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Review Article

Osgood-Schlatter Disease: A New Perspective and Update of the Classification based on Ultrasonography

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Abstract

This paper describes Ultrasonographic (US) features characteristically seen in the traction type of Osgood-Schlatter Disease (OSD), such as patellar tendon microtears with increased vascularity of the entesis zone, fracture of the tibial tuberosity cartillage even before formation of the beak process ossification center, delamination of the epiphyseal beak process ossification center, fracture of the ossification center's overlying cartilage, patellar ligament tear and mineral formations within it, both deep and superficial infrapatellar bursitis/fibrosis, and soft tissue oedema around the tibial tuberosity. Author offers insight into the pathophysiologic sequence of events and their cause, and propose an updated classification of OSD based on US, which has proven useful in evaluation and treatment of the disease for the last 16 years.

Keywords: Osgood-Schlatter Disease; Tibial Tuberosity; Trauma; Ultrasonography (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 causing pain. One such reason was direct 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 [4]. This paper contains a full insight to the ultrasonographic features of the disease with updated classification. The classification which was presented 16 years ago by Czyrny and Greenspan is supported with 2 new types of the disease.

Physiopathology of The Tibial Tuberosity Region

The tibial tuberosity develops in four stages, beginning with cartilage occupying the whole anterior part of the upper tibia. This cartilage may undergo a fracture even before the appearance of the epiphyseal beak process ossification center. At the age 10-14 the ossification center of the beak process of the epiphysis begins to be formed by a fusion of multiple foci of ossification. Calcium deposits within the ossification center of the tibial tuberosity, appear as scattered cloud-like ultrasound-transparent structures that are often mistaken for fragmentation but merely represent an incomplete fusion of the ossifying center. The productive zone of the tibial tuberosity consists of cellular columns that fit within the cartilage, which undergoes ossification beginning with production of unstructured early bone [5]. This zone, a cellular gap between the layers of solid cartilage, and the newly formed bone lacks reinforcing structures such as the collagen fibers present in the surrounding cartilage. As a result, these cellular columns and unstructured bone, deprived of collagen reinforcement, constitute the weakest structural link of the tuberosity [4]. Next, the ossified cartilage transforms into early bone more or less scattered in the area of the tibial tuberosity (Figure 1); this bone is, at best, only partially transparent to the ultrasound beam. Once the bone of the beak-like process gradually fuses (Figures 2,3), it gives final shape to the physeal part of the tibial tuberosity (Figures 4,5). 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, incomplete bone fusion. At

this point, the tibial tuberosity does not possess a cortical layer. 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 [6].

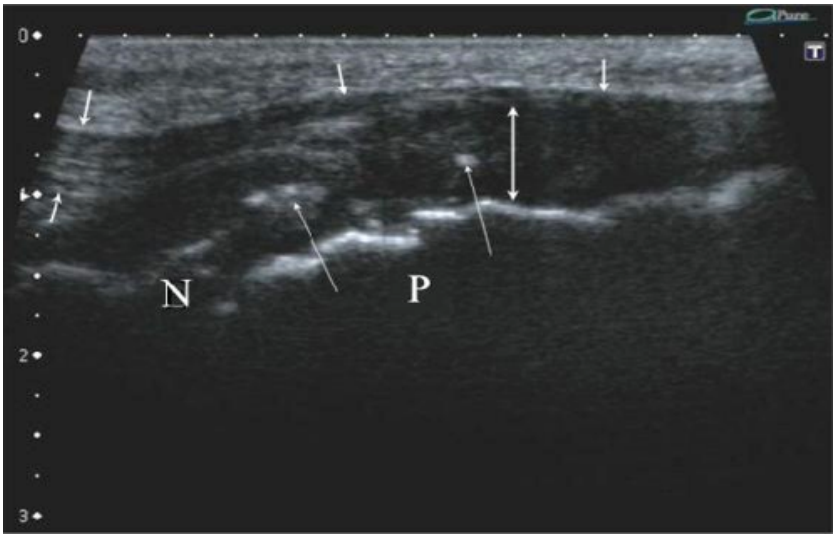

Figure 1: Longitudinal US image of the early stage of bone formation in the tibial tuberosity. Short arrows, patellar ligament; long arrows, ossifying cartilage (ossification center is transparent to the US beam at this stage); E, epiphysis; M, metaphysis; double arrow, thickness of the tibial tuberosity cartilage.

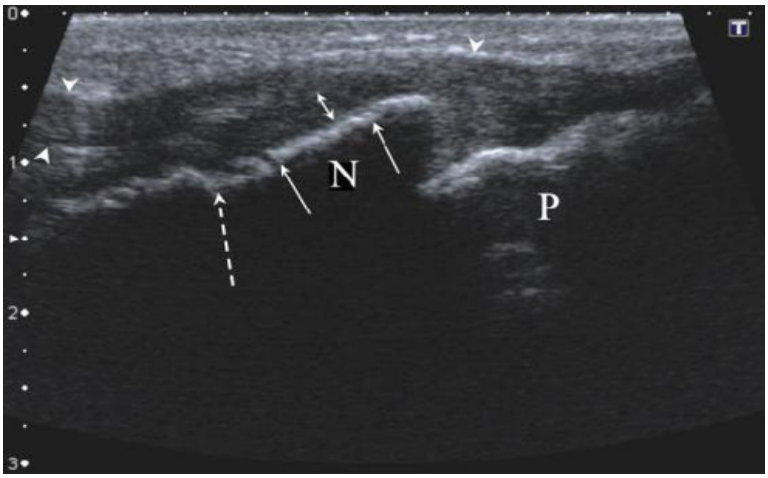

Figure 2: Longitudinal US image of the middle stage of tibial tuberosity bone formation. Arrowheads, patellar ligament; long arrows, ossification center anterior margin (ossified enough to be no longer transparent to the US beam); E, beak-like process of the epiphysis; M, metaphysis; double arrow, cartilage thickness between the ossification center and the patellar ligament attachment; dashed arrow, level of the proximal patellar ligament's attachment.

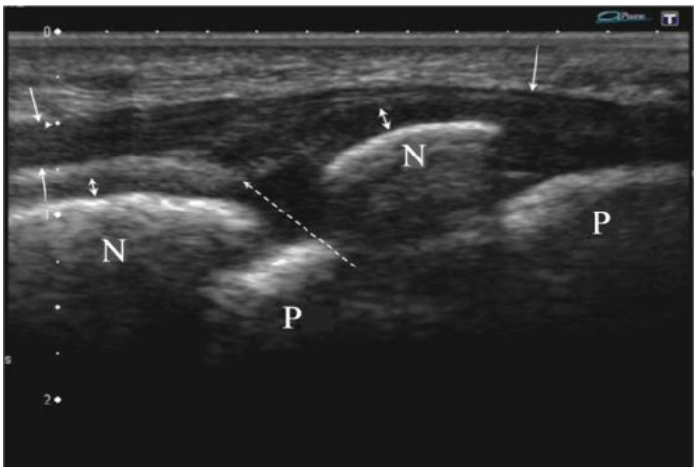

Figure 3: Longitudinal US image of the middle stage of tibial tuberosity bone formation. Short arrows, patellar ligament; E, epiphysis; M, metaphysis; double arrows, cartilage thickness between the ossification center and the patellar ligament attachment (right) and in the area of the deep infrapatellar bursa (left); dashed arrow, the level of the patellar ligament's proximal attachment.

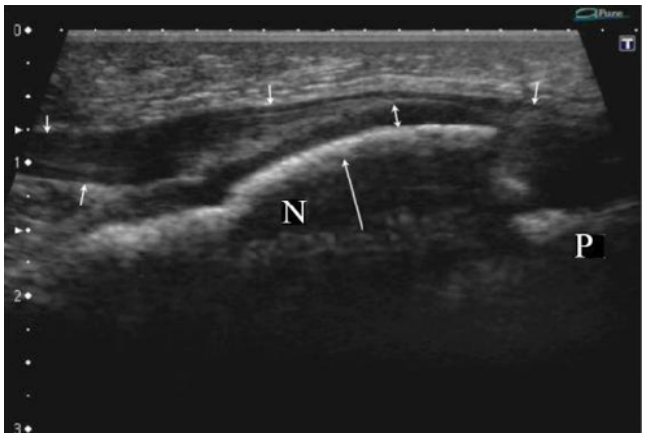

Figure 4: Longitudinal US image of the late stage of tibial tuberosity bone formation with ossification center fusion (long arrow). Short arrows, patellar ligament; E, epiphysis; M, metaphysis; double arrow, cartilage thickness between the ossification center and the patellar ligament attachment.

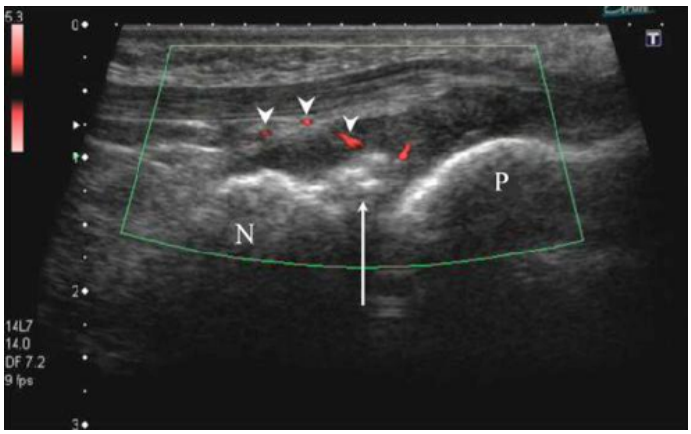

Figure 5: Longitudinal US image of the tibial tuberosity. Arrowheads indicate the same artery (seen clearly as continuous in real-time imaging) within and around the ossification center (arrow), the cartilage of the tibial tuberosity, and the synovial lining of the deep infrapatellar bursa. Increased vascularity of the patellar tendon insertion zone and the tibial tuberosity cartillage is the last feature of the disease that reduces to normal.

The vascularity of the tibial tuberosity is also an important functional/metabolic feature of active OSD. To provide a sufficient supply of nutrients and oxygen to the distorted cartilage or ossification center, the vascularity of this region is, of necessity, extremely rich in a normal state and in some healthy patients single vessel within patellar ligament insertion and within the tibial tuberosity may be observed. These should be compared to the contralateral side as it usually is a normal vascularity of the patellar ligament and the tuberosity. Patellar ligament increased blood supply reflecting increased metabolism/healing is the second most important feature of active disease. Not only it is the earliest sign of the disease but also the last to disappear in control studies meaning the end of the disease. In the previous paper I mentioned the importance of the deep infrapatellar bursa effusion and/or fibrosis (Figure 11). These features are frequently present but have little importance to the wellbeing of the tibial tuberosity region so I will not talk about them here. Osgood-Schlatter disease may be classified into five types:

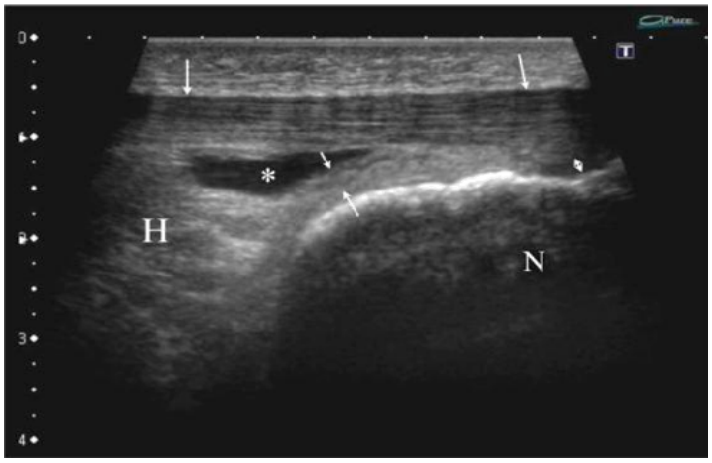

Figure 11: Effusion (*) and fibrosis (short arrows) can be observed within the deep infrapatellar bursa. This side-effect of the injury to the tuberosity is of little importance as far as the come back to normal loads is concerned. Long arrows, patellar ligament; H, Hoffa's fat pad retracted by fluid; E, tibial epiphysis; double arrowhead, cartilage layer.

Type 0

Increased vascularity of the patellar ligament insertion zone with normal structure of the ligament and the tibial tuberosity (Figure 6).

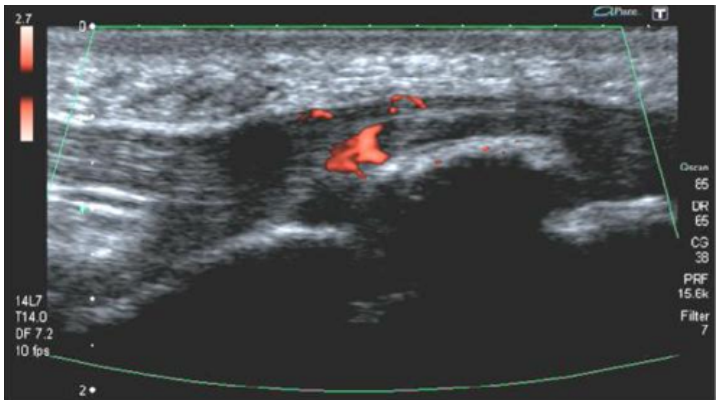

Figure 6: Tibial tuberosity longitudinal view – OSD Type 0. Increased vascularity of the patellar ligament and the tuberosity cartilage without any changes of the tuberosity zone structures.

Type I

Fracture of the tibial tuberosity cartilage before appearance of the beak process ossification center. Increased vascularity of the patellar ligament insertion zone and the tibial tuberosity cartilage (Figures 7,8).

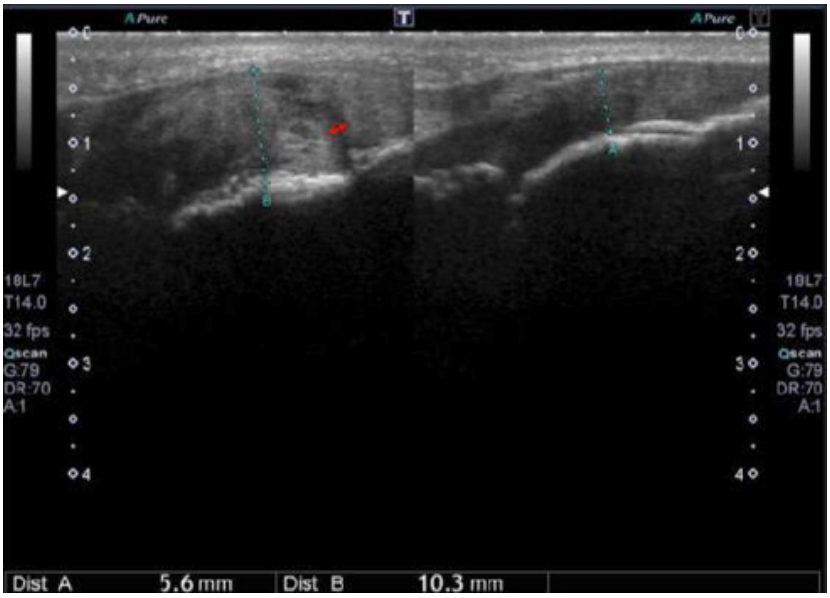

Figure 7: OSD Type I. Fracture (double red arrow on the left image) and disseminated oedema (the whole tuberosity is hyperechogenic and much thicker than the other side) of the tibial tuberosity cartilage before appearance of the ossification center, compared to the contralateral side.

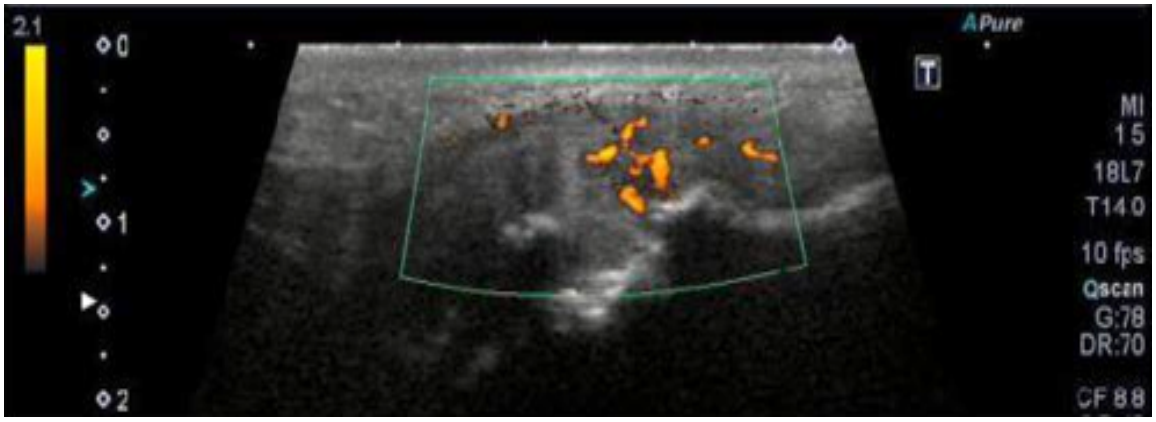

Figure 8: OSD Type I after 9 months. There are signs of cartilage healing with the presence of hypervascularity at the fracture zone – healing in progress.

Type II

Delamination of the beak-process of the tibial tuberosity, without any injury to the host cartilage. Increased vascularity of the patellar ligament insertion zone and the tibial tuberosity cartilage (Figures 9,10)

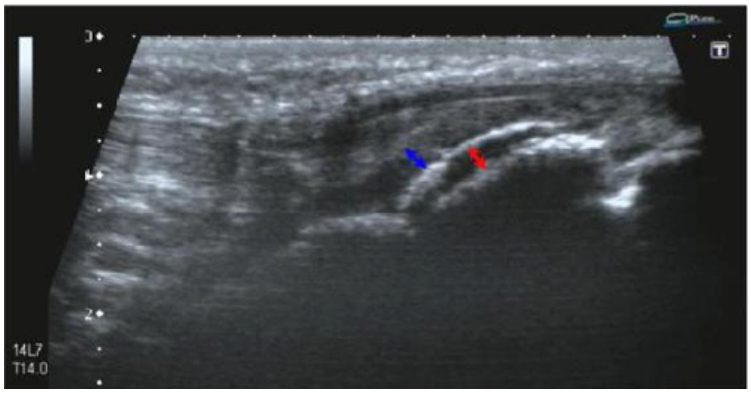

Figure 9: Type II OSD, longitudinal US image. Delamination tear of the beak-process of the tuberosity (double red arrow) with no pathology of the overlying cartilage (blue double arrow).

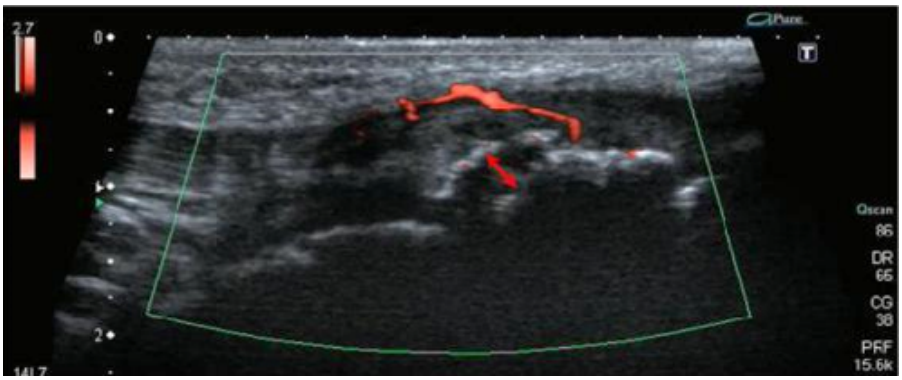

Figure 10: OSD Type II. Delamination (double red arrow) of the beak-process of the tuberosity with increased vascularity/healing of the tuberosity zone.

Type III

Delamination of the beak-process of the tibial tuberosity, with a fracture of the host cartilage directly proximally to the patellar ligament insertion footprint. Increased vascularity of the patellar ligament insertion zone and the tibial tuberosity cartilage (Figures 12,13).

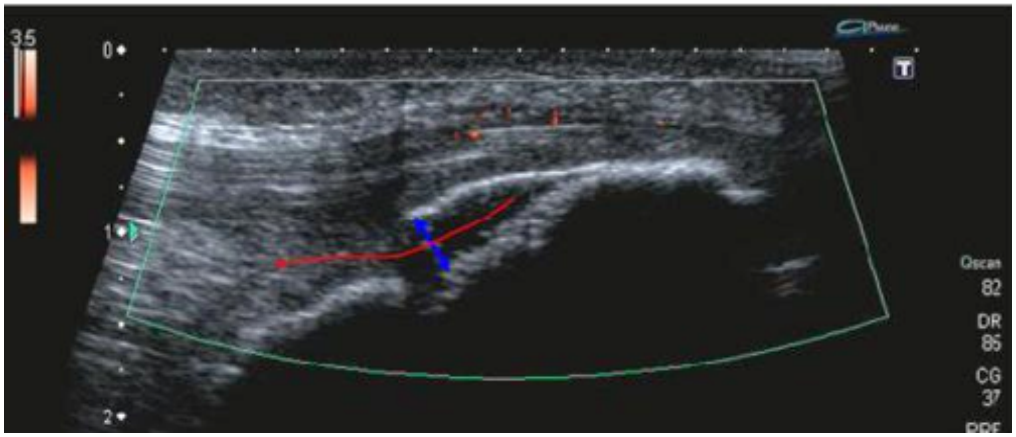

Figure 12: OSD Type III. Delamination tear of the beak-process with the overlying cartilage fracture at the proximal border of the patellar ligament insertion. In this type the fibrosis of the deep infrapatellar bursa is imminent as the cartilage fracture opens the way for the bleeding pour from the ripped ossification center into the bursa.

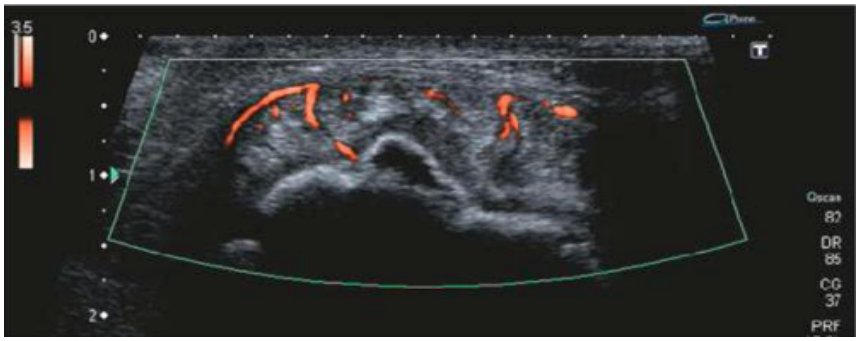

Figure 13: OSD Type III transverse US image (patient from Figure12) showing increased blood supply of the patellar ligament and the tuberosity cartilage.

Type IV

Delamination of the beak-process of the tibial tuberosity, with a fracture of the host cartilage within the patellar ligament insertion footprint. Increased vascularity of the patellar ligament insertion zone and the tibial tuberosity cartilage (Figure 14).

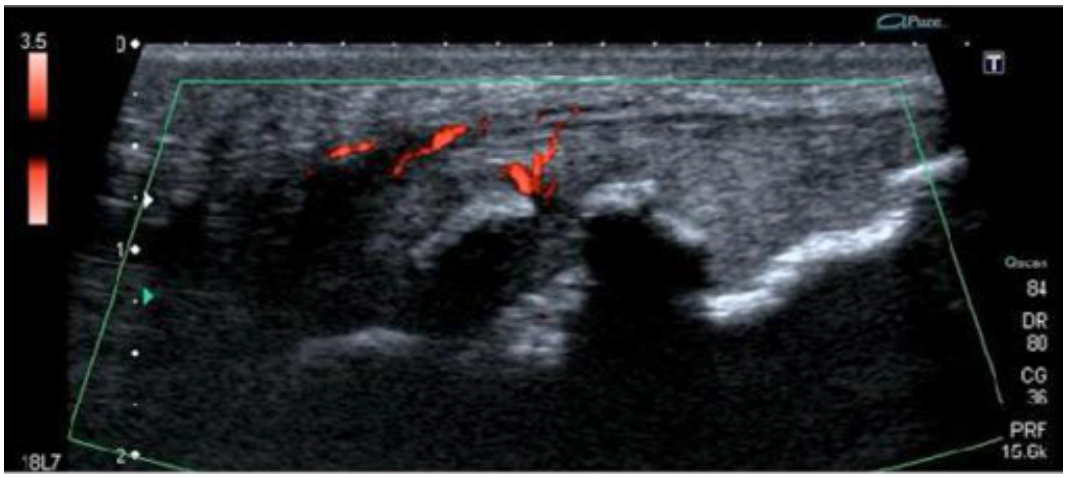

Figure 14: OSD Type IV. Delamination of the ossification center of the beak-process with the overlying cartilage fracture located within the footprint of the patellar ligament enthesis.

In the ossification center delamination types of OSD (II-IV), healing of the ossification center progresses well when not complicated by subsequent injuries. Sequelae consist of an anterior bulging of the tuberosity (Figure 15). The observation of the disease seases at the moment of normalization of the patellar ligament insertion zone vascularity – the end of increased metabolism/healing. When the cartilage fracture is located within the footprint of the patellar ligament's attachment the prognosis is uncertain. It may heal like type II and III. It may also generate an ectopic form within the torn (by the enthesal cartilage fracture) ligament insertion. Those ectopic forms (Figures 16,17) which are detached from the tuberosity consist of a bone, cartilage and fibers – a full enthesis in a mineral shell formed by ossification center cells within a ligament.

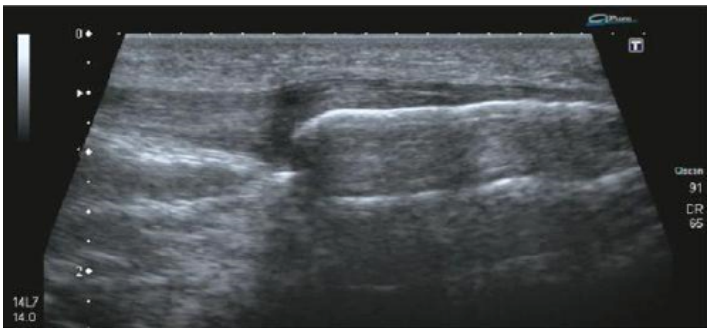

Figure 15: Sequelae of uncomplicated type II-IV as an anterior bulging of the tibial tuberosity.

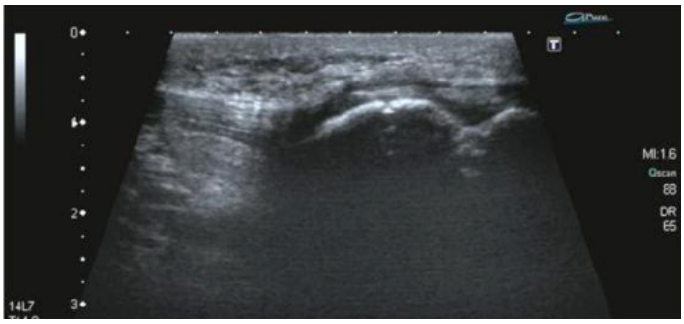

Figure 16: Ectopic form at the tibial insertion zone of the patellar ligament as sequelae of type IV OSD.

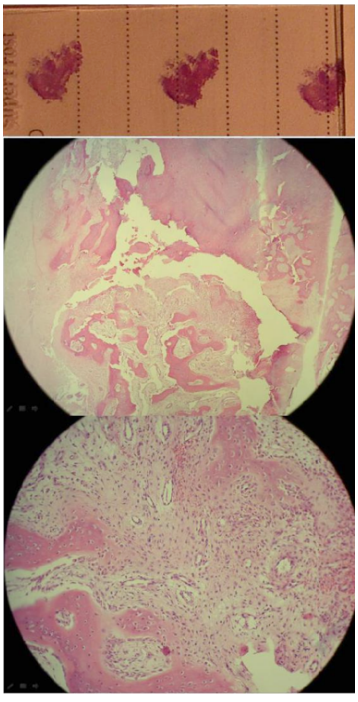

Figure 17: Histological specimen of the ectopic formation of the patellar ligament separated from the tibial tuberosity. Macroscopically it looks like an oval pearl. The specimen shows bone, cartilage and fibers – a full enthesis in a shell.

Conclusion

Ultrasound shows very early and subtle pathologic changes of OSD within the tibial tuberosity complex. US provides excellent visualization of the patellar ligament, the cartilage of the tibial tuberosity and the beak-process ossification center. Even if some doubt exists regarding the tibial tuberosity's structure, the absence of increased vascularity of the tibial tuberosity region (especially the patellar ligament) gives the examiner a margin of safety in excluding an actual disease. In contrast, radiography may not give as final and definitive an answer as US, especially in 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 or metabolical 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 simple and 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 but also in monitoring its course which should be ending when the increased vascularity of the tibial tuberosity region comes back to normal (max. single vessel).

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