Osgood Schlatter syndrome

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Osgood Schlatter syndrome

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Purpose of review
Osgood Schlatter syndrome presents in growing children (boys, 12–15 years; girls, 8–12 years) with local pain, swelling and tenderness over the tibial tuberosity. Symptoms are exacerbated with sporting activities that involve jumping (basketball, volleyball, running) and/or on direct contact (e.g. kneeling). With increased participation of adolescent children in sports, we critically looked at the current literature to provide the best diagnostic and treatment guidelines.

Recent findings
Osgood Schlatter syndrome is a traction apophysitis of the tibial tubercle due to repetitive strain on the secondary ossification center of the tibial tuberosity. Radiographic changes include irregularity of apophysis with separation from the tibial tuberosity in early stages and fragmentation in the later stages. About 90% of patients respond well to nonoperative treatment that includes rest, icing, activity modification and rehabilitation exercises. In rare cases surgical excision of the ossicle and/or free cartilaginous material may give good results in skeletally mature patients, who remain symptomatic despite conservative measures.

Summary
Osgood Schlatter syndrome runs a self-limiting course, and usually complete recovery is expected with closure of the tibial growth plate. Overall prognosis for Osgood Schlatter syndrome is good, except for some discomfort in kneeling and activity restriction in a few cases.

Keywords
diagnosis, management, Osgood Schlatter syndrome

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Abbreviations

MRI magnetic resonance imaging
OSS Osgood Schlatter syndrome

1040-8703

Introduction

In 1903, Osgood [1] and Schlatter [2] separately described a painful condition of the anterior tibial tubercle characterized by partial separation of the tongue-like epiphysis of the tibial tuberosity, apparently caused by continued strain placed upon it by the patellar tendon. Osgood Schlatter syndrome (OSS) involves the tibial tuberosity in growing children and presents with local pain, swelling and tenderness of the tuberosity. The common age of presentation in boys is between the ages of 12 and 15 years and in girls is between the ages of 8 and 12 years [3,4]. The occurrence is reported to be greater in boys than girls [5,6] and it frequently presents bilaterally (20–30%) [7–9].

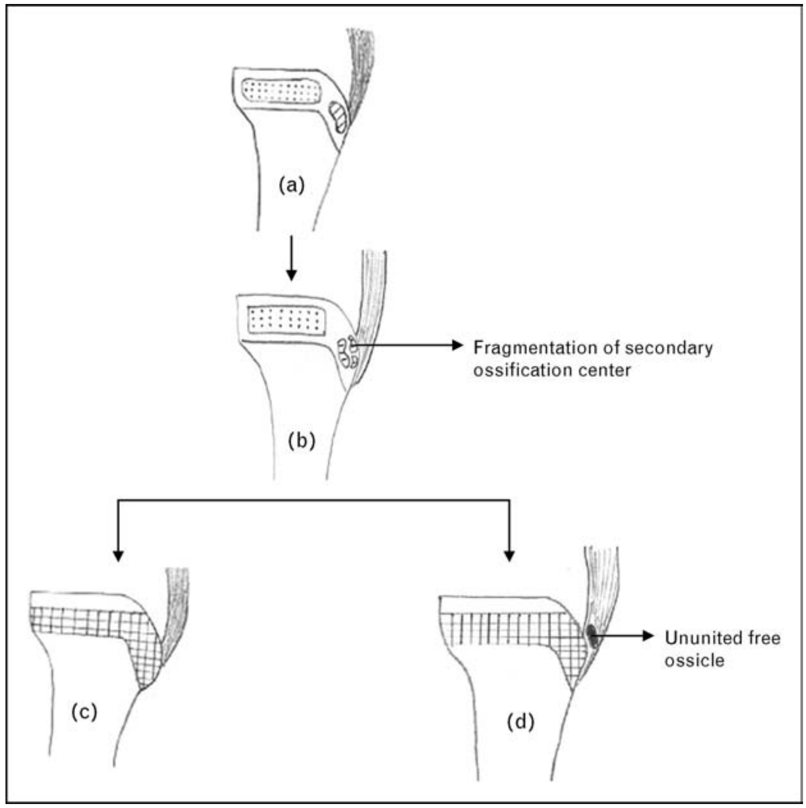
Etiopathogenesis

Currently it is widely accepted that OSS is a traction apophysitis of the tibial tubercle due to repetitive strain and chronic avulsion of the secondary ossification center of the tibial tuberosity. The repetitive strain is from the strong pull of the quadriceps muscle produced during sporting activities. The tibial tuberosity avulsion may occur in the preossification phase or the ossified phase of the secondary ossification center. Once the bone or cartilage is pulled away it continues to grow, ossify and enlarge. The intervening area may become fibrous, creating a localized nonunion (separate persistent ossicle), or may show complete bony union with mild enlargement of the tibial tuberosity (Fig. 1).

The theory of traction apophysitis and traumatic avulsion of the secondary ossification center of tibial tuberosity is supported by Ehrenborg and Engfeldt [5,10–12] and Ogden and Southwick [13]. Ehrenborg and Lagergren [14] described four radiological stages in the maturation of tibial apophysis (Fig. 2): cartilaginous stage (aged 0–11 years); apophyseal stage (aged 11–14 years); epiphyseal stage, the tibial apophysis coalesces with tibial epiphysis (aged 14–18 years); and bony stage, the epiphysis is fused (aged >18 years). On correlating the clinical findings, most OSS cases were seen in the apophyseal stage [5]. With work on cadaveric specimens Ehrenborg [10] suggested that ligamentum patellae fibers are attached distally to the fibrocartilage, which has high tensile strength, and hence a traumatic force causes fracture of the secondary ossification center of tibial tuberosity and not a fracture through the proximal growth plate.

Figure 1 Pathogenesis of an Osgood Schlatter lesion

(a) Appearance of the secondary ossification center of tibial tuberosity. (b) Fragmentation of the bony ossification center. (c) Complete healing and fusion of the apophysis with prominent tuberosity. (d) Ununited free ossicle. Adapted and modified from [4].



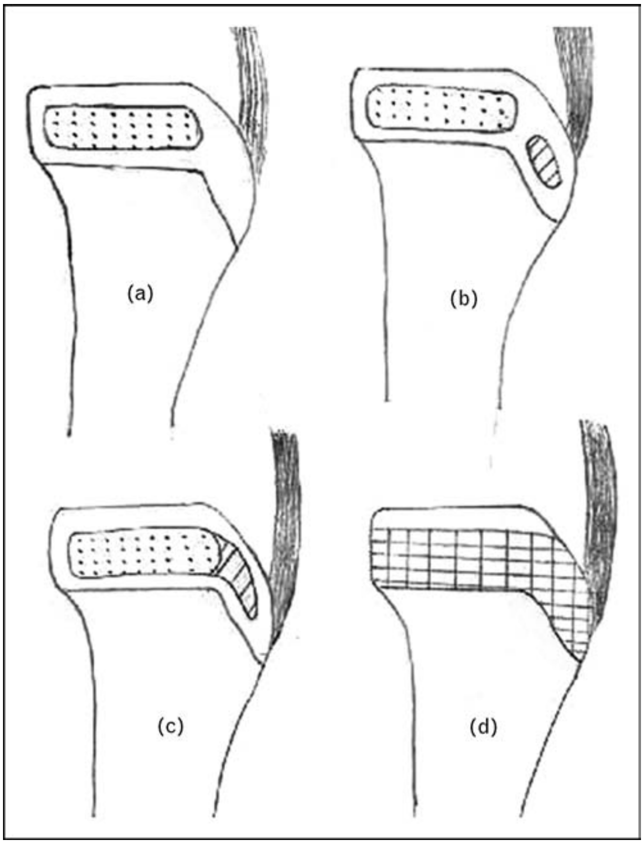
Ogden and Southwick [13] further expanded this work. They studied autopsy specimens of proximal tibiae of children aged from few days to 16 years. Histological analysis of the tibial tuberosity growth plate revealed three zones that gradually merge into one another. The proximal zone is analogous to the upper tibial growth plate and is formed of short cell columns. The middle zone is made of fibrocartilage (high tensile strength) with interposed layers of hyaline cartilage. The distal zone is formed mainly of fibrous tissue. As the tuberosity matures through the apophyseal to epiphyseal stages, the most important change is distal migration of the short cell columns replacing fibrocartilage. Based on these findings Ogden and Southwick suggested OSS to be caused by the developing secondary ossification center's inability to withstand the forces from the patellar tendon, resulting in avulsion of the center and later formation of extra bone between the fragments.

Pathological or normal variants of anatomy also have been suggested as etiological factors. Aparicio *et al.* [15] reported an association between OSS and patella alta in their radiological study. Jacob *et al.* [16] reported 125 patella alta in 185 knees with OSS. Almost all patients in their series healed, and they believed rectus femoris contracture caused the patella alta. Contradicting the above findings, Lancourt and Christini [17] reported patella infera in skeletally immature adolescents with OSS. They explained increased stress on tibial tuberosity due to the short length of the patellar ligament. Willner [18] reported pronated feet, genu valgum and internal rotation in all 78 patients with OSS. Demirag *et al.* [19], in their magnetic resonance imaging (MRI) study, compared patellar tendon insertion of 15 OSS patients with that of 15 normal adolescents. Their study showed that the patellar tendon was attached more proximally and had broader insertion above the tibial physis in OSS as compared with the controls. Gigante *et al.* [20] studied the relationship between OSS and torsional abnormalities of the lower limbs. They measured rotational profiles on computed tomography scans in 20 control knees and 21 knees affected with OSS. A significantly increased condylomalleolar angle and external tibial rotation was found in patients with OSS. Despite all the above reports, none of these variations have been universally accepted as etiological factors for lack of sufficient evidence.

Clinical features and natural history

Boys become symptomatic between the ages of 12 and 15 years and girls between the ages of 8 and 12 years [3,6]. Bilateral symptoms are observed in 20–30% of patients [7–9]. Kujala *et al.* [21] in a study of 389 adolescent athletes reported OSS in 21% of those actively participating in sports, as compared with only 4.5% in

Figure 2 Ehrenborg and Lagergren radiological stages in maturation of tibial tuberosity



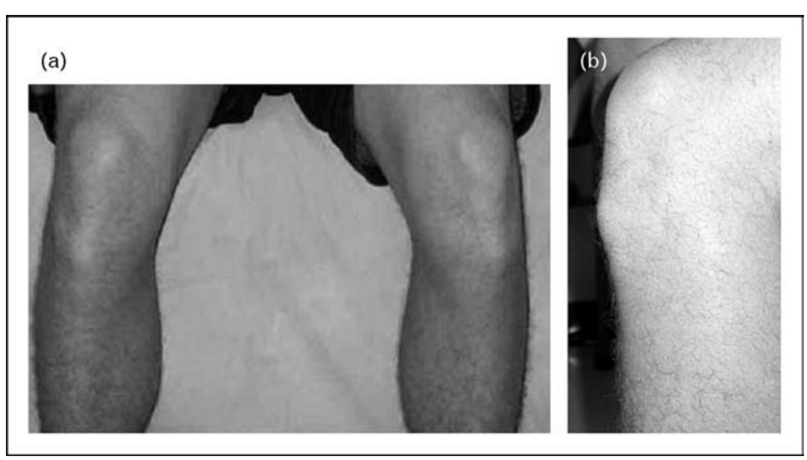
(a) Cartilaginous stage (aged 0–11 years). (b) Apophyseal stage (aged 11–14 years). (c) Epiphyseal stage, the tibial apophysis coalesces with tibial epiphysis (aged 14–18 years). (d) Bony stage, the epiphysis is fused (aged > 18 years). Adapted and modified from [4].

nonparticipants. Although complaints vary, they usually present with a vague history of gradual onset pain and swelling in the region of tibial tuberosity. Pain is mild and intermittent initially. In acute phase the pain is severe and continuous in nature. Pain exacerbates after sporting activity involving jumping (basketball, volleyball, running) and/or on direct contact (e.g. kneeling) [4]. Physical examination reveals tenderness, local swelling and prominence in the area of the tibial tuberosity (Fig. 3). Pain can be reproduced with extension of the knee against resistance. Once the acute phase heals, the pain and tenderness subside, and the only positive physical finding may be an anterior mass. Most investigators [3,4,6,22–24] claim spontaneous resolution and self-limiting nature of the symptoms, with recovery expected in about 90% of patients. Symptoms, however, may continue to wax and wane for 12–24 months before complete resolution. In approximately 10% of patients the symptoms continue unabated into adulthood, despite all conservative measures.

Krause *et al.* [23] reported natural history of OSS in 69 knees of 50 patients. They identified two groups of patients: those with radiological fragmentation who had separated ossicles or abnormally ossified tuberosity at review, and the other group with soft tissue swelling but no radiological abnormality at review. The mean age at onset of the symptoms was 12 years 3 months (range, 10–14 years) and at review the mean age was 21 years 6 months (range, 16–33 years). Seventy-six percent of patients did not have any limitation of activity, although 60% were not able to kneel without discomfort. They reported low incidence of anterior knee pain and no cases of premature proximal tibial epiphyseal arrest. There were no differences between those with or without radiological abnormality.

Ross and Villard [24] criticized the Krause *et al.* study for not mentioning the current level of activity and for a lack of any objective scores. Ross and Villard assessed the disability levels of 25 college-aged male individuals who had history of OSS and compared this with 25 healthy

Figure 3 Prominent tibial tuberosity



(a) Frontal view of the prominent tibial tuberosity in a 14-year-old boy with bilateral Osgood Schlatter syndrome. (b) Lateral view of the same prominent tibial tuberosity.

college-aged men with no previous history of OSS matched by age and intercollegiate sport. The disability was objectively reported on the Knee Outcome Survey Activities of Daily Living Scale and the Sports Activity Scale. The mean time from being diagnosed with OSS to participation in their study was 7.6 ± 2.4 years. Their results demonstrated significantly lower scores in the OSS group as compared with the normal healthy adults. One of the important weaknesses of this study was that all patients and normal participants were enrolled in the US Air Force Academy and led an active sporting life. Authors recommend caution while generalizing the above results in a less active population.

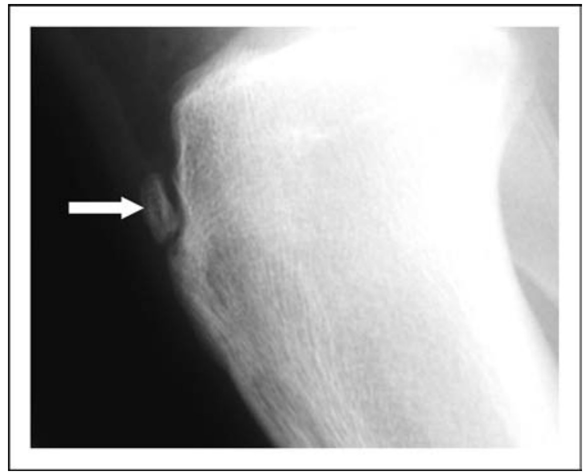
Radiographic features

OSS is a clinical diagnosis. Plain radiographs of the knee are recommended in all unilateral cases of OSS to rule out other conditions such as acute tibial apophyseal fracture, infection, or tumor (Fig. 4).

Plain radiographs

Plain radiographs (lateral view of the knee with the leg internally rotated 10–20°) show irregularity of apophysis with separation from the tibial tuberosity in early stages of OSS and fragmentation in the later stages. A persistent bony ossicle may be visible in a few cases after fusion of the tibial epiphysis (Fig. 5). Anterior soft tissue swelling may be the only sign observed very early in the acute phase when avulsion occurs through the cartilaginous portion of the secondary ossification center.

Figure 5 Tibial tuberosity showing ununited free ossicle

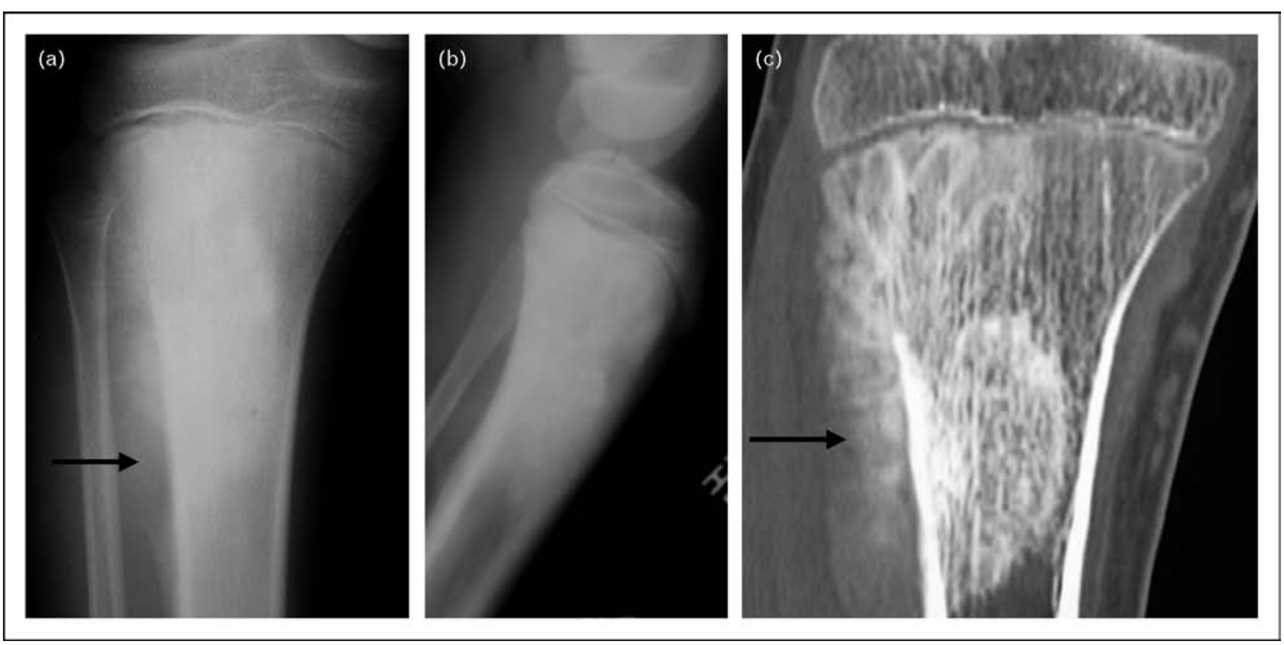


Lateral radiograph of tibial tuberosity showing ununited free ossicle (white arrow) in a 15-year-old girl with unilateral Osgood Schlatter syndrome.

Magnetic resonance imaging

Hirano *et al.* [25] studied MRI scans of 30 boys (40 knees) with diagnosis of OSS. Twenty-two boys were followed longitudinally for 1 year 6 months and MRI was performed every 6 months. They proposed five stages of the disease based on MRI scan appearances: normal, early, progressive, terminal, and healing. In the normal stage the MRI scan is normal, although the patient has

Figure 4 A 12-year-old boy initially treated (in an outside institution) as unilateral Osgood Schlatter syndrome



Subsequent radiographs at 6 months. (a) Anteroposterior and (b) lateral radiographs of his knee reveal sclerosis, new bone formation and periosteal elevation (arrow). (c) Coronal computed tomography scan reveals new bone formation (arrow). He was diagnosed with osteogenic sarcoma.

developed symptoms. The early stage did not reveal any MRI evidence of inflammation or avulsed portion of the secondary ossification centre. The progressive stage revealed the presence of partial cartilaginous avulsion from the secondary ossification centre. The terminal stage was characterized by the existence of separated ossicles. The healing stage was defined as osseous healing of the tibial tuberosity without separated ossicles.

MRI may assist in diagnosis of an atypical presentation. In future, with more understanding, it may play a role in staging of the disease and prognosticating the clinical course. The role in diagnosis, prognostication and management is currently limited.

Differential diagnosis

The important differentials are discussed with pertinent findings.

Sinding–Larsen–Johansson syndrome

Sinding–Larsen–Johansson syndrome is a traction apophysitis of the inferior patellar pole. The pathology is analogous to OSS except for the involvement of the inferior pole of the patella. Children present between ages 10 and 12 years with complaints of knee pain localized to the inferior patella. Slight separation and elongation or calcification is noted radiographically at the inferior patellar pole on the lateral view of the knee [26].

Hoffa's syndrome

The infrapatellar fat pad is a richly innervated tissue. Any injury to the fat pad can cause pain. Patients present with complaints of anterior knee pain, and maximal tenderness is noted in the anterior joint line lateral to the patellar tendon. The plain radiographs are usually normal. MRI scans characteristically reveal a low signal on all sequences within the fat pad due to fibrin, hemosiderin and/or calcification [27].

Synovial plica injury

Synovial plicas are normal synovial folds within the knee joint. They are remnants from embryological development of the knee. The mediopatellar or infrapatellar plica connects the lower pole of the patella to the intercondylar notch. Trauma and repetitive motion cause thickening, fibrosis and hemorrhage in this plica, giving rise to anterior knee pain. It can be diagnosed by MRI, which shows a curvilinear high T2 signal intensity within Hoffa's fat pad in the line of infrapatellar plica [28•].

Tibial tubercle fracture

Tibial tubercle fracture usually occurs in boys between the ages of 12 and 17 years. The mechanism of injury is violent contraction of the quadriceps or forceful flexion of the knee when the quadriceps is contracted. Patients present with complaint of pain, local swelling, knee effusion and an inability to actively extend the knee. Lateral radiographs of the tibia in 10–20° of internal rotation best reveal the fracture. Watson-Jones [29] classified fractures of the tibial tubercle into three types. In type I, a small distal portion of the tubercle is avulsed. In type II, the secondary center of the tubercle is hinged upward with the apex of the angulation being at the level of the proximal tibial physis. In type III, the fracture line extends through the proximal tibial physis into the knee joint. The presenting history and plain radiographs of the knee differentiate tibial tubercle fracture from OSS.

Other differentials to be considered include idiopathic anterior knee pain, tumor and infection.

Treatment

There are no prospective, randomized, controlled, interventional studies evaluating the treatment of OSS. The data and evidence are therefore available from retrospective studies. Once the diagnosis is made and other pathologies are ruled out, nonoperative treatment is initially recommended.

Nonoperative treatment

The standard nonoperative treatment of OSS includes application of ice, limitation of activities, oral antiinflammatory medications, protective knee padding and physical therapy. Those with mild pain and no weakness are allowed to continue sporting activity with the use of antiinflammatory medication and knee padding. Those who have moderate to severe pain may benefit with activity modification, rest and antiinflammatory medication. Cast immobilization has fallen out of favor due to the risk of quadriceps wasting. A few patients with severe prolonged pain, however, may benefit from a short period of rest in a knee immobilizer.

Physical therapy and a conditioning program form an important part in the management of OSS. Physical therapy is commenced once the acute symptoms have abated. Ross and Villard [24] recommended exercises for strengthening and improving flexibility of the surrounding musculature, including the quadriceps, hamstring, iliotibial band and gastrocnemius muscle. High-intensity quadriceps-strengthening exercises increase stress across tibial tuberosity and are initially avoided. Low-intensity quadriceps-strengthening exercises, such as multiple-angle quadriceps isometric exercise, are therefore instituted earlier in the conditioning program. High-intensity quadriceps exercises and hamstring stretching are introduced gradually.

Mital *et al.* [30], in a series of 118 patients, reported pain relief in 88% of the patients treated nonoperatively with intermittent limitation of activity or immobilization in a cylinder cast. In a study by Beovich and Fricker [31], 91% of adolescent athletes had symptomatic relief with ice, aspirin and mild activity modification, and only two patients out of 22 needed to stop playing all sports for any period of time – none of them required surgery. Hussain and Hagroo [32] followed 261 patients with OSS for 1–2 years and reported that 237 (91%) patients responded well to activity modification, rest and nonsteroidal antiinflammatory medication.

Surgical treatment

The symptoms continue unabated in 5–10% of patients, despite all conservative measures. These patients complain of local pain, difficulty in kneeling and restricted activity into adulthood. Depending on the symptomatology and affected quality of life, surgical intervention may be considered after skeletal maturity [4,30,33,34].

In the literature we identified different surgical procedures such as drilling of the tibial tubercle [6], excision of the tibial tubercle (decreasing the size) [4,34], longitudinal incision in the patellar tendon [35], excision of the ununited ossicle and free cartilaginous pieces (tibial sequestrectomy) [4,36], insertion of bone pegs [37] and/or a combination of any of these procedures. All investigators have reported their techniques with variable results.

Bosworth [37] considered that the problem was in the apophyseal plate, and aimed to expedite the fusion by needling or drilling across the growth plate and/or pegging it with bone graft. Cole [35] assumed a presence of venous hypertension within the tendon as the cause of the condition and proposed longitudinal incisions in the patellar tendon to decompress it. Orava *et al.* [6] reported surgical results of 70 OSS knees: 62 knees had removal of the free ossicle, while the remaining eight knees had excision of the prominent tibial tubercle and/or drilling of the tubercle. The mean age at surgery was 19.2 years. The final results were excellent or good in 56 cases, moderate in nine cases, poor in three cases and unknown in two cases. No significant differences were observed between the procedures.

Mital *et al.* [30] reported surgical results on 15 knees with OSS. These knees had a free separated ossicle, pain and direct tenderness over the ossicle. The patients were symptomatic despite conservative management for a mean period of 3.8 years. These knees underwent excision of the ossicle with the adjacent bursa and all patients reported pain relief following surgery. Glynn and Regan [36] reported two surgically treated groups for OSS. Group 1 (22 knees) underwent drilling of the tuberosity with or without removal of the prominence, while Group 2 (22 knees) underwent excision of the loose pieces of bone and cartilage without drilling or excision of the prominent tuberosity. The results showed a much higher incidence of excellent or good results in Group 2. The patients in Group 2 had rapid mobilization and returned to full activity. Importantly, this was not a prospective study but a report following a change in the authors' practice.

Ferciot [34] and Thompson [33] believed the tibial tuberosity excisional procedure (decreasing the size) preferable as it dealt with the prominence of tuberosity. The nonexcisional procedures made the prominence worse [33,37]. It was later that Krause *et al.* [23] described the natural history of OSS and the importance of difficulty in kneeling partly due to prominent tuberosity. Flowers and Bhadreshwar [4] reported surgical results of 42 knees in 35 patients. They used a modified Ferciot technique of excision of the prominent tibial tuberosity with removal of any osteocartilaginous material. Ninety-five percent of the patients revealed relief of pain and 86% reported relief from the prominence of tibial tuberosity.

Conclusion

OSS runs a self-limiting course with resolution of symptoms in greater than 90% of patients. In rare cases, surgical excision of the ossicle and/or free cartilaginous material may give good results in skeletally mature patients, who remain symptomatic despite conservative measures. The overall prognosis for OSS is good, except for some discomfort in kneeling and activity restriction in a few cases.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:
- • of special interest
- •• of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 117).

1 Osgood RB. Lesions of the tibial tubercle occurring during adoloscence. Boston Med Surg J 1903; 148:114–117.

2 Schlatter C. Verletzungen des schnabelformigen Fortsatzes der oberen Tibiaepiphyse. Beitr Klin Chir 1903; 38:874–887.

3 Blankstein J, Cohen I, Heim M, *et al.* Ultrasonography as a diagnostic modality in Osgood–Schlatter disease: a clinical study and review of the literature. Arch Orthop Trauma Surg 2001; 121:536–539.

4 Flowers MJ, Bhadreshwar DR. Tibial tuberosity excision for symptomatic Osgood–Schlatter disease. J Pediatr Orthop 1995; 15:292–297.

5 Ehrenborg G. The Osgood–Schlatter lesion: a clinical study of 170 cases. Acta Chir Scand 1962; 124:89–105.

6 Orava S, Malinen L, Karpakka J, *et al.* Results of surgical treatment of unresolved Osgood–Schlatter lesion. Ann Chir Gynaecol 2000; 89: 298–302.

7 Bloom OJ, Mackler L. Clinical inquries. What is the best treatment for Osgood–Schlatter disease? J Fam Pract 2004; 53:153–156.

8 Marx JA, Hockberger RS, Walls RM, Adams J. Rosen's emergency medicine: concepts and clinical practice. 5th ed. St Louis, MO: Mosby; 2002.

9 Wall EJ. Osgood–Schlatter disease: practical treatment for a self-limited condition. Phys Sport Med 1998; 26:29–34.

10 Ehrenborg G. The Osgood–Schlatter lesion. A clinical and experimental study. Acta Chir Scand Suppl 1962; (Suppl 288):1–36.

11 Ehrenborg G, Engfeldt B. The insertion of the ligamentum patellae on the tibial tuberosity. Some views in connection with the Osgood–Schlatter lesion. Acta Chir Scand 1961; 121:491–499.

12 Ehrenborg G. Etiology of Osgood–Schlatter's disease. Nord Med 1961; 66:1269–1273.

13 Ogden JA, Southwick WO. Osgood–Schlatter's disease and tibial tuberosity development. Clin Orthop Relat Res 1976; 116:180–189.

14 Ehrenborg G, Lagergren C. Roentgenologic changes in the Osgood–Schlatter lesion. Acta Chir Scand 1961; 121:315–327.

15 Aparicio G, Abril JC, Calvo E, Alvarez L. Radiologic study of patellar height in Osgood–Schlatter disease. J Pediatr Orthop 1997; 17:63–66.

16 Jacob RP, Von Gumppenberg S, Engelhard P. Does Osgood–Schlatter disease influence the position of the patella? J Bone Joint Surg Br 1981; 63:579–582.

17 Lancourt JE, Cristini JA. Patella alta and Patella infera. J Bone Joint Surg Am 1975; 57:1112–1115.

18 Willner P. Osgood–Schlatter's disease: etiology and treatment. Clin Orthop Relat Res 1969; 62:178–179.

19 Demirag B, Ozturk C, Yazici Z, Sarisozen B. The pathophysiology of Osgood–Schlatter disease: a magnetic resonance investigation. J Pediatr Orthop B 2004; 13:379–382.

20 Gigante A, Bevilacqua C, Bonetti MG, Greco F. Increased external tibial torsion in Osgood–Schlatter disease. Acta Orthop Scand 2003; 74:431–436.

21 Kujala UM, Kvist M, Heinonen O. Osgood–Schlatter's disease in adolescent athletes. Retrospective study of incidence and duration. Am J Sports Med 1985; 13:236–241.

22 Barbee J. What is the best treatment for Osgood–Schlatter disease? J Fam Pract 2004; 53:153–156.

23 Krause BL, Williams JP, Catterall A. Natural history of Osgood–Schlatter disease. J Pediatr Orthop 1990; 10:65–68.

24 Ross MD, Villard D. Disability levels of college-aged men with a history of Osgood–Schlatter disease. J Strength Cond Res 2003; 17:659–663.

25 Hirano A, Fukubayashi T, Ishii T, Ochiai N. Magnetic resonance imaging of Osgood–Schlatter disease: the course of the disease. Skeletal Radiol 2002; 31:334–342.

26 Medlar RC, Lyne ED. Sinding–Larsen–Johansson disease. Its etiology and natural history. J Bone Joint Surg Am 1978; 60:1113–1116.

27 Jacobson JA, Lenchik L, Ruhoy MK, *et al.* MR imaging of the infrapatellar fat pad of Hoffa. Radiographics 1997; 17:675–691.

28 • Peace KA, Lee JC, Healy J. Imaging the infrapatellar tendon in the elite athlete. Clin Radiol 2006; 61:570–578.
This is a good review, discussing the ultrasound and MRI findings in different pathologies affecting the infrapatellar tendon.

29 Watson-Jones R. Injuries of the knee (revised by Trickley EL). In: Wilson JN, editor. Fractures and joint injuries. Vol 2, 5th ed. Edinburgh: Churchill Livingston; 1976. pp. 1047–1050.

30 Mital MA, Matza RA, Cohen J. The so-called unresolved Osgood–Schlatter lesion: a concept based on fifteen surgically treated lesions. J Bone Joint Surg Am 1980; 62:732–739.

31 Beovich R, Fricker PA. Osgood–Schlatter's disease a review of the literature and an Australian series. Aust J Sci Med Sport 1988; 20: 11–13.

32 Hussain A, Hagroo GA. Osgood–Schlatter disease. Sports Exer Injury 1996; 2:202–206.

33 Thompson JEM. Operative treatment of osteochondritis of the tibial tubercle. J Bone Joint Surg Am 1956; 38:142–148.

34 Ferciot CF. Surgical management of anterior tibial epiphysis. Clin Orthop 1955; 5:204–206.

35 Cole JP. A study of Osgood–Schlatter disease. Surg Gynecol Obstet 1937; 65:55–67.

36 Glynn MK, Regan BF. Surgical treatment of Osgood–Schlatter's disease. J Pediatr Orthop 1983; 3:216–219.

37 Bosworth DM. Autogenous bone pegging for epiphysitis of the tibial tubercle. J Bone Joint Surg Br 1934; 16:829–838.