

# 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

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## 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 sequela, 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

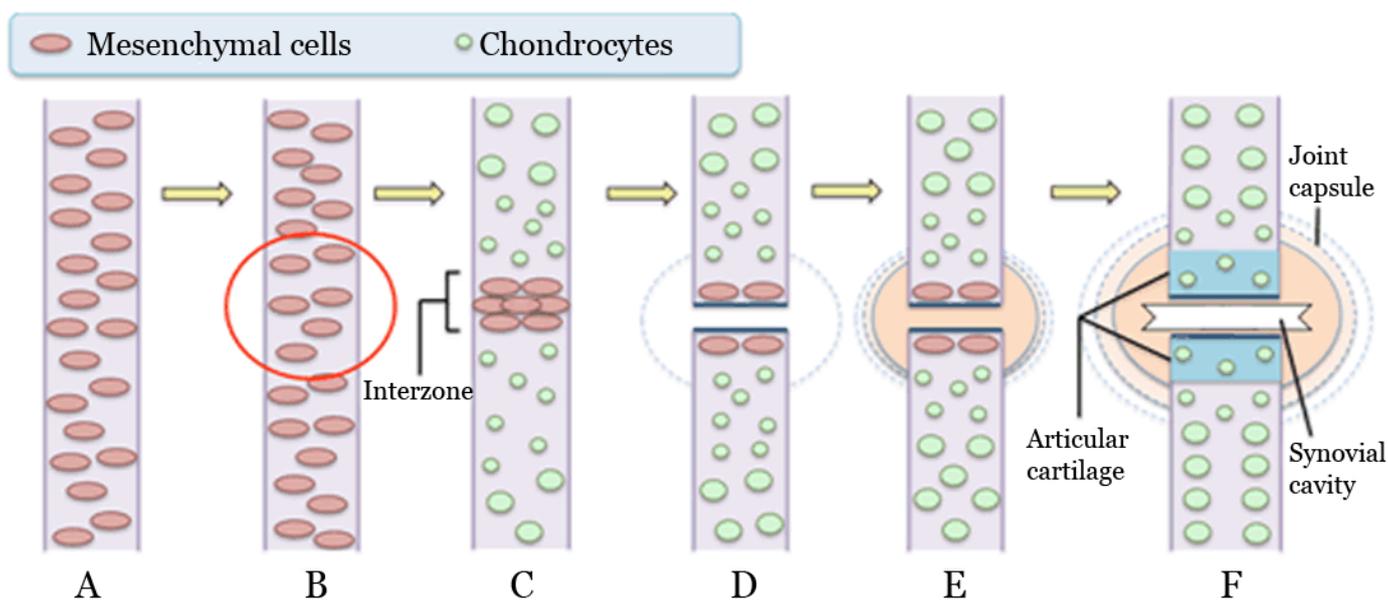


Figure 1: Synovial Joint Morphogenesis

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 morphogenetic 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

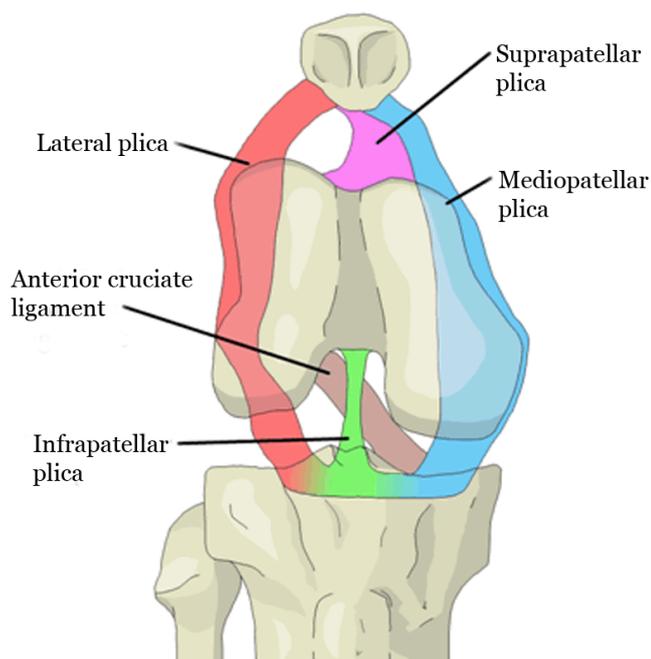


Figure 2: An anatomical depiction of the various manifestations of plicae in the knee joint.

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.

as an extension of the suprapatellar plica, while in other cases it may continue inferiorly as the infrapatellar plica [5].

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 femoral 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 synovialis suprapatellaris, 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 adiposynovial 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 desiccans, 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 decreased 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

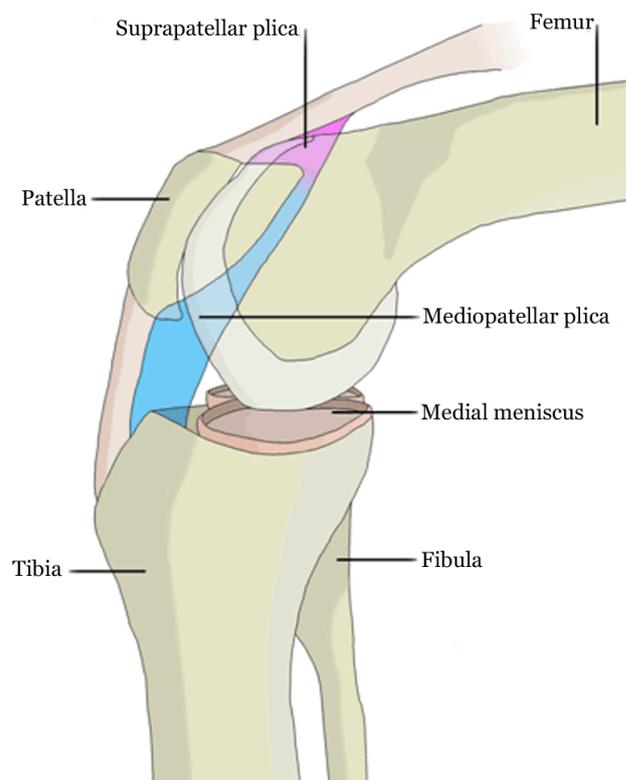


Figure 3: Medial view of a knee joint with mediopatellar and suprapatellar plicae.

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 indispensable 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 desiccans, patellar maltracking, loose bodies, osteophyte formation, fractures, and arthritis

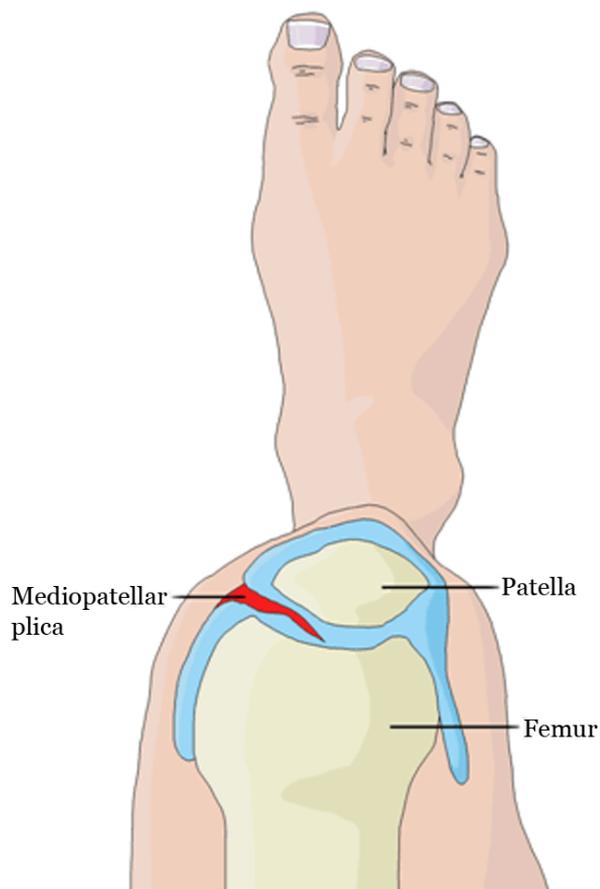


Figure 4: Superior view of the knee joint with pathologic mediopatellar plica. As the knee moves into flexion, a pathologic mediopatellar plica can impinge between the femur and patella. The impingement more commonly occurs when the knee is flexed 30 to 45 degrees [1]. This contributes to the knee pain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

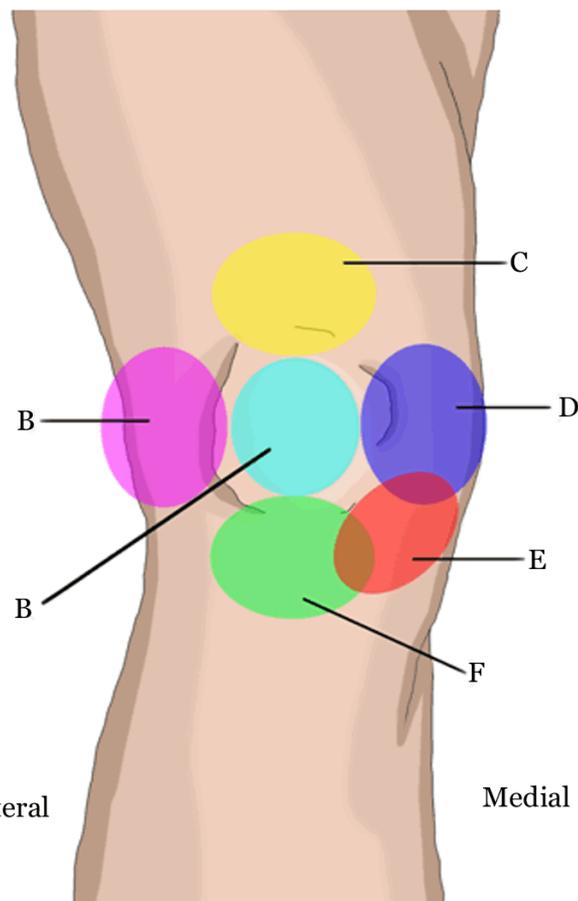


Figure 5: Differential diagnosis of knee pain based on location. Knee pain is a common complaint and has a wide differential diagnosis. The location of pain relative to the patella can hint at the etiology of the pain. The left side of the image represents the lateral side of the leg, and the right side 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 desiccans, 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 a 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 permeation by the NSAID ketoprofen with phonophoresis, however, an unexpected decrease in skin permeation 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].

## 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.

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## 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.

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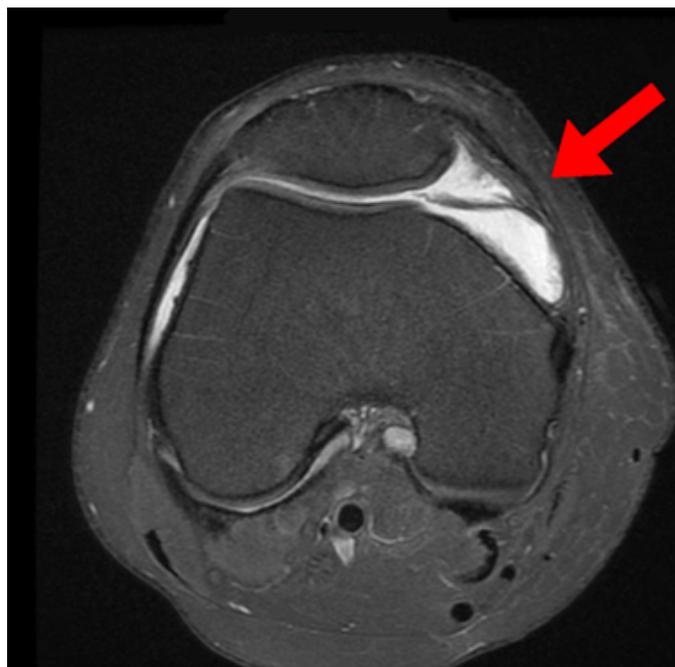


Figure 6: MRI radiograph depicting medial patellar plica syndrome.

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].

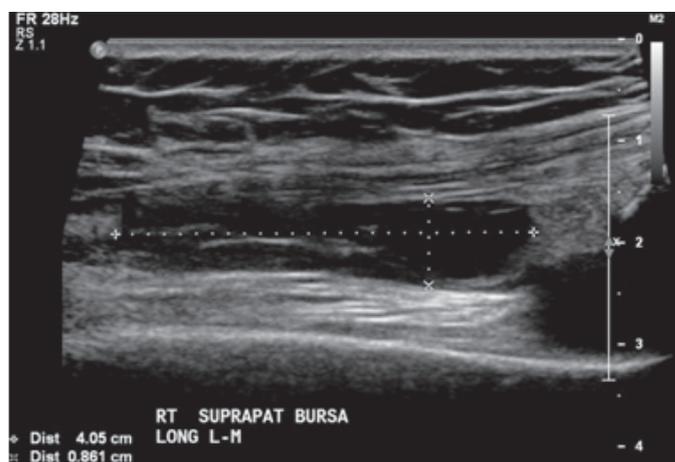


Figure 7: Ultrasonographic radiograph depicting localized fluid and synovitis in the suprapatellar recess secondary to suprapatellar plica syndrome.

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].

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