Choroba Osgood-Schlattera: nowe spojrzenie i nowa klasyfikacja w oparciu o diagnostykę ultrasonogaficzną

Osgood-Schlatter Disease: a new Perspective and Classification Based on Ultrasonography

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Streszczenie

Cel pracy. Celem niniejszej pracy jest opisanie charakterystycznych dla choroby Osgood-Schlattera (OS) cech ultrasonograficznych (usg) widywanych po urazach powstałych w mechanizmie pociągania przez aparat wyprostny za guzowatość kości piszczelowej. Dodatkowo autorzy proponują podróż po przyczynach widywanych zjawisk, ich podłożu histologicznym i patofizjologicznym oraz proponują nową koncepcję i klasyfikację choroby OS w oparciu o obrazy usg, które mogą być pomocne w prawidłowej, wczesnej diagnostyce tej choroby.

Materiał i Metoda. Ocenie retrospektywnej przez dwóch diagnostów specjalizujących się w diagnostyce układu mięśniowo-szkieletowego poddano na zasadzie konsensusu wyniki badań 90 pacjentów z cechami klinicznymi choroby OS (82 osób płci męskiej, 8 płci żeńskiej w wieku 10-17 lat, średnia wieku 13,3). Każde badanie usg było oceniane pod względem obecności delaminacyjnego złamania jądra kostnienia nasadowej części guzowatości kości piszczelowej oraz statusu powlekającej jądro kostnienia chrząstki w obrębie i okolicy stopy przyczepu piszczelowego więzadła rzepki (obecność lub brak szczeliny złamania chrząstki). Na podstawie kombinacji tych dwóch podstawowych cech chorobę OS podzielono na trzy typy. Dodatkowe elementy takie jak: uszkodzenie i bliznowacenie więzadła rzepki, obecność zapalenia kaletki podrzepkowej powierzchownej i głębokiej (wysięk, obrzęk i wzmożone unaczynienie błony maziowej, jej włóknienie), wzmożone unaczynienie i włóknienie więzadła rzepki w strefie dystalnego przyczepu więzadła rzepki oraz obrzęk i wzmożone unaczynienie tkanek okołowięzadłowych strefy nasadowej przyczepu piszczelowego więzadła rzepki oceniano jako elementy dodatkowe we wszystkich trzech typach.

Wyniki. Zmiany zaobserwowano po stronie prawej u 37 pacjentów, lewej u 53 pacjentów. Dwudziestu trzech pacjentów (25,5%) prezentowało strukturalne cechy typu I, 15 pacjentów (16,6%) typu II i 52 pacjentów (57,9%) typu III.

Konkluzje. Wysoka rozdzielczość obrazów więzadła rzepki, kaletek aparatu wyprostnego oraz stanu chrząstki, w której zlokalizowane jest jądro kostnienia nasady kości piszczelowej udostępniają nieporównywalne do innych metod obrazowania dane dotyczące tej choroby stawiając usg w ocenie choroby OS na czele metod diagnostycznych. Kluczowa rola usg dotyczy nie tylko diagnostyki potwierdzającej obecność zmian chorobowym, ale ma również kapitalne znaczenie w ich wykluczeniu. Ostatecznie należy usg uznać za metodę z wyboru w diagnostyce tego typu patologii.

Abstract

Objective. The aim of this study was to describe the ultrasonographic (US) features characteristically seen in the traction type of Osgood-Schlatter disease (OSD). In addition, the authors wanted to offer insight into the pathophysiologic sequence of events and their cause, and to propose a new concept and classifica-

tion of OSD based on US findings, which may facilitate early diagnosis of this disorder and its treatment. **Materials and methods.** Two musculoskeletal radiologists retrospectively reviewed in consensus US findings in 90 patients with clinical features of OSD (82 males and 8 females, age range 10-17 years, mean age 13.3 years). Each US study was evaluated for the presence of delamination of the ossification center of the tibial tuberosity and the status of overlying cartilage at the site of patellar ligament "footprint" (presence or absence of fracture). Based upon the combination of these features, OSD was classified into three types. In addition, patellar ligament tear and scar formation; presence of bursitis (effusion) of both deep and superficial infrapatellar bursae; presence of synovial edema, hypervascularity, and fibrosis; condition of vascularity at the zone of insertion of the patellar ligament; and presence of soft tissue inflammation around the tibial tuberosity was assessed in all three groups. **Results.** The right knee was affected in 37 patients, the left knee in 53 patients. Twenty-three patients (25.5%) presented with type I, 15 patients (16.6%) with type II, and 52 patients (57.9%) with type III disease. **Conclusions.** 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.

Słowa kluczowe
choroba Osgood-Schlattera, guzowatość kości piszczelowej, uraz, ultrasonografia (usg)

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

Introduction

Osgood-Schlatter disease (OSD) was first described in 1903 by Robert Osgood of Boston (1) and in 1903 and then 1908 by Carl Schlatter of Zurich (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). This paper examines the events occurring within and around the tibial tuberosity that are specifically produced by traction on the patellar ligament.

Osgood and Schlatter based their diagnoses on sound medical practice: the patient's history, clinical evaluation, and radiography, at that time the only technique available for diagnostic imaging. In interpreting the radiographic features, both physicians agreed that disease of the tibial tuberosity's enthesis zone manifested in patients between 8 and 15 years of age (in girls, usually between 8 and 13; in boys, usually between 10 and 15). Despite the limited ability of radiography to image primarily calcified structures, however, this pathologic condition was described as a fracture of the tibial tuberosity (1-3). Ultrasonography (US), an imaging method primarily useful in the diagnosis of soft tissue abnormalities, sheds new light on the 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 in common 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 is difficult, though, to find in the world literature definitive answers to why certain features appear and what they actually signify. 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 (see Fig. 14) 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 (see Fig. 15).

Materials and methods

Two musculoskeletal radiologists retrospectively reviewed in consensus US findings in 90 patients with clinical features of OSD. Ultrasound high-end equipment of different manufacturers (Siemens, Toshiba, Philips) was used with linear 5- to 14-MHz transducers and power Doppler options. The patients ranged in age between 10 and 17 years, with a mean age of 13.3 years. There were 82 males and 8 females. The left knee was affected in 53 patients, the right knee in 37 patients. Each US study was evaluated for:

- the presence of delamination tear of the ossification center of the tibial tuberosity,
- the status of overlying cartilage (extent of fracture in relation to the site of the patellar ligament's "footprint" and deep infrapatellar bursa) and
- the presence of patellar ligament tear or scar including ossified/calcified scars within the ligament as a result of the cartilage tear.

Based upon these features, OSD was classified into three types:

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 abscence of the following features was assessed in all three groups:

- deep infrapatellar bursa effusion defined as even faint amount of fluid but present within the whole width of the bursa or hematoma defined as bright fluid with visible dense material or fibrotic clot movement;
- bursal synovial edema 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. It may be difficult at times to distinguish inflammatory edema from fibrosis but after a week of inflammatory process the fibrosis is an inevitable sequelae so the duration of the symptoms may be a key to classification of the image;
- 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. The Tissue Doppler (Toshiba) shows in my experience the presence of vessels in asymptomatic and structurally normal insertions of the patellar ligament which makes it difficult at times not to overdiagnosed insertional pathology. So the presence of a single vessel at the patellar ligament's tibial insertion does not automatically mean pathology. It seems easier with the use of less sensitive Doppler techniques where a presence of a vessel within a tendon/ligament seems always pathological.

Results

Twenty-three patients (25.5%) presented with type I, 15 patients (16.6%) with type II, and 52 patients (57.9%) with type III disease. The distribution of abnormal findings in each type of OSD, including deep infrapatellar fibrosis, deep infrapatellar bursa effusion/hematoma, deep infrapatellar bursa synovitis, and deep infrapatellar tissue hypervascularity (hyperperfusion), is shown in Table 1. It is of interest that deep infrapatellar fibrosis was present in all 67 patients with type II and III OSD. Deep infrapatellar synovitis was a rare feature, seen only in 5 patients (4 with type I and 1 with type II) probably due to the fact that it is a rarity to see a fresh injury to the tibial insertional zone. Patients usually come to see a doctor weeks or months after the first onset of the disease and that is when fibrosis has been a longstanding element of the bursal disease already. Hyperperfusion at the site of the patellar ligament insertion was a common feature in type I and III disease, and deep infrapatellar tissue hyperperfusion indicating active inflammation was more common in type I.

Table 1

	Deep infrapatellar fibrosis	Deep infrapatellar effusion	Deep infrapatellar synovitis	Deep infrapatellar tissue hyperperfusion	Patellar ligament hyperperfusion
Type I (N=23)	19 (82.6%)	11(47.8%)	4 (17.4%)	4 (17.4%)	9 (39.1%)
Type II (N=15)	15 (100%)	10 (66.7%)	1 (6.7%)	1 (6.7%)	3 (20%)
Type III (N=52)	52 (100%)	20 (38.4%)	0	3 (5.8%)	23 (44.2%)

Discussion

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 (or, to be more accurate, Schlatter disease) 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 (10). 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. 1). 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 (10). 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.

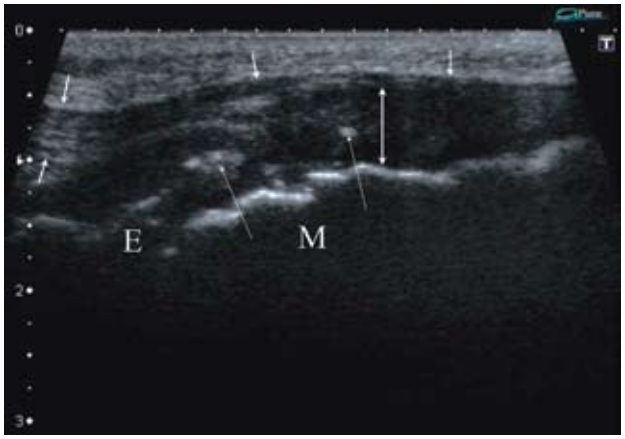


Figure 1. Longitudinal US image of the early stage of bone formation in the tibial tuberosity. Short arrows, patellar ligament; long arrows, ossifying cartilage (ossification center is transparent to the US beam at this stage); E, epiphysis; M, metaphysis; double arrow, thickness of the tibial tuberosity cartilage.
Ryc. 1. Obraz usg wczesnego okresu formowania się struktury kostnej guzowatości kości piszczelowej w przekroju podłużnym. Krótkie strzałki, więzadło rzepki; długie strzałki, zmineralizowana chrząstka (jądro kostnienia jest w tym stadium tworzenia przejrzyste dla ultradźwięków); E - nasada; M – przynasada; Strzałka z dwoma grotami, chrząstka guzowatości.

Next, the ossified cartilage transforms into mature bone more or less scattered in the area of the tibial tuberosity (Figs. 2 and 3); 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 part of the tibial tuberosity (Figs. 4 and 5). 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 (11,12). 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.

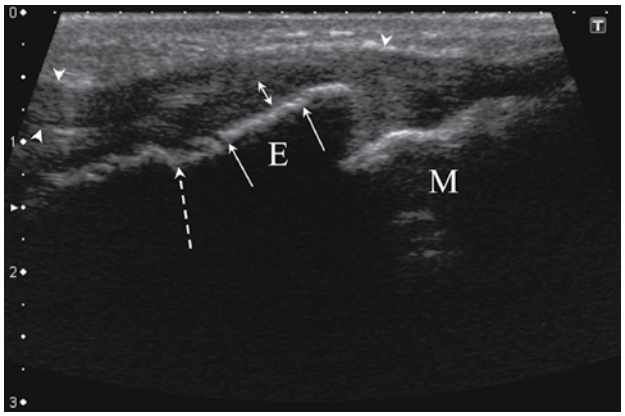


Figure 2. Longitudinal US image of the middle stage of tibial tuberosity bone formation. Arrowheads, patellar ligament; long arrows, ossification center anterior margin (ossified enough to be no longer transparent to the US beam); E, beak-like process of the epiphysis; M, metaphysis; double arrow, cartilage thickness between the ossification center and the patellar ligament attachment; dashed arrow, level of the proximal patellar ligament's attachment.
Ryc. 2. Obraz usg guzowatości k. piszczelowej w stadium pośrednim tworzenia jej kostnej struktury. Groty strzałek, więzadło rzepki; długie strzałki, przedni zarys jądra kostnienia (stopień uwapnienia jądra nie pozwala już na penetrację ultradźwięków); E, Wyrostek dziobiasty nasady; M, przynasada; Podwójna strzałka, grubość chrząstki pomiędzy jądrem kostnienia i przyczepem więzadła rzepki; Przerywana strzałka, poziom bliższej granicy przyczepu więzadła rzepki.

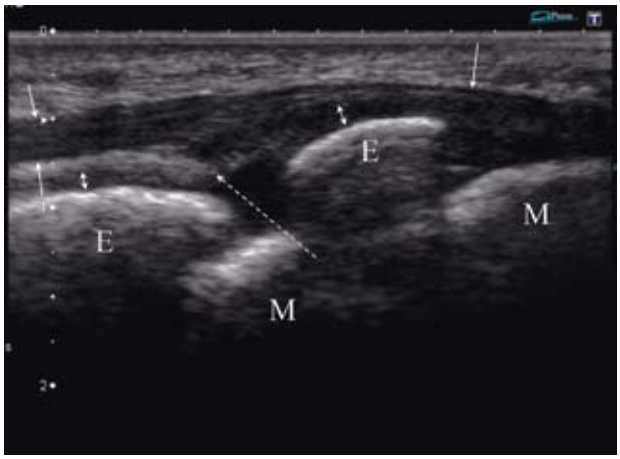


Figure 3. Longitudinal US image of the middle stage of tibial tuberosity bone formation. Short arrows, patellar ligament; E, epiphysis; M, metaphysis; double arrows, cartilage thickness between the ossification center and the patellar ligament attachment (right) and in the area of the deep infrapatellar bursa (left); dashed arrow, the level of the patellar ligament's proximal attachment.
Ryc. 3. Obraz usg guzowatości k. piszczelowej w stadium pośrednim tworzenia jej kostnej struktury. Krótkie strzałki, więzadło rzepki; E, nasada i wyrostek dziobiasty nasady; M, przynasada; Podwójna strzałka, grubość chrząstki pomiędzy jądrem kostnienia i przyczepem więzadła rzepki (prawa) w rejonie ściany piszczelowej kaletki podrzepkowej głębokiej; Przerywana strzałka, poziom bliższej granicy przyczepu więzadła rzepki.

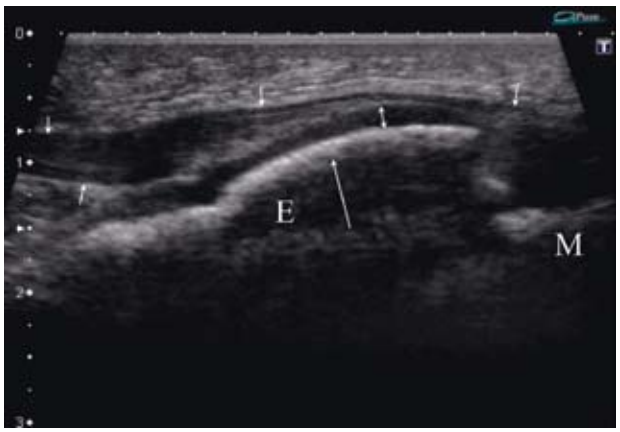


Figure 4. Longitudinal US image of the late stage of tibial tuberosity (beak-like process) bone formation with ossification center fusion (long arrow). Short arrows, patellar ligament; E, epiphysis; M, metaphysis; double arrow, cartilage thickness between the ossification center and the patellar ligament attachment.
Ryc. 4. Obraz usg późnego stadium formowania kostnej struktury jądra kostnienia nasadowej części guzowatości kości piszczelowej (wyrostka dziobiastego) z cechami fuzji całego jądra kostnienia (długa strzałka). Krótkie strzałki, więzadło rzepki; E, nasada; M, przynasada; podwójna strzałka, grubość warstwy chrząstki pomiędzy jądrem kostnienia i przyczepem więzadła rzepki.

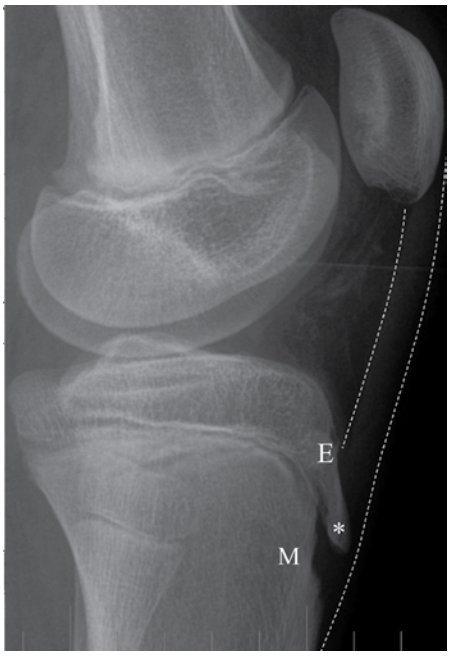


Figure 5. Lateral radiograph of normal late-stage formation of the beak-like process of the epiphyseal part of the tibial tuberosity (*). E, epiphysis; M, metaphysis; dashed lines, approximate anterior and posterior margins of the patellar ligament.
Ryc. 5. Prawidłowy radiogram późnego stadium formowania kostnej struktury wyrostka dziobiastego nasady; E, nasada; M, przynasada; przerywane linie, przybliżone zarysy przedni i tylny więzadła rzepki.

Key to understanding pathologic events involving the growing tibial tuberosity is the "footprint" of the patellar ligament's tibial insertion (Fig. 6). 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 (Fig. 7). 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 (13). 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 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.

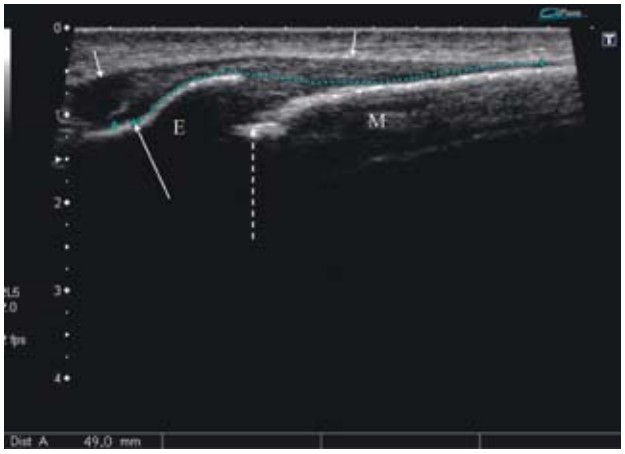


Figure 6. Normal longitudinal US image of the patellar ligament attachment to the tibial tuberosity in an adolescent. Short arrows, the patellar ligament's margin; long arrow, the proximal border level of the patellar ligament's insertion; dashed long arrow, the level of the cartilage zone between the beak-like process of the epiphysis (E) and the metaphysis (M); curved dotted line, approximate total length of the patellar ligament's tibial insertion.
Ryc. 6. Prawidłowy obraz usg pola przyczepu więzadła rzepki do guzowatości kości piszczelowej u osoby młodocianej. Krótkie strzałki, zarys więzadła rzepki; długa strzałka, bliższa granica przyczepu piszczelowego więzadła; Przerywana długa strzałka, poziom granicy chrzęstnej pomiędzy wyrostkiem dziobiastym nasady (E) i przynasady (M); Kropkowana krzywa, przybliżona całkowita długość pola przyczepu piszczelowego więzadła obejmująca nasadę i przynasadę – 49mm.

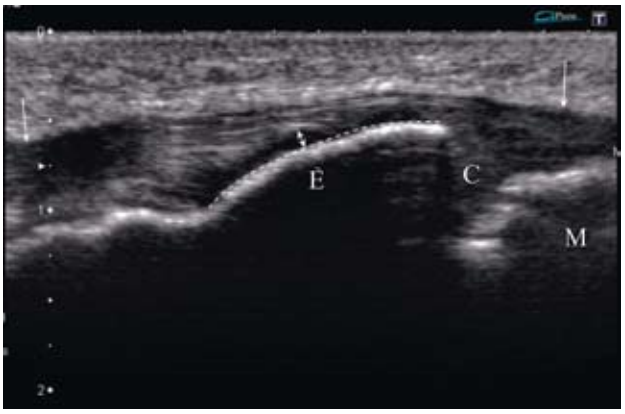


Figure 7. Sagittal US image of the midproximal normal tibial tuberosity. M, metaphysis; C, cartilage separating the epiphysis from the metaphysis (in normal conditions the cartilage tends to be dark, whereas in OSD it becomes bright due to edema); E, epiphysis; dashed line, surface area of the ossification center equal in length to patellar ligament's insertion to the epiphyseal part of the tibial tuberosity, where cartilage fracture and delamination can be seen in OSD; double arrow, cartilage covering the beak-like process of the epiphysis; arrows, anterior surface of the patellar ligament.
Ryc. 7. Obraz usg przekroju strzałkowego środkowo-bliższej strefy guzowatości kości piszczelowej. M, przynasada; C, chrząstka oddzielająca uwapnione elementy nasady i przynasady (w normalnych warunkach echogeniczność chrząstki jest niska/bezechowa, podczas gdy w chorobie OS jej echogeniczność jest średnia/wysoka, wtórnie do obrzęku pourazowego); E, nasada; przerywana linia, powierzchnia jądra kostnienia równa długości przyczepu więzadła rzepki do nasadowej części guzowatości kości piszczelowej, gdzie obserwuje się rozwarstwieniowe złamania jądra kostnienia w chorobie OS; podwójna strzałka, chrząstka powlekająca wyrostek dziobiasty/jądro kostnienia nasady; strzałki, przedni zarys więzadła rzepki.

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. 8). 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.

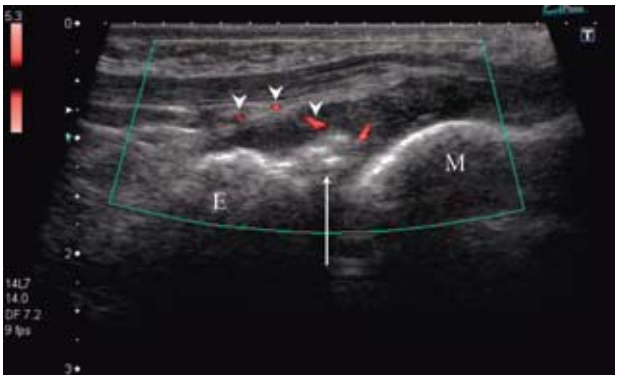
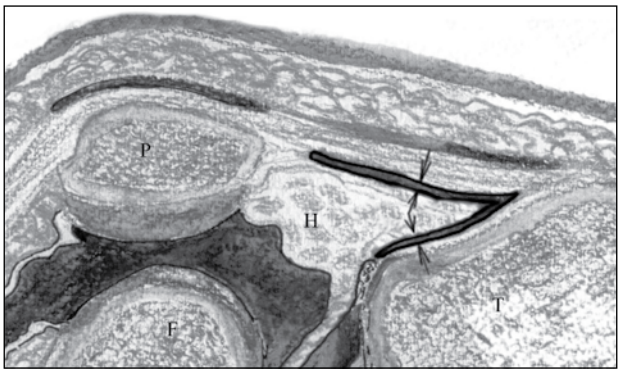


Figure 8. Longitudinal US image of the tibial tuberosity; Tissue Doppler gate. Arrowheads indicate the same artery (seen clearly as continuous in real-time imaging) within and around the ossification center (arrow), the cartilage of the tibial tuberosity and the synovial lining of the deep infrapatellar bursa. E, epiphysis; M, metaphysis.
Ryc. 8. Obraz podłużny usg guzowatości kości piszczelowej; bramka Tissue Doppler. Groty strzałek wskazują to samo naczynie tętnicze (jego ciągłość widoczna jest podczas badania w czasie rzeczywistym) wewnątrz i poza strefą słabo, w tej fazie rozwoju, zorganizowanego jądra kostnienia (strzałka), w obrębie chrząstki guzowatości oraz błony maziowej kaletki podrzepkowej głębokiej. E, nasada; M, przynasada.

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 (Fig. 9), 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.

Figure 9. Anatomic drawing of the deep infrapatellar bursa. Arrows, four walls of the bursa; P, patella; H, Hoffa's fat pad; T, tibia; F, femur. Note that a significant part of Hoffa's fat pad is a moving element of the bursa. It does not get compressed between the ligament and the tibial wall in normal nonfibrotic conditions – it glides in and out of the space between the ligament and the tibia. In the case of serious bursal fibrosis, the movement of that part during flexion-extension is disturbed and it may become a compressed element of the bursa.
Ryc. 9. Rysunek anatomicznego przekroju strzałkowego kaletki podrzepkowej głębokiej. Strzałki oznaczają cztery ściany kaletki – więzadłową, fałdu tłuszczowego ciała Hoffy oraz piszczelową; P, rzepka; H, ciało Hoffy; T, piszczel; F, kość udowa. Zwróć uwagę na fakt, że znaczna część ciała tłuszczowego Hoffy stanowi element ruchomy kaletki. Nie ulega on kompresji pomiędzy więzadłem rzepki i ścianą piszczelową kaletki w warunkach prawidłowego ślizgu jej ścian, lecz wślizguje (wyprost) i wyślizguje (zgięcie) się z przestrzeni więzadło-kość. W przypadku pozapalnych lub powstałych w przebiegu krwiaka zwłóknień, ruchomość tego fałdu tłuszczowego jest zaburzona podczas ruchów zgięcia/wyprostu w stawie kolanowym i w takich warunkach może on ulegać uwięzieniu i kompresji.

Classification and features of Osgood-Schlatter disease

Based upon the US findings in our patients, 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, we classified OSD 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 (Figs. 10 and 11), or a "blurred" delamination (Figs. 12 and13), 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 (Fig. 14), but are much obvious on US (Fig. 15, see also Figs. 10 and 11).

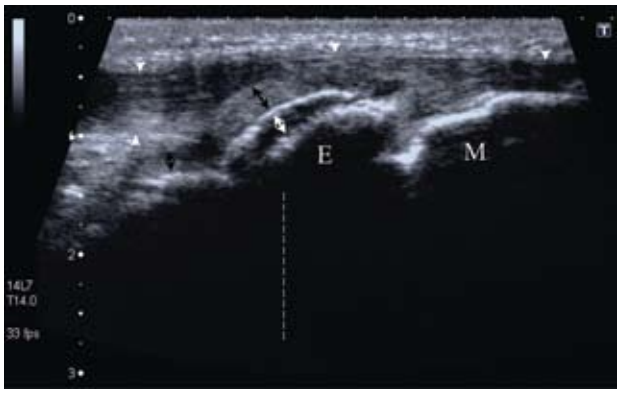


Figure 10. Type I OSD, longitudinal US image. Arrowheads, margins of the patellar ligament; black double arrows, cartilage thickness in the tibial tuberosity over the ossification center (hyperechogenic cartilage – edema or/ and fibrosis); E, epiphysis; M, metaphysis. Note the clear -cut margins of the delaminated layers of the ossification center (white double arrow) – a clear transparent igloo. Dashed line indicates the level of the image from Figure 11.
Ryc. 10. Typ I choroby OS, przekrój podłużny obraz usg. Groty strzałek, zarysy więzadła rzepki; podwójna czarna strzałka, grubość chrząstki guzowatości kości piszczelowej nad jądrem kostnienia (hiperechogeniczna chrząstka – obrzęk i/lub włóknienie); E, nasada; M, przynasada. Zwróć uwagę na ostro odgraniczone zarysy rozwarstwionego jądra kostnienia (biała strzałka z dwoma grotami) – „czyste Igloo" z przeziernym dachem. Przerywana linia wskazuje na poziom przekroju poprzecznego z następnej ryciny (11).

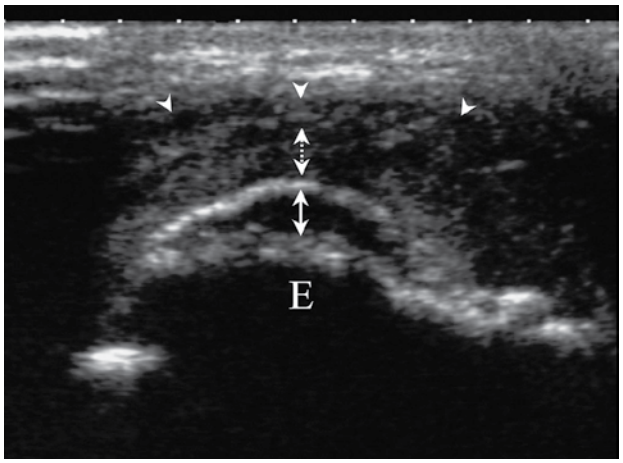


Figure 11. Type I OSD, transverse US image, patient from figure 10. E, epiphysis; arrowheads, margins of the patellar ligament; dotted double arrow, cartilage thickness in the tibial tuberosity over the ossification center. Note the clear -cut margins of the delaminated layers of the ossification center (white double arrow) – a clear transparent igloo.
Ryc. 11. Typ I choroby OS, przekrój poprzeczny, obraz usg na poziomie przerywanej linii z ryc 10. E, nasada; groty strzałek, zarysy więzadła rzepki; kropkowana strzałka z dwoma grotami, grubość chrząstki guzowatości kości piszczelowej nad jądrem kostnienia. Zwróć uwagę na ostro odgraniczone zarysy rozwarstwionego jądra kostnienia (biała strzałka z dwoma grotami) – „czyste Igloo" z przeziernym dachem.

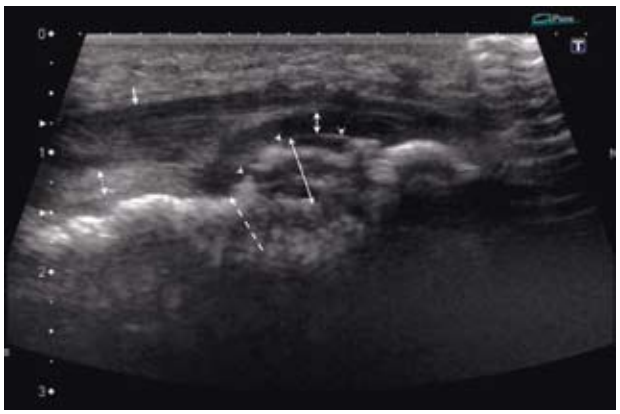


Figure 12. Type I OSD, longitudinal US image. No fracture and anterior transfer of the proximal part of the tuberosity is present (dashed arrow at the level of the proximal ligament's attachment). Short arrow, the anterior margin of the patellar ligament; short double arrow, cartilage thickness in the tibial tuberosity; short double dotted arrow, fibrosis within the deep infrapatellar bursa; arrowheads, anterior margin of the delaminated ossification center. Note the uneven distribution of calcified tissue within the delaminated area (long double arrow) – a blurred igloo. Such a finding suggests profound disturbance of the ossification center and immature bone.
Ryc. 12. Typ I choroby OS, przekrój podłużny usg. Brak cech złamania i istotnego przedniego przemieszczenia bliższego końca nasadowej części guzowatości (strefa oznaczona przerywaną strzałką na poziomie linii bliższego przyczepu piszczelowego więzadła rzepki), zamknięta delaminacja. Krótka strzałka, przedni zarys więzadła rzepki; krótka podwójna strzałka, grubość chrząstki guzowatości kości piszczelowej; krótka podwójna kropkowana strzałka, zwłóknienia kaletki podrzepkowej głębokiej; groty strzałek, przedni zarys rozwarstwionego jądra kostnienia (przezierny dach Igloo). Zwróć uwagę na nierówną dystrybucję elementów zmineralizowanych rozerwanego jądra kostnienia (długa podwójna strzałka) – zatarte wnętrze Igloo. Taki obraz wskazuje na głębokie rozerwanie struktury jądra kostnienia wraz z niedojrzałą kością jądra.

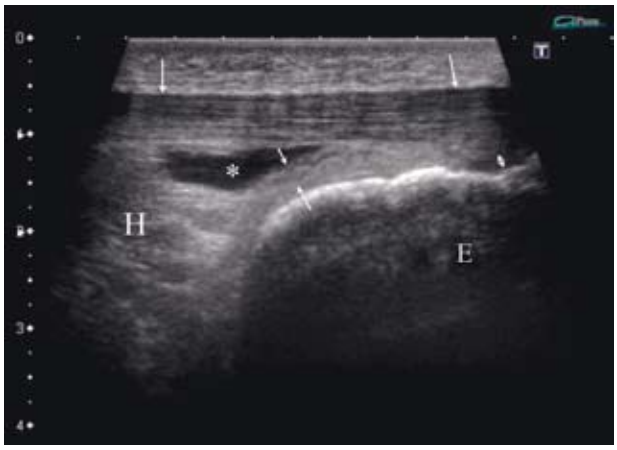


Figure 13. Same patient as in Figure 12. Effusion (*) and fibrosis (short arrows) due to inflammation can be observed within the deep infrapatellar bursa. Long arrows, patellar ligament; H, Hoffa's fat pad retracted by fluid; E, tibial epiphysis; double arrowhead, cartilage layer.
Ryc. 13. Pacjent z ryc. 12. Wysięk (*) i zwłóknienia (krótkie strzałki) powstałe wtórnie do zapalenia, widoczne w świetle kaletki podrzepkowej głębokiej. Długie strzałki, więzadło rzepki; H, ciało Hoffy zepchnięte przez wysięk i sklejone ze zwłóknieniami ściany piszczelowej kaletki; E, nasada; krótka strzałka z dwoma grotami, warstwa chrząstki jądra kostnienia.

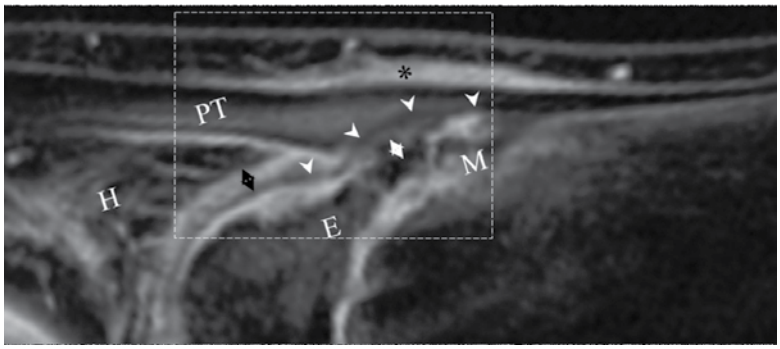


Figure 14. Magnetic resonance imaging (MRI), FLASH 2D, of Osgood-Schlatter disease (OSD) type I in a 14-year-old boy 4 months after the first onset of symptoms. PT, patellar ligament edematous at the distal end; asterisk, pretibial soft tissue edema; H, Hoffa's fat pad (the moving part of the deep infrapatellar bursa); black double arrowheads, edema and fibrosis over the tibial wall of the deep infrapatellar bursa; E, epiphysis; M, metaphysis; white double arrowheads, approximate thickness of the delamination zone; arrowheads, margin of the cartilage. Note the diffused edema of the anterior epiphysis and superior metaphysis. Dotted line frame denotes the field of view in Figure 15.
Ryc. 14. Obraz rezonansu magnetycznego (RM), sekwencja FLASH 2D, choroby Osgood-Schlattera typ I u 14 letniego pacjenta 4 miesiące po pierwszym epizodzie dolegliwości. PT, więzadło rzepki, obrzęknięte na poziomie dystalnym (jasne/podwyższony sygnał); gwiazdka, obrzęk tkanek miękkich okolicy przed guzowatością kości piszczelowej; H, ciało Hoffy (część ruchoma kaletki podrzepkowej głębokiej); czarna podwójna strzałka, obrzęk i włóknienie na powierzchni ściany piszczelowej kaletki podrzepkowej głębokiej; E, nasada; M, przynasada; białe podwójne groty strzałek, przybliżona grubość strefy delaminacji jądra kostnienia; groty strzałek, zarys chrząstki. Zwraca uwagę rozlany obrzęk przedniej strefy nasady i przedniej górnej strefy przynasady. Ramka z przerywanych linii określa zakres pola widzenia z ryc. 15.

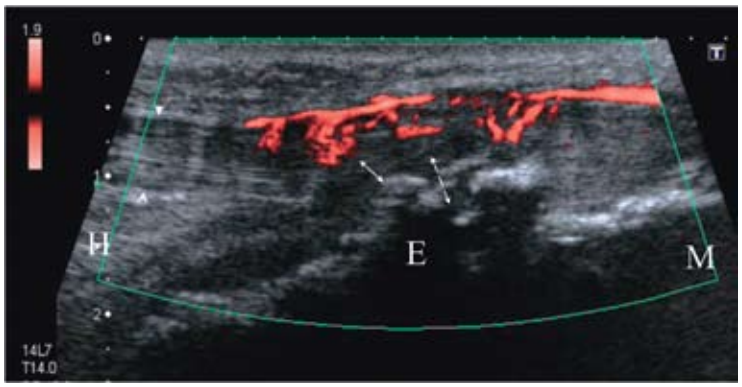


Figure 15. Ultrasonographic (US) image of the same patient as in Figure 14. Arrowheads, patellar ligament edematous and hyperperfused (Doppler frame) at the distal end/insertion, most likely representing a reparative process; H, Hoffa's fat pad; E, epiphysis; M, metaphysis; double arrow, thickness of the intact cartilage overlying the ossification center; double dashed arrow, approximate thickness of the delamination zone.
Ryc. 15. Obraz usg pacjenta z ryc. 14. Groty strzałek, więzadło rzepki z cechami obrzęku i wzmożonego unaczynienia (bramka Dopplera) na poziomie przyczepu dystalnego, najprawdopodobniej jako manifestacja procesu naprawczego; H, ciało Hoffy; E, nasada; M, przynasada; podwójna strzałka, grubość nieuszkodzonej chrząstki wokół jądra kostnienia; podwójna przerywana strzałka, przybliżona grubość strefy delaminacji/rozerwania jądra kostnienia.

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 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 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. 16) as the roof and the inside of it has undergone ossification and is no longer transparent to the ultrasound beam.

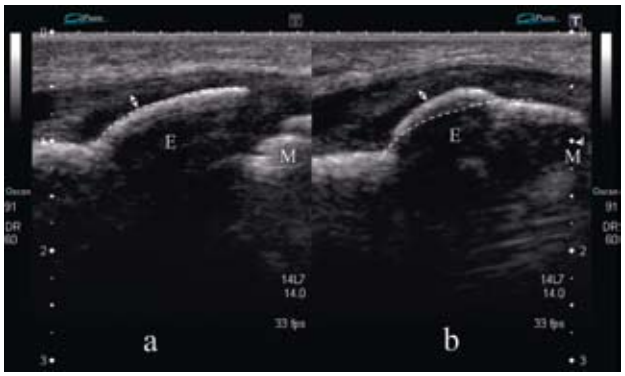


Figure 16. a, Normal tibial tuberosity. b, Ossified end-stage after type I delamination tear of the beak-like process of the tuberosity. Double arrows, cartilage thickness over the ossification center; dashed line on Figure "a", normal outline of the ossification center transferred directly (same scale) 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.
Ryc. 16. a, Obraz usg prawidłowej guzowatości kości piszczelowej. b, Obraz usg końcowego etapu kostnienia jądra guzowatości w przebiegu choroby OS typu I (rozwarstwieniowego uszkodzenia wyrostka dziobiastego nasadowej części guzowatości). Podwójne strzałki, grubość chrząstki nad jądrem kostnienia; przerywana linia na ryc. "a", prawidłowy kształt zarysu guzowatości "przeniesiony" w skali 1:1 na ryc. "b", wskazujący na różnicę kształtu jądra kostnienia/guzowatości kości piszczelowej po przebytej „zamkniętej" delaminacji; E, nasada; M, przynasada. Zwraca uwagę brak (skostnienie) chrzęstnej przestrzeni pomiędzy nasadą i przynasadą na ryc. „b", odzwierciedlający typowy proces wcześniejszego niż w warunkach prawidłowych kostnienia tej strefy na skutek wzmożonego metabolizmu tej okolicy w przebiegu uszkodzenia jądra kostnienia.

→ Deep infrapatellar bursitis and/or fibrosis due to a shared arterial supply with the tibial tuberosity and local inflammation/reparative process.
→ Disseminated edema/hyperperfusion 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 (Figs. 17, 18 and 19).

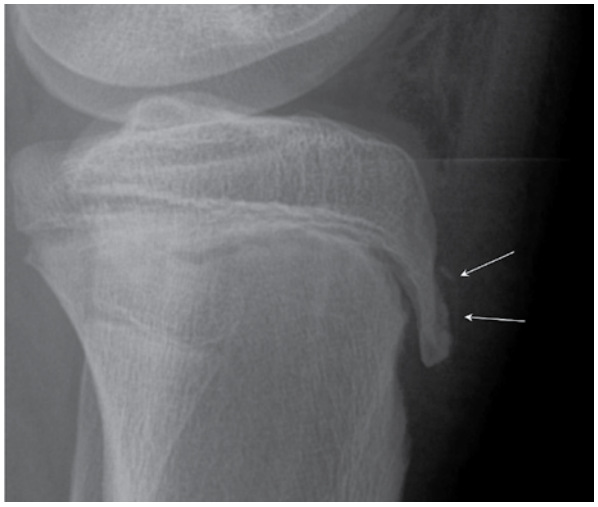


Figure 17. Lateral radiograph showing a type II OSD delamination injury to the tibial tuberosity ossification center and a cartilage tear of the beak-like process (seen only on US) just above the patellar ligament insertion level. Arrows show a faint image of the anteriorly displaced calcified part of the anterior ossification center (See Figure 18).
Ryc. 17. Zdjęcie boczne RTG choroby OS typu II pokazujące rozwarstwieniowe uszkodzenie jądra kostnienia nasady guzowatości kości piszczelowej z pęknięciem chrząstki wyrostka dziobiastego nasady (widocznym wyłącznie na zdjęciu usg) bezpośrednio powyżej linii przyczepu więzadła rzepki. Strzałki wskazują na linię oderwanej od macierzy i przemieszczonej ku przodowi zmineralizowanej przedniej warstwy jądra kostnienia guzowatości (patrz ryc. 18).

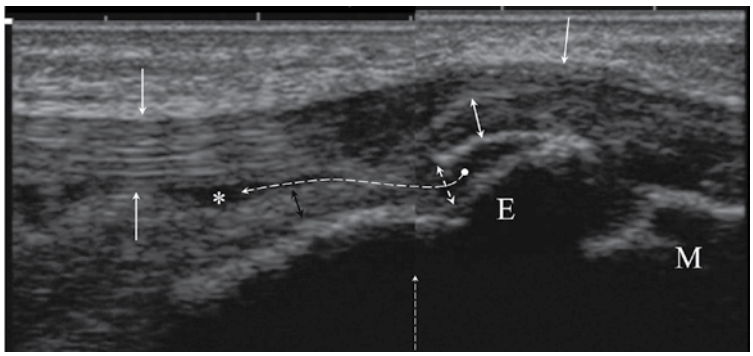


Figure 18. US image of the patient from Figure 17. Longitudinal US image (stitched of two images for better panoramic view) of a delamination tear and cartilage fracture at the patellar ligament's proximal insertion border of the tibial tuberosity ossification center (OSD type II). Arrows, margins of the patellar ligament; double solid arrow, thickness of cartilage between the ossification center and the patellar ligament's insertion; 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; double black arrow, fibrosis on the tibial wall of the deep infrapatellar bursa; E, epiphysis; M, metaphysis; asterisks, effusion within the deep infrapatellar bursa; long dashed arrow, image stitching level.

Ryc. 18. Obraz usg pacjenta z ryc. 17. Obraz podłużny usg choroby OS typu II ("sklejony" z dwóch zdjęć) pokazujący rozwarstwieniowe uszkodzenie jądra kostnienia nasady guzowatości kości piszczelowej z pęknięciem chrząstki wyrostka dziobiastego nasady, bezpośrednio powyżej linii bliższego przyczepu więzadła rzepki do guzowatości. Strzałki, zarysy więzadła rzepki; podwójna strzałka, grubość chrząstki pomiędzy zarysem jądra kostnienia i entezą więzadła rzepki; podwójna przerywana strzałka, maksymalna grubość delaminacji jądra kostnienia/przedniego przemieszczenia bliższej strefy wyrostka dziobiastego/przyczepu więzadła rzepki – wrota łączące wnętrze rozerwanego jądra kostnienia z jamą kaletki podrzepkowej głębokiej; zakrzywiona przerywana strzałka, droga krwawienia z rozerwanych naczyń jądra kostnienia/chrząstki do kaletki podrzepkowej głębokiej; podwójna czarna strzałka, warstwa zwłóknień na ścianie piszczelowej kaletki podrzepkowej głębokiej; E, nasada; M, przynasada; gwiazdka, ślad płynu w jamie kaletki podrzepkowej głębokiej; długa przerywana strzałka, poziom połączenia dwóch zdjęć w celu uzyskania panoramicznego obrazu.

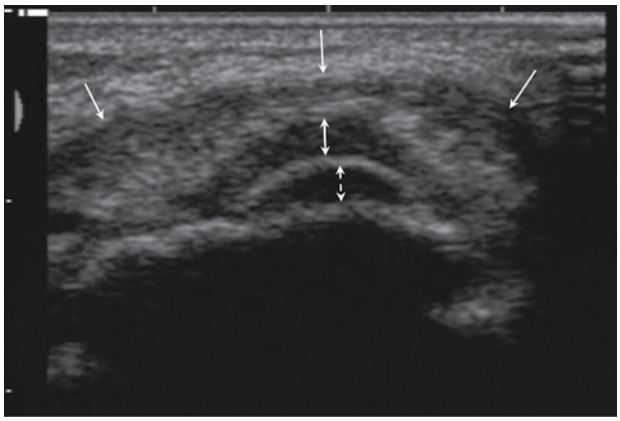


Figure 19. Same patient as in Figures 17 and 18. Transverse US image of a type II injury to the tibial tuberosity ossification center. Arrows, anterior margin of the patellar ligament; double solid arrow, thickness of cartilage between the ossification center and the patellar ligament's insertion; double dashed arrow, delamination thickness in the ossification center. In the transverse plane, the delaminated layer usually resembles a snow Igloo, in most instances not wider than 10mm. In this case the Igloo is sharply delineated, indicating evenly distributed delamination of the very anterior ossification center layer with little internal derangement of its interior.

Ryc. 19. Ten sam pacjent co na ryc. 17 i 18. Przekrój poprzeczny usg uszkodzenia jądra kostnienia guzowatości kości piszczelowej typu II w chorobie OS. Strzałki, zarysy więzadła rzepki; podwójna strzałka, grubość chrząstki pomiędzy zarysem jądra kostnienia i entezą więzadła rzepki; podwójna przerywana strzałka, grubość delaminacji jądra kostnienia. W przekroju poprzecznym, linie rozwarstwionego jądra kostnienia przypominają śnieżne Igloo z przejrzystym dachem. Jego szerokość najczęściej nie przekracza 10mm. W przypadku tego uszkodzenia, ściany i podłoga Igloo są bardzo gładkie co wskazuje na bardzo powierzchowne odlaminowanie jądra kostnienia bez cech rozerwania jego wnętrza. jądra kostnienia. W przekroju poprzecznym, linie rozwarstwionego jądra kostnienia przypominają śnieżne Igloo z przejrzystym dachem. Jego szerokość najczęściej nie przekracza 10mm. W przypadku tego uszkodzenia, ściany i podłoga Igloo są bardzo gładkie co wskazuje na bardzo powierzchowne odlaminowanie jądra kostnienia bez cech rozerwania jego wnętrza.

→ Deep infrapatellar bursitis or fibrosis (or both) due to bleeding from the torn cartilage/ossification center.
→ Disseminated edema/hyperperfusion 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 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.

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.
→ 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.
→ Disseminated edema/hyperperfusion or diffused fibrosis (or both) of the patellar ligament as in type I and II with additional 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 most likely communicating the damaged ossification center with the torn patellar ligament.
→ 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.

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 (Figs. 10 to 15). 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 (Fig. 12). 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 (Fig. 13), 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. In interpreting US studies, it was occasionally difficult to distinguish fibrosis within the bursa from organized synovitis. Three factors determined our interpretation of pathologic changes as representing fibrosis rather than organized synovitis. First, only a few (8 out of 90) patients had detectable tissue hyperperfusion, which is an expected event in the case of an active process such as synovitis. The tissues that exhibited hyperechogenic reflections were not edematous, which usually is the case in other synovial proliferation sites, such as bursae, joints, and tendinous sheets. Second, the impaired movement of the deep infrapatellar fat pad could be caused only by fibrotic adhesions, and this feature was frequently observed during dynamic examination, and was used as one of the criteria. Third, surgeons operating on some patients with OSD described the tissue present within the bursae as "scar-like or fibrotic with adhesions" (personal communication). The presence of fibrosis could also be supported, especially in types II and III, by the fact that the primary reason for pathologic findings within the bursa is bleeding from the fractured cartilage/ossification center into the bursal space; thus fibrosis, rather than a secondary synovitis, is the most natural sequela. That is probably why patients with no sign of fibrosis (4 out of 23) were detected only in type I OSD.

Many delamination tear lesions occur in combination with a cartilage fracture just proximal to the patellar ligament footprint – type II (Figs. 17 to 19). The delaminated tuberosity resembles an igloo, as in type I (Figs. 18 and 19). 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 (Fig. 19). 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 (Figs. 20 to 27) 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 (Figs. 24 and 25). 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 (thus direct contact is made with the tendinous wall of the deep infrapatellar bursa) (Figs. 26 and 27). 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. The term is inaccurate, however, since the calcifications are not dead fragments of previously living bone tissue.

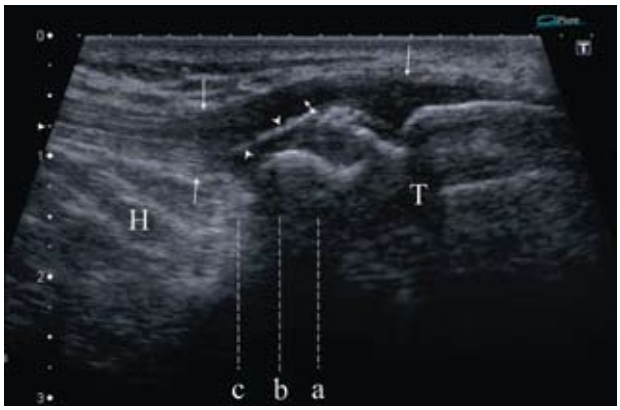


Figure 20. Longitudinal US image of a type III OSD injury to the epiphyseal beak-like area of the tibial tuberosity. Arrows, anterior and posterior margins of the distal patellar ligament; double arrow, cartilage of the tibial tuberosity; arrowheads, line of the cartilage crack and patellar ligament scar in the early ossification phase; a, b, c, levels of the transverse images of this area, shown in Figures 21 to 23.
Ryc. 20. Obraz usg w przekroju podłużnym typu III choroby OS/uszkodzenia jądra kostnienia wyrostka dziobiastego nasady kości piszczelowej. Strzałki, przedni i tylny zarys dystalnego odcinka więzadła rzepki; podwójna strzałka, chrząstka guzowatości k. piszczelowej; groty strzałek, linia pęknięcia chrząstki i blizny więzadła rzepki we wczesnej fazie mineralizacji/kostnienia; a, b, c, poziomy przekrojów poprzecznych tej strefy na ryc. 21 do 23.

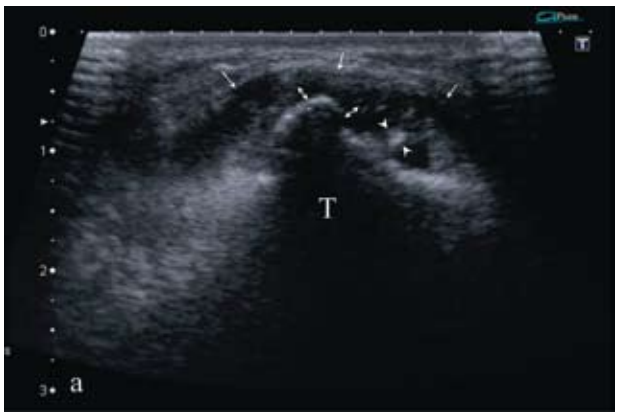


Figure 21. Transverse US image at level "a" from Figure 20. Arrows, anterior margin of the patellar ligament; double arrows, cartilage layer over the deformed tibial tuberosity ossification center; arrowheads, ossifying scar of the torn cartilage.
Ryc. 21. Przekrój poprzeczny usg na poziomie "a" z ryciny 20. Strzałki, przedni zarys więzadła rzepki; podwójne strzałki, warstwa chrząstki nad zdeformowanym jądrem kostnienia nasady; groty strzałek, mineralizująca/kostniejąca blizna uszkodzonej chrząstki.

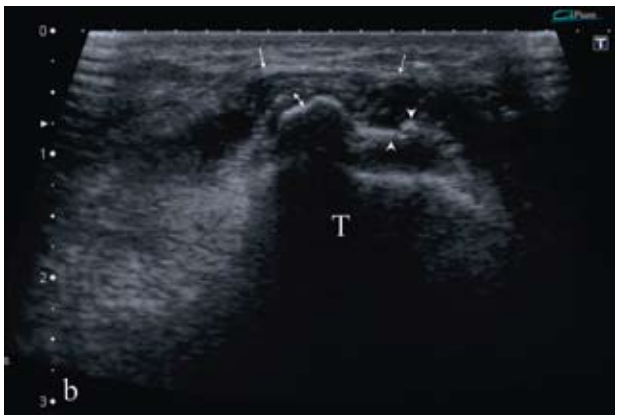


Figure 22. Transverse US image at level "b" from Figure 20. Arrows, anterior margin of the patellar ligament; double arrow, cartilage layer over the deformed tibial tuberosity ossification center; arrowheads, ossifying scar of the torn cartilage and patellar ligament – the possible start of an ectopic ossified or calcified scar within the patellar ligament.
Ryc. 22. Przekrój poprzeczny usg na poziomie „b" z ryciny 20. Strzałki, przedni zarys więzadła rzepki; podwójne strzałki, warstwa chrząstki nad zdeformowanym jądrem kostnienia nasady; groty strzałek, mineralizująca/kostniejąca blizna uszkodzonej chrząstki i więzadła rzepki – prawdopodobny zalążek ektopowego zwapnienia lub skostnienia w obrębie blizny więzadła rzepki.

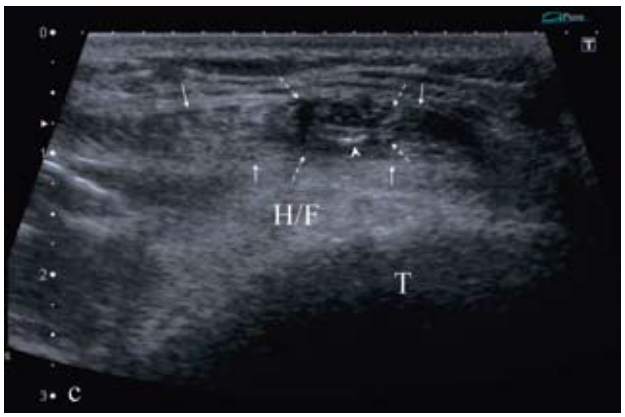


Figure 23. Transverse US image at level "c" from Figure 20. Arrows, anterior and posterior margins of the patellar ligament; arrowhead, calcified area within the scar of the patellar ligament (dashed arrows) – the possible start of an ectopic ossified or calcified scar within the patellar ligament; H/F, Hoffa's fat pad and fibrosis within the deep infrapatellar bursa; T, tibia.
Ryc. 23. Przekrój poprzeczny usg na poziomie "c" z ryciny 20. Strzałki, przedni i tylny zarys więzadła rzepki; grot strzałki, zmineralizowana strefa w bliźnie więzadła rzepki (przerywane strzałki) – prawdopodobny zalążek ektopowego zwapnienia lub skostnienia w obrębie blizny więzadła rzepki; H/F, Ciało tłuszczowe Hoffy i zwłóknienia w kaletce podrzepkowej głębokiej; T, kość piszczelowa.

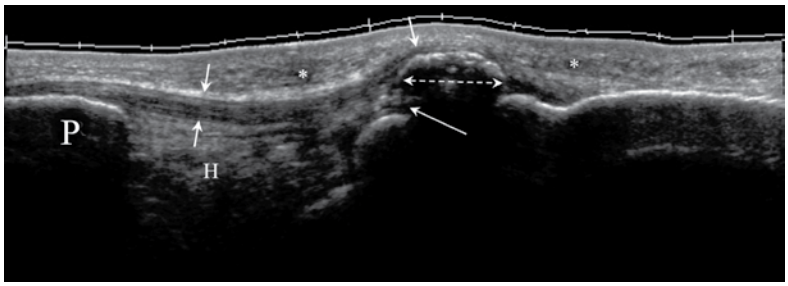


Figure 24. Longitudinal US panoramic continuous reconstruction image of the type III OSD. P, patella; short arrows, patellar ligament margins; H, Hoffa's fat pad. Subcutaneous tissue edema is seen over the patellar ligament and tibial tuberosity (*). Double dashed arrow, massive local calcium deposits infiltrating the entire thickness of the patellar ligament; long arrow, proximal level of the patellar ligament's insertion.
Ryc. 24. Podłużna panoramiczna rekonstrukcja usg typu III choroby O-S. P, rzepka; krótkie strzałki, zarysy więzadła rzepki; H, ciało Hoffy. Obrzęk tkanki tłuszczowej podskórnej ku przodowi od więzadła rzepki i guzowatości kości piszczelowej (*). Podwójna przerywana strzałka, masywne lokalne zwapnienia infiltrujące całą grubość więzadła rzepki; długa strzałka, bliższa granica przyczepu więzadła rzepki.

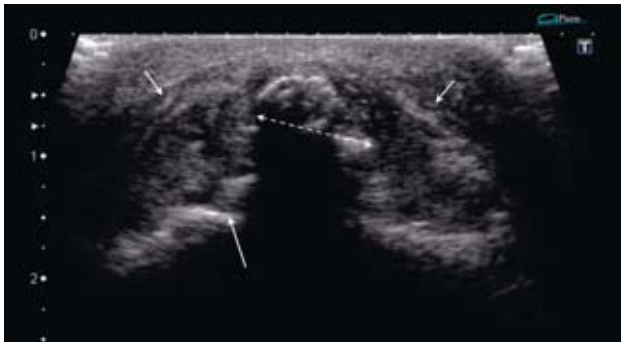


Figure 25. Transverse US image of the patient from Figure 24 at the level of the patellar ligament's ossification. Arrows, margins of the scarified patellar ligament; double dashed arrow, width of a massive local calcium infiltration of nearly the entire thickness of the patellar ligament; long arrow, blurred outline of the disrupted original anterior margin of the tibial tuberosity.
Ryc. 25. Przekrój poprzeczny nad zwapnieniem infiltrującym więzadło rzepki – pacjent z ryciny 24. Strzałki, zarysy zmienionego bliznowato więzadła rzepki; podwójna przerywana strzałka, szerokość zwapnienia infiltrującego niemal całą grubość więzadła rzepki; Długa strzałka, zniekształcony/zatarty przedni zarys guzowatości kości piszczelowej.

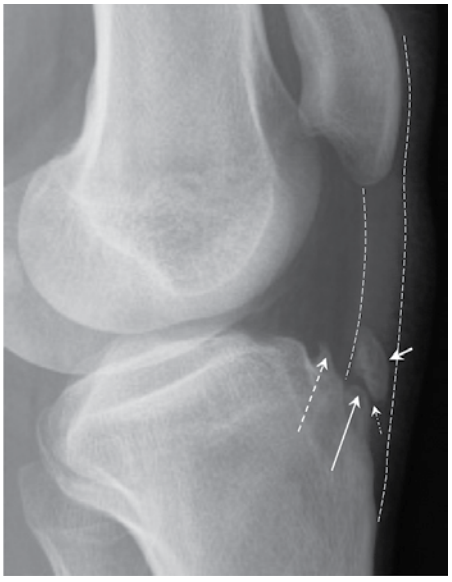


Figure 26. Lateral radiograph of an adult male patient with a history of OSD, type III. Dashed lines, approximate anterior and posterior margins of the patellar ligament; short arrow, ectopic ossification formed within the patellar ligament's scar; short dotted arrow, possible past fracture line between the ectopic ossification and the main part of the tibial tuberosity; long solid arrow, distorted shape of the tibial tuberosity within the epiphyseal footprint of the patellar ligament; long dashed arrow, distorted shape of the tibial wall of the deep infrapatellar bursa, proximal to the patellar ligament insertion, indicating a past serious disruption in the ossification center and multidirectional cartilage fracture.
Ryc. 26. Boczny radiogram dorosłego mężczyzny z przebytą chorobą O-S, typ III. Przerywane linie, przybliżone przedni i tylny zarysy więzadła rzepki; Krótka strzałka, ektopowe skostnienie uformowane w bliźnie więzadła rzepki; krótka kropkowana strzałka, najprawdopodobniej linia przebytego dawniej złamania w strefie łączącej skostnienie z nasadą guzowatości; długa ciągła strzałka, zniekształcony zarys guzowatości kości piszczelowej w obrębie nasadowej części pola przyczepu więzadła rzepki; długa przerywana strzałka,↱

zniekształcony zarys kostny kaletki podrzepkowej głębokiej proksymalnie od bliższej linii przyczepu więzadła , wskazujące na przebyte istotne uszkodzenie zarówno jądra kostnienia jak i jego otoczenia włącznie z wielomiejscowym uszkodzeniem chrząstki powyżej wyrostka dziobiastego.

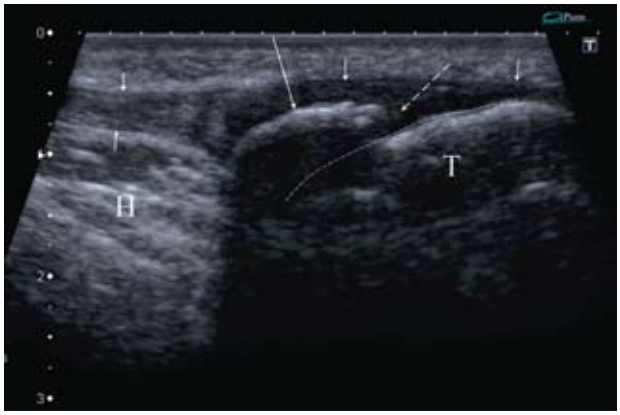


Figure 27. US image of the type III OSD with an ectopic ossification (another patient) shows the following: short arrows, anterior and posterior margins of the patellar ligament; long arrow, ectopic ossification formed within patellar ligament's scar; dashed arrow, distal segment of the possible fracture 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.
Ryc. 27. Obraz usg pacjenta z typem III choroby O-S (inny pacjent niż na ryc. 26) z uformowanym ektopowym zwapnieniem/skostnieniem. Krótkie strzałki, zarysy więzadła rzepki; długa strzałka, ektopowe skostnienie/zwapnienie w bliźnie więzadła rzepki; przerywana strzałka, najprawdopodobniej szczelina po przebytym dawniej złamaniu w strefie łączącej skostnienie z nasadą guzowatości; Przerywana zakrzywiona linia, przedni zarys guzowatości kości piszczelowej częściowo przesłonięty przez zwapnienie/skostnienie. H, ciało Hoffy; T, kość piszczelowa.

A word on 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, stretching 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. Only 4 out of 90 patients did not exhibit fibrosis within the bursa, and all were from the type I group – the mildest form of the disease. In contrast, radiography may not give as final and definitive an 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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