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Treatment of Schlatter's disease

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It is likely that Schlatter's disease can be attributed to incompletely repaired microfissures along the fibres that anchor the patellar ligament to the tibial tuberosity. We believe that sufficient unloading is necessary to repair the damage.

Illustration: Kristine Lie Øverland

Osgood-Schlatter disease (Schlatter's disease) is a common cause of pain, tenderness and swelling over the tibial tuberosity in physically active children and adolescents. The cause of the problems is probably repetitive strain on the apophysis from the quadriceps femoris muscle, leading to structural changes in the bone. The changes appear on X-rays as fragmentation of the bone structure, and an X-ray scan will verify the diagnosis if the clinical picture is not clear. The disease is relatively common in the early teens. In the area covered by the local hospital in our region, with about 50 000 inhabitants, 33 patients were referred for an X-ray scan because of Schlatter's disease in 2016–2017 (Kristian Kolnes, Volda Hospital, personal communication).

Schlatter's disease resolves spontaneously with time, but normally causes discomfort for 1–2 years before patients recover (1). Conservative treatment is generally recommended, in the form of reduced physical activity, pain-relieving drugs, non-steroidal anti-inflammatory drugs (NSAIDs) and ice packs for pain (2). A retrospective study at a sports medicine clinic in Finland found that pain in young athletes with Schlatter's disease led to 3–4 months cessation of training and training restrictions for 7–8 months (3). Long-term problems are unusual, but enlarged tibial tuberosities may cause some discomfort when kneeling for several years afterwards. A small loose bone shard under the patella ligament may also irritate the bursa under the ligament to the extent that the bone shard has to be removed surgically.

A type of fracture?

When, as a young doctor interested in sports, I arrived in a municipality where football was a major sporting activity for both adults and children, I met many youngsters who had dropped out of active sport for a long time on account of Schlatter's disease. I thought that the changes in the tibial tuberosity must be a type of fracture, and attempted to treat the patients with a plaster cast from thigh to ankle for six weeks. Those I treated got better. I presented the results for the period 1976–1982 at the World Congress on Sports Medicine in Vienna in 1982. However, plaster casting for six weeks was a rather drastic treatment, and in the period 1982–1996 I treated Schlatter's disease by locking the knee in slight flexion during the day with an orthoplastic splint on the dorsal side of the thigh and calf, fastened with an elastic bandage. Most of those treated got better after six weeks. None were better after only four or five weeks of splint treatment. It was difficult to mould plastic splints without using a kettle to make extra hot water, and it was also a rather cumbersome form of treatment. As a result, I started to recommend ordinary conservative treatment instead of splinting.

Treatment with knee orthosis

Since retiring from general practice, I have remained in close contact with the football community in the municipality. I have met several people who I treated for Schlatter's disease and who now have their own children with Schlatter's problems. They have asked me why nobody treats the disease any longer, thereby enabling patients to become free of pain relatively quickly.

I took up the matter with a colleague at Ulstein Medical Centre, and he wanted to try treating patients with Schlatter's disease with a knee orthosis that locked knee movement for 6–8 weeks. The orthosis was used during the day. It was locked with 10 degrees of flexion for three weeks. After three weeks, full knee extension was permitted, but still only 10 degrees of flexion. Our aim was to reduce the loss of muscular strength, particularly in the vastus medialis muscle, during the treatment period. The orthosis was used until there was no pain on palpation over the tibial tuberosity or on extending the knee against resistance with the knee in 90 and 45 degrees of flexion. Pain when these tests are conducted is the most common clinical finding when X-ray or ultrasound shows Schlatter's changes in the tibial tuberosity (4). If patients were not free of pain after six weeks, they were advised to use the orthosis for eight weeks. If freedom from pain had not been achieved after eight weeks, the treatment was discontinued. All patients were counselled to allow the knee free movement but to engage only in light physical training the first week after finishing treatment, and normal training with cautious strength training for the quadriceps, but no plyometric training, the following week. After that it was standard training without restrictions.

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Over the past three years, 20–30 patients with Schlatter's disease have been treated with knee orthoses. We chose to treat only those with such severe problems that they had

difficulty taking part in physical activity without a great deal of pain. Almost all got better. Their medical histories varied from a couple of months to several years of pain.

Sharpey's fibres and microfissures

In Quain's *Elements of Anatomy* from 1867, William Sharpey describes special connective tissue fibres that anchor the teeth to the sockets in the jawbones - Sharpey's fibres. A century and a half later, there is renewed interest in these fibres, not only as anchors for muscles, tendons and ligaments to periosteum and bone, but also as structures that affect actual bone formation, structure and resorption (5). The fibres spread out over the periosteum, and pass through the bone and into the endosteum. They contain various types of collagen that make them very stable (6). This results in the fibres forming channels in the bone; they are kept open and affect the bone structure. The fibres also contain elastin, which gives them a certain elasticity (7). Forces from muscles, tendons and ligaments are transferred to Sharpey's fibres, which affect bone formation and modulate the trabecular bone structure (8). The greater the forces that are transmitted via Sharpey's fibres, the more numerous and stronger the fibres become (9). Under great stress, microfissures may develop in the bone tissue near the fibres – stress fractures or fatigue fractures (10). Unrepaired microfissures and persistent strain may lead to local release of hydrolytic enzymes, causing structural changes in the bone tissue and potentially local tissue destruction (11).

X-ray scans show fragmentation of the tibial tuberosity bone structure with Schlatter's disease. The area is the anchor for the anterior thigh muscles through the patella and patella ligament. During puberty the length of the thigh bone and strength of the anterior thigh musculature increase relatively rapidly. This could conceivably increase the tendency to overloading of the tendon anchor and to microfissures near Sharpey's fibres in the tibial tuberosity. The fragmentation of the bone structure may then be a result of a number of incompletely repaired microfissures in the tibial tuberosity.

In our experience, some form of knee immobilisation is needed for rapid healing.

Jean Aaron in Leeds has done considerable research on Sharpey's fibres and how they affect bone building, structure and breakdown. She is of the view that unrepaired microfissures along Sharpey's fibres can explain the changes one finds in the apophysis with Schlatter's disease, and that our treatment may have unloaded the fibres and allowed enough time for the microfissures to mend (Jean Aaron, personal communication).

Permanent relief from symptoms?

Those of our patients who were treated and got better resumed their previous physical activity after only a couple of weeks. Many were still in their early teens, and at an age where one might perhaps expect that Schlatter's symptoms would recur. None of those we have been in contact with had experienced a recurrence of their problems some months after discontinuing treatment. This led to our contacting Jean Aaron again. Her response was that the local osteocyte network can be "primed" to improved tissue stability against microfissures when the latter have been repaired, and that this could explain why nobody experienced recurrence (Jean Aaron, personal communication).

Sufficient unloading

Many Schlatter's sufferers have problems with both legs. We have treated several of them. The orthosis was used on the side that was worst. This side got better, but the patient still had problems on the other side. This may indicate that the reduced physical activity during the period the patient was using the orthosis was not sufficient for the microfissures in the tibial tuberosity to mend on the side that was not treated. In our experience, some form of knee immobilisation is required to achieve rapid healing. Daytime treatment with an orthosis and an almost rigid knee for 6–8 weeks is not something active teenagers enjoy.

However, given some hope of becoming free of pain, few of them indicated that the treatment period was difficult to get through.

If Schlatter's disease is a result of microfissures along Sharpey's fibres, fatigue fractures in the apophysis, it is logical to treat the disease in the same way as fatigue fractures in other parts of the body: adequate unloading of the anchor for long enough for the bone damage to be repaired.

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