Does Osgood-Schlatter Disease exist in the dog?

Review of human and canine literature and proposed classification system for tibial tuberosity avulsions in the immature dog.

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Keywords

Osgood-Schlatter disease, avulsion, dog, tibial tuberosity, apophysis

Summary

Osgood-Schlatter disease (OSD) is a condition affecting human adolescents in which there is partial separation of bone fragments from the tibial tuberosity at the site of insertion of the patellar ligament to the tibial tuberosity. Tensile trauma seems to be the most likely aetiology. Clinical signs in people consist of swelling and pain at the proximal part of the tibial tuberosity and around the distal end of patellar ligament. Radiographs frequently show small ossicles at the patellar ligament insertion. Conservative treatment is usually curative. The term OSD has also been used for the canine patient. However, radiographs of these patients typically show an enlarged radiolucent line at the apophyseal plate of the tibial tuberosity. This finding is consistent with a mild avulsion fracture of the canine tibial tuberosity. Based on the radiographic differences between the two species, it seems more appropriate to use the term OSD only for people. The purpose of this paper is to review the literature on OSD in people and the reports of injuries to the proximal tibial tuberosity in dogs. In addition, a new classification system for tibial tuberosity avulsion injuries in the immature dog is proposed, with an algorithm for management of this injury.

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Introduction

The first medical descriptions of a painful condition affecting the tibial tuberosity in young human active adolescents were provided by the American surgeon Robert Bayley Osgood (1873–1956) and the Swiss surgeon Carl Schlatter (1864–1934) in 1903 (1, 2). As this specific injury was reported often, Osgood-Schlatter Disease (OSD) became a commonly used term in human orthopaedics (3,4). Other names used in the past were traction apophysitis, enthesopathy of the tibial tuberosity, and aseptic necrosis of the apophysis of the tibia (5–7). Osgood-Schlatter Disease is commonly diagnosed in athletically active adolescents (1, 9). Conservative treatment usually alleviates discomfort (1–9). Although numerous aetiological theories are proposed, trauma seems to be the most likely cause (1–9).

The diagnosis of OSD is well established in people, but reports on similar conditions in dogs are rare (5, 10–14). The purpose of this paper is to review the literature on OSD in humans and to evaluate the use of this term in the canine patient. In addition, a new classification system for tibial tuberosity avulsions in the immature dog is proposed, with an algorithm for management of this injury.

The development of the tibial tuberosity

The development of the tibial tuberosity can be divided into four stages: cartilagenous, apophyseal, epipyseal and bony stage. In the cartilaginous stage an ossification centre is not radiographically visible. People remain in this stage until about 11 years of age (15). In the dog, this stage lasts from four to six weeks of age (16). The apophyseal stage in people is from 11–14 years, and in the dog from four to six weeks until five to six months of age (15, 16). During this stage, one ossification centre of the tibial tuberosity appears in both, people and dogs (8, 19). The epiphyseal stage is characterised by fusion of visible tuberosity bone with the proximal tibial epiphysis. This stage is seen in people between 14–18 years, and in dogs between six to 10 months of age (10, 15, 16). The bony stage is characterised by closure of the physes; this occurs in people at more than 18 years of age and in average sized dogs at 10–12 months of age (10, 15, 16). Ossification of the

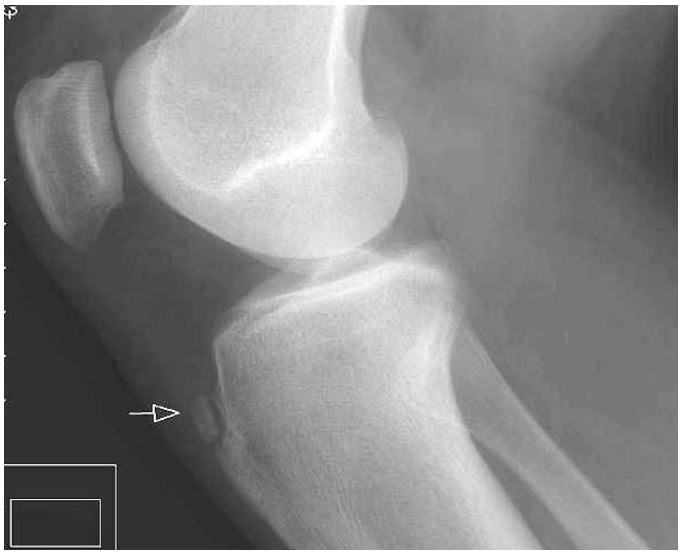

Fig. 1
Radiograph of a 12-year-old boy with Osgood-Schlatter Disease. Note the small avulsed bone at the cranial and proximal aspect of the tibial tuberosity (white open arrow) and swelling of the knee joint.

tibial tuberosity takes place about two years earlier in girls than in boys (17). Completion of the bony stage may be delayed until 18–22 months of age, especially in giant and large breed dogs, but delayed complete fusion of the tibial tuberosity apophysis with the proximal tibial epiphysis is also possible in smaller breeds (19). In people, symptoms of OSD can initiate in stages 1–3 (girls 8–12 years, boys 12–15 years of age) and can persist in stage 4 (15, 16). Dogs are most frequently presented with injuries to the tibial tuberosity in stages 2, 3, and early stage 4 (3–11 months of age) (10).

Aetiology

Most authors support the original theory of Osgood and Schlatter, that forceful contraction of the quadriceps muscle in young human individuals may exceed the tensile strength of the insertion of the patellar ligament, resulting in avulsion of small pieces of cartilage or ossified bone from the tibial tuberosity (▶Fig.1) (8, 16, 20). This theory is supported by findings from an experimental study and various histological examinations (15, 16, 21). Local desmitis of the patellar ligament at its insertion site has also been reported as a possible aetiology for OSD (22).

Other proposed theories such as congenital or developmental malformation, leading to an alteration of the biomechanics of the affected area, have been supported by studies using computed tomography (CT) and magnetic resonance imaging (MRI) (20, 23).

Avascular changes may represent another aetiology. Minor trauma, chronic trauma, genetic predisposition, fat emboli, or thrombi may impair blood supply, resulting in multicentric impairment of the ossification process within the tibial tuberosity and subsequent development of OSD (7). This theory is supported by a microangiographic study in affected children, which showed avascularity of the epiphyseal plate persisted until physis closure had occurred (24). The importance of diminished blood supply has also been described in reports of a combination of OSD and aseptic necrosis of the calcaneal apophysis (7, 9). Finally, osteomyelitis, hypothyroidism, a genetic predisposition, underlying osteochondrosis and infrapatellar bursal osteochondromatosis have been suggested as aetiologies (7, 25–27). However, scientific support for these theories is scarce.

In the case of dogs, there are different opinions on the aetiology for traction injuries to the tibial tuberosity. One paper, describing a litter of Greyhounds, suggests that this breed may be genetically predisposed to the development of osteochondrosis at the apophyseal plate with subsequent mild displacement or complete avulsion of the tibial tuberosity (12). However, another report about racing Greyhounds clearly shows that trauma is the precipitating cause for avulsion (11). Avulsions seem to occur especially in dogs with steep angles of inclination of the tibial plateau, in the range of 35–55° (13). Experimental and clinical studies suggest that, as in people, tensile trauma is the most common cause for injuries to the tibial tuberosity in the dog (10, 16, 17).

History, incidence, and clinical signs

Regardless of the underlying aetiology, recurring trauma and high tensional stress at the insertion at the patellar ligament results in pain (1–9). Boys present commonly in the age range of 11 to 15 years, with a peak at 13 years; in girls the peak age is 11 years (7, 8, 17). This difference may be due to the fact that ossification occurs approximately two years earlier in girls compared to boys (17). The male to female-ratio is 7:1, likely due to the greater participation in specific risk activities by boys in comparison to girls (9). Patients often have a history of recent rapid growth, or vigorous participation in sports (7, 17). The overall incidence in people is reported to be 12.9% (9). Bilateral symptoms are observed in 20–30% of affected individuals (29). Clinical signs usually consist of intermittent or chronic pain in one or both knees while standing, going up and down stairs, running, or when kneeling (1–4, 7–9). Soft-tissue swelling, erythema, and warmth over the proximal tibia are present. Pain can be localised at the insertion of the patellar ligament, and elicited upon flexion of the knee (1–4, 7, 8). Soft tissue-damage seems to be the main reason for the occurrence of clinical signs (22, 20).

While OSD is a well characterised condition in people, reports of traction injury to the canine tibial tuberosity apophysis with respect to OSD are rare (5, 10–14). All dogs in one study were between three to 11 months of age when they were presented with various degrees of lameness; additionally, the dogs exhibited signs of pain upon palpation of the cranial tibia (10). The same study reports an incidence of 0.08% (17 dogs out of 25,500 cases, which were admitted to a hospital over a 10-year period) (10). The incidence of bilateral occurrence is not described.

Diagnostic imaging

While the clinical signs were well known when Osgood and Schlatter first described the disease, the increased use of radiography

in the early 20th century facilitated the diagnosis. Since then, further diagnostic imaging techniques have been used, including CT, MRI, ultrasound, and scintigraphy (22, 30, 31). Radiographs of human adolescents show soft tissue swelling around the tibial tuberosity and the patellar ligament. Joint effusion and *patella alta* may also be present (7, 17, 22). Ossification and calcification at the base or within the patellar ligament can be seen (▶Fig.1) (7, 17, 22). Frequently, these signs are visualised in the later stages of OSD, supporting the hypothesis that initially avulsed chondral fragments enlarge by endochondral ossification as the patient matures (17, 28). Rarely, the injury involves more than just the proximal aspect of the tibial tuberosity (15, 17, 20, 22). An irregular ossification of the proximal tibial tuberosity may be appreciated after healing of avulsed fragments has occurred (22, 31). Elongation of the patella, secondary to relatively rapid lengthening of the femur in relation to the quadriceps muscle during rapid growth, may also be seen with OSD (32).

In contrast to a lesion of the insertion of the patellar ligament (Sharpey's fibres) in OSD, avulsion fractures of the tibial tuberosity are today classified separately in human patients (33). In a type Ia injury only the most distal part of the tibial tuberosity is affected (33). Type Ib shows mild cranio-proximal deviation (33). Type IIa consists of separation of the entire tibial tuberosity with possible propagation of the fracture line into the proximal tibial epiphysis (33). In type IIb injuries, the avulsed fragment is comminuted (33). Type IIIa is described as extension of the fracture line to the stifle joint with discontinuity of the joint surface, while type IIIb shows additional comminution of the avulsed tibial tuberosity (33). It is important to differentiate avulsion fractures of different degrees in people from OSD to prevent confusion (33).

Compared to radiography, MRI has been shown to be more sensitive for the diagnosis of OSD (30). Typical findings include soft-tissue swelling cranial to the tibial tuberosity, loss of the sharp demarcation of the infrapatellar fat pad and surrounding soft tissues, thickening and oedema of the inferior patellar ligament, and infrapatellar bursitis (30). Ultrasonography has also been shown to be a valuable diagnostic method, and may provide similar findings as MRI (31). Bone scintigraphy, however, did not prove to be a valuable diagnostic tool (22).

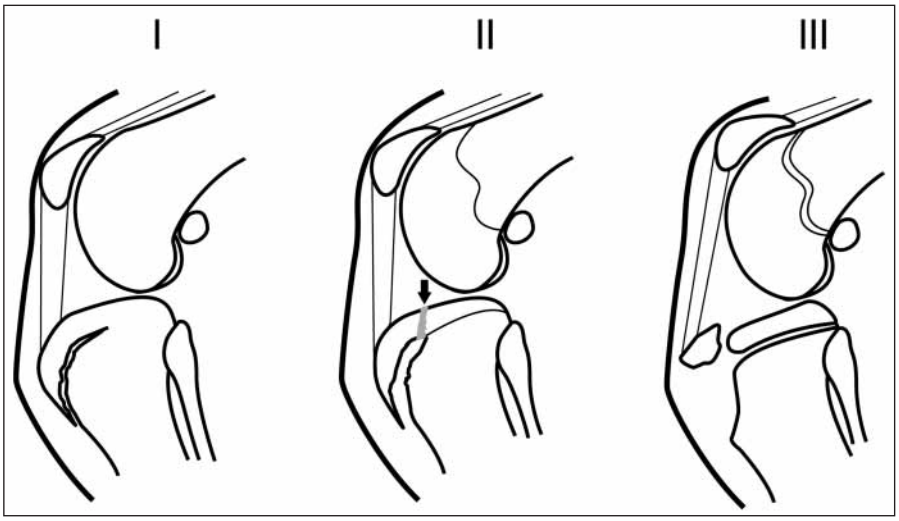

Fig. 2 Suggested classification system for tibial tuberosity avulsions in the immature dog. Type I avulsions show minimal displacement of the tibial tuberosity (<2 mm) with an increased width of the apophyseal plate in dogs, typically just prior to closure of this physis. Type II lesions consist of a fracture through the apophysis. Occasionally, the fracture line may reach caudally into the epiphysis. There may also be moderate displacement of the tibial tuberosity (>2 mm). Infrequently, this fracture may progress into the stifle joint (gray line and black arrow). There is no, or only a very small intra-articular step off (<2 mm). Mild proximal displacement of the patella (*patella alta*) is possible. With type III lesions, the apophysis is widely detached from the tibial epiphysis, resulting in *patella alta*. There is an intra-articular step off (>2 mm). This lesion is typically seen in dogs between three to eight months of age. Type I and II lesions in the canine patient have been previously called Osgood-Schlatter Disease, despite the different radiographical changes seen between dogs and people.

In dogs, damage to the soft tissues is observed infrequently. In contrast to the radiographic findings in people, none of the radiographs of clinically affected dogs seen in our institution (n = 8, unpublished data) or those reported in the literature, showed cartilaginous or bony fragments at the insertion of the patellar ligament to the tibial tuberosity (5, 10–14, 16). Instead, avulsions of the tibial tuberosity of variable degrees can be observed radiographically (▶Fig. 2, Fig. 3A and 3B) (10, 12). In addition, it has been suggested that there is not any relation between the radiographic findings in dogs affected by traction injuries to the tibial tuberosity, and the lesions observed in human patients with OSD (10, 16). As a consequence, it was concluded by Ehrenborg et al. in 1961 that OSD does not occur in dogs, and that this description should be reserved for human patients only (10, 16).

However, if tensile trauma to the insertion of the patellar ligament at the tibial tuberosity is the cause for lesions in both people and dogs, the question remains why different radiographic findings are observed between the two species. Possible explanations could be developmental and anatomical differences. Gillert considers an insufficient anchorage of the patellar ligament to the tibial tuberosity as being the aetiology for OSD for people (7). Dogs might possibly have stronger Sharpey's fibres compared to people. In addition, the site of insertion of the patellar ligament ossifies earlier in dogs than in people (10). In the apophyseal stage, patellar ligament fibres insert into apophyseal cartilage in people, whereas they insert into a remarkably thick bone plate in the dog (28). Finally, quadrupeds may generate different forces through their patellar ligament and tibial tuberosity compared to bipeds, and the angle of patellar ligament insertion at the tibial tuberosity may be different between people and dogs. All these differences could result in different lever arms to the tibial tuberosity when traction is applied to the patellar ligament (▶Fig. 4).

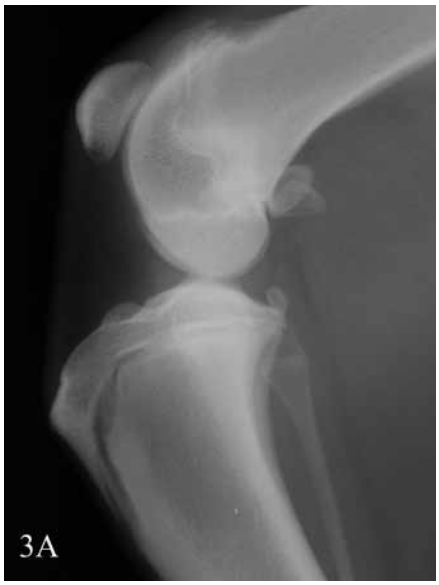

Fig. 3A Lateral radiograph of the left stifle of a 13-month-old Scottish Deerhound, presented for hindlimb lameness. The radiograph shows a none to minimal displaced traction injury of the tibial tuberosity apophyseal plate. According to the suggested new classification system, this appearance may be called a type I avulsion injury. Type I is difficult to diagnose radiographically as well as to differentiate from the final stage of normal closure. Dogs affected from type I injury present lame and react with signs of pain upon palpation of the tibial crest. Thus clinical signs, findings upon examination, and radiographs are all needed to diagnose type I avulsions.

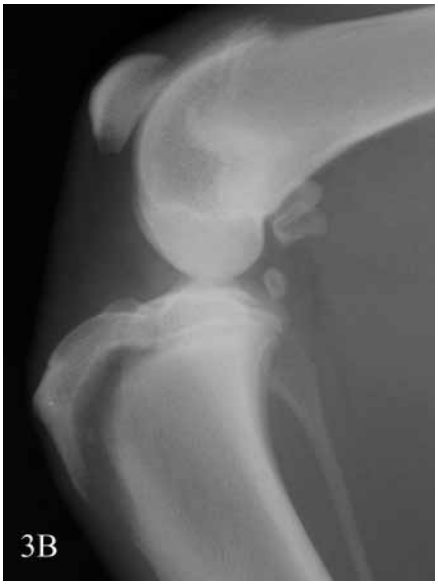

Fig. 3B Lateral radiograph of the right stifle of the same dog, showing a mild displaced traction injury of the tibial tuberosity apophyseal plate, mild soft tissue opacity within the joint space, and mild *patella alta*. The fracture line reaches into the joint. According to the suggested new classification system, this lesion may be called a type II avulsion injury.

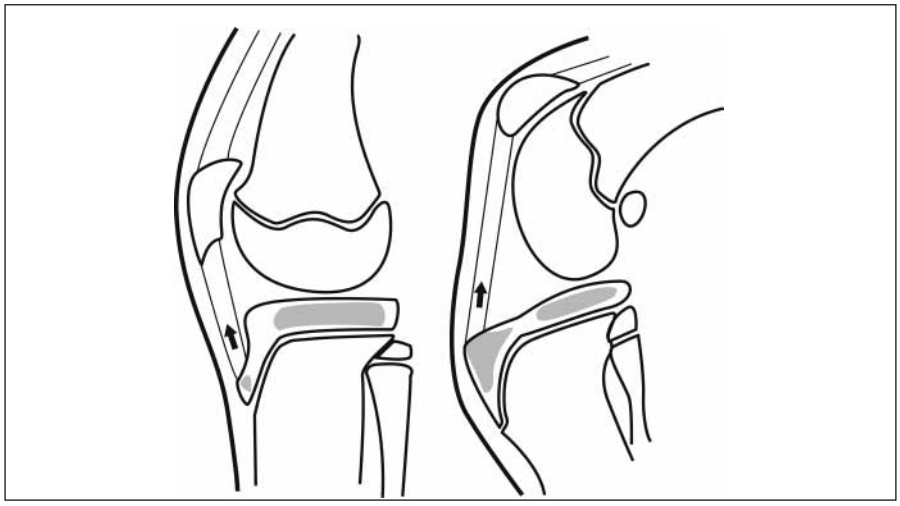

Fig. 4 Schematic illustration, demonstrating the differences in development and anatomy of the tibial tuberosity in people (left), and dogs (right). Ossification of the patellar ligament insertion occurs later in people compared to dogs. Patellar ligament fibres insert into apophyseal cartilage in people, whereas they insert into a remarkably thick bone plate in the dog. The angle of patellar ligament insertion between the two species may differ. These factors may result in different lever arms, causing the typical, species-specific lesions associated with traction injuries to the tibial tuberosity in people and dogs.

Previous classification system for avulsion fractures of the canine tibial tuberosity

Ehrenborg and Olsson described three types of avulsion lesions in the dog based on a clinical case series (10). A severe avulsion of the secondary ossification centre of the tibial tuberosity was described as type I, and less displaced avulsions were described as types II and III (10). This classification may be misleading, because the level of type increased (I to III) despite a progressive reduction in degree of displacement. In addition, the authors of that study did not distinguish between a non-displaced to minimal displaced tuberosity, and a mildly displaced traction injury of the tibial tuberosity (▶Fig. 3A and 3B). However, exactly the same types of lesions were called OSD, or an OSD-similar lesion in dogs described in previously published reports (5, 10–14). In order to improve on the previously described system we suggest using a different classification system for tibial avulsion fractures of the immature dog (▶Fig. 2). The suggested new system is based on data from the previous study of Ehrenborg and Olsson, and on our own clinical observations (n=8, unpublished data) (10). Because these injuries are rare, a large, possibly multi-centre based, evaluation on incidence and radiographic appearance of these injuries would provide a thorough data base to support the herein proposed classification system.

Proposed new classification

Type I avulsions show minimal or no displacement of the tibial tuberosity with an increased width of the apophyseal plate (<2 mm displacement of the tibial tuberosity). This is typically observed in dogs just prior to complete closure of the apophyseal physis (8–13, and up to 18 months of age in giant breeds). Type II avulsions consist of a fracture through the apophysis with the fracture line occasionally reaching into the epiphysis with mild displacement of the tibial tuberosity (>2 mm displacement of the tibial tuberosity). Infrequently, this fracture may also progress into the epiphysis and stifle joint, but there is

no, or only a very small intra-articular step off (<2 mm). Mild proximal displacement of the patella (*patella alta*) may be observed. Type II avulsions are typically observed in eight- to 11-months-old dogs (19). A type III avulsion is an avulsion of the apophysis with a fracture extending through the epiphysis into the stifle joint, causing intra-articular step-off (>2 mm), marked displacement of the tibial tuberosity, and *patella alta*. This avulsion is typically seen in dogs between three to eight months of age (10).

We suggest using the terms type I or II avulsion fractures for conditions that have been previously called OSD in dogs (▶Fig. 3A and 3B). These avulsions should not be confused with a late closure of this physis, as commonly seen in healthy, large breed dogs (▶Fig. 5) (19). Type I is difficult to diagnose radiographically, and to differentiate from the final stage of normal closure. Dogs affected with a type I injury are presented with the complaint of lameness and react with signs of pain upon palpation of the tibial crest. Thus clinical signs, findings upon examination, and radiographic examination are all needed to diagnose type I avulsions. On the other hand, a type I injury is easy to differentiate from more severe avulsions, such as a type III injury (▶Fig. 6).

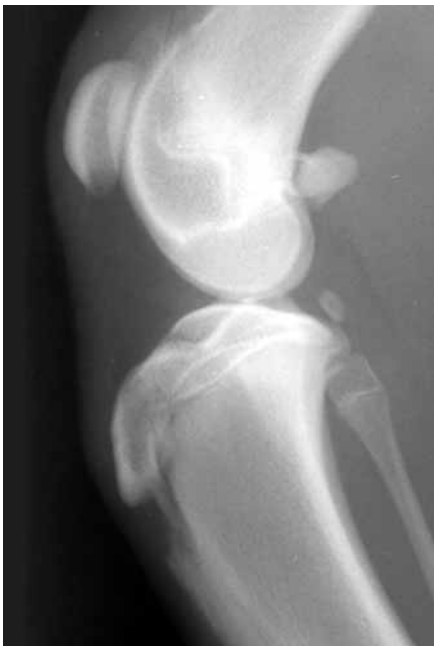

Fig.5 Lateral radiograph of a 14-month-old Newfoundland with a radiolucent area at the distal aspect of the tibial tuberosity. This is the normal appearance of the physis of the tibial tuberosity in large or giant breed dogs. The authors have observed that cartilage of the physis may remain present in large and giant breed dogs until as late as 18 months of age.

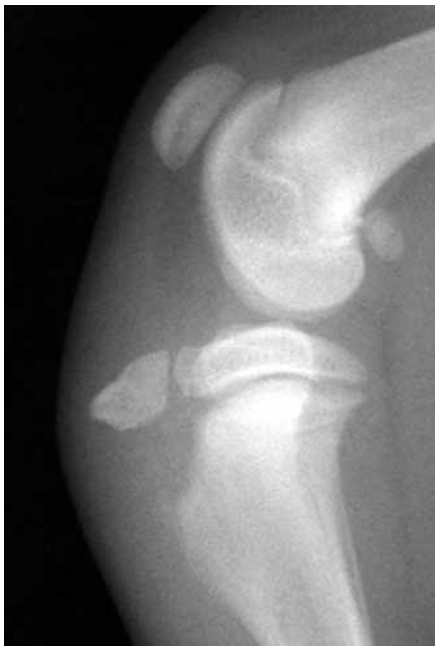

Fig. 6 Severe avulsion of the tibial tuberosity in a four-month-old Labrador. Note the difference in the degree of avulsion compared to the dog in figures 3A and 3B. According to the suggested new classification system, this lesion may be called a type III avulsion injury. Note proximal displacement of the patella (*patella alta*).

Treatment and prognosis

In people, conservative management for OSD is always considered to be the first treatment option (1–4, 8,9, 34, 36). Immobilisation, cold therapy, compression bandages, and the administration of anti-inflammatory agents have been suggested (29). The chances of returning to unlimited activity are reported to be as good as 76–91% (29). However, ununited ossicles may lead to chronic irritation, and the bone prominence that developed during the disease process at the anterior aspect of the tibial tuberosity, may cause difficulties in kneeling activities (15, 26). In these situations, surgery is indicated (26, 34, 36, 37). Resection of the ununited ossicles, excision of the tibial tuberosity or drilling of the tibial tuberosity have been reported (36, 37). Resection of the ossicles or excision of the tibial tuberosity to decrease the bone prominence appear to be the most successful procedures, with a success rate of 88–90% (36). For actual avulsion injuries of the tibial tuberosity in adolescent children, the application of a tension band wire is indicated (33, 34).

Conservative therapy for minimally displaced avulsion fractures (Type I of our proposed classification system) is usually also successful in the dog (10). If a conservative approach is chosen, progression of fusion should be monitored closely. This may help to detect proximal displacement of the tuberosity and malformation of the cranial tibia over time. Such changes could potentially result in chronic lameness, warranting surgical treatment.

Surgery, such as the application of a tension-band-wire (▶Fig. 7), should be considered if there is severe lameness and extreme pain upon orthopaedic examination, if radiographs show moderate to significant displacement of the tibial tuberosity (Type II and type III of our classification system), if fusion of the apophysis is not apparent on follow-up radiographs, if there is consistent lameness, or if there is some combination of

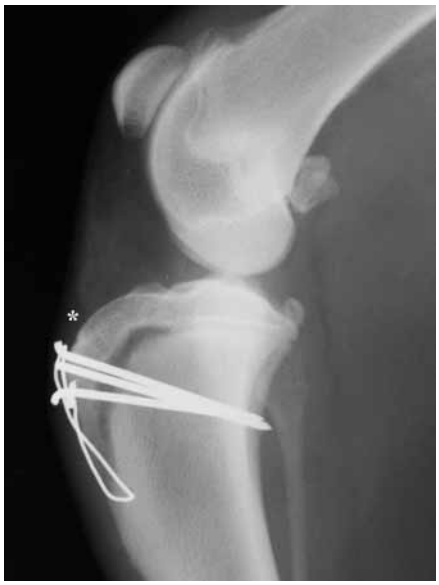

Fig. 7 Lateral radiograph of the right stifle after surgical fixation of the dog in figure 3B. Note the radiolucent line at a 45° angle through the reattached tibial tuberosity (starting at the white asterix), demonstrating the harvesting site of a biopsy core.

these problems (10, 11, 38). A biopsy specimen of the affected area collected at the time of surgery may help to diagnose a potential underlying disease process at the level of the apophyseal plate (▶Fig. 8). Postoperative treatment for avulsion fractures of the tibial tuberosity in dogs consists of application of a light bandage or an off-weight-bearing sling for 10–14 days, as well as the administration of analgesics, non-steroidal anti-inflammatory medication or a combination of both. Activity should be restricted for a minimum of four weeks. The prognosis is usually good (38). However, complications such as delayed union, avulsion of the implants from the tuberosity, or the tuberosity from the implants, implant breakage or bending, patella luxation, deformity of the proximal tibial plateau, or flattening of the tibial tuberosity with distal translocation have been reported (39).

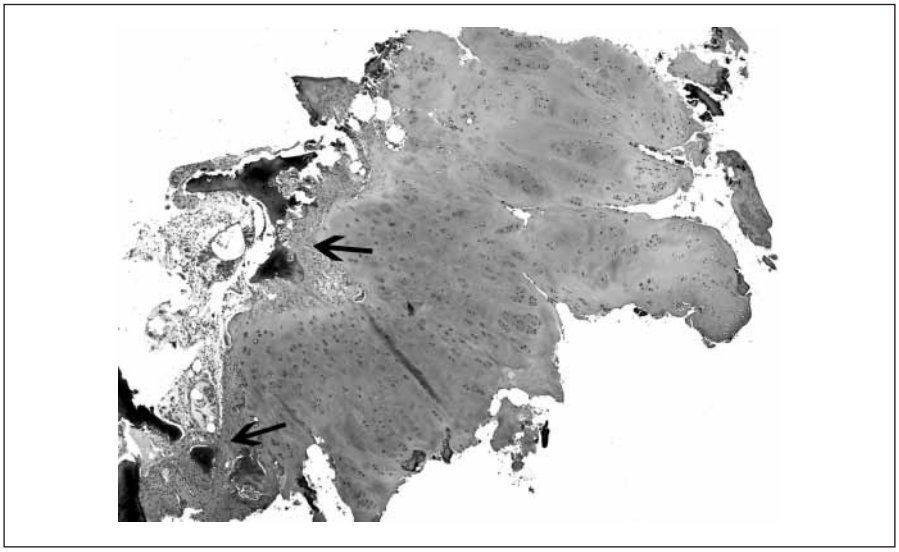

Fig. 8 Photomicrograph of the biopsy specimen from the limb of the dog shown in figures 3B and 7. Note the irregular, sometimes clumped arrangement of chondrocytes in a generally hypocellular and widened physis. There are multiple areas of fibroplasia (black arrows) surrounding small areas of devitalised bone. These changes suggest trauma and repair processes. Hematoxylin and eosin, 4X.

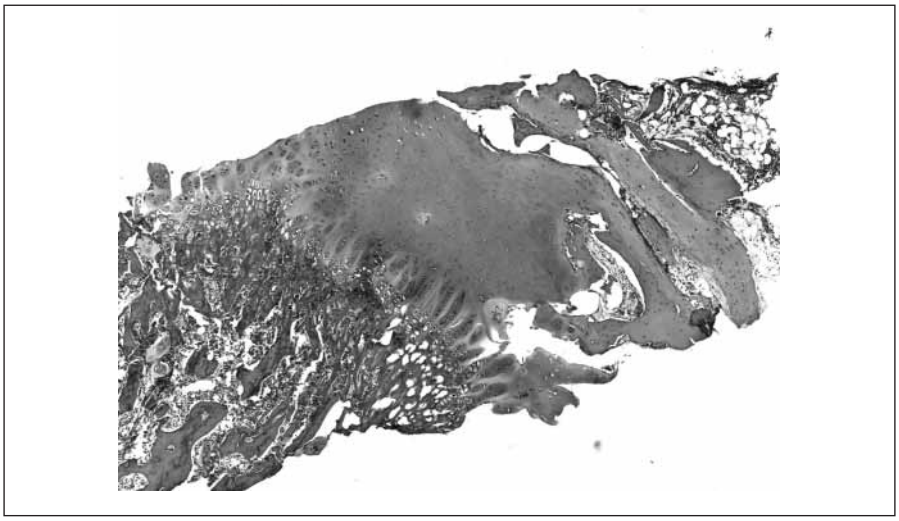

Fig. 9 Comparison photomicrograph of a tibial tuberosity physis shows more healthy tissue in a dog of similar maturity to the dog referred to in figures 3B and 7. This sample was obtained from a nine-month-old German Shorthair Pointer, presented for bilateral grade-3 patella luxation. The owner gave permission to harvest a biopsy of the physis of the tibial tuberosity when performing a transposition of the tibial tuberosity. Note the regular linear arrangement of chondrocytes with orderly ossification. Hematoxylin and eosin, 4X.

Histology

Common histopathological descriptions of samples from people with OSD are scar tissue, aseptic necrotic bone, bone that is undergoing resorption or creeping replacement, changes similar to non-union with pseudoarthrosis in larger bones, and reparative vascularisation (15, 21, 28). These changes occur mainly at the insertion of the patellar ligament to the tibial tuberosity, suggesting a response to trauma (21, 28). However, three reports from 1932 and 1944 described the characteristic scar tissue on the posterior surface of the apophysis in people (15, 21). These histological descriptions could be consistent with mild avulsion injuries. Today, OSD is considered a lesion that is mainly confined to the patellar ligament insertion at the tibial tuberosity (8, 26, 28, 33, 40). While osteochondrosis as an underlying aetiology has been suggested, there is only minimal histological evidence for this theory (41). In contrast, comparably large and detailed histological studies on OSD showed no signs of osteochondrosis in the examined clinical cases (15, 28).

The same seems to be true for dogs. While histology of two out of seven puppies from a litter of Greyhounds suggested osteochondrosis as the underlying cause for avulsion of the tibial tuberosity, the majority of histological samples in dogs support a purely traumatic aetiology for traction injuries to the tibial tuberosity (10, 12, 16). Typical findings for a response to trauma are provided in a clinical case example (▶Fig.8): The physis of the tibial tuberosity in this 13-month-old Scottish Deerhound was wide, hypocellular, and disorganised, with delayed closure. The epiphysis featured scattered areas of osteonecrosis and chondronecrosis. Minimally reactive fibrosis was present along the epiphyseal aspect of the physis, and woven bone formation was associated with more prominent fibrosis along the metaphysis. The histological diagnosis was chronic fibrosing phy-

seal chondritis, resulting from trauma with attempted reparative processes. For comparative purposes, a sample of a control dog is provided, showing the appearance of the physis in a normal dog of similar maturity (▶Fig.9).

The histological evidence of trauma in samples from people with OSD, and in the few available samples from dogs with traction injuries to the tibial tuberosity, support the theory that trauma may also be the most likely aetiology in the majority of the canine patients, presenting not only with type III, but also with type I or II avulsions of the tibial tuberosity (14–16, 21, 25, 28).

Conclusion

Osgood-Schlatter Disease is a traction injury to the patellar ligament–tibial tuberosity complex of young active adolescents. The anterior surface of the tuberosity is affected, and there is not any physeal involvement. Calcified particles within the distal aspect of the patellar ligament are pathognomonic for OSD (▶Fig. 1). In contrast, dogs with similar traction injuries typically show complete avulsion of the entire tibial tuberosity, with physeal involvement (▶Fig. 3A, 3B, and 6). A classification system for tibial tuberosity avulsion injuries in the immature dog is proposed to better describe these minimally displaced injuries (▶Fig. 2). Also, a proposed scheme of treatment is described based on the suggested classification system. Based on the radiographic differences between the two species, it seems more appropriate to use the term OSD only for people.

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