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Causes, short- and long-term consequences and treatment options of Osgood Schlatter's disease

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Introduction and summary

Osgood-Schlatter's disease (MOS or OSD) is a common cause of knee joint pain in growing age. It is an inflammation of bone and cartilage on the shin head (tibia) immediately under the growth disc. This disease is sometimes caused by overuse of the leg. In the process, pieces of bone can detach from the shin and die (necrosis). The disease is therefore counted among the aseptic (i.e., non-infection-related) osteonecrosis. Typical symptoms are pain, swelling and tenderness of the knee. This article discusses etiology, the natural course, as well as the long-term consequences and treatment options.

Symptoms and predisposing factors

Robert Osgood and Carl Schlatter first described the disease independently in 1903 as a partial avulsion of the tuberosities' tibiae.[1,2] The disease occurs mainly in adolescents who are active in sports. The male sex is preferred. Depending on the author, the age peak is between the ages of 11 and 14 for boys and between the ages of 10 and 13 for girls. In 25–50% both sides are affected.[3,4] In the literature, several predisposing factors are mentioned. The excessive sporting activity, such as football, hand- and basketball, athletics, and gymnastics in combination with a growth spurt are most often mentioned. According to Itoh,[5] landings on one leg and rapid changes of direction are the most rigorous loads. Watanabe[6] noted that pathogenic factors, associated with OSD in the supporting bone of adolescent male football players, include height, weight, body mass index, quadriceps femoris muscle tension in the kick and support legs and gastrocnemius muscle tension, soleus muscle tension, and medial longitudinal arch in the supporting bone. Other factors included the diagnosis of Sever's disease and the distance from the lateral malleolus to the center of gravity during kicking.

Although several possible causes are listed in the literature,[7] the etiology of OSD is unknown. Sarcevic[7] reported that pronated feet, genu valgum and internal rotation are associated with OSD. In their magnetic resonance imaging (MRI) study, Demirag et al.[8] compared the patellar tendon insertion of 15 OSD patients with that of 15 healthy adolescents. Gigante et al.[9] investigated the relationship between OSD and torsional abnormalities in the lower extremities and found significantly increased condylo-malleolar angle and external tibial rotation. Sarcevic[7] also mentions that pathological findings in this condition are most likely to coincide with a traumatically induced disorder somewhere along the side of the patellar tendon attachment to the tuberosities' tibiae. Pain worsens after jumping (basketball, volleyball, running) and/or direct contact (e.g., knees). Most common findings are tenderness, local swelling, and prominence in the area of tuberosities' tibiae. Pain may be reproduced by stretching the knee against resistance. Simple X-rays show an irregularity of the apophysis with separation from the tuberosities' tibiae. Sarcevic[7] also concluded in a clinical study that 42 out of 45 boys and girls tested had impaired dorsal flexion of the ankle. As the quadriceps femoris muscle contracts eccentrically during the standing phase of running until the beginning of propulsion, when the knee has reached the highest flexion, limited dorsal flexion in the ankle joint is associated with increased knee flexion, tibia internal rotation and foot pronation during the standing phase of running. In his study, Sarcevic[7] found that the cohort of participants diagnosed with OSD had limited dorsal flexion of the ankle joint. In fact, these mechanisms could cause an increased load on the attachment of the quadriceps muscle to the tuberosities' tibia and be a predisposing factor for OSD.

Natural course and long-term consequences

As early as 1990, Krause et al.[10] stated in a retrospective study with 69 knees in 50 patients what the natural course of untreated Osgood-Schlatter disease was in this group. Seventy-six percent of patients believed they had no restriction on activity, although 60% still could not kneel without discomfort. Two groups of patients were identified: those who had radiological fragmentation and had either separate ossicles or abnormally ossified tuberosity at the screening, and those who had soft tissue swelling without radiological fragmentation and were asymptomatic at the screening. According to Cakmak,[11] the prognosis of OSD is usually self-limiting course. Long-term outcome is good for the majority of OSD patients. Complications (e.g., pseudarthrosis, genu recurvatum, patella alta, fragmentation/migration of the ossicle) can occur in the chronic stage. Although OSD disease is a well-known clinical entity, management with long-term follow-up remains controversial. E.g., Cakmak presented the long-term outcome of a patient with untreated OSD. A 21-year-old man had been seen for the past 10 years for mild swelling in his left knee (which did not interfere with his daily activities). He denied any episode of trauma and the medical history was otherwise not contributing. The physical examination revealed tenderness and swelling in the left knee. Active and passive knee movements provoked pain and moderate restriction of movement in the left knee (30° flexion and 5° extension delay) was present. The neurological examination was inconspicuous. X-rays of the patient showed a large, fragmented bone of a proximal part of the tuberosity. The patient was consulted with the orthopedic surgeons, and they suggested surgery due to the presence of limitations in his knee movements, but the patient did not accept surgery.

Krause,[10] in 1990, followed fifty subjects with 69 affected knees for 9 years (range 3–30). During the acute phase of OSD, patients were re-evaluated and followed under conservative treatment. Forty-seven subjects had varying degrees of knee tension, and only three patients had no limitation in the acute phase, and by the end of their study, all patients were free of limitations. Ozer Kaya[12] also described medium-term symptoms. The study was conducted in eighteen Osgood-Schlatter patients with unilateral involvement and 14 age-

adapted healthy controls. The De Flaviis classification and patellar tendon characteristics were observed with a GE Logiq 9 scanner. Wide and vertical jump tests were used for jump performance. The coordination, proprioception, strength, and endurance functions were evaluated using the Functional Squat System. For quality of life, the SF-36 questionnaire was used. The Wilcoxon test was used for the first- and second-year evaluation of patients and the Mann-Whitney U test for the comparison between the patient and control groups. By the end of the second year, 38.9% of patients had fully recovered. The patellar tendon lengthened, the distal diameter and distal area of the tendon had decreased, and no significant difference was observed between patient and control groups (n.s.). Improvements were noted in bilateral broad jump test results, quality of life and patient coordination after 2 years ($p < 0.05$). Average endurance performance and total strength work were significantly higher in the control group ($p < 0.05$). According to the sonography results 2 years after diagnosis, almost half of the patients had fully recovered. Coordination was the only parameter that improved over the period of 2 years. The strength and endurance function of the patient group remained lower than that of the control group (Evidence Level III).

Current status of treatment options

Circi[13] claimed, that Osgood-Schlatter is a self-limiting disease and usually stops with skeletal maturity. Treatment is usually symptomatic. Adults with persistent symptoms may need surgical treatment if they don't respond to conservative treatment. Surgical procedures include open, bursoscopic and arthroscopic techniques. Arthroscopic surgery is advantageous via an open procedure due to early postoperative recovery, no incision scar before tuberosity, which usually causes discomfort when kneeling with a better cosmetic result, and the ability to treat concomitant intra-articular pathology. Osgood-Schlatter syndrome proceeds self-limiting, and as a rule, a full recovery with a closure of the tibial growth plate is expected. The overall prognosis for Osgood-Schlatter syndrome is good, apart from some discomfort with kneeling and activity restrictions in some cases. Cirsi[13] believes that arthroscopic techniques are the best choice for the treatment of unresolved Osgood Schlatter lesions.

However, since symptoms occur in childhood, the question is which therapies are and can be used. Gaulrapp[14] claims that a pure clinical diagnosis is sufficient. Imaging is only necessary if simultaneous secondary diagnoses are suspected, as it does not provide clear results and is neither therapy-supporting nor prognostic. Patients reduce their activities on their own. Late pain occurs at the apophyseal protrusion due to local compression. Restrictive recommendations to patients to give up their sporting activity are not justified. This contradicts the recommendations of Gerulis[15] and Lyng[16] among others. Rathleff[17,18] also recommends a more active form of treatment, namely an intervention consisting of activity modification, pain monitoring, progressive strengthening, and a paradigm of returning to exercise,[19,20] which were associated with improved self-reported outcomes, hip and knee muscle strength, and jumping performance. This approach can provide an alternative to passive approaches such as rest or wait and see, which are often prescribed for teens with OSD. Arakelyan[21] recommends stretching the quadriceps of the thigh under physiotherapeutic guidance, which can help reduce tension where the kneecap- (patella-) tendon is attached to the shin. A patellar tendon belt can also help release the tension. Strengthening exercises for the quadriceps and legs in general can help stabilize the knee joint. Surgery is only necessary in very rare cases. When[21] the pain is debilitating and does not subside after the growth spurt, then surgery to remove the bony overgrowth can be considered.

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