CASE REPORT

Acute Post-Traumatic Locked Knee - An Unmasking of a Rare Knee Disorder

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ABSTRACT

Locked knees are commonly caused by meniscal tears, floating osteochondral bodies, ruptured anterior cruciate ligament (ACL) stump, or other mechanical origins in the knee. Some locked knees occur spontaneously, while in most cases, by a preceding knee trauma. Locked knees are rarely caused by a pathological growth in the knee. More unusually is the occurrence of locked knee caused by a pre-existing pathological entity after a traumatic event. We report a rare case of locking in the knee by a pre-existing knee condition presented only after trauma to the knee. This case emphasizes that locking in the knee can be caused by a pathology that may be asymptomatic until it is revealed by a traumatic event.

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INTRODUCTION

Locked knee is a sudden incapacitation of knee extension despite a normal flexion range. It is a clinically-important symptom and warrants urgent intervention. Meniscal tears, ruptured ACL with prominent stump tissue and loose bodies are common causes of this condition (1). Less common causes include inflamed or ruptured plicae, intra-articular tumours, extruded fixation, gouty tophi, osteophytes in the joint and, in some reported cases, the cause is unknown (2,3). We report a rare case of acute traumatic locked knee, caused by a preexisting intra-articular knee pathology that only became symptomatic after the trauma. Surgeons should be aware of this phenomenon and be vigilant on possible preexisting pathologies in the knee, as a cause for locking.

CASE REPORT

A 42-year-old male car driver was admitted after a headon collision with another car, complaining of pain and locking in his left knee since the accident, despite his ability to bear weight with his left knee. Examination of the knee revealed mild effusion with tenderness over the medial joint line and the medial femoral articular surface. Range of motion was 10° to 90° and attempt at full extension caused pain in the knee. Ligaments were noted to be intact.

Magnetic resonance imaging showed knee effusion with intra-articular blood collection, features of osteochondral lesion in the lateral tibial plateau, and a lobulated lesion within the infrapatellar fat pad in keeping with haematoma formation (Fig 1).

In view of persistent locking, a decision to perform arthroscopic examination of the knee was made. The knee remained locked despite anaesthesia. Arthroscopy revealed a full-thickness 10 x 6 mm chondral injury in the medial femoral condyle and a lobulated mass measuring 15 x 15 mm just anterior to the medial femoral condyle, arising from the infrapatellar fat pad; while other structures in the knee were noted to be intact. There were no floating bodies noted in the joint. The locking was due to the lobulated swelling obstructing full extension (Fig 2). Excision of the mass was done piecemeal due to its size, and rusty-brown fluid was noted coming out from the mass during this process. Care was taken to remove all of the mass along with its margin, until normal synovial tissue was noted around the excision margin. After removal of



Figure 1: Sagittal MRI of the knee showing the infrapatellar PVNS on two slices (arrow)



Figure 2: Arthroscopic images of the PVNS with the knee in flexion (2A) and in extension (2B)

the mass, full extension in the knee was achieved and debridement followed by marrow stimulation procedure was performed for the chondral lesion. Other parts of the knee were also examined and no similar pathology was discovered. Washout and wound closure were done in the usual manner.

Histopathological examination revealed haemorrhagic fibrocollagenous tissue having hyperplastic synovium with papilliferous projections. It is composed of diffuse histiocytic infiltration some of haemosiderin-laden and foamy macrophages. Scattered multinucleated giant cells are also noted. The histiocytes were strongly and diffusely positive for CD68 immunohistochemical staining. All these features were consistent with tenosynovial giant cell tumour - localized type, or also known as pigmented villonodular synovitis (PVNS) (Fig 3).

Post-operatively he was started on immediate passive range of motion exercises and was taught non-weightbearing ambulation for 6 weeks to protect the chondral treatment. His swelling improved albeit slowly due to the chondral injury, and his knee movements were fully restored after the surgery. At 3 months follow-up, full weight bearing was allowed, his swelling recovered fully and he never had any recurrence of the locking.

DISCUSSION

Acute locked knee is an urgent condition requiring early



Figure 3: HPE images of the PVNS

intervention, as treatment delay will result in articular damage, joint contractures and persistent limp during walking (2). Even though there are many known causes of locking in the knee including PVNS (3,4), we believe this is the first report of acute locked knee caused by PVNS which had been asymptomatic prior to a traumatic episode, and the symptoms first appeared after a traumatic event. There were acute locked knee cases caused by PVNS described in the literature previously, but the presentations were atraumatic in nature.

PVNS affecting the knee is rare (3). PVNS causing locking is even more uncommon, but not unusual. It can be localized or diffuse and may typically arise from the menisco-capsular junction in the anteromedial part of the knee (3). Macroscopically, PVNS has a typical yellow-brown appearance from haemosiderin deposition. Histologically, just like this patient, it is characterized by synovial proliferation with presence of multinucleated giant cells and macrophages (5).

MRI findings in this patient were somewhat misleading in identifying the cause of his locking. Due to the preceding trauma, the mass in the infrapatellar fat pad was misinterpreted as haematoma collection. Furthermore, the reported osteochondral lesion in the lateral tibial plateau had suggested floating osteochondral fragments in the knee a more-likely cause to the locking.

There are a few possible explanations why this patient's locking occurred after his vehicular mishap. Firstly, the HPE showed some changes suggestive of acute traumatic bleeding within the tumour. Bleeding and the resultant increase in size had caused locking due to the PVNS's expansion. A localized PVNS, if large enough, is known to cause locking in the knee (4). Secondly, the chondral damage which occurred during the trauma may cause the PVNS to rub against the injured cartilage surface, and subsequent pain had prevented the patient from extending his knee fully. Therefore, excising the PVNS as well as treatment of the cartilage injury were thought to be beneficial for the patient in preventing future locking in his knee. Excision of the tumour can be done arthroscopically or through an open surgery, with some advocating a margin-free open excision (3, 4).

CONCLUSION

Surgeons need to be aware that an asymptomatic PVNS may exist long before it can manifest as a mechanical symptom in the knee, and that trauma may trigger its symptoms, leading to an unmasking of this rare pathology in the knee.

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