CASE REPORT

Acta Orthop Traumatol Turc 2013;47(3):208-211
doi:10.3944/AOTT.2013.2754

Mucoid hypertrophic mass of the anterior cruciate ligament causing knee extension block

Ji-Hun PARK, Dae-Hee LEE

Department of Orthopaedic Surgery, Korea University Anam Hospital, Korea University College of Medicine, Seoul, Korea

We report on the case of a patient with a knee extension block caused by a mucoid hypertrophic mass on the anterior cruciate ligament (ACL). Magnetic resonance imaging showed diffuse thickening of the ACL with a mass-like lesion anterior to the tibial attachment. Arthroscopy revealed a huge amorphous mass arising from ACL fibers at the tibial attachment that impinged on the femoral intercondylar notch on knee extension. The treatment included excision of the mass and accompanying abnormal ACL fibers and notchplasty, and resulted in complete recovery of knee motion and symptom relief without instability. This case shows that a degenerative mass in the form of a mucoid hypertrophied ACL can cause a knee extension block.

Key words: Anterior cruciate ligament; extension block; mucoid hypertrophy.

Mucoid hypertrophy of the anterior cruciate ligament (ACL) is an unusual entity and a rare cause of knee pain and motion restriction. The pathogenesis and pain mechanisms underlying mucoid hypertrophy remain unclear.[1-3] It is likely that the hypertrophic mass fills the space around the ACL resulting in a mechanical block. To date, ACL masses that produce a mechanical impingement have been reported to be associated with intra-articular ganglion cysts, synovial chondromatosis or cyclops lesions.[4,5]

The present report describes a case with a limitation of knee motion in extension due to a huge mass originating from mucoid hypertrophied ACL fibers. To our knowledge, there is only one other report describing such pathology.[6,7]

Case report

A 48-year-old female presented with increasing left knee pain on motion, which had begun 2 years ago. There was no history of major knee trauma. She reported a history of occasional swelling, but no feeling of giving way. Six months prior to presentation, she underwent an arthroscopic examination at a local hospital, but no formal diagnosis was made. The pain progressively worsened. At presentation, she had a 30° extension block. Pain was exacerbated at the end of the range of motion, especially during extension. There was no clicking or swelling. Tests for Lachman's sign, anterior drawer, pivot shift, and posterior drawer returned negative findings, indicating that there was no knee instability. A marked wasting of the left quadriceps muscles was observed. Plain radiographic findings were normal. Magnetic resonance imaging (MRI) of the knee showed a diffuse thickening of the ACL with high inhomogeneous signal intensity on both T1- and T2- weighted images. In addition, there was a mass-like lesion anterior to the tibial attachment of the ACL that showed high signal intensity on both T1- and T2-weighted images (Figs. 1a and b). Although the mass

Correspondence: Dae-Hee Lee, MD. Department of Orthopaedic Surgery, Korea University Anam Hospital, Korea University College of Medicine, 126-1, Anam-dong 5-ga, Seongbuk-gu, Seoul 136-705, Korea

Tel: 82-2-920-5924 e-mail: eoak22@empal.com

Submitted: September 9, 2011 **Accepted:** June 8, 2012

Available online at
www.aott.org.tr
doi:10.3944/AOTT.2013.2754
QR (Quick Response) Code:

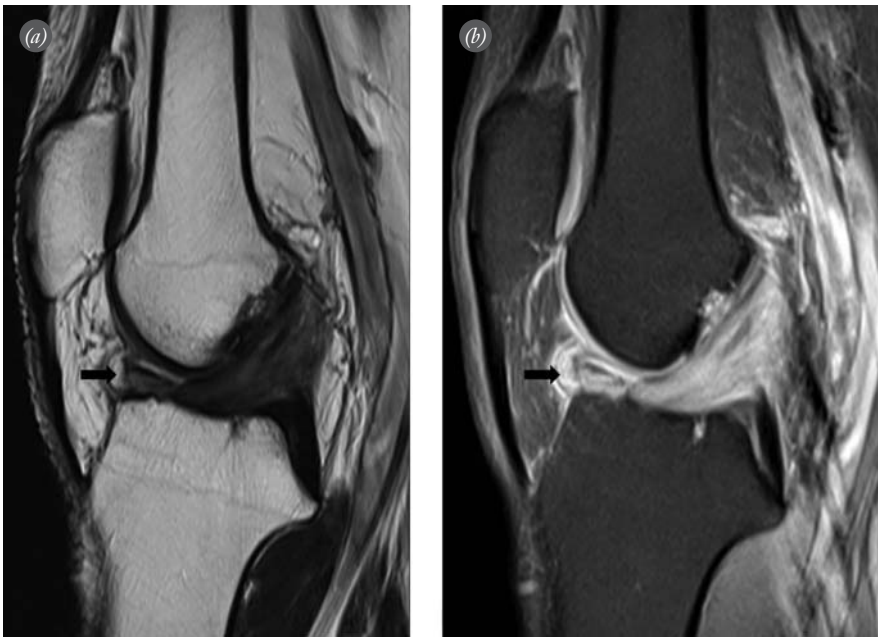

Fig. 1. Sagittal T2-weighted image **(a)** and T1-weighted image **(b)** showing a thickened and ill-defined ACL with high inhomogeneous signal intensity and a huge mass (arrow) anterior to the tibial attachment of the ACL, which impinged on the intercondylar notch.

was suspected to be the cause of the knee motion limitation, the MRI findings alone were not sufficient to make a diagnosis. Therefore, we performed an arthroscopic excision of the mass with a biopsy in order to obtain a definite diagnosis.

Arthroscopy revealed an unusual and amorphous mass arising from the ACL fibers at the tibial attachment. The mass was around the whole ACL fiber, with an impingement on the femoral intercondylar notch on knee extension, and thus it appeared to be the reason for the extension block (Fig. 2a). The ACL was diffusely hypertrophied, and it had yellowish discolored patches. The amorphous shape of the mass allowed it to fit into the contour of the notch on knee extension (Fig. 2b). There was no meniscal pathology medially or laterally. The posterior cruciate ligament (PCL) was normal.

We performed arthroscopic treatment consisting of debridement of the ACL and notchplasty. We progressively excised the hypertrophied ACL with the mass until we achieved impingement-free motion between the ACL and intercondylar notch. Following debridement, we tested ACL stability, and found that the joint was stable.

The excised ACL mass was examined histologically (hematoxylin-eosin staining). The examination revealed distorted collagen fibers with fibrin deposits and multifocal myxomatous changes indicative of mucoid hypertrophy of the ligament (Fig. 3).

Postoperatively, the patient was allowed to bear weight as tolerated with crutches and without limiting the knee motion. At her first follow-up visit on postoperative day 7, the patient showed full knee motion without pain. There were no signs of knee instability. At postoperative month 22, the patient showed maintenance of full knee motion, and he was pain free.

Discussion

The present report describes an unusual case with a mechanical block of knee extension due to ACL hypertrophy. The restricted knee motion appeared to be caused by an ACL mass being caught in the notch space upon extension. The mass was large and amorphous, and surrounded all of the ACL fibers at the tibial attachment, not just one bundle. The mass was found to fit into the contour of the intercondylar notch in knee extension. The pathological process appeared to be that a hypertrophic bulge was molded by repeated friction with the intercondylar notch in flexion-extension of the knee, and finally formed a huge mass at the tibial attachment of the ACL. Arthroscopic and histological findings indicated that the mass was degenerative in nature.

A search of peer-reviewed literature revealed 17 cases with mucoid hypertrophy of the ACL in middle-aged patients.[1,3,6,8-13] Restricted knee motion was present in 15 of those cases. In 12 cases, there was a restriction of knee flexion, while 3 cases had restriction of knee extension, as in the present case.[6,10] Motmans and Verheyden[12]

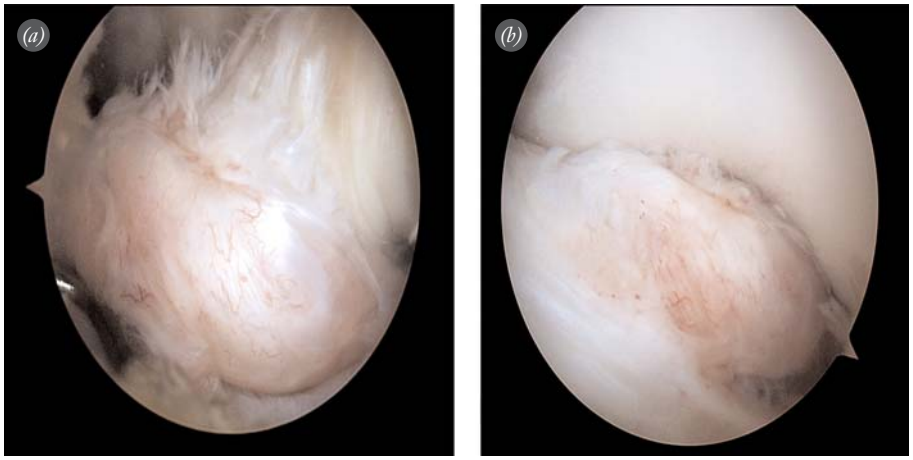

Fig. 2. **(a)** Arthroscopic view of a huge deformed mass arising from ACL fibers at the tibial attachment. **(b)** The mass impinged on the femoral intercondylar notch on knee extension, and the deformed shape allowed the mass to fit into the contour of the notch. [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]

found a mass interspersed within the hypertrophied ACL at the femoral insertion. The mass impinged on the PCL and posterior capsule, and caused flexion limitation. Hsu et al.[6] reported on a mass of the hypertrophied ACL at the posterolateral bundle, which impinged on the tibiofemoral joint and caused extension block. It is the only reported case with a histologically confirmed degenerative mass. The present case was similar to that case in terms of clinical presentation and degenerative nature of the mass. However, the present case differed in that it involved a huge amorphous mass at the tibial attachment and around the whole ACL fiber.

Differential diagnoses in the current case may have included an intra-articular ganglion cyst, giant cell tumor or synovial chondromatosis arising from the cruciate ligament. Those conditions may also cause pain, swelling, tenderness and loss of joint function. Previous reports demonstrated that intra-articular ganglion cysts and synovial chondromatosis can cause a mechanical block of knee motion similar to the present case.[4,7,14] In the current case, although MRI was very useful in locating the lesion, the findings did not allow us to identify the type of lesion. Having viewed the MRI data, we initially considered a tumorous condition such as a ganglion cyst or giant cell tumor, given that a degenerative mass is a rare entity and has no distinguishing MRI features.

Our arthroscopic treatment consisted of careful excision of the mass and debridement of the hypertrophic ACL bulging and notchplasty. We excised the mass piece by piece along with other bulbous portions of the ACL. This debulking procedure probably led to pain relief due to the decreased volume and tension within the ligament.[1] Although the necessity for a notchplasty remains debatable,[1,12] in the present case excision of the abnormal ACL tissue did not result in impingement-free movement. Therefore, a notchplasty was performed, achieving a normal movement without any effect on ACL stability. Lintz et al.[15] emphasized the unreliability of earlier reports claiming that patients were satisfied and did not complain of instability after ACL resection. The cited authors recommended notchplasty as an alternative to ACL resection to avoid postoperative laxity. Judicious debridement combined with notchplasty may be a better option than excessive ACL debridement alone.

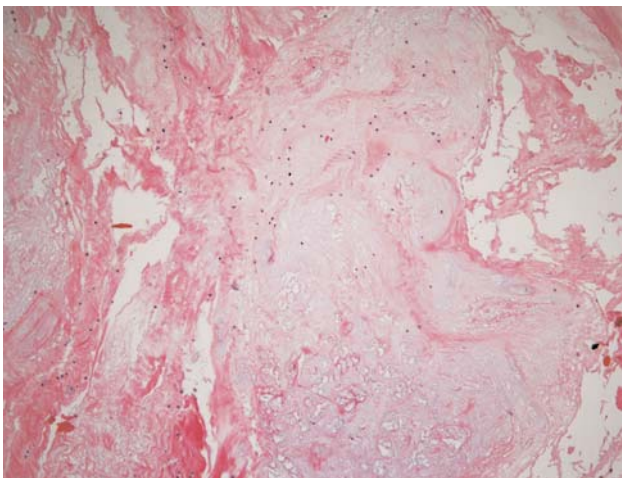

Fig. 3. Histological evaluation of a biopsy specimen from the excised ACL mass. Note the distorted collagen fibers with fibrin deposits and multifocal myxomatous changes (hematoxylin-eosin staining, original magnification ×100). [Color figure can be viewed in the online issue, which is available at www.aott.org.tr]

Although the pathogenesis of mucoid hypertrophy remains to be established, it appears it is the result of a continuum of degenerative changes triggered by repetitive minor trauma and the aging process.[2,6] Several recent studies support the idea that the degenerative pathophysiology of ACL features mucoid hypertrophy; the histological findings are in accord with this notion. Lintz et al.[15] described the histologic characteristics of ACL mucoid degeneration and distinguished the condition from a mucoid cyst. Makino et al.[16] also compared mucoid degeneration with post-traumatic ACL hypertrophy. The cited authors found that true histologic mucoid degeneration usually accompanied an intraosseous tibial cyst or a ganglion cyst; the two conditions share the same pathology arising secondarily to global degeneration of the joint. In the present case, the histological findings included the presence of an amorphous granular matrix located among thin collagen fibrils of the ACL, and myxomatous distorted fibers. These data suggest that there was a degeneration in the ACL rather than a simple traumatic hypertrophy per se.

Although limitation of knee flexion has been the commonest symptom of previously described cases, our case differs in that a limitation of knee extension was evident. This unique symptom is attributable to the anterior mass effect of the ACL. A possible mechanism of the symptom is the repetitive frictional interactions between the ACL and the intercondylar notch. Earlier, it was suggested that ACL degeneration might be caused by mechanical irritation by the intercondylar notch.[17,18] The cited authors proposed that the degenerative process was exacerbated upon repetitive microtrauma. In our case, notch irritation was arthroscopically observed, indicating that repetitive friction can result in formation of a deformed mass, in addition to simple hypertrophic degeneration. Although we do not know the mechanisms underlying the ACL mucoid hypertrophy formation in the present case, an important observation was that under arthroscopy the mass was found to impinge on joint motion of the knee. Our case supports the contention that mucoid hypertrophy of the ACL is part of a degenerative process, and it may explain knee symptoms in patients with mucoid hypertrophy.

In conclusion, the present report shows that knee extension limitation can result from the presence of a degenerative mass in the form of a mucoid hypertrophied ACL. Treatment of the mass involving the ACL with a careful debridement and notchplasty can result in complete symptom relief without any effect on knee stability.

Conflicts of Interest: No conflicts declared.

References

1. Narvekar A, Gajjar S. Mucoid degeneration of the anterior cruciate ligament. Arthroscopy 2004;20:141-6.
2. Kim TH, Lee DH, Lee SH, Kim JM, Kim CW, Bin SI. Arthroscopic treatment of mucoid hypertrophy of the anterior cruciate ligament. Arthroscopy 2008;24:642-9.
3. Fealy S, Kenter K, Dines JS, Warren RF. Mucoid degeneration of the anterior cruciate ligament. Arthroscopy 2001;17: E37.
4. Andrikoula SI, Vasiliadis HS, Tokis AV, Kosta P, Batistatou A, Georgoulis AD. Intra-articular ganglia of the knee joint associated with the anterior cruciate ligament: a report of 4 cases in 3 patients. Arthroscopy 2007;23:800.e1-e6.
5. Runyan BR, Bancroft LW, Peterson JJ, Kransdorf MJ, Berquist TH, Ortiguera CJ. Cyclops lesions that occur in the absence of prior anterior ligament reconstruction. Radiographics 2007;27: e26.
6. Hsu CJ, Wang SC, Fong YC, Huang CY, Chiang IP, Hsu HC. Mucoid degeneration of the anterior cruciate ligament. J Chin Med Assoc 2006;69:449-52.
7. Chung JW, Lee SH, Han SB, Hwang HJ, Lee DH. A synovial osteochondroma replacing the anterior cruciate ligament at the intercondylar notch. Orthopedics 2011;34:136.
8. Niki Y, Matsumoto H, Enomoto H, Toyama Y, Suda Y. Mucoid degeneration of the anterior cruciate ligament associated with subclinical instability in young patients. J Orthop Sci 2010;15:251-6.
9. Kumar A, Bickerstaff DR, Grimwood JS, Suvarna SK. Mucoid cystic degeneration of the cruciate ligament. J Bone Joint Surg Br 1999;81:304-5.
10. Melloni P, Valls R, Yuguero M, Saez A. Mucoid degeneration of the anterior cruciate ligament with erosion of the lateral femoral condyle. Skeletal Radiol 2004;33:359-62.
11. Nishimori M, Sumen Y, Sakaridani K. Mucoid degeneration of the anterior cruciate ligament - a report of two cases. Magn Reson Imaging 2004;22:1325-8.
12. Motmans R, Verheyden F. Mucoid degeneration of the anterior cruciate ligament. Knee Surg Sports Traumatol Arthrosc 2009;17:737-40.
13. Lancaster TF, Kirby AB, Beall DP, Wolff JD, Wu DH. Mucoid degeneration of the anterior cruciate ligament: a case report. J Okla State Med Assoc 2004;97:326-8.
14. Otsuka Y, Mizuta H, Nakamura E, Kudo S, Inoue S, Takagi K. Tenosynovial giant-cell tumor arising from the anterior cruciate ligament of the knee. Arthroscopy 1996;12:496-9.
15. Lintz F, Pujol N, Boisrenoult P, Bargoin K, Beaufils P, Dejour D. Anterior cruciate ligament mucoid degeneration: a review of the literature and management guidelines. Knee Surg Sports Traumatol Arthrosc 2011;19:1326-33.
16. Makino A, Pascual-Garrido C, Rolon A, Isola M, Muscolo DL. Mucoid degeneration of the anterior cruciate ligament: MRI, clinical, intraoperative, and histological findings. Knee Surg Sports Traumatol Arthrosc 2011;19:408-11.
17. Hernigou P, Garabedian JM. Intercondylar notch width and the risk for anterior cruciate ligament rupture in the osteoarthritic knee: evaluation by plain radiography and CT scan. Knee 2002;9:313-6.
18. Cha JH, Lee SH, Shin MJ, Choi BK, Bin SI. Relationship between mucoid hypertrophy of the anterior cruciate ligament (ACL) and morphologic change of the intercondylar notch: MRI and arthroscopy correlation. Skeletal Radiol 2008;37:821-6.