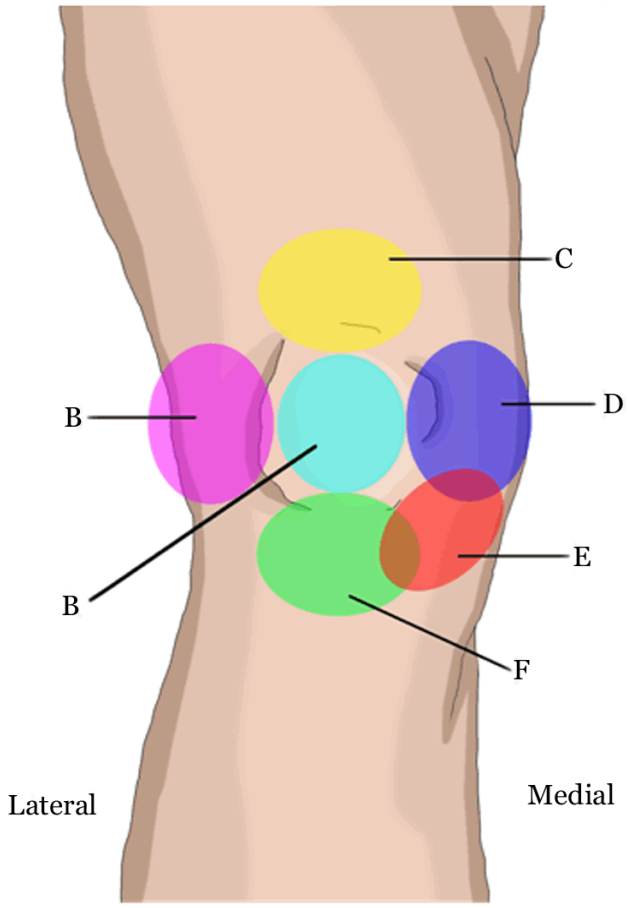

Figure 5: Differential diagnosis of knee pain based on location.
Knee pain is a common complaint and has a wide differential diagnosis. The location of pain relative to the patella can hint at the etiology of the pain. The left side of the image represents the lateral side of the leg, and the right side of the image represents the medial side of the leg. Each colored circle indicates an area of the knee that encompasses a distinct differential diagnosis. This list is not all-inclusive, as other pathologies may exist. The pathologies listed may also present atypically and cause pain at different territories of the knee. (A) Pain located in the area marked by the cyan circle, or at the patella, is typically worse when walking down stairs and can reveal the presence of chondromalacia, patellofemoral syndrome, patella maltracking, arthritis, or bursitis. Pain behind the knee at this location may suggest a Baker's cyst. (B) Pain located in the area marked by the pink circle, or the lateral margin of knee, suggests iliotibial band syndrome. This pain can stretch up the side of the leg all the way to the hip. More rarely, pain at the lateral margin of the knee may also indicate a lateral meniscus tear, lateral collateral ligament injury, or arthritis. (C) Pain located in the area of the yellow circle, or above the patella, is suspicious for quadriceps tendinopathy. (D) Pain located in the area marked by the blue circle, or the medial margin of the knee, can indicate medial meniscus tear, medial collateral ligament injury, pes anserine bursitis, or arthritis. (E) Pain located in the area marked by the red circle, or the medial aspect of the knee below the joint line, is the classic location of intermittent pain associated with plica syndrome. (F) Pain located in the area marked by the green circle, or below the patella, can indicate osteochondritis dessicans, Osgood-Schlatter disease, patellar tendinitis, Sinding-Larsen and Johansson syndrome, or patellofemoral instability.

[1]. Computed tomography arthrography, which has some ability to identify impingement of the medial plica, is generally not reliable enough to warrant regular usage. Additionally, it leaves the patient vulnerable to radiation exposure [4, 5, 51]. Contrast arthrography and pneumoarthrography are antiquated techniques that are not commonly used [2, 5, 51]. Magnetic resonance imaging may be helpful in the diagnosis as it can depict both the thickness of plicae, as well as the degree to which the plicae extends between the medial femoral condyle and patella, however, not all studies have been successful in demonstrating its usefulness (Figure 6) [4, 51–53]. Additionally, dynamic ultrasonography exists as powerful tool when investigating for plicae as it has a sensitivity as high as 90% and a specificity of 83% (Figure 7) [2, 4, 50].

Knee arthroscopy remains the gold standard for identifying the presence of plicae [2, 3, 4, 31, 52, 54]. Specifically, the medial plica can be appreciated via the routine anterolateral portal, but is better visualized from the superolateral portal [1, 8]. For the suprapatellar plica, proximal visualization is best achieved through the lateral suprapatellar portal [29]. Despite this, arthroscopy is not recommended to use as a diagnostic tool for plica syndrome because it can cause additional irritation and scarring of the medial plica [1]. It is important to consider that the presence of plicae in the context of a constellation of symptoms consistent with plica syndrome does not necessarily confirm the disease; plicae are often asymptomatic and a number of other intra-articular pathologies can cause synovitis similar to plica syndrome [1, 4, 5, 51].

MANAGEMENT

Conservative management

The prognosis of this condition is variable, as some cases are amenable to conservative management, while others require surgical intervention [1, 2]. The initial management of plica syndrome is conservative treatment including physiotherapy and anti-inflammatory agents [1, 2, 4, 16, 24, 55]. Quadriceps strengthening and stretching of the hamstring, quadriceps, and gastrocnemius may be suggested [5, 7]. An exercise program should be implemented for six to eight weeks once the diagnosis of plica syndrome is made [1]. The patient should also be educated to avoid symptom-aggravating activities [2, 5]. Non-steroidal anti-inflammatory drugs (NSAID) and cryotherapy should be utilized for pain relief, with intra-articular or intra-plical steroid injections reserved for those who do not improve with or tolerate NSAID's [2, 7, 37, 56]. It is currently unclear how effective conservative management is when managing plica syndrome [4, 5, 37]. Most studies demonstrate a low rate of recovery, except for younger patients with a brief episode of symptoms secondary to trauma [5].

Iontophoresis and phonophoresis exist as interesting options available for the conservative management of plica syndrome. Iontophoresis utilizes a small electrical current to enhance the penetration of topically applied medicine to deeper tissues. Phonophoresis functions similarly, except that ultrasound technology is utilized instead of an electrical charge. Research in this field has been controversial thus far. A study performed by Crevenna et al., was successful in demonstrating detectable plasma levels of diclofenac after iontophoresis, however, it failed to show an increase in the efficacy of the topical medication [57]. Xin et al., proposed that the low efficiency of this technique is due to the particularly small fraction of the total current contributed by the drug ions [58]. New methods are being investigated to increase the efficacy of this technique [59–62]. Similarly, a study performed by Souza et al., demonstrated an increase in skin permutation by the NSAID ketoprofen with phonophoresis, however, an unexpected decrease in skin permutation was observed with sodium diclofenac [63]. More research is indicated regarding these therapeutic modalities in the context of plica syndrome.

Surgical management

After conservative treatment has failed for at least six months, surgical intervention may be considered [1, 2]. Knee arthroscopy can be utilized to excise the entire pathological plica and can simultaneously address other pathologic components of the knee joint [2, 5, 51]. This brief procedure is associated with a quick recovery time and a low morbidity [1]. Range of motion exercises beginning 3–4 days after the procedure accompanied by NSAIDs, especially indomethacin, may reduce intra-articular scar formation [5]. Most patients will be able to participate in sports after a 3–6 weeks recovery period [5]. Furthermore, torn plicae respond exceptionally well to surgical treatment, which may be the only effective means of addressing this specific pathology [2, 64–66].

During knee arthroscopy to treat plica syndrome, a superolateral view, or direct medial portal view, should be utilized with a 70-degree scope to provide a panoramic view that allows optimal visualization of the impinging plica during dynamic movements of the knee [2, 5, 51]. Usually it is the mediopatellar plica that must be resected [1, 2, 5, 51]. Transection of the plica should include the entire plica down to its base, with careful dissection not to injure the medial retinaculum, as it would predispose to patellar subluxation [5]. The major complication associated with plica surgery is postoperative hemarthrosis. Therefore, resection of the plica should not extend to the capsule, as it incorporates a rich vascular territory [5]. Furthermore, aggressive use of hemostasis using electrocautery should be implemented to prevent hemarthrosis [5]. Additional complications related to arthroscopic surgery of the knee, but not specifically related to surgery for plica syndrome, should be considered as well. Such complications include septic arthritis, wound dehiscence, neurapraxia, neuromas, synovial fistulae, pulmonary embolus and complex

regional pain syndrome [67]. These complications are rare as most are associated only in patients with distinct risk factors, such as diabetes, steroid dependence, or a history of contracting these conditions [67].

Surgical intervention is a robust treatment method with a good prognosis as only mild symptoms may remain in the majority cases. However, surgical intervention may fail in the context of other concomitant knee pathology [3, 4, 7, 9, 24, 36, 42, 46, 49, 55, 68]. These pathologies may be secondary to the plica syndrome itself, such as chondromalacia or synovitis, and may persist despite addressing the pathological plica [4, 51]. Additionally, unrelated pathologies may coexist with an asymptomatic plica, such as medial meniscus tears, osteochondritis dissecans, ligament ruptures, or patellofemoral maltracking [51, 53]. They could be the primary source of symptoms and will not respond to surgical treatment directly aimed at plica syndrome, unless they are specifically and additionally addressed [4, 5, 51]. During a procedure to address any intra-articular pathology, it is worthwhile to consider also excising an asymptomatic plica to prevent a future occurrence of plica syndrome [4, 24, 55].

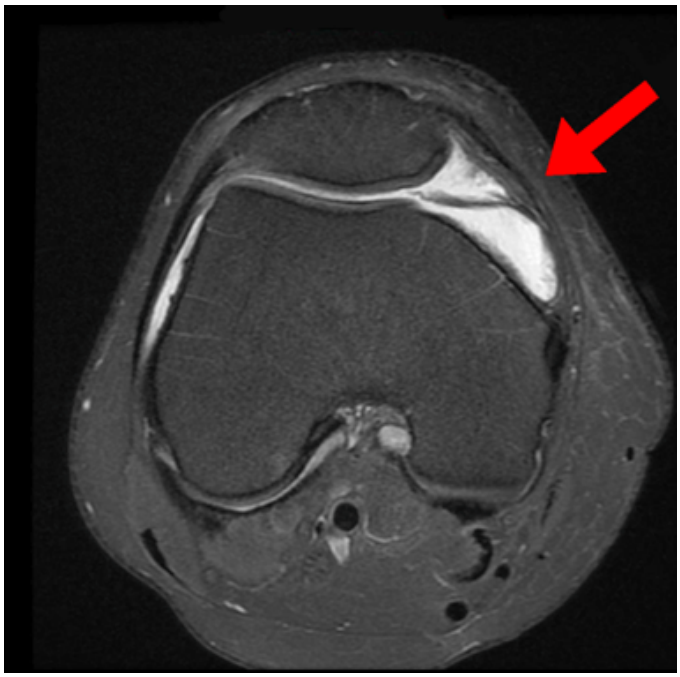

Figure 6: MRI radiograph depicting medial patellar plica syndrome.
Radiograph depicts medial patellar plica syndrome investigated with magnetic resonance imaging. The red arrow highlights thickened medial patellar plica with a large focal near full thickness medial patella facet cartilage defect. Associated joint effusion, synovial thickening and synovitis are also present.
[Case courtesy of Dr Andrew Dixon, Radiopaedia.org, rID: 18022].

CONCLUSION

This brief review has explored the embryology of the synovial knee joint, the anatomical classification of plicae, pertinent information regarding the development and diagnosis of plica syndrome, as well as the management for this pathology. A deeper understanding of the embryological origins of plica syndrome may assist the clinician in treating this common condition.

****

Acknowledgements
I would like to thank Joshua A. Cuoco for his critical review of the manuscript.

Author Contributions
Christopher L. Hoehmann – Substantial contributions to conception and design, Acquisition of data, Analysis and interpretation of data, Drafting the article, Revising it critically for important intellectual content, Final approval of the version to be published

Guarantor
The corresponding author is the guarantor of submission.

Conflict of Interest
Authors declare no conflict of interest.

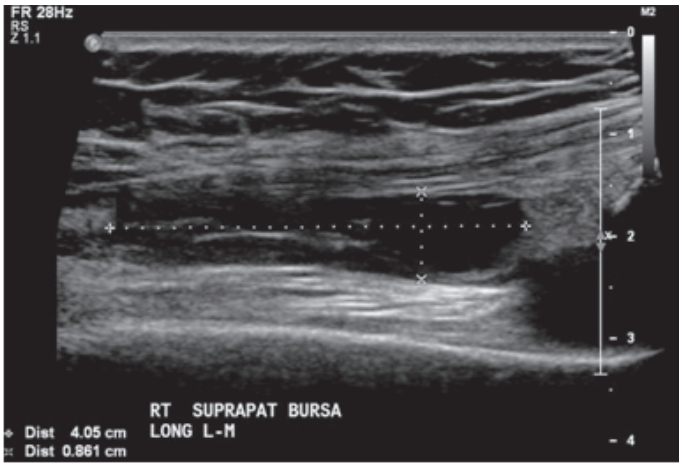

Figure 7: Ultrasonographic radiograph depicting localized fluid and synovitis in the suprapatellar recess secondary to suprapatellar plica syndrome.
Radiograph depicts fluid within the suprapatellar recess of the knee joint as a result of suprapatellar plica syndrome as investigated with ultrasonographic technology. The size of the fluid is 0.861 x 4.05 cm and is indicated by the white markers demonstrating the borders of the localized fluid. Associated synovial thickening and hypervascularity indicates concomitant synovitis.
[Case courtesy of Dr Andrew Dixon, Radiopaedia.org, rID: 41239].

REFERENCES

1. Sznajderman T, Smorgick Y, Lindner D, Beer Y, Agar G. Medial plica syndrome. Isr Med Assoc J 2009 Jan;11(1):54–7.
2. Al-Hadithy N, Gikas P, Mahapatra AM, Dowd G. Review article: Plica syndrome of the knee. J Orthop Surg (Hong Kong) 2011 Dec;19(3):354–8.
3. Bellary SS, Lynch G, Housman B, Medial plica syndrome: A review of the literature. Clin Anat 2012 May;25(4):423–8.
4. Dupont JY. Synovial plicae of the knee: Controversies and review. Clin Sports Med 1997 Jan;16(1):87–122.
5. Schindler OS. Synovial plicae of the knee. Curr Orthop 2004;18:210–19.
6. Irha E, Vrdoljak J. Medial synovial plica syndrome of the knee: A diagnostic pitfall in adolescent athletes. J Pediatr Orthop B 2003 Jan;12(1):44–8.
7. Dorchak JD, Barrack RL, Kneisl JS, Alexander AH. Arthroscopic treatment of symptomatic synovial plica of the knee. Long-term followup. Am J Sports Med 1991 Sep–Oct;19(5):503–7.
8. Boles CA, Martin DF. Synovial plicae in the knee. AJR Am J Roentgenol 2001 Jul;177(1):221–7.
9. Ewing JW. Plica: Pathologic or not? J Am Acad Orthop Surg 1993;1(2):117–21.
10. Berendsen AD, Olsen BR. Bone development. Bone 2015;80:14–18.
11. Goldring SR, Goldring Kelley MR. Biology of the Normal Joint. In: Firestein GS, Budd RC, Gabriel SE, McInnes IB, O'Dell JR, editors. Kelley and Firestein's Textbook of Rheumatology. 10ed. Philadelphia: Elsevier; 2017. p. 1–19.
12. Foster NC, Henstock JR, Reinwald Y, El Haj AJ. Dynamic 3D culture: Models of chondrogenesis and endochondral ossification. Birth Defects Res C Embryo Today 2015 Mar;105(1):19–33.
13. Wang Q, Green RP, Zhao G, Ornitz DM. Differential regulation of endochondral bone growth and joint development by FGFR1 and FGFR3 tyrosine kinase domains. Development 2001 Oct;128(19):3867–76.
14. Ogata S, Uhthoff HK. The development of synovial plicae in human knee joints: An embryologic study. Arthroscopy 1990;6(4):315–21.
15. Khan IM, Redman SN, Williams R, Dowthwaite GP, Oldfield SF, Archer CW. The development of synovial joints. Curr Top Dev Biol 2007;79:1–36.
16. Kent M, Khanduja V. Synovial plicae around the knee. Knee 2010 Mar;17(2):97–102.
17. Mérida-Velasco JA, Sánchez-Montesinos I, Espín-Ferra J, Mérida-Velasco JR, Rodríguez-Vázquez JF, Jiménez-Collado J. Development of the human knee joint ligaments. Anat Rec 1997 Jun;248(2):259–68.
18. Hartmann C, Tabin CJ. Wnt-14 plays a pivotal role in inducing synovial joint formation in the developing appendicular skeleton. Cell 2001 Feb 9;104(3):341–51.
19. Jin L, Li X. Growth differentiation factor 5 regulation in bone regeneration. Curr Pharm Des 2013;19(19):3364–73.
20. Edwards JC, Wilkinson LS, Jones HM, et al. The formation of human synovial joint cavities: A possible role for hyaluronan and CD44 in altered interzone cohesion. J Anat 1994 Oct;185 (Pt 2):355–67.
21. Mérida-Velasco JA, Sánchez-Montesinos I, Espín-Ferra J, Rodríguez-Vázquez JF, Mérida-Velasco JR, Jiménez-Collado J. Development of the human knee joint. Anat Rec 1997 Jun;248(2):269–78.
22. García-Valtuille R, Abascal F, Cerezal L, et al. Anatomy and MR imaging appearances of synovial plicae of the knee. Radiographics 2002 Jul–Aug;22(4):775–84.
23. Dandy DJ. Anatomy of the medial suprapatellar plica and medial synovial shelf. Arthroscopy 1990;6(2):79–85.
24. Tindel NL, Nisonson B. The plica syndrome. Orthop Clin North Am 1992 Oct;23(4):613–8.
25. Sakakibara J. Arthroscopic study on linos band (plica synovia is mediopatellaris). J Jpn Orthop Assoc 1976;50:513–22.
26. Munzinger U, Ruckstuhl J, Scherrer H, Gschwend N. Internal derangement of the knee joint due to pathologic synovial folds: The mediopatellar plica syndrome. Clin Orthop Relat Res 1981 Mar–Apr;(155):59–64.
27. Pekmezci M, Atay OA, Kerimoglu U, Aydingöz U, Tetik O, Doral MN. A complete supra-patellar plica with an unusual presentation. Knee Surg Sports Traumatol Arthrosc 2006 Sep;14(9):872–4.
28. Pipkin G. Knee injuries: The role of the suprapatellar plica and suprapatellar bursa in simulating internal derangements. Clin Orthop Relat Res 1971 Jan;74:161–76.
29. Strover AE, Rouholamin E, Guirguis N, Behdad H. An arthroscopic technique of demonstrating the pathomechanics of the suprapatellar plica. Arthroscopy 1991;7(3):308–10.
30. Demirag B, Ozturk C, Karakayali M. Symptomatic infrapatellar plica. Knee Surg Sports Traumatol Arthrosc 2006 Feb;14(2):156–60.
31. Shetty VD, Vowler SL, Krishnamurthy S, Halliday AE. Clinical diagnosis of medial plica syndrome of the knee: A prospective study. J Knee Surg 2007 Oct;20(4):277–80.
32. Kim SJ, Choe WS. Arthroscopic findings of the synovial plicae of the knee. Arthroscopy 1997 Feb;13(1):33–41.
33. O'Dwyer KJ, Peace PK. The plica syndrome. Injury 1988 Sep;19(5):350–2.
34. Blok A, Weiss W, Dolata T, Szczepaniec M. Medial synovial plica. Ortop Traumatol Rehabil 2005 Aug 30;7(4):397–400.
35. Kurosaka M, Yoshiya S, Yamada M, Hirohata K. Lateral synovial plica syndrome. A case report. Am J Sports Med 1992 Jan-Feb;20(1):92–4.
36. Boyd CR, Eakin C, Matheson GO. Infrapatellar plica as a cause of anterior knee pain. Clin J Sport Med 2005 Mar;15(2):98–103.
37. Amatuzzi MM, Fazzi A, Varella MH. Pathologic synovial plica of the knee. Results of conservative treatment. Am J Sports Med 1990 Sep–Oct;18(5):466–9.

38. Hardaker WT, Whipple TL, Bassett FH III. Diagnosis and treatment of the plica syndrome of the knee. J Bone Joint Surg Am 1980 Mar;62(2):221–5.
39. Lyu SR, Tzeng JE, Kuo CY, Jian AR, Liu DS. Mechanical strength of mediopatellar plica: The influence of its fiber content. Clin Biomech (Bristol, Avon) 2006 Oct;21(8):860–3.
40. Farkas C, Hargitai Z, Gáspár L, Kuki A, Csernátony Z, Szepesi K. Histological changes in the symptomatic mediopatellar plica. Knee 2004 Apr;11(2):103–8.
41. Pessler F, Dai L, Diaz-Torne C, et al. The synovitis of "non-inflammatory" orthopaedic arthropathies: A quantitative histological and immunohistochemical analysis. Ann Rheum Dis 2008 Aug;67(8):1184–7.
42. Kasim N, Fulkerson JP. Resection of clinically localized segments of painful retinaculum in the treatment of selected patients with anterior knee pain. Am J Sports Med 2000 Nov–Dec;28(6):811–4.
43. Lyu SR. Relationship of medial plica and medial femoral condyle during flexion. Clin Biomech (Bristol, Avon) 2007 Nov;22(9):1013–6.
44. Lyu SR, Hsu CC. Medial plicae and degeneration of the medial femoral condyle. Arthroscopy 2006 Jan;22(1):17–26.
45. Magee D. Orthopedic Physical Assessment. 2ed. Philadelphia: WB Saunders; 1992; p. 564.
46. Kinnard P, Levesque RY. The plica syndrome. A syndrome of controversy. Clin Orthop Relat Res 1984 Mar;(183):141–3.
47. McCarthy MM, Strickland SM. Patellofemoral pain: An update on diagnostic and treatment options. Curr Rev Musculoskelet Med 2013 Jun;6(2):188–94.
48. Kim SJ, Jeong JH, Cheon YM, Ryu SW. MPP test in the diagnosis of medial patellar plica syndrome. Arthroscopy 2004 Dec;20(10):1101–3.
49. Muse GL, Grana WA, Hollingsworth S. Arthroscopic treatment of medial shelf syndrome. Arthroscopy 2010 Mar;26(3):391–2.
50. Paczesny L, Kruczynski J. Medial plica syndrome of the knee: Diagnosis with dynamic sonography. Radiology 2009 May;251(2):439–46.
51. Shetty VD, Vowler SL, Krishnamurthy S, Halliday AE. Clinical diagnosis of medial plica syndrome of the knee: A prospective study. J Knee Surg 2007 Oct;20(4):277–80.
52. Jee WH, Choe BY, Kim JM, Song HH, Choi KH. The plica syndrome: Diagnostic value of MRI with arthroscopic correlation. J Comput Assist Tomogr 1998 Sep–Oct;22(5):814–8.
53. Monabang CZ, De Maeseneer M, Shahabpour M, Lenchik L, Pouliart N. MR imaging findings in patients with a surgically significant mediopatellar plica. JBR-BTR 2007 Sep–Oct;90(5):384–7.
54. Jemelik P, Strover AE, Evans G. Results of resection of medial patellar plica through a supero-lateral portal as a main arthroscopic procedure. Acta Chir Orthop Traumatol Cech 2008 Oct;75(5):369–74.
55. Mine T, Ihara K, Kawamura H, Seto T, Umehara K. Shelf syndrome of the knee in elderly people: A report of three cases. J Orthop Surg (Hong Kong) 2012 Aug;20(2):269–71.
56. Rovere GD, Adair DM. Medial synovial shelf plica syndrome. Treatment by intraplical steroid injection. Am J Sports Med 1985 Nov–Dec;13(6):382–6.
57. Crevenna R, Burian A, Oesterreicher Z, et al. Iontophoresis driven concentrations of topically administered diclofenac in skeletal muscle and blood of healthy subjects. Eur J Clin Pharmacol 2015 Nov;71(11):1359–64.
58. Xin C, Li-hong W, Yue Y, et al. A novel method to enhance the efficiency of drug transdermal iontophoresis delivery by using complexes of drug and ion-exchange fibers. Int J Pharm 2012 May 30;428(1–2):68–75.
59. Tomoda K, Terashima H, Suzuki K, Inagi T, Terada H, Makino K. Enhanced transdermal delivery of indomethacin-loaded PLGA nanoparticles by iontophoresis. Colloids Surf B Biointerfaces 2011 Dec 1;88(2):706–10.
60. Zuo J, Du L, Li M, Liu B, Zhu W, Jin Y. Transdermal enhancement effect and mechanism of iontophoresis for non-steroidal anti-inflammatory drugs. Int J Pharm 2014 May 15;466(1–2):76–82.
61. Rigby JH, Mortensen BB, Draper DO. Wireless Versus Wired Iontophoresis for Treating Patellar Tendinopathy: A Randomized Clinical Trial. J Athl Train 2015 Nov;50(11):1165–73.
62. Srinivasa A, Marshall JM. Effects of cyclooxygenase inhibition on vascular responses evoked in fingers of men and women by iontophoresis of 1- and 2-adrenoceptor agonists. J Physiol 2011 Sep 15;589(Pt 18):4555–64.
63. Souza J, Meira A, Volpato NM, Mayorga P, Gottfried C. Effect of phonophoresis on skin permeation of commercial anti-inflammatory gels: Sodium diclofenac and ketoprofen. Ultrasound Med Biol 2013 Sep;39(9):1623–30.
64. Gerbino PG II, Micheli LJ. Bucket-handle tear of the medial plica. Clin J Sport Med 1996 Oct;6(4):265–8; discussion 268–9.
65. Adachi N, Ochi M, Uchio Y, Kawasaki K, Yamasaki K. The complete type of suprapatellar plica in a professional baseball pitcher: Consideration of a cause of anterior knee pain. Arthroscopy 2004 Nov;20(9):987–91.
66. Madhusudhan TR, Kumar TM, Bastawrous SS, Sinha A. Clinical examination, MRI and arthroscopy in meniscal and ligamentous knee Injuries: A prospective study. J Orthop Surg Res 2008 May 19;3:19.
67. Salzler MJ, Lin A, Miller CD, Herold S, Irrgang JJ, Harner CD. Complications after arthroscopic knee surgery. Am J Sports Med 2014 Feb;42(2):292–6.
68. Broom MJ, Fulkerson JP. The plica syndrome: A new perspective. Orthop Clin North Am 1986 Apr;17(2):279–81.