Osgood-Schlatter disease in ultrasound diagnostics – a pictorial essay

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Abstract

This paper tries to describe reasons, pathophysiological sequence of events, including the sequelae, and location of the traumatic tears of the ossification center of the tibial tuberosity in adolescents. Ultrasonographic features characteristically seen in the traction type of Osgood-Schlatter disease (OSD) are presented in a pictorial. The classification and definitions used is based on the three types of OSD described by Czyrny&Greenspan in 2009.

Keywords: ultrasonography, Osgood-Schlatter disease, tibial tuberosity

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. The main reason was traction trauma to the tibial tuberosity that led to avulsion of a proximal part of the tuberosity or to incomplete fracture.

OSD disease occurs in patients between 8 and 15 years of age [1-3]. Features of OSD revealed by US, CT and MRI [4-9] 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. Ultrasound has unmatched ability to show the structure of the physeal part of the tibial tuberosity (fig 1).

Fig 1. Longitudinal US image of the late stage of tibial tuberosity (beak-like process) bone formation with ossification center fusion (long arrow). Arrowheads, patellar ligament; E, epiphysis; M, metaphysis; double arrow, cartilage thickness between the ossification center and the patellar ligament attachment.
Probably second most fitted for the diagnostic accuracy would be MRI. It is much more sensitive in the depiction of the bone/cartilage edema (fig 2) and can even sometimes be a more sensitive tool for exclusion of the disease due to subtle oedematic changes which may appear even before presenting a delamination tear of the ossification center.

**Definitions**

**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 (increased perfusion) 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 but after a week of inflammatory process the fibrosis is an inevitable sequela 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 distal ligament. In borderline images comparing to the asymptomatic side, if not affected by the disease, is important. The presence of a single vessel at the patellar ligament’s tibial insertion does not automatically mean pathology.

**Anatomy and histology of the tibial tuberosity**

The anatomic location of the traction injury in OSD is the ossification center of the beak-like process of the tibial epiphysis. It is an endochondral ossification center – 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 terminates it’s growth. This zone, a cellular gap between the layers of solid cartilage and 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.

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 10-15 mm zone is where traction on the patellar ligament generates most of the delaminating force [11]. 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.

**The tibial tuberosity development** may be divided into three stages: **early** (fig 3), **middle** (fig 4, fig 5), and **late-stage** (fig 6, fig 7) of bone formation of the tibial tuberosity.

Fig 8-11 show the normal US aspect of the patellar ligament attachment to the tibial tuberosity

**Classification and features of Osgood-Schlatter disease** (based on Czyrny&Greenspan 2009 [12]).
Fig 3. Longitudinal US image of the early stage of bone formation of the tibial tuberosity beginning with the appearance of cloud-like ossified tissue within the anterior tibial cartilage that may be mistaken for fragmentation but in fact merely represent an incomplete fusion of the ossifying cartilage. 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.

Fig 4. Longitudinal US image of the middle stage of tibial tuberosity bone formation. The ossified cartilage transforms into mature bone more or less scattered in the area of the tibial tuberosity; this bone is usually not transparent to the ultrasound beam any more and presents as white line that is irregular or discontinued. 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.

Fig 5. Longitudinal US image of the middle stage of tibial tuberosity bone formation. Double arrows, cartilage thickness between the ossification center and the patellar ligament attachment (right), cartilage thickness of the tibial wall of the deep infrapatellar bursa. Asymptomatic patient; note the normal arteries within the patellar ligament insertion (a) and cartilage of the ossification center (b).
Fig 6. Lateral radiograph of normal late-stage formation of the beak-like process of the epiphyseal part of the tibial tuberosity (*). The epiphyseal part of the tibial tuberosity overlaps the anterior proximal part of the tibial metaphysis anteriorly. The product of this ossification center was named the “beak-like” process of the tibial tuberosity by Carl Schlatter. E, epiphysis; M, metaphysis; dashed lines, approximate anterior and posterior margins of the patellar ligament.

Fig 7. Sagittal US image of the normal late-stage formation of the beak-like process of the tibial tuberosity. M, metaphysis; 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 (enthesis), 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.

Fig 8. Normal longitudinal US image of the patellar ligament attachment to the tibial tuberosity in an adolescent representing the sagittal crossection of the ligament’s “footprint”. Usually the length and width of the ligament’s footprint in ultrasonographical curved line measurements range between 20 and 40 mm and 20 and 30 mm respectively. The type of OSD discussed in this paper is found only within approximately the 10-15 mm long zone of the proximal tibial insertion of the patellar ligament, which corresponds to the length of the beak-like process. 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.
Longitudinal US image of the tibial tuberosity. The vascularity of this region is, of necessity (supply of nutrients and oxygen to the ossification center) extremely rich. That is why a concept of avascular necrosis in this area seems extremely out of place. There is a common arterial supply to the tuberosity and to the synovium of the deep infrapatellar bursa. This arterial architecture may explain why, even in minor injuries, the inflammatory-reparative process of and around the ossification center may still cause inflammation and later fibrosis in the bursa.

The deep infrapatellar bursa is 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. The fat pad of the bursa is a part of Hoffa’s fat pad. Because of these particular anatomic characteristics the pathologies of the bursa are a part of the OSD in most cases. Arrows, four walls of the deep infrapatellar bursa bursa; P, patella; H, Hoffa’s fat pad; T, tibia; F, femur. Red, the bursae of the extensor apparatus.

US images from full extension (a) to full flexion (c) of the knee. 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.
The pathologic features and clinical consequences of the three types of OSD:

**Type I** (fig 12-20)
- 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.
- Deep infrapatellar bursitis and/or fibrosis due to a shared arterial supply with the tibial tuberosity and local inflammation/reparative process.
- Disseminated inflammation/fibrosis of the patellar ligament secondary to the main injury and deep infrapatellar bursitis. Main character after the acute phase is reparative process.
- Superficial infrapatellar bursitis secondary to deep infrapatellar bursitis and to changes involving the patellar ligament.

**Fig 12.** Type I OSD, longitudinal US image. A “clean” delamination tear, with only a thin anterior layer of the ossification center displaced with no calcified material present within the delamination zone. 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 transversal section in the next figure.

**Fig 13.** Type I OSD, transverse US image, patient from previous figure. 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.

**Fig 14.** Type I OSD, longitudinal US image. A slightly blurred minimal delamination tear of the ossification center. No displacement of the cartilage at the beginning of the patellar ligament’s footprint (long arrow) is observed; Dotted double arrows, cartilage of the ossification center and the tibial wall of the deep infrapatellar bursa; solid double arrows, thickness of the delamination of the ossification center.

**Fig 15.** Type I OSD, transverse US image, patient from previous image. A slightly blurred minimal delamination tear of the ossification center; dotted double arrow, thickness of the delamination of the ossification center; solid double arrows, cartilage of the ossification center and the tibial wall of the deep infrapatellar bursa; arrowheads, patellar ligament margin.
Fig 16. **Type I OSD**, longitudinal US image, patient from previous image. Hyperperfusion of the patellar ligament at the insertion. Most likely the “cousin” of the reparative process at the delaminated ossification center.

Fig 17. **Type I OSD**, longitudinal US image. A “blurred” delamination of the epiphyseal ossification center 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. 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) which is a key difference from type II. 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.

Fig 18. **Type I OSD**, longitudinal US image. Patient from previous figure. Effusion (*) and fibrosis (arrows) due to inflammation can be observed within the deep infrapatellar bursa. Arrowheads, patellar ligament; Hoffa’s fat pad retracted by fluid; double arrowhead, ossification center cartilage layer.

Fig 19. US images of the **type I OSD** with a “blurred igloo”. Patellar ligament edematous and hyperperfused (Doppler frame) at the distal end/insertion, most likely representing a reparative process. A lot of vessels enter the ligament from the superficial arterial network but there is no inflammation of the superficial infrapatellar bursa. They seem to reach the depth of the internal arteries of the ossification center. Double arrow, thickness of the intact cartilage overlying the ossification center; double dashed arrow, approximate thickness of the delamination zone.
Type II (fig 21-27)

- 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.
- Deep infrapatellar bursitis or fibrosis (or both) due to bleeding from the torn cartilage/ossification center.
- Disseminated inflammation/fibrosis of the patellar ligament secondary to the main injury and deep infrapatellar bursitis. Main character after the acute phase is reparative process.
- Superficial infrapatellar bursitis secondary to deep infrapatellar bursitis and inflammation/fibrosis 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.
Fig 23. Patient from previous figure. **OSD type II**, a further close-up. *Arrowheads*, patellar ligament; *double arrow*, thickness of the fracture at the line of the proximal patellar ligament insertion - the gate for the blood to pour from the torn ossification center into the deep infrapatellar bursa. *P*, patella; *T*, tibia; *H*, Hoffa’s fat pad; *HE*, hematoma within the deep infrapatellar bursa; *F*, fibrotic clot separating the fractured ossification center cartilage from the deep infrapatellar bursa.

Fig 24. US longitudinal **type II OSD**. *Arrowheads*, patellar ligament margin; *double black arrow*, the thickness of the anteriorly displaced part of the ossification center; *double white arrow*, distance of anterior displacement of the proximal fractured cartilage; *long arrow*, proximal, anteriorly transferred, insertion of the patellar ligament. Note the disturbance of tibial wall of the deep infrapatellar bursa filled with bright fibrotic tissue (*short arrows*).

Fig 25. US longitudinal **type II OSD**. Patient from previous image. *Double arrow right*, distance of anterior displacement of the proximal fractured proximal cartilage of the beak-like process; *double arrows (two left)*, fibrotic layers induced by previous hematoma from fractured cartilage and ossification center; *long dashed arrow*, the route of blood from the fractured ossification center into the deep infrapatellar bursa.

Fig 26. US transverse **type II OSD**. Patient from previous image. *Double arrow*, maximum delamination distance of the beak-like process; *arrowheads*, patellar ligament margins.

Fig 27. US longitudinal **type II OSD**. Patient from previous image. *b*: contralateral side.
Type III (fig 28-43)

- 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 inflammation/reparative process of the patellar ligament secondary to the main injury and deep infrapatellar bursitis.
- 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 torn tibial insertion.

Fig 28. 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 levels of the transverse images of this area, shown in next figures.

Fig 29. Transverse US image at level “a” from previous figure; type III OSD. 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.

Fig 30. Transverse US image at level “b” from figure 28; type III OSD. Arrows, anterior and posterior margins of the patellar ligament; arrowheads, calcified area within the scar of the patellar ligament; T, tibia.

Fig 31. Normal tibial tuberosity (left) and OSD featuring type III (right). Arrows show margins of the horizontally fractured/torn ossification center. This kind of injury must have torn the insertion of the patellar ligament. Not clearly seen on this image is the fracture and anterior displacement of the proximal beak-like process opening the connection between the ossification center and the deep infrapatellar bursa.
Fig 32. OSD type III. Patient from the previous image. Arrows show margins of the horizontally fractured/torn ossification center; double arrow, the size of fracture and anterior displacement of the proximal beak-like process opening the connection between the ossification center and the deep infrapatellar bursa; F, fluid in the bursa; FI, fibrosis of bursal walls, due to the hemorrhage/inflammation in the bursa.

Fig 34. Longitudinal US panoramic continuous reconstruction image of the mature ossified 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.

Fig 35. Transverse US image of the patient from previous figure 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.

Fig 33. OSD type III, patient from the previous image. F, fluid in the bursa; FI, fibrosis of bursal walls, due to the hemorrhage/inflammation in the bursa.

Fig 36. Longitudinal US image of the OSD type III. Patient 1 month after the first symptoms. There was no effusion or fibrosis of the deep infrapatellar bursa, no evidence of the cartilage tear at the proximal border of the patellar ligament’s insertion (no communication between the torn/fractured ossification center and the bursa. Double dotted arrow, the size of the ossification center fracture; Double long solid arrow, anterior displacement of the fractured/delaminated part of the ossification center; Small solid double arrows, cartilage thickness of the ossification center and the tibial wall of the deep infrapatellar bursa. Note that cartilage over the ossification center is bright indicating it’s oedema. Hyperperfusion within the patellar ligament, cartilage and the floor of the ossification center – reparative process.
• Superficial infrapatellar bursitis secondary to deep infrapatellar bursitis and to a 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.
Fig 41. US longitudinal image of the type III OSD with an ectopic ossification. Arrowheads, anterior and posterior margins of the patellar ligament; long arrows, ectopic ossifications 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, theoretical anterior margin of the tuberosity, partly obscured by the ossification. H, Hoffa’s fat pad; T, tibia.

Fig 42. US longitudinal image of the type III OSD at the border of an ectopic ossification (patient from previous image) shows rebuilt scar of the patellar ligament at the border of the intraligamentous ossification. Deformed anterior margin of the tuberosity.

Fig 43. US transverse of the type III OSD over an ectopic ossification (arrow) (patient from previous image).

References

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