TREATMENT OPTIONS FOR JUVENILE OSTEOCHONDRAL KNEE DEFECTS

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Abstract
Articular cartilage is a avascular, aneural and alymphatic tissue that covers the articular ends of bones. It serves as al low friction high wear resistance surface to ideal provide mobility and strength. However due to its poor vascularization it has rather limited regenerative capabilities.

Cartilage lesions of the knee are rather common in children and adolescents representing a more difficult pathological entity due to the young age of the patients and the risk of secondary degenerative disease in case of a poor management. Osteochondral fractures (OCF) are most prevalent in this age group due to a higher risk of direct knee trauma, skeletal immaturity in girls predisposing them to lateral patellar dislocation with secondary OCF and a more elastic ligamentary apparatus. Juvenile osteochondritis dissecans (JOCD) has been a recognized entity for more than 100 years and despite extensive research there is no clear explanation of the cause of this disease, although repetitive trauma seems to play an important role in its pathogenesis.

Treatment options vary from conservative treatment in minor lesions to arthroscopic or open knee surgery. Arthroscopic surgery has the advantage of a minimally invasive technique, but there are cases where a conversion to open or a combined approach are necessary. Different techniques are available, depending on the size and stability of the defect and the stage of the disease such as internal fixation, bone marrow stimulation, allograft and autograft transplants (OATS) and ACI.

We propose a clinical presentation of the techniques used for arthroscopic management of osteochondral defects in skeletally immature patients.

Introduction
Lesions to the articular cartilage are a more frequent pathological entity with a potentially disabling outcome on the affected joint and overall life and activity level modifying results on the skeletally immature patient which can in turn result in a sedentary lifestyle. The most important lesions that affect the articular cartilage of the knee are osteochondral fractures, acute, traumatic lesions that regularly involve the condylar or patellar cartilage or juvenile osteochondritis dissecans, a chronic pathology that is believed to be caused by repetitive trauma(1-3)

JOCD has been recognised as a pathological entity for more than 100 years, but its pathogenesis is still widely debated, with no certain cause having been pinned down.

Youth is involved in a wide array of sports that require intensive family support, thus early recognition of osteochondral lesions of the knee is easier to make.(3, 4) Osteochondritis dissecans OCD of the knee can be classified in an adult form and a juvenile form. However, these two forms of OCD have a different natural evolution in terms of the disease and the outcomes that they produce. JOCD has a better outcome under conservative treatment than the adult form with high degree of family involvement and commitment, the "compliance triad" of physician, parent, and child being the key element when a conservative treatment plan for JOCD is initiated. JOCD can be further subdivided in a adolescent form a juvenile form, depending on the status of the growth physis:(1, 5, 6)

- Juvenile Osteochondritis Dissecans: wide open physis
- Adolescent Osteochondritis Dissecans: closing physis
- Adult Osteochondritis Dissecans: closed physis.

The incidence of JOCD in global population is of about 15 – 29 per 100 000 acording to Kocher et. al. and it is more frequent in children than in adults, with a reported prevalence of approximately 6 cases per 10,000 men and 3 cases per 10,000 women.(7)

Even though the name of the disease wrongly suggests an inflammatory etiology and it probably should have been better named “osteochondrosis” it stuck since in 1888, Konig used this term to describe the pathologic process that led to atramautic loose bodies of the knee and hip joints.

The etiology of JOCD was the center of a decades of debate with many hypotheses postulated, the cause of OCD was believed to be inflammatory, genetical, ischamical, repeated microtraumatisms on the articular cartilage or heterotopic ossification. Much discussion and arguments have been advocated for all of these but more recently most authors seem to agree to a multifactorial etiology centered around microtrauma. Trauma has been described as a potential etiology of OCD but the result of direct trauma is often a OCF but the predilection of OCD for the posterolateral portion of the medial femoral condyle suggests indirect trauma as a more likely cause, this is also supported by the repetitive impingement of the tibial spine on the lateral portion of the medial femoral condyle that takes place during the internal rotation movements of the tibia during gait.(7-10)

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The typical presentation of a young patient with knee OCD includes dull, nonspecific, poorly localized pain in the early stages of the disease, without knee trauma as opposed to a osteochondral fracture that produces sharp pain over the knee, caused by a traumatic event. Classic OCD pain will be antero-medial to the patella, accompanied by joint effusion and joint stiffness. Later stages of the disease can be accompanied by more severe symptoms such as locking, catching and giving away, usually indicative of a loose body if symptoms are constant and severe. The progressive nature of the lesion is indicated by symptoms that are increasing in frequency and intensity.

Method

Imaging of osteochondral lesions of the knee is regularly started with a X-ray in standard incidences (anteroposterior and lateral) and a special views, the tunnel view for the notch and the sunrise view for investigating the patellar articular surface. This can provide some information on the level of the injury, OCFs can be detected by standard radiographs take in the emergency department(fig.1), but in the early stages of OCD radiographic modifications can be absent. Knee MRI would be the next imaging modality that is often used in evaluating the osteochondral injured knee. It is useful both in acute lesions and in OCD detecting lesions invisible on standard radiographs and it also visualized chondral loose bodies, bone edema, joint fluid accumulation and associated pathology. Ultrasound has the advantage of dynamic scanning and decreased cost over MRI, in the hands of an experienced musculoskeletal operator it can prove to be a reliable investigation. Bone scintigraphy with technetium (99mTc scintigraphy) it can detect subclinical bilateral lesions as well as the degree of healing, which is proportionally higher with 99mTc uptake in the bone. This makes scintigraphy a useful tool in evaluating the joint status in a conservatively treated juvenile patient.(11-15)

Two classification systems are widely used for staging JOCD, the Hefti classification, based on MRI aspects and the arthroscopic Guhl classification.

The Hefti classification:
- Stage 1: Small change without clear margins of fragment
- Stage 2: Osteochondral fragment with clear margins but without fluid between fragment and underlying bone
- Stage 3: Fluid is visible partially between fragment and underlying bone
- Stage 4: Fluid is completely surrounding the fragment, but the fragment is in situ.
- Stage 5: Fragment is completely detached and displaced (loose body).

The Guhl classification
- Stage I: Intact Lesions
- Stage II: Lesions showing signs of early separation
- Stage III: Partially detached lesions
- Stage IV: Craters with loose bodies (fig 2.3 and 4)

Treatment of osteochondral lesions of the juvenile knee should be coordinated with the degree of articular cartilage impairment and the size of the lesion and most importantly the type of lesion. While conservative treatment for JOCD has a demonstrated success rate, with a secondary surgical indication, for acute OCF the primary indication is arthroscopic, open or combined refixation of the fragment while chronic lesions benefit from arthroscopic treatment as well.

For juvenile patients with OCD the first line of treatment is conservative therapy for which relatively good outcomes have been reported, Cahill reported a 56% success rate on a series of 92 knees followed scintigraphically for an average of 4.2 years with no knee immobilization and a complete restriction of athletic activities.

Other authors report similar success rates, with or without immobilization the higher number of stable lesions and healing under conservative treatment demonstrates that juvenile OCD has a much better prognosis than adult OCD. There is still debate over the role and length of immobilization but a general phased conservative treatment protocol has been produced (13, 16-19)

**Phase 1 (Weeks 0 – 6):**
- Knee immobilisation (cast or brace)
- Partial weight bearing with crutches
- Pain free knee is a criteria for phase 2

**Phase 2 (Weeks 6 – 12):**
- No knee immobilization
- Weight bearing as tolerated
- Physical therapy: ROM and low impact strengthening (quads, hams).
- No sports
- X-ray - healing with clinical improvement - phase 3.

**Phase 3 (from 3 – 4 months):**
- A complete absence of symptoms allows for gradually loading the knee (running, jumping and eventually sports) with progressively increased intensity
- Follow-up MRI/scintigraphy shows the degree of lesion healing.

**Treatment and results**

While conservative treatment is indicated for most patients with JOCD as the first line of treatment and surgical therapy is usually reserved for cases that failed to show signs of healing at follow-up, there are situations that imply surgery as the first line of treatment such as unstable lesions on MRI evaluation and articular loose bodies from a lesion with a detached osteochondral fragment. Also the surgical indication is definitive in cases of acute OCF.

Surgical therapy for osteochondral lesions includes several options such as bone marrow stimulation techniques, subchondral drilling, loose fragment fixation, cartilage abrasion, loose body removal, allograft transplant, OATS and ACI. Treatment algorithms have been conceived to address theese lesions, one of the most usefull is the one described by Garrido et. al., presented below (fig.5)(17, 20)
Fig. 1 Osteochondral fracture in a skeletally immature patient

Fig. 2, fig.3 and fig.4 This MRI shows a OCD Stage IV lesion on coronal T1 GFE, coronal STIR and sagital T1.
Fig. 5 Treatment algorithm for OCD (Garrido et al).

Fig. 6 Stable JOCD treated antegrade drilling.
Bone marrow stimulation include drilling the lesion for grade I and II stable JOCD and microfractures for chronic JOCD and OCF. Microfractures represent the piercing of small holes in the area of the defect to allow for nutrient and growth factor rich blood to penetrate trough to the joint space. This will allow the cartilaginous defect to be filled with fibrous cartilage. Subchondral drilling creates vascular channels in low grade stable OCD lesions or as a supplement to fragment fixation. Antegrade drilling (Fig. 6 Antegrade drilling) is done orthogonally under direct arthroscopic visualization using the standard portals or separate accessory portals. Retrograde driling necessitates C-arm radioscopic control to avoid joint penetration or the secondary mobilisation of the osteochondral fragment or ACL tibial aiming device with or without sonography. Iliac crest bone graft supplementation has also been added to help in healing. Good and excellent results have been reported in literature. The healing rate was of 80% in adolescent JOCD patients, with 70% or more being able to return to sports. (2, 17, 21-26)

Higher grade lesions (3&4) with unstable osteochondral fragments with a totally or partially detached fragment (trap door) and acute OCF’s have an indication for fixation of the fragment. This can be achieved via metal screws (Herbert), metal or bioabsorbable pins or bio-screws (fig. 7). Each have their advantages, some authors prefer the use of metal implants that need hardware removal surgeries to benefit from this second look arthroscopy to check the status of the cartilage.(15, 20, 24, 26-29)

Grafting the lesion is the most complex treatment method, reserved for the chronic, large size JOCD lesions. Autograft transplantation is a surgical option but availability is reduced to a small number of patients. It represents the harvesting of a osteochondral plugs from nonweightbearing areas of the joint and implanting them in the bed of the defect. One plug is sufficient for 2cm2 of lesion surface. Allografting is permitted in some countries by legislation, with instrumentation systems available to size and match the defect. NWB rehabilitations is started postoperatively. Good rates of survival have been reported for osteochondral grafts.

Autologus condhrocite implantation allows the repair of defects up to 10cm2 and up to 8mm in depth. Chondrocytes are harvested, grown in a cell culture in a lab and inserted in the defect in a pouch made by autologus periosteum or a synthetic colagen patch (fig. 8)(2, 5, 20, 30).

Conclusions

In conclusion, while JOCD if detected in a timely fashion with a compliant patient and adult supervision has a generally good outcome by conservative treatment, surgical options are available even for cases with advanced destruction of the articular cartilage with similary good results.

References


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