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Mucoid Degeneration of Anterior Cruciate Ligament – A Review of Literature

Sujit N Korday[1], Pushkar V Khandekar[1], Suhail S Kantawala[2], Nicholas Antao[2]

Abstract

Mucoid degeneration of the anterior cruciate ligament (MD-ACL), once thought to be a rare entity, is seen not so uncommonly in present day orthopaedic practice. Several hypotheses attempt to explain this entity. Clinically, it presents as knee pain with limitation of flexion or extension. There is no specific test available to clinically diagnose MD-ACL. Magnetic Resonance Imaging (MRI) remains the imaging modality of choice to diagnose MD-ACL. Therefore, a high index of suspicion is necessary to be able to diagnose this condition. This article reviews the available literature about the etio-pathological aspects of MD-ACL, its clinical features, radiological and arthroscopic findings, as well as various treatment modalities available.

Keywords: mucoid degeneration, anterior cruciate ligament, knee, arthroscopy, MRI.

Introduction

Mucoid degeneration, a rare disease of connective tissues, was first described in the cardiovascular system in the 1950's [1]. The first ligamentous mucoid degeneration was reported in the patellar ligament [2]. Kumar [3] first described mucoid degeneration of the anterior cruciate ligament (MD-ACL) in 1998. MD-ACL is characterized by infiltration of mucoid like substance (glycosaminoglycans) interspersed within the substance of the ACL [4, 5]. The ACL is hypertrophied causing impingement in the intercondylar notch resulting in chronic knee pain with restriction of knee motion [6]. Initially considered to be a rare entity, it is now considered to be under-diagnosed or mis-diagnosed, as it is often mistaken for partial ACL tears on Magnetic Resonance Imaging (MRI) [7–9]. This is reflected in the various names used to describe the same entity: Kumar [3] called the lesion "Mucoid cystic degeneration of the cruciate ligament"; Kim et al [10] described it as "Mucoid Hypertrophy", Bergin [11] named it "Mucoid Degeneration" & Salvati called it 'mucoid metaplastic degeneration' [12]. Cho et al [13] have even described concomitant mucoid degeneration of both anterior and posterior cruciate ligaments (PCL).

It is often thought to be due to degenerative changes in the knee and exists on a spectrum of ACL disease ranging from ganglion cyst to mucoid degeneration [14].

This article reviews the controversies and the current concepts regarding MD-ACL.

[1]Department of Orthopedics, V. N. Desai Hospital, Santacruz East, Mumbai, Maharashtra, India.
[2]Department of Orthopedics,Holy Spirit Hospital, Mahakali Caves Road, Andheri East, Mumbai, Maharashtra, India.

Address of Correspondence
Dr. Pushkar Khandekar,
Department of Orthopaedics, V. N. Desai Hospital, Santacruz East, Mumbai, Maharashtra, India.
E-mail: drpushkarkhandekar@gmail.com

Etio-pathogenesis

There is no universally accepted theory explaining the etiopathogenesis of MD-ACL [15] (Fig. 1). The "degenerative theory" suggests that this lesion may simply represent a continuum of the senescent age-related degeneration of the ligament [8, 16]. The presence of degenerative changes like meniscal tears, chondral lesions, osteoarthritis along with a predominant middle-aged group of population holds support for this theory [17]. Kwee et al [17] reported that knees with MD-ACL showed a greater progression of medial tibio-femoral joint space reduction, as compared to knees with a normal ACL. The "traumatic theory" hypothesizes that repeated microtrauma [18] due to impingement of a synovium deficient ACL on the intercondylar notch due to notch stenosis [19, 20] or due to prominent tibial eminences causes microcyst formation and eventual mucoid degeneration [21, 22]. This explains the presence of MD-ACL in younger, athletic patients. The deleterious effects of the synovial fluid and hemarthrosis on the synovectomized ACL have been documented by Amiel et al [23]. The "Synovial theory" [4, 16, 24, 25] postulates accumulation of synovial fluid inside the substance of ACL in a herniated pouch of synovium. This theory considers that mucoid degeneration and mucoid cyst lie on a continuum [14], and explains their coexistence [16]. The "ectopic synovial theory" describes the presence of congenitally displaced synovial tissue inside the ACL substance forming microcysts

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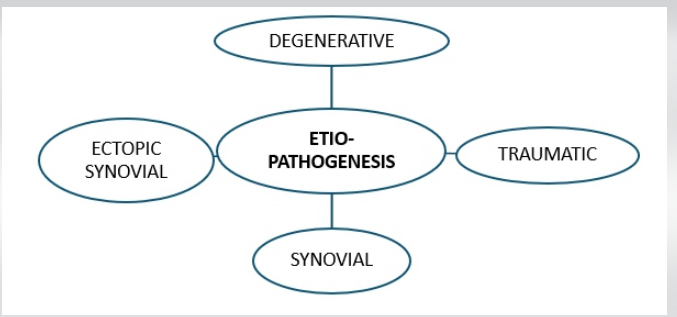

Figure 1: Theories of etio-pathogenesis of MD-ACL

[26]. Anatomical abnormalities like an increased posterior tibial slope [27, 28], steep notch with a smaller sagittal notch angle [29] or a narrower notch [15, 29] are associated with mucoid degeneration. Jung et al [27] found a correlation between increased posterior tibial slope and presence of MD-ACL.

Pathology

The characteristic feature of MD-ACL is the presence of a yellowish homogenous mass interspersed within the substance of a hypertrophied ACL with intact parallel oriented fibers, and with a frequently deficient synovial lining [4, 6].

On histopathological staining, eosinophilic mucoid materials were seen in between thin, fragile degenerated collagen fibers along with glycosaminoglycan deposition [13, 27, 30]. Alcian blue staining positivity is characteristic of mucoid degeneration as it has a high affinity for the glycosaminoglycan secreted by fibroblasts in the mucoid lesion [10, 20]. Nelissen et al [31] classified mucoid degeneration on conventional light microscopy findings as follows: stage 0, no mucoid degeneration with slight fibrosis in some areas; stage 1, slight degeneration starting between the collagen bundles, and collagen bundles still in a normal parallel arrangement; stage 2, moderate focus of mucoid degeneration with irregular structure of the collagen fibers; and stage 3, extensive muco-myxoid degeneration with total loss of fiber structure and bundle integrity [5].

Epidemiology

Bergin et al [11] screened 4221 knee MRIs for mucoid degeneration as well as mucoid cyst of the ACL (MC-ACL). Of these, 74 knees had radiological features consistent for MC-ACL or MD-ACL. The mean age of the study population was 42 years, with 76% of patients having discrete intra-ligamentous ganglia, 24% having features of MD-ACL and 35% having both. They calculated a prevalence rate of 1.8%. Salvati et al [12] examined 1215 knee MRIs for mucoid metaplastic degeneration of ACL (MMD-ACL) and found a total of 64 patients with MMD-ACL with a mean age of 44. The posterolateral bundle was affected in 17% of the patients while 83% had diffuse involvement with a prevalence rate of 5.3%.

In terms of age, there are two discrete groups of patients representing the two theories of MD-ACL: i) younger than 50 years of age with a more active lifestyle, and ii) above 50 years of age with sedentary lifestyle[32]. Pandey et al [4] reported a median age of 40 (21-59) with a sex ratio of 1.2:1, while Morice [33] reported a median age of 50.4 years with a sex ratio of 0.85. Ventura et al [34] reported a mean age of 57 (31-78) [34]. Saravanan [22] reported a mean age of 33 (31-70) while Khanna [7] reported median age of 36 (28-52) in females and 42 (30-54) in males. Kim et al [10] reported an age range of 42-80 years. Clearly, this is a disease of the middle-age [35].

Clinical Features

The main presenting complaints of MD-ACL are knee pain and loss of terminal flexion or extension. The most common complaint is pain [16] which is insidious in origin and can be posterior [5]or centrally in the knee behind the patella, and is often described by patients as "dull aching". Characteristically, there are no complaints of instability [36] and the Lachmann's, pivot shift and anterior drawer tests are negative with firm endpoint [5, 18, 30, 37]. There is usually no history of trauma [15]. The mean duration of symptoms was 5 months on an average [4]. The etiology of pain is often debated. Narvekar and Gajjar [38] attributed this pain to the increased volume and tension in the ligament stimulating nociceptor receptors in the substance of the ligament [18]. The pain can also originate from mechanical impingement of the hypertrophied ACL in the intercondylar notch [10] especially in patients with associated notch stenosis. Knee pain during flexion or extension can be explained due to involvement of the anteromedial or posterolateral bundle of the ACL which tightens during knee flexion or extension, and can cause restricted terminal flexion or extension respectively [13, 27, 29, 30].

MRI Findings

The gold standard investigation for diagnosing MD-ACL is MRI [1, 18, 19]. This is because MRI can identify intra-ligamentous pathologies [8, 11]. Bergin [11] set the following criteria for diagnosis of MD-ACL on MRI:

1. ACL fibers seen poorly on T1 weighted or proton density sequences but seen on T2 weighted images
2. Increased intensity signal seen on both T1 and T2 weighted images.
3. Intact fibers of ACL seen from origin to insertion.

The poorly defined fibers in T1 weighted images are often mistaken for partial or complete ACL tears [9]. McIntyre [9] described the appearance of intact fibers with low signal separated from each other by a prominent high signal within thickened ACL on fat-saturated PD-weighted images as "celery stalk" sign. Cha et al [29] measured notch width, notch index,

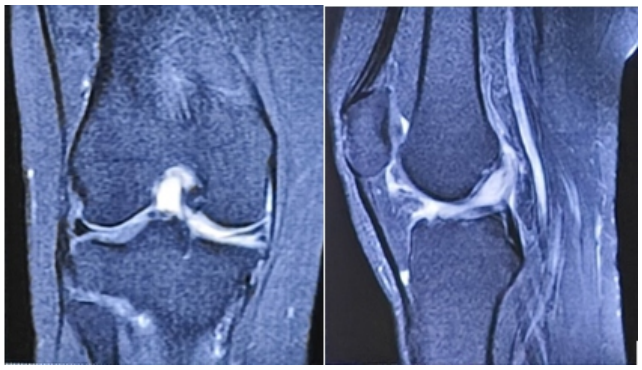

Figure 2: MRI images of MD-ACL showing hypertrophy of the ACL

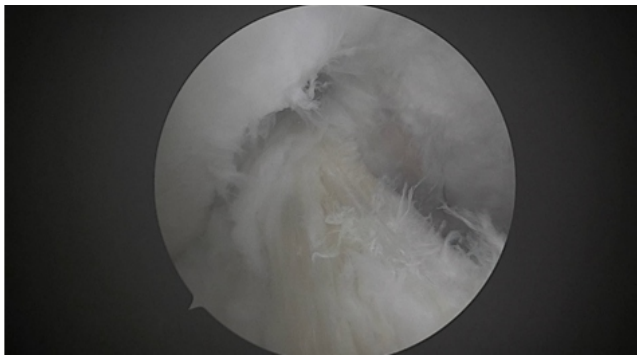

Figure 3: Arthroscopic appearance of MD-ACL showing absence of synovial lining and separation of ACL bundles and fibres

sagittal notch angle (SNA) and ACL diameter to record notch stenosis, vertical notch angle and ACL hypertrophy respectively. They concluded that patients with MD-ACL show a narrower notch, a steeper notch angle, and a smaller notch area than control groups. The ACL thickening was defined by Celikyay et al [36] when the thickness was not uniform and more than 7 mm. (Fig. 2)

Arthroscopic Findings

The characteristic finding of MD-ACL is the presence of a yellowish mass interspersed within the intact fibrillated substance of the ACL which can be expressed using a probe [4, 5, 7, 10, 20, 22, 27, 29, 33, 35, 39]. Often, this necessitates a posterior portal as they might not be visible through the standard anterior portals [11]. The hypertrophy of the ACL can either be in the anteromedial bundle, posterolateral bundle or both [7, 20, 30]. The hypertrophied ACL bulges in the intercondylar notch and causes impingement resulting in a flexion or extension deficit, depending on the position of the impingement [10, 13, 27]. The tension in the ACL is adequate as evidenced on probing as well as during anterior drawer test done intraoperatively [27, 30]. The synovial lining and the ligamentum mucosum are often conspicuous by their absence [4, 6, 7, 13, 14, 19, 27, 33, 40]. Based on these findings, McIntyre postulated the following arthroscopic diagnostic criteria for MD-ACL [9]:

1. Presence of continuity of the ACL with an increase in its volume
2. Presence of yellowish coloured material in the ACL which can be expressed on probing
3. Loss of synovial lining of the ACL

(Fig. 3)

Treatment

The initial treatment [26] for MD-ACL consists of conservative methods with oral analgesics and physiotherapy. Failure of conservative treatment is an indication for arthroscopic surgical management. Ahuja et al [41] described ultrasound-guided aspiration, fenestration, with local and steroid injection as an effective and minimally invasive technique for management of MD-ACL with excellent immediate to long-term results. The goal of the surgical treatment is to debulk the lesion without compromising the integrity of the ACL [7, 18]. Shelly [42] reported a case of metastatic adenocarcinoma of lung masquerading as MD-ACL and advocated taking a biopsy for histopathological examination for all cases. The treatment methods include partial or complete ACL debridement using a motorised shaver, or volume reductionplasty using radiofrequency ablation [33]. Partial debridement consists of excision of the mucoid degeneration and the degenerated ACL, along with notchplasty if deemed necessary [10, 13, 19]. The mucoid degeneration is probed out and removed using motorized shavers or basket forceps [38]. Kadi et al [43] described a longitudinal incision to split the ACL to look for intra-ligamentous mucoid degeneration. The adequacy of the debridement was decided by disappearance of impingement in flexion and extension of the knee while maintaining integrity of the ACL. Choukimath et al [44] described a systematic approach to debulking of MD-ACL. They suggested debulking at the femoral insertion of ACL followed by intra-ligamentous debridement using a motorised shaver in low power mode without suction. This is followed by radiofrequency ablation (RFA) of anteromedial bundle near the lateral femoral condyle. Next, adhesions are released between ACL and PCL using RFA in coagulation mode. They then recreated the Howell's triangle (interval between and ACL & PCL) proceeded with notchplasty using a motorised burr if osteophytes were present. The hypertrophic posterolateral bundle is examined in a "figure of 4" position [45] and debrided until the posterior horn of the lateral meniscus and the lateral tibial spine are visible. Regular flexion-extension maneuvers are performed to evaluate the presence of impingement and effectiveness of the debridement. Using an awl, they then performed microfractures at medial wall of lateral femoral condyle to promote healing. Finally, they performed Lachmann and anterior drawer tests to look for instability and the necessity for ACL reconstruction.

Narvekar and Gajjar [38] concluded that notchplasty was to be done if impingement persists in the notch after debulking of ACL, while Kim et al [10] & Lee et al [19] advocate performing a notchplasty of the lateral femoral condyle with osteotome [10] or a burr [38] in cases of notch narrowing followed by partial debulking of ACL. Partial resection of ACL results in immediate alleviation of pain with improvement in range of motion which was attributed to a decrease in volume and tension of ACL [1, 18, 30, 35, 38, 46]. A systematic review undertaken by Sweed et al [47] reported postoperative ACL laxity as a common finding after ACL arthroscopic debridement without symptomatic instability. The need for delayed ACL reconstruction should be discussed preoperatively with the patient. Kusano et al [40] reported delayed rupture in two patients with partial resection of MD-ACL in active sportsmen and recommend staged ACL reconstruction in patients who undergo total ACL excision.

Kumar [3] performed total excision of ACL. Himpe [1] recommended total ACL resection without notchplasty or reconstruction in elderly or sedentary population. Ventura et al [34] reported on a series of 25 cases and performed total ACL resections in 18 (72%) patients with only one patient needing reconstruction secondarily due to instability. Saravanan [22] performed complete ACL resection with primary reconstruction in a patient due to complete involvement of the ligament. Loganathan et al [20] reported arthroscopic ACL reconstruction as a better alternative to debridement in middle aged patients as well as elderly patients with active lifestyle.

Morice [33] proposed volume reductionplasty as an alternative to partial excision of ACL. They did not report the need for any ACL reconstruction. Hotchen [14] has also described the use of radiofrequency ablation for removal of synovial covering overlying the ACL, clearance of the Howell's triangle as well as the peripheries of the ACL which are causing impingement. They also advocated the use of RFA over shaver as it provides added benefits of coagulation. (Fig. 4)

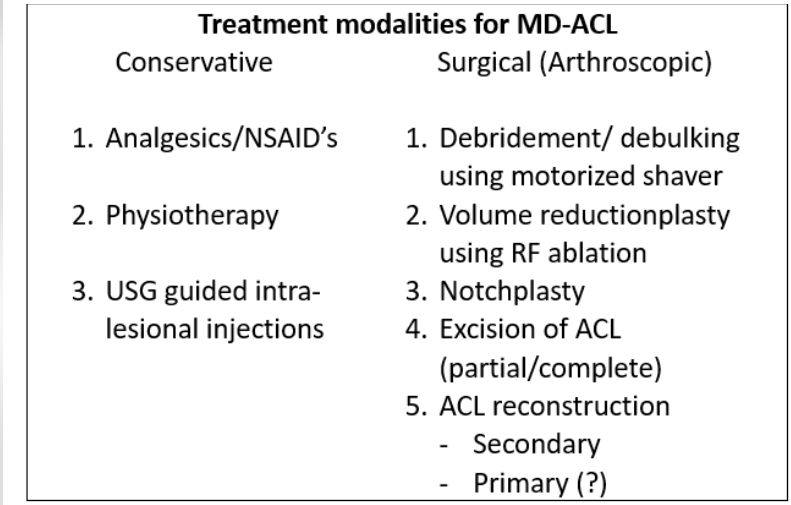

Figure 4: Treatment modalities for MD-ACL

Rehabilitation

Postoperatively, all patients were allowed to bear weight as tolerated with crutches or a walker and advised knee range of motion exercises [7, 10, 22, 30, 38, 40.] Kusano et al [40] and Ventura et al [34] both advocated rapid mobilization of the knee with no restraints on weight-bearing even for patients who had undergone complete ACL resection. Saravanan [22] advised a knee immobilizer for patients who had undergone an ACL reconstruction for protection of the graft. Morice [33] also did not restrict weight-bearing and knee range of motion in his series of volume reductionplasty.

Conclusion

MD-ACL, once thought to be a rare condition, is not as uncommon as was thought of in the past. It should be suspected in any patient with knee pain with limitation of knee flexion and/or extension. The ACL is stable on clinical examination. MRI is the 'gold standard' in clinching the diagnosis. Conservative treatment in the form of NSAID's and physiotherapy has been described but the literature on this is scanty. Surgical options described are arthroscopic debridement/de-bulking using a motorized shaver, volume reductionplasty using RFA, with or without a notchplasty, and partial or complete excision of ACL. There are no guidelines for the extent of debulking or excision of the ACL and notchplasty to be done. Most authors agree on titrating the extent of ACL excision and notchplasty to achieve an 'impingement-free' excursion of the ACL in the notch. Primary ACL reconstruction is rarely required and is reserved for young active individuals with documented ACL laxity at the end of the ACL debridement. There is no universal agreement on the indication for a primary ACL reconstruction following complete ACL excision. Most authors agree that young active patients should be counseled for the need for a second stage ACL reconstruction if they present with instability.

There is a need for further research to study the outcomes of conservative treatment and the role of intra-lesional steroids. The natural history of MD-ACL with and without treatment also needs to be studied in longer-term studies.

Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the Journal. The patient understands that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.
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