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Knee Arthroscopy Abnormal Findings

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Abstract:

Although arthroscopy of the knee is a minimally invasive procedure with less risk than endoscopy of the visceral cavities complications can and do occur. The operation is performed in very large numbers worldwide and although the percentage of patients experiencing problems is relatively small the total number is not insignificant. Complications are more likely to occur with more complex procedures such as meniscal repair, synovectomy and intraarticular reconstruction of both the anterior (ACL) and posterior (PCL) cruciate ligaments

Keywords: Knee, Arthroscopy, Osteoarthritis.

Introduction:

Arthroscopy of the knee is the most common orthopaedic procedure performed in the United States. Given the frequency of this procedure, it is hard to fathom that knee arthroscopy did not enter into the mainstream of orthopaedic surgery until the 1970s **(1)**.

Arthroscopy of the knee became an accepted practice in the 1970s. Several forward-thinking orthopaedists contributed to the early evolution of knee arthroscopy, developing the techniques and tools that are still used today **(2)**.

Over the past 40 years, knee arthroscopy has evolved from a rudimentary diagnostic tool to a state-of-the-art system of fiber optics and precision equipment. Knee arthroscopy has become a standard part of orthopaedics **(3)**.

It is the foundation for procedures ranging from the simple meniscectomy, to the multiligamentous knee injury, to cartilage restoration **(4)**.

<u>Meniscal Injuries</u>

Most meniscal tears appear due to degenerative causes, only few of them deriving from traumatic actions. Degenerative tears occur in older patients and are usually the result of normal tissue ageing, whereas traumatic injuries occur in younger people due to shear forces that the tibio-femoral joint creates during sport activities and trauma **(5)**.

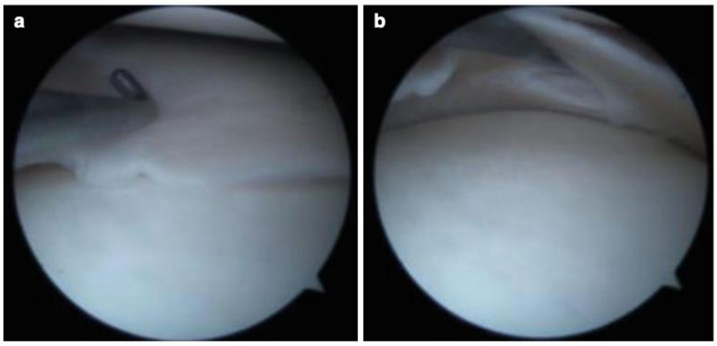


Figure (1): Occult lesion of the medial meniscus. The femoral surface can be viewed with no lesions (**a**). Longitudinal meniscal rupture on the tibial aspect (**b**) **(3)**.

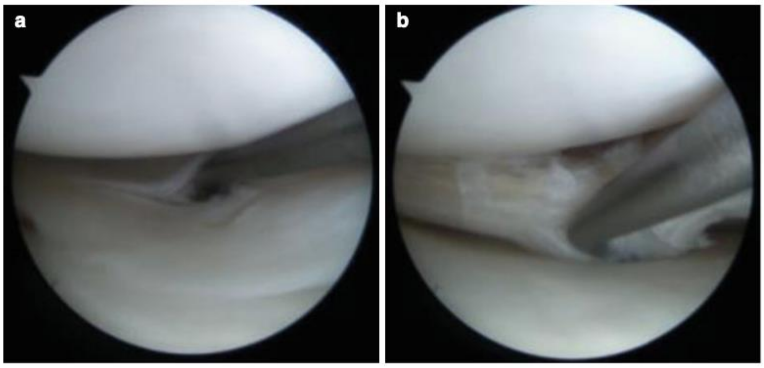


Figure (2): Apparently an incomplete longitudinal rupture of the medial meniscus (**a**). Upon further inspection the full extent of the rupture can be seen expanding further (**b**) **(3)**.

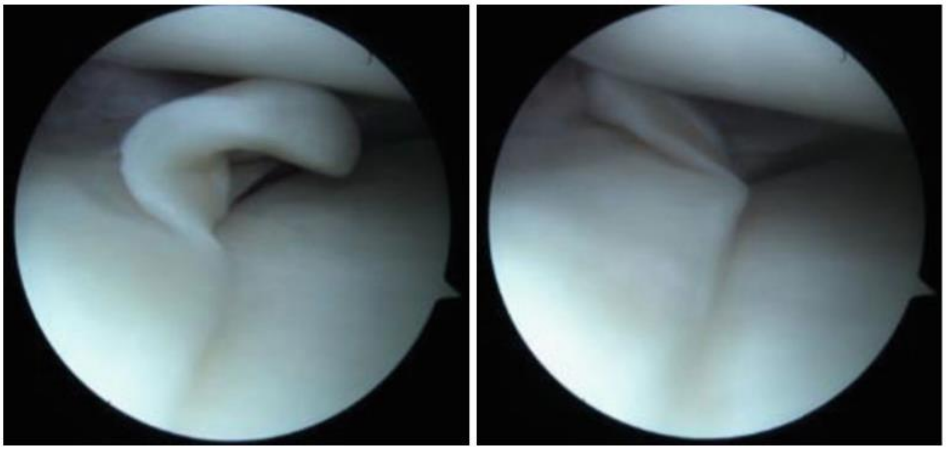


Figure (3): “Parrot-beak” medial meniscal rupture **(3)**.

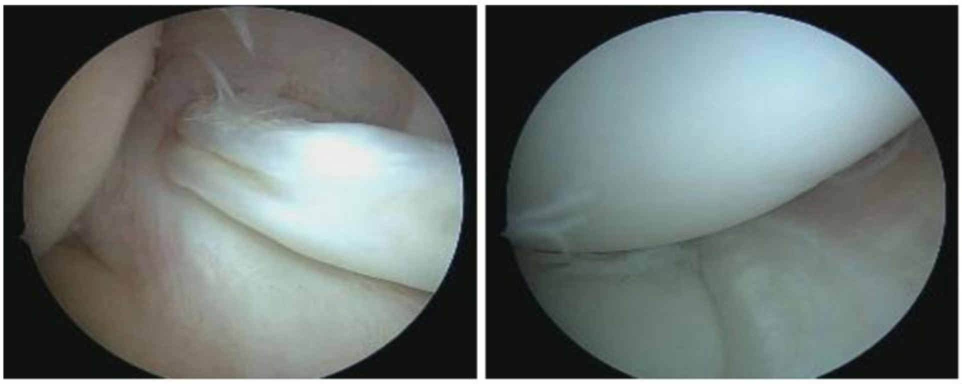


Figure (4): "Bucket-handle" lesion of the medial meniscus associated with an intercondylar dislocation of the torn piece **(3)**.

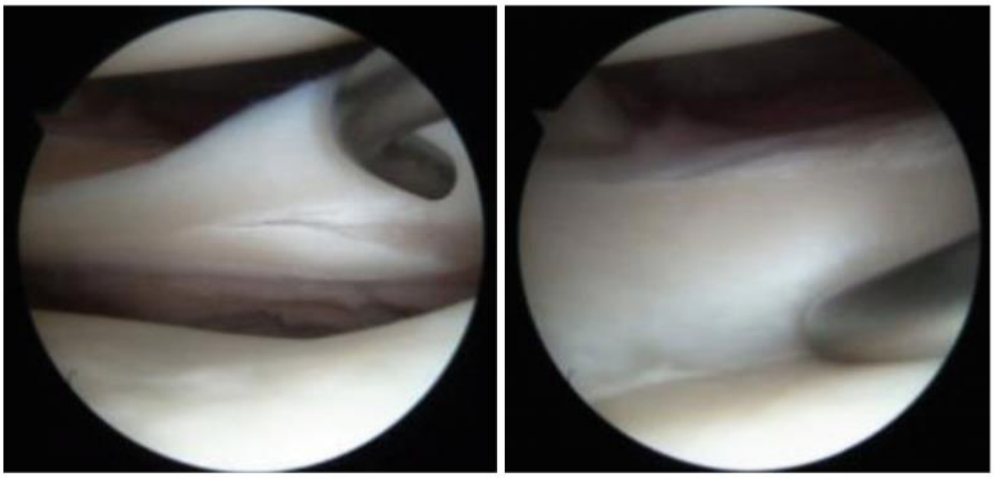


Figure (5): Stable longitudinal tear of the lateral meniscus **(3)**.

These two pathogenic paths also divide the meniscal tears in acute (first 2–3 weeks) and old (older than 3 weeks) lesions. Meniscal tears can also be classified by their shape, location and extent. Most traumatic tears are "bucket-handle" and flap lesions. Horizontal tears are usually created by a degenerative background **(1)**.

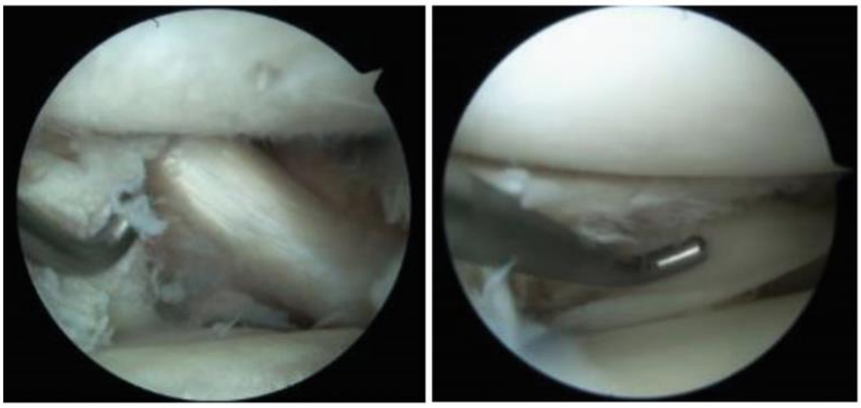


Figure (6): A complex degenerative tear of the external meniscus. Secondary condral pathology Outerbridge II. The popliteal tendon can be seen in the posterior aspect **(3)**.

Medial Meniscus Injury

A medial meniscus root tear can be visualized via a standard anterolateral portal. In addition, degenerative changes of the posterior meniscus horn and associated cartilage damage can be assessed. However, in the case of a posterior medial root tear, the torn margins may be closely approximated and overlooked without thorough evaluation. Therefore, examination of the root using a probe is mandatory. In case of a meniscal root tear, the root can be lifted up by the probe ("Lift-up test") and displaced anteriorly into the joint space. The arthroscopic surgeon should also assess the quality of the meniscal tissue and the ability to reduce the torn root to its insertion zone by manual probing **(6)**.

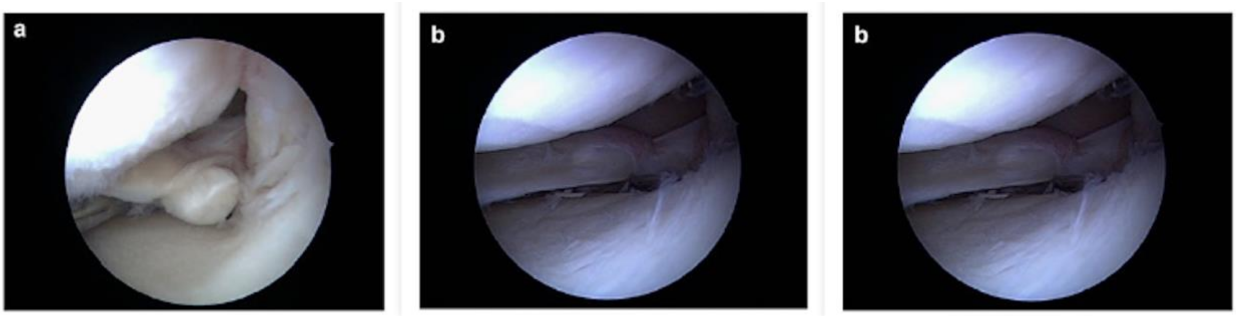


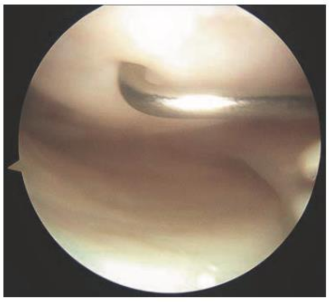
Figure (7): a Arthroscopic view of the posterior medial meniscus root via the anterolateral portal. Probing is essential to identify the tear and to determine the amount of impingement. **b** Large linear defect. **c** Medial meniscus root tear with accompanying degenerative changes of the meniscal body **(7)**.

A general problem in visualization of the posterior medial root is that the insertion zone is covered by the posterior cruciate ligament (PCL). One solution is to pass the arthroscope underneath the posteromedial bundle of the PCL into the posteromedial compartment. Another option is to visualize the posterior medial meniscus root using an additional posteromedial portal. For arthroscopic refixation techniques, an accessory posterolateral transseptal portal may be useful **(8)**.

Knee Osteoarthritis

Routine diagnostic arthroscopy is then carried out. Visualization of the suprapatellar pouch may reveal synovitis common in degenerative conditions and cartilaginous loose bodies. Routine synovectomy in this area is not typically carried out. The facets of the patella and trochlea are visualized, and grading of cartilage injury is noted using the Outerbridge classification **(1)**.

Table (1): Outerbridge grading of chondral surface lesions **(9)**.

Grade	Pathology	Arthroscopic Findings
Grade 1	Softening and swelling of cartilage.	

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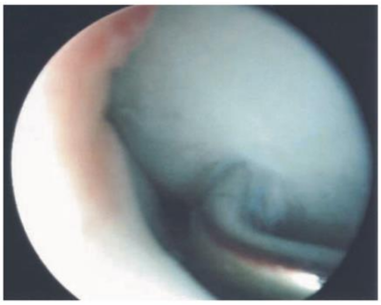
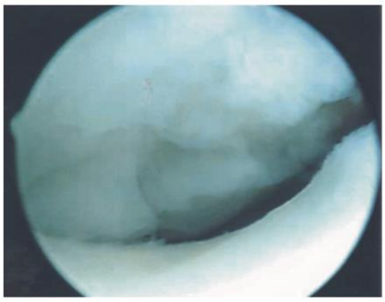
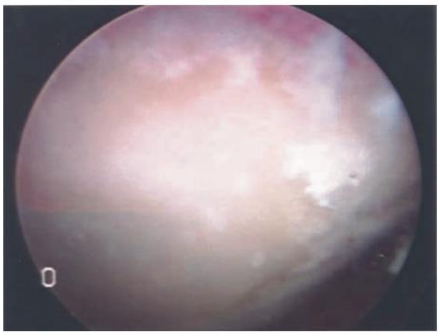
Grade 2	Fragmentation and fissuring, less than 0.5-inch-diameter lesion.	
Grade 3	Fragmentation and fissuring, greater than 0.5-inch-diameter lesion.	
Grade 4	Erosion of cartilage down to exposed subchondral bone.	

It is generally believed that the pain sources are the sub- chondral bone, synovial membrane, joint capsule ligament, patellofemoral joint, femoral notch, tendon insertions, and muscles, with synovium-derived pain and subchondral osteogenic pain as the main sources **(10)**.

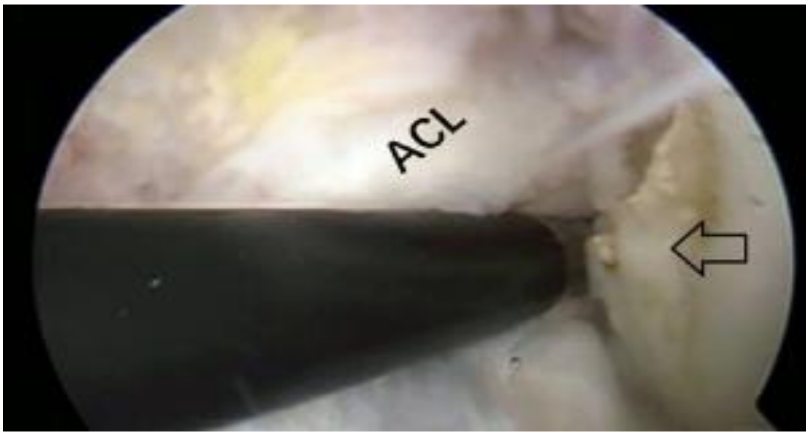


Figure (8): Arthroscopic view of left knee through anterolateral portal. An osteophyte (arrow) impinging on the anterior cruciate ligament (ACL) is defined **(9)**.

Changes in Knee Osteoarthritis

Inflammation

By initiating inflammatory processes and inducing a catabolic state of the cartilage, early surface changes seen as fibrillations extend distally, forming deep fissures, leading to cartilage delamination uncovering the calcified cartilage and the subchondral bone. The thinning of hyaline articular cartilage is accompanied by the expansion of underlying calcified cartilage, which additionally contributes to increased mechanical stress and the further production of catabolic factors **(11)**.

Furthermore, an enlarged layer of calcified cartilage advances into the overlying AC together with a duplication of the tidemark as another finding in osteoarthritic knee joints. These changes are caused by the penetration of vascular channels from the bone marrow, through the subchondral bone, calcified cartilage and tidemark to the articular cartilage, accompanied by sympathetic and sensory nerves **(12)**.

Subchondral Bone

Even though progressive cartilage damage and its eventual loss were the most mentioned features of the OA in the past, it is now well accepted that subchondral bone alterations and synovial inflammation also influence all other structures in the joint. Their structural changes are important disease characteristics, confirming that OA is a whole joint disease. Moreover, numerous studies suggest that interactions between cartilage and subchondral bone are fundamental for joint homeostasis, but also disease progression. Relative to the slower turnover rate of the AC, subchondral bone undergoes more rapid modeling and remodeling in response to changes in the mechanical environment **(13)**.

In established OA, the subchondral bone plate increases in volume and thickness. These changes are accompanied by alterations in subchondral trabecular bone in terms of its deterioration in the early stage and sclerosis in the late stage of OA. Furthermore, modifications of osteoblastic and osteoclastic activity result in bone turnover, causing subchondral bone lesions, cysts and osteophyte formation **(13)**.

Synovium

Synovitis is recognized as an important feature in patients with OA and has been associated both with symptoms and with structural progression. The inflammation in OA causes synovia to proliferate and induces the infiltration of T and B lymphocytes as well as mast cells. Synovial hypertrophy is defined as a synovial thickening of ≤4mm and effusion depth of fluid ≤4 mm or ≤4 mm in the suprapatellar recess. In addition, synovitis causes the extensive production of proteolytic enzymes, causing cartilage damage, while cartilage matrix catabolism produces molecules that propagate synovial inflammation **(14)**.

Synovial thickening is associated with radiographic and clinical progression of knee OA, pain and dysfunction that predominantly occurs posterior to the ACL and in the suprapatellar region. It is commonly identified during arthroscopy in approximately 50% of patients with OA. Moreover, areas of inflamed synovium tend to relate to locations of cartilage degradation **(15)**.

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