Osgood-Schlatter Disease: effectiveness of US Imaging

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Abstract: The aim of this paper is to describe the ultrasonographic (US) features characteristically seen in the traction type of Osgood-Schlatter disease (OSD). In addition, the insight into the pathophysiologic sequence of events and their cause is given. and a new concept as well as classification of OSD based on US findings is provided, which may facilitate early diagnosis of this disorder and its treatment. US assessment of OSD yields unparalleled findings because it provides excellent visualization of the fine structures of the patellar ligament, the superficial and deep infrapatellar bursae, and the status of the cartilage of the ossification center of the tibial tuberosity. US plays a key role not only in the confirmation of the disease but also in its exclusion. It is clearly a method of choice in diagnosing OSD and monitoring its treatment.

Key words: Osgood-Schlatter disease, tibial tuberosity, trauma, ultrasound (US)

AZ ULTRAHANGOS KÉPALKOTÁS HATÉKONYSÁGA OSGOOD-SCHLATTER-KÓRBAN

Ennek a közleménynek a trakciós típusú Osgood-Schlatter betegségben (OSD) tipikusan észlelt ultrahang-jellemzők ismertetése a célja. Ezen felül áttekinti a kórélettani események sorozatát és azonosítja azok okát. Vázolja az OSD új, ultrahangleleteken alapuló koncepcióját és osztályozását, mely megkönnyítheti a kórkép korai kórismézését és kezelését. Az OSD ultrahangvizsgálata páratlan diagnosztikai adatokat szolgáltat, mert kitűnően ábrázolja a ligamentum patellare finom képleteit, a felszínhez közeli és a mélyen fekvõ infrapatelláris nyáktömlőket, továbbá a tuberositas tibiae csontosodási centrumában lévő porcszövet állapotát. Az ultrahangvizsgálat szerepe nem csupán a betegség fennállásának igazolásában, hanem a kizárásában is döntő – egyértelmûen ez a választandó módszer az OSD kórismézésére és kezelésének monitorozására.

Osgood-Schlatter disease (OSD) was originally described by *Robert Osgood* of Boston in 1903(1), and by *Carl Schlatter* of Zurich in 1903 and 1908 (2,3). Whereas Osgood focused on full avulsion fractures of the tibial tuberosity, with loss of patellar ligament continuity, Schlatter noted a wide variety of reasons for the disease. One such reason was traction trauma to the tibial tuberosity that led to avulsion of a portion of the tuberosity or to incomplete fracture due to contraction of the quadriceps muscle. Subsequent investigators implicated other causes, including rickets, infection, endocrinologic disturbances, avascular necrosis, tendinitis, and hereditary predisposition (4). At present, it is generally accepted that OSD is traumatic in origin and can be regarded as a specific kind of adolescent enthesopathy (5). The disease occurs in patients between 10 and 15 years of age (in girls, usually between 8 and 13; in boys, usually between 10 and 15).This paper describes the events occurring within and around the tibial tuberosity that are the result of traction exerted on the patellar ligament, effectively demonstrated by ultrasonography (US).

US, an imaging method primarily useful in the diagnosis of soft tissue abnormalities, sheds new light on the

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complex pathophysiology of OSD. Many authors have described the use of diagnostic imaging methods other than conventional radiography in the diagnosis of OSD, such as US (4-8), magnetic resonance imaging (MRI), computed tomography (CT), and radionuclide bone scan (scintigraphy) (5,9). Features of OSD revealed by US and MRI have included pretibial soft tissue swelling, cartilage swelling, fragmentation of the tibial tuberosity's ossification center, thickening at the insertion of the patellar ligament, and inflammation of the deep infrapatellar bursa.

It is generally agreed that US should be a modality of choice in the diagnosis and follow-up of OSD (4-8). It seems that MRI can reveal many features of OSD seen in US. However, paradoxically, the edema of the bone and cartilage that is generally so effectively depicted by MRI, frequently is either not present or not so obvious at the delamination zone of the ossification center, probably due to the healing process. Conversely, US puts that anatomic site in the "front row" of the image, easily detected by the observer.

There are several characteristic features helpful in diagnosis and classification of OSD:
- the presence of delamination tear of the ossification center of the tibial tuberosity,
- the appearance of a fracture of overlying cartilage (its extent in relation to the site of the patellar ligament's "footprint" and deep infrapatellar bursa is important consideration in classification), and
- the presence of patellar ligament tear or scar including ectopic ossified/calcified scars within the ligament as a result of the cartilage tear.

Based upon these features, OSD can be classified into three types (10):

Type I – pure delamination tear/fracture of the ossification center of the tibial tuberosity;

Type II – delamination tear/fracture of the ossification center with fracture of the overlying cartilage situated outside (usually proximal) the patellar ligament's footprint, but extending into the deep infrapatellar bursa;

Type III – delamination tear of the ossification center with the cartilage fracture line within the patellar ligament's footprint, causing delamination injury to the patellar ligament; in most cases the fracture line also extended into the deep infrapatellar bursa.

Additionally the presence or absence of the following features is helpful in precise classification of this disorder:
- deep infrapatellar bursa effusion or hematoma,
- bursal synovial edema, which is defined as layer of more than 1mm thickness of hypo or hyperechogenic tissue overlying the tibial wall of the bursa (a layer of tissue <1mm thickness overlying the tibial wall of the bursa is frequently seen in asymptomatic bursas),
- hypervascularity (hyperperfusion) of the bursal tissues defined as presence of even single vessel in

Tissue Doppler Mode (Toshiba) set on maximum sensitivity at the synovial tibial bursal lining,
- fibrosis within the bursal space defined as layer of more than 1mm thickness of hyperechogenic tissue overlying the tibial wall of the bursa and/or improper bursal fat apron movement between the patellar ligament and tibial bursal wall,
- hypervascularity of the zone of insertion of the patellar ligament indicating active inflammation/reparative processes defined as the presence of at least two vessels within the ligament. In borderline images comparing to the asymptomatic side, if not affected by the disease, is important.

ANATOMY AND HISTOLOGY OF THE TIBIAL TUBEROSITY

To understand the pathophysiologic sequence of events in OSD, the histologic type and architecture of the tibial tuberosity and the anatomic structures surrounding it must be fully elucidated. The anatomic location of the traction injury in OSD should first be precisely defined, because it is always the same – the ossification center of the beak-like process of the tibial epiphysis. This endochondral ossification center, by which the beak-like process of the tibial tuberosity ossifies, is a layer of cellular columns responsible for calcium production and the later transformation of calcium into bone (11). The anterior (cellular) surface of the tuberosity ossification center is a metabolically active zone until the tuberosity becomes solid bone. The epiphyseal part of the tibial tuberosity in boys and girls between the ages of 11 and 15 years is mainly a fibrocartilaginous structure that hosts a unique type of ossification center belonging to the epiphysis but also overlapping the anterior proximal part of the tibial metaphysis. The product of this ossification center was named the "beak-like" process of the tibial tuberosity by Carl Schlatter.

The tibial tuberosity development may be divided into three stages, beginning with the appearance of cloud-like ossified tissue within the anterior tibial cartilage (Fig. 1A). Calcium deposits within the ossification center of the tibial tuberosity, seen first at or just below the level of the border between the epiphysis and metaphysis, appear as these scattered cloud-like ultrasound-transparent structures that are often mistaken for fragmentation but merely represent an incomplete fusion of the ossifying cartilage. The productive zone of the tibial tuberosity consists of cellular columns that fit within the cartilage, which undergoes ossification (11). This zone, a cellular gap between the layers of solid cartilage (later cartilage-bone), lacks reinforcing structures such as the collagen fibers present in the surrounding cartilage. As a result, these cellular columns, deprived of collagen reinforcement, constitute the weakest structural link of the tuberosity.

Next, the ossified cartilage transforms into mature bone more or less scattered in the area of the tibial tuberosity
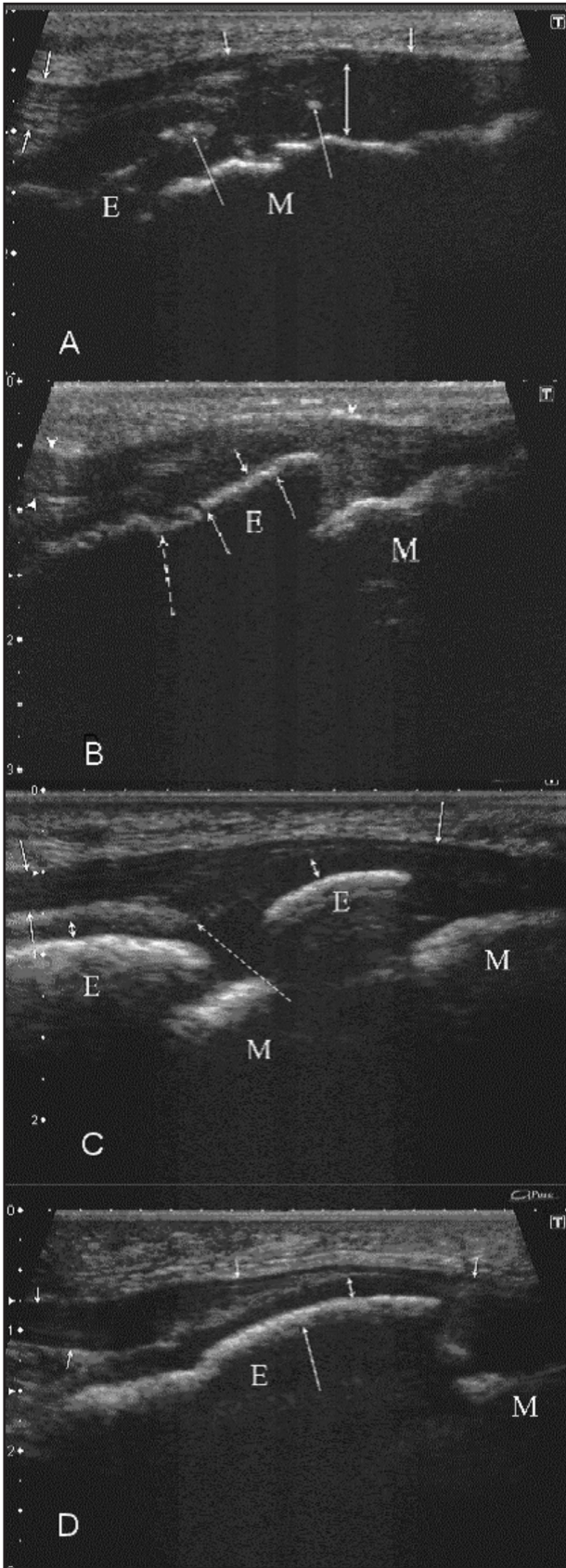
Figure 1. Longitudinal US image of the early stage (A), middle stage (B,C) and late stage (D) of bone formation in the tibial tuberosity. Short arrows, patellar ligament; long arrows, ossifying cartilage (ossification center from transparent (A) to non-transparent (D) to the US beam); E, epiphysis; M, metaphysis; double arrow, thickness of the tibial tuberosity cartilage, dashed arrow, level of the proximal patellar ligament's attachment.

part of the tibial tuberosity *(Fig.1D)*. At this late stage, the bone is still covered by a cartilage layer or, more exactly, the bone is still within cartilage, as it has not yet completed the ossification process. It is important to realize that neither of these stages represents fragmentation but, rather, incomplete bone fusion. At this point of development, the tibial tuberosity does not possess a cortical layer, a fact unfortunately misunderstood by some authors (12,13). The anterior bright margin of the physeal beak-like process that is seen on US is not cortex. It is the most active cellular layer of the growing ossification center, and the weakest link of the tuberosity.

Physiology and pathology of tibial tuberosity

Key to understanding pathologic events involving the growing tibial tuberosity is the "footprint" of the patellar ligament's tibial insertion *(Fig. 2)*. The total length of the insertion may, in author's experience, exceed 40 mm and the width, 30 mm. Usually, however, the length and width range between 20 and 40 mm and 20 and 30 mm, respectively, as determined ultrasonographically using curved line measurements. The type of OSD discussed in this paper is found only within approximately the 10-15mm long zone of the proximal tibial insertion of the patellar ligament, which corresponds to the length of the beak-like process. Primary injuries to the tuberosity in the remaining distal part of the ligament's insertion that crosses the cartilage zone between the epiphyseal beak-like process and the metaphysis, as well as the metaphyseal part of the patellar ligament insertion, are extremely rare and result from direct trauma rather than traction.

Three factors account for the specific location of the traction type of injury in OSD. First, there is a difference in durability between the epiphyseal and metaphyseal parts of the patellar ligament insertion; this can be explained by the type of ossification at the epiphysis (endochondral) versus that at the metaphysis (membranous) (10). Second, compared with the distal 20 to 25 mm, the proximal 10-15 mm of the tibial tuberosity has a larger angle between its surface and the longitudinal fibers of the patellar ligament. This approximately 10-15mm zone is where traction on the patellar ligament generates most of the delaminating force (14). Third, the epiphyseal beak-like process, with its ossification center of cellular columns, is the weakest structural link. These three factors prove to be the "fatal combination" that

(Figs.1B and 1C); this bone is, at best, only partially transparent to the ultrasound beam. Once the bone of the beak-like process fuses, it gives final shape to the physeal
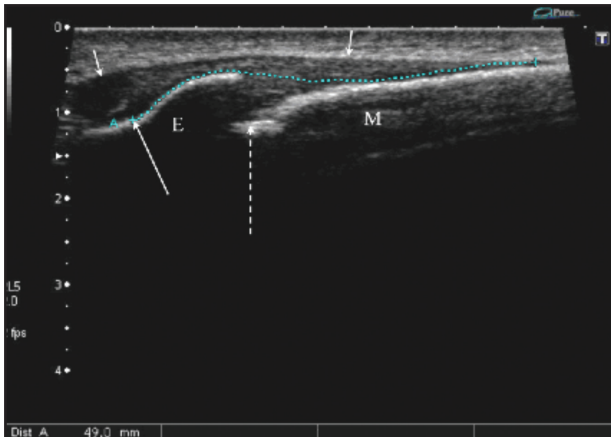
Figure 2. Normal longitudinal US image of the patellar ligament attachment to the tibial tuberosity in an adolescent. Short arrows, the patellar ligament's margin; long arrow, the proximal border level of the patellar ligament's insertion; dashed long arrow, the level of the cartilage zone between the beak-like process of the epiphysis (E) and the metaphysis (M); curved dotted line, approximate total length of the patellar ligament's tibial insertion.
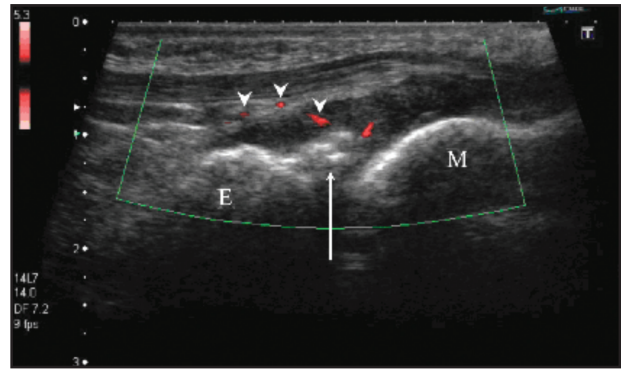

Figure 3. Longitudinal US image of the tibial tuberosity. Arrowheads indicate the same artery (seen clearly as continuous in real-time imaging) within and around the ossification center (arrow), the cartilage of the tibial tuberosity and the synovial lining of the deep infrapatellar bursa. E, epiphysis; M, metaphysis.

results in injury to this specific area. Just distal to this area – at the cartilage junction between the epiphysis and metaphysis – the tuberosity-ligament angle has already decreased and no cellular columns are present in the ossification center of the epiphysis. At the level of the metaphyseal part of the patellar ligament insertion, the tuberosity-ligament angle is small and the type of ossification is different (membranous); no beak-like process is present, just the upper shaft of the tibia. Therefore, the zone of edema will be present within a wide area around the beak-like process, representing collateral damage rather than "core injury."

The vascularity of the tibial tuberosity is also an important anatomic and functional feature of OSD. To provide a sufficient supply of nutrients and oxygen to the ossification center, the vascularity of this region is, of necessity, extremely rich. That is why a concept of avascular necrosis in this area seems extremely out of place. Of special note is the common arterial supply to the tuberosity and to the synovium of the deep infrapatellar bursa *(Fig.3)*. This arterial architecture may explain why, even in minor, closed-delamination injuries without bleeding into the deep infrapatellar bursa, the inflammatory-reparative process of and around the ossification center may still cause inflammation and later fibrosis in the bursa. Common vascularity may mean shared inflammatory problems.

In addition to the tibial tuberosity itself, which at this stage represents cartilage hosting the ossification center, surrounding soft tissue structures that may undergo significant pathologic changes in OSD are:

1. The patellar ligament
2. The deep infrapatellar bursa
3. The superficial infrapatellar bursa
4. The subcutaneous pretibial fatty tissue

Of these, the most pivotal are the patellar ligament and the deep infrapatellar bursa. The deep infrapatellar bursa – occupying a large space between the posterior margin of the patellar ligament and the anterior margin of the tibial epiphysis, right above the proximal patellar ligament's insertion line – may in the authors' experience reach as much as 20 cc in volume. The fat pad of the bursa is an important part of Hoffa's fat pad. Because of these particular anatomic characteristics, pathologic changes affecting the bursa will have clinical impact.

Classification and features of Osgood-Schlatter disease.

Based upon the US findings regarding the presence of delamination tear of the ossification center of the tibial tuberosity, the status of overlying cartilage, and the presence of patellar ligament tear/scar, OSD can be classified into three general types. The pathologic features and clinical consequences of these three types are delineated as follows:

Type I

- Delamination of the internal ossification center, resulting in an "igloo"-like deformation of the physeal part of the tibial tuberosity, with hump-like anterior displacement of the proximal attachment of the patellar ligament. It can be a "clean" delamination tear, with only a thin anterior layer of the ossification center displaced or a "blurred" delamination, which occurs when ossified tissue of the center is torn within and displaced in a scattered manner with only the "roof" of the igloo being smooth. The above-described features can be seen on MRI examination, but are much obvious on US *(Figs.4 and 5)*.

Delamination of the ossification center is called the "double cortical sign" by some authors (11, 12). It is a misnomer, since the tibial tuberosity in adolescents does
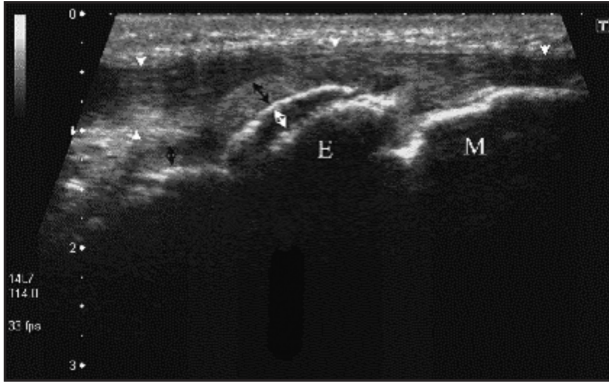
Figure 4. Type I OSD, longitudinal US image. Arrowheads, margins of the patellar ligament; black double arrows cartilage thickness in the tibial tuberosity over the ossification center (hyperechogenic cartilage – edema or/and fibrosis); E, epiphysis; M, metaphysis. Note the clear-cut margins of the delaminated layers of the ossification center (white double arrow) – a clear transparent igloo.

Figure 5. Type I OSD, longitudinal US image. Note ossified tissue between delaminated layers indicating a profound fracture of the ossification center of the beak-like process. Patellar ligament and cartilage edematous and hyperperfused (Doppler frame) at the distal end/insertion, most likely representing a reparative process; H, Hoffa's fat pad; E, epiphysis; M, metaphysis; double dashed arrow, thickness of the cartilage overlying the ossification center; double arrow, approximate thickness of the delamination zone.

not possess cortex and delamination/tear occurs within the ossification center, which has critically different histologic and mechanical properties than bone cortex. Moreover, the presence of two lines of the delaminated ossification center can be seen only in the early stages after delamination injury, when the space between the delaminated margins is not yet filled with ossified US-nontransparent tissue. Then we see an "igloo" with a transparent roof consisting of little calcified tissue, and also the "floor" can be seen in the image representing the rest of the torn ossification center. In the late ossified stage, one can see only the outside margin of the lesion – a true, solid igloo *(Fig. 6)* as the roof and the inside of

it has undergone ossification and is no longer transparent to the ultrasound beam.

- Deep infrapatellar bursitis and/or fibrosis due to a shared arterial supply with the tibial tuberosity and local inflammation/reparative process.
- Disseminated inflammation or diffused fibrosis (or both) of the patellar ligament secondary to the main injury and deep infrapatellar bursitis.
- Superficial infrapatellar bursitis secondary to deep infrapatellar bursitis and to tendinopathy involving the patellar ligament.

The prognosis is very favorable for this type of disease as it leaves minimal disturbance to the shape of the tuberosity and the state of the patellar ligament.

Type II

- Igloo deformation (delamination tear/fracture) of the epiphyseal part of the tibial tuberosity, with fracture of cartilage overlying ossification center and significant anterior displacement of the proximal attachment of the patellar ligament due to the displacement of the fractured cartilage *(Fig. 7)*.
- Deep infrapatellar bursitis or fibrosis (or both) due to bleeding from the torn cartilage/ossification center.
- Disseminated inflammation or fibrosis (or both) of the patellar ligament secondary to the main injury and deep infrapatellar bursitis.
- Superficial infrapatellar bursitis secondary to deep infrapatellar bursitis and tendinopathy involving enthesis zone of the patellar ligament.

The prognosis is moderately favorable for this type of disease as it tends to create significant bursal fibrosis including impairment of dynamic behavior of the fatty apron of the deep infrapatellar bursa/Hoffa's fat pad, however doesn't leave significant scarring within the patellar ligament.
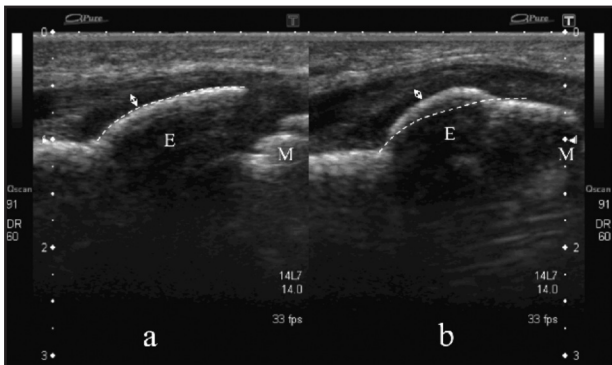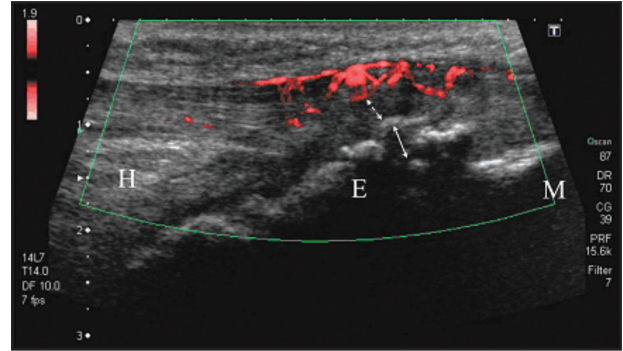

Figure 6. a, Normal tibial tuberosity. b, Ossified end-stage after type I delamination tear of the beak-like process of the tuberosity. Double arrows, cartilage thickness over the ossification center; dashed line on Figure "a", normal outline of the ossification center transferred directly (same scale) onto Figure "b", indicating the difference between the normal outline and the final effect of the ossification center delamination tear; E, epiphysis; M, metaphysis. Note that there is no gap between the epiphysis and the metaphysis on Figure "b", reflecting typical earlier than normal ossification of that area due to an important metabolic boost.
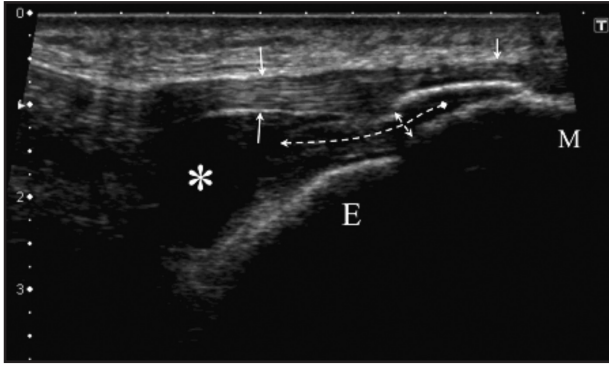
Figure 7. Longitudinal US image of a delamination tear and cartilage fracture at the patellar ligament's proximal insertion border of the tibial tuberosity ossification center (OSD type II). Arrows margins of the patellar ligament; double dashed arrow, maximum delamination thickness/ligament's insertion anterior transfer – the opening connecting the ossification center with the deep infrapatellar bursa space; curved dashed arrow, the bleeding route from the torn ossification center vessels (dot) into the bursa; E, epiphysis; M, metaphysis; asterisks, hematoma within the deep infrapatellar bursa

Type III

- Delamination tear of the ossification center resulting in irregular deformation of the tuberosity, cartilage fracture with or without significant anterior displacement of the proximal attachment of the patellar ligament. Cartilage fracture at least in part within the footprint of the patellar ligament insertion *(Figs.8 and 9).*

- Deep infrapatellar bursitis or fibrosis (or both) due to a shared arterial supply with the tibial tuberosity and bleeding from the torn patellar ligament and fractured cartilage.

- Focal scarring and possible ectopic calcium or bone formation due to a tear (usually a longitudinal delamination tear) in the patellar ligament arising from its tibial insertion.

- Superficial infrapatellar bursitis secondary to deep infrapatellar bursitis and to tendinopathy/tear and later scar formation involving the patellar ligament.

For this type of disease, the prognosis is unfavorable, with a high probability of chronic symptoms due to fibrosis of the deep infrapatellar bursa and ectopic bone or calcium formation within the patellar ligament's scar.

In our opinion, based on the clinical findings and follow-up examinations the most important prognostic factor is whether the cartilage fracture line crosses the patellar ligament's footprint. The main difference in outcome between types II and III is the possibility of ectopic calcification or bone formation within the patellar ligament's tear/scar when the fracture line does cross its insertion, because that kind of fracture results in delamination injury to the patellar ligament.

CORRELATION OF CLINICAL AND ULTRASONOGRAPHIC FINDINGS IN OSGOOD-SCHLATTER DISEASE

The mildest form of OSD is a closed (i.e., without cartilage fracture) delamination of the physeal part of the tuberosity – type I *(see Figs.4 and 5).* This variant is in many cases not symptomatic enough to warrant a visit to the physician. It is, therefore, highly unusual to see a patient with such type in an early stage of injury.

The shape of the delaminated tuberosity resembles an igloo (especially on transverse images), usually approximately 1 cm in width. In some cases, the delamination is not smooth. Some new bone of the ossification center may be torn within, or dislodged, so that the igloo image is blurred. In the delamination type of OSD, healing of the ossification center progresses well when not complicated by subsequent injuries. Sequelae consist of a bulging of the tuberosity and fibrosis in the deep infrapatellar bursa, which constitute the two basic diagnostic features of OSD. It is worthy noting that this is the only type of OSD in which fibrosis within the bursa may not be present, although it is a rarity not to see it.

Many delamination tear lesions occur in combination with a cartilage fracture just proximal to the patellar ligament footprint – type II. The delaminated tuberosity resembles an igloo, as in type I. The cartilage fracture, just proximally to the patellar ligament's insertion, allows bleeding from the torn vessels of the ossification center area directly into the deep infrapatellar bursa. Later, the organizing hematoma becomes an area of fibrosis in the bursa. This process may also result in symptomatic chronic bursitis.

When the cartilage fracture ends at the footprint of the patellar ligament's attachment – a type III injury – the prognosis is much worse. A fracture line ending in the distal patellar ligament footprint area will produce calcified or ossified, somewhat irregular scars within the broken cartilage (later bone) or/and the torn distal patellar ligament which alone is then a source of free blood pouring into the deep infrapatellar bursa. If the fracture also extends into the deep infrapatellar bursa, and it usually does, it will cause bleeding into the bursa from the torn blood vessels of the extremely well perfused ossification center and thus altogether generate substantial inflammation and later fibrosis within the bursa.

Calcified scar within the patellar ligament usually produces two unfortunate results:
- Local extensive infiltration of calcium deposits at the level of the patellar ligament's insertion, usually reaching the anterior margin of the patellar ligament. These deposits may be difficult to remove because of their infiltrative nature.
- Single or multiple ossified or calcified areas within the patellar ligament at the level of and proximal to its insertion *(see Figs. 8 and 9);* thus direct contact is made with the tendinous wall of the deep infrapatellar
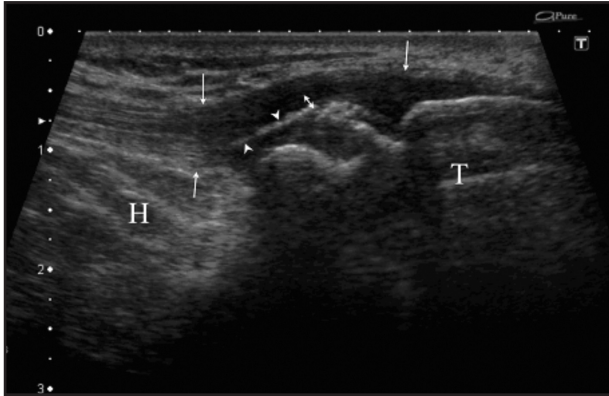
Figure 8. Longitudinal US image of a type III OSD injury to the epiphyseal beak-like area of the tibial tuberosity. Arrows, anterior and posterior margins of the distal patellar ligament; double arrow, cartilage of the tibial tuberosity; arrowheads, line of the cartilage crack and patellar ligament scar in the early ossification phase.
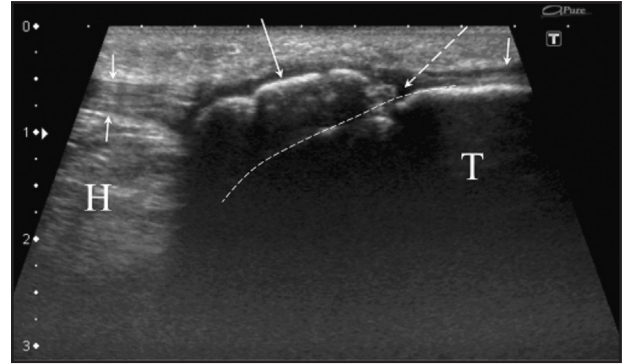

Figure 9. US image of the type III OSD with an ectopic ossification shows the following: short arrows, anterior and posterior margins of the patellar ligament; long arrow, ectopic ossification formed within patellar ligament's scar; dashed arrow, distal segment of the possible fracture line between the ectopic ossification and the main part of the tibial tuberosity; dashed curved line, anterior margin of the tuberosity, partly obscured by the ossification. H, Hoffa's fat pad; T, tibia.

bursa. Some calcified scars form small stones within the ligament detached from the tibial tuberosity at the outset; some are so massive that they become continuous with the tibial tuberosity. These large areas, however, may fracture, becoming separate ectopic formations. They may be wrongly interpreted as pieces of the fractured tibia that have moved upward. This misinterpretation may, in fact, account for the confusion sometimes surrounding the nature of the disease. Even at present, OSD is still occasionally called "avascular necrosis," possibly because of these ectopic calcifications which may contain areas of necrotic substance. The term is inaccurate, however, since the calcifications are not dead fragments of previously living bone tissue.

Treatment

The detailed description of treatment of OSD is beyond the scope of this paper, however, it is worthwhile to note that US examination is helpful in the decision-making process of therapy, as well as in monitoring the healing process. Some conclusions regarding treatment of OSD can be drawn from the features of the disease. Osgood already proved with his anatomic dissections that even with complete detachment of the patellar ligament from the tibial tuberosity, the strength of other patello-tibial supporting structures is sufficient to bear significant weight. In OSD there is no total detachment of the patellar ligament; therefore, immobilization of the limb, if used at all, should be limited to a very short period in the acute stage of the disease, simply to protect the patient from unexpected weight loaded on the extensor apparatus. Long immobilization, in fact, seems potentially harmful, as it creates muscle atrophy and contracture. When the acute pain has significantly decreased, stretch-

ing exercises are more appropriate, as they result in a more elastic load on the tibial tuberosity. Simple supportive anti-inflammatory treatment, such as the use of nonsteroidal anti-inflammatory drugs (NSAIDs) and ice, is an obvious necessity.

Conclusions

Although very early and subtle pathologic changes of OSD within the tibial tuberosity complex can be detected by MRI, ultrasonographic assessment of OSD yields unparalleled findings. US provides excellent visualization of the bursae, the fine structure of the patellar ligament, and the cartilage within the ossification center. Even if some doubt exists regarding the tibial tuberosity structure, the absence of deep infrapatellar bursitis or fibrosis on US gives the examiner a margin of safety in excluding more serious disease, as it is rare not to see this pathologic feature. In contrast, radiography may not give as final and definitive answer as US, especially in the early stages of the disease or if the changes are subtle. In this respect, US plays a key role not only in the confirmation of disease but also in its exclusion. For example, a major clinical feature of OSD – pain – may be present simply due to overload of the extensor apparatus and may not automatically mean that a structural disturbance of the tibial tuberosity complex actually exists. In such cases, no aggressive treatment is necessary, and the prognosis for a quick recovery is very good. This exclusion factor is of importance to the young athlete or any other teenager, not to mention the parents. In addition, US is an examination quick to perform, is relatively cost-effective, and does not expose the growing skeleton to ionizing radiation. Therefore, US is clearly a method of choice in diagnosing OSD and monitoring its treatment.

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