Clinical Commentary

Physitis in the horse

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In this issue, Jackson et al. (2011) describe the clinical, radiographic, computed tomographic and post mortem findings in 2 foals with severe bilateral physitis of the distal third metatarsal bones. The term ‘physitis’ is used to indicate inflammation of the physis, or growth complex, at the end of an immature long bone (Baxter and Turner 2002). The physis contributes to the growth in length of long bones as a horse matures and is a physiologically very active site in the growing animal, and even more so in the horse than in animals of lesser size because of the amount of growth in length that it must accomplish in a very short time before maturity (Fretz et al. 1983). Physeal inflammation has been termed ‘epiphysitis’, although it would be more accurate to use ‘metaphysitis’ because it is the metaphyseal side of the growth plate that is most active and most often becomes inflamed. However, since some growth occurs on both sides of the physis and inflammation eventually affects the entire complex, ‘physitis’ is the more correct term.

With the exception of a small amount of growth in the proximal physis of the proximal phalanx, the linear growth of the phalanges is nearly complete at birth in the horse. The distal cannon bones grow for approximately 4 months. The most active physis in the distal limbs in the horse is the distal radial physis, where the distal aspect of the radius contributes the majority of growth in length of the mature radius (Fretz et al. 1983). The tibia likewise has a distal physis but here the proximal physis contributes the majority of growth in length, in contrast to the radius. Physes more proximally in the limb continue to grow slightly later, but most of the growth in limb length is completed by age 2 years. More proximal physis rarely get physitis to the degree that the distal physis are affected as they are well supported by the soft tissues and not subjected to the same biomechanical forces as the distal physis where the bone is not surrounded by muscular support and the long axis of the limb acts as a lever to increase the mechanical force.

The epiphysis, the end of the bone and the side of the growth plate most remote from the nutrient foramen, has a growth complex similar to the metaphysis, but rather than contributing primarily to length, the epiphyseal growth creates circumferential enlargement in size, enlarging the joint surfaces on the ends of the long bones (Kawcak and Bramlage 1992). Enlargement of the epiphysis continues until the horse is mature in most joints whereas cessation of longitudinal bone growth on the metaphyseal side of the physis and maturation of the physis occurs sequentially from distal to proximal in the limb as the horse ages, starting in utero and continuing until maturity.

The pathophysiology of physitis is multi-factorial and not all physitis is the same disease as has been suggested (Firth 1990). Inflammation of the physis occurs as a result of discordance in the process of longitudinal growth and then maturation, which occurs in concert with increasing body size and activity level, creating a process where growth finishes “just in time” to accept the increased mechanical load of an increasing body size and activity level. If a physis is not capable of accepting the mechanical load asked of it, structural trauma and subsequent inflammation result.

The distal metacarpus, the distal most active physis in the newborn foal, will grow for approximately 4 months. The distal radius and tibia grow for approximately 18–20 months and the more proximal physis of the tibia and radius as well as the physis of the proximal limb will grow for a slightly longer period. Physitis can occur anytime before the physis disappears radiographically. Even when the growth has stopped, the process of maturation of the new bone growth remains vulnerable to structural weakness, inflammation and disturbance in normal physisal maturation.

Normal growth is a 2 stage endeavor: the cartilage cells of the physis proliferate, hypertrophy and degenerate (Bahlage 1993). As the cells degenerate the intercellular matrix calcifies, forming the calcified cartilage layer of the physis. Calcified cartilage is the weakest layer of the physis biomechanically. This is the mineralisation first step of bone formation, where the cartilage growth is gradually mineralised. The calcified cartilage is then converted to trabecular bone by the very active bone remodelling process; this is the ossification step of physisal maturation. Osteoclasts remove approximately two-thirds of the columns of degenerating cells and calcified cartilage matrix, and replace every 3 columns of cells with one
Disturbances in growth can be brought about by a number of causes. By far the most frequent in the foal is infectious. The physis is predisposed to localisation of bacteria and initiation of their growth by the very large amount of vasculature and the low flow vascular organisation. The physis have a plethora of ‘hairpin loops’ where the growth is most active (Baxter and Turner 2002). These loops sample a circulating bacteraemia at a much higher rate than most tissues. The ‘hairpin loops’ create a low flow state near the physis where the flow reverses from the arterial input to the venous drainage allowing bacteria to ‘sludge’ the normal flow, obstruct it, proliferate and establish infection. However, infection is another topic that will not be discussed here.

There are also metabolic causes of physitis where nutritional deficiencies or imbalances can be sufficient to slow or stop the process of maturation of the growing bone. Calcification/mineralisation must be rapid after cartilage proliferation, followed closely by conversion to bone, ossification, to preserve structural strength. The hypertrophy and degeneration of the chondrocytes and mineralisation of the intercellular matrix weakens the structure of the physis. If not rapidly followed by trabeculae formation and ossification, the weaker calcified cartilage begins to accumulate. This increase in the weaker calcified cartilage is vulnerable to physical insult, creating physitis because the stronger trabeculae were not formed rapidly enough to strengthen the bone and withstand the stress. This can occur when the nutritional building blocks for the hydroxyapatite crystals were not available or were not available in the right proportions to form trabecular bone, and it can occur when the cartilage production and degeneration accelerates faster than the ossification process can increase, causing an accumulation of the weaker mineralised cartilage, eventually disrupting the cartilage degeneration causing further accumulation of cartilage (Fig 2).

A period of abundant nutrient intake and very rapid growth increases the foal’s cartilage growth, degeneration and calcified cartilage production, as well as increasing the foal’s body size. This is very common when very rapid periods of grass growth with its excessive content of soluble carbohydrates causes marked weight gain in the foal or yearling. If this process exceeds the ability of the physis to ossify and structurally adapt fast enough, structural damage begins to occur and physitis results.

There are physical insults that can create physitis when the trabecular bone has been formed in a configuration and strength sufficient for one exercise status, and then the exercise is abruptly increased, overloading the unprepared bone. This type of physitis will occur after a period of under stimulation where the foal has been restricted due to disease or lameness, either his or his dam’s. This causes under stimulation of the trabecular bone being formed during the period of inactivity, resulting in relative weakness and lack of preparation of the metaphyseal bone for resumption of normal exercise. A similar situation can occur if a sudden increase in exercise causes overload of the normally prepared trabeculae. This occurs when the amount of exercise is markedly increased. This situation can occur when warm weather approaches and the field exercise is switched from all day to all night.
doubling the amount of exercise time for the foal. The physes may become inflamed because they are not prepared for this rapid increase in exercise stress. Increased exercise stress damages the trabecular bone because it was not prepared (Fig 3).

Direct trauma to the physis can cause physeal inflammation. Salter type I physeal fractures are predisposed by the anatomy of the physis (Jackson et al. 2011). The physis is surrounded by a tough ring of fibrous tissue that supports the weak cartilage growth complex by connecting the epiphysis to the metaphysis. Since the fibrous tissue of the ‘periphyseal ring’ is more flexible than the calcified cartilage of the physis it is possible to disrupt the calcified cartilage layer of the physis without disrupting the periphyseal ring. This injury is a Salter Harris type I physeal fracture, but without disruption of the periphyseal tissue (Fig 4).

A second type of traumatic physeal will occasionally occur when the physis has been manipulated surgically and the growth complex is disturbed sufficiently to create physeal inflammation.

Clinical signs of physitis can be generalised (multiple physes) or localised (one physis, or one pair of physes). Localised physeis is often paired in similar anatomic sites when predisposed to by conformation deformities that overload one or a pair of physes in both fore- or both hindlimbs. Generalised physeis is normally caused by nutrition or exercise alterations that result in overload of all of the immature physes.

Neonatal foals that exercise with their dams during the day will often have transient generalised physeal inflammation commonly noted as quivering of the forelimbs, especially the carpus. When the foal is standing quietly, the carpus appears somewhat contracted. The foal will not lock the limb into a normal weightbearing position, but quivers in the anterior to posterior direction. The carpal discomfort indicates increased muscle tone and discomfort relating to the pain of the physes. After a period of stall rest the discomfort normally disappears as the foal rests recumbent. As long as this discomfort does not accumulate from one exercise period to the next in a 24 h period, this is physiological and the discomfort suffered by the foal will be negated by the aggressive and stronger adaption response of the physis to the exercise level, eventually adapting to the point where the quivering disappears. Generalised physeis of this type in the neonate is common after exercise restriction and then resumption (Fig 1). In severe cases, the contraction becomes cumulative and does not disappear during rest, requiring exercise reduction until it resolves.

In the weanling, especially in commercial horse operations, a physeis commonly encountered affects the distal metacarpal and metatarsal physes preferentially, because these physes chronologically mature at approximately the time when the foal is weaned and switched to a higher concentrate diet. If the weanling foal becomes physically heavier than would be expected for the chronological age and activity level, thickening of the metacarpal/metatarsal physis will occur. Rarely is much inflammation present, just the thickening, which is a physiological adaptation to the increased load (Fig 3).

Yearling physeis generally affects the distal radius and distal tibia; most commonly the distal radius is affected because of the larger amount of growth occurring there. The physis in the metacarpi and metatarsi are already mature in the yearling and therefore the most distal, actively growing physis in the limbs is just proximal to the carpus and tarsus.
The clinical signs of physitis include those of inflammation, pain on palpation or ambulation, heat on palpation, swelling either disseminated around the margin of the physis or localised in the case of conformation predisposition. Thickening will be generalised in the more disseminated causes of physitis and localised in the traumatic or conformational causes.

Radiographic confirmation of the presence of physitis is important to assess the severity of the inflammation and to determine treatment. Physiological physitis where the foal has attained too heavy a bodyweight or the exercise level has exceeded the strength of the under prepared physis, will have normal radiographic findings initially and only become pathological radiographically if chronic. If significant disturbance of bone formation accompanied by physis inflammation begins to affect the growth complex, it is seen as thickening of the cartilage plate due to slowed degeneration and calcification of the hypertrophying cartilage. This situation becomes cyclic as progressive damage disturbs the bone structure, which damages the blood supply, further disturbing the normal sequence of bone growth, resulting in progressive accumulation of the immature layers of cartilage and degenerating and calcifying cartilage (Fig 2). This changes the radiographic appearance of the physis, increasing physisal width and metaphyseal lysis and sclerosis. Metaphyseal sclerosis is seen when calcified cartilage accumulates and the trabecular trauma causes the metaphyseal trabeculae to produce callus, or secondary woven bone, rather than trabecular bone, in an attempt to heal the trauma (Fig 5). This creates metaphyseal sclerosis, loss of trabecular detail with occasional presence of irregular areas of lysis within the metaphyseal bone. The lysis and sclerosis can be disseminated across the entire physis with nutritional or generalised insult or focal in the instance of a traumatic, iatrogenic or conformation predisposed physitis.

Treatment of physitis consists primarily of addressing the primary cause and preventing any secondary damage or permanent angular deformity. This is normally accomplished with exercise restriction, nonsteroidal anti-inflammatory medication, correction of angular limb deformity, and correction of any mis-match between the body size and maturity of the physis.
Correction of the inciting cause is obvious in cases of inappropriate nutrition, inappropriate exercise stress or under preparation of the physis for the current exercise load. It might be necessary to reduce the exercise stress or at least modulate it to a level at which it does not result in pain. Exercise intensity and duration combine to determine the level of exercise stress encountered by the physis. In foals, it is useful to break exercise periods into small segments giving a few hours of exercise multiple times a day rather than one long period of continuous exercise, until the foal adapts. Normal physseal re-adaptation to abrupt increases in exercise stress takes approximately 3–4 weeks after a prolonged period of restricted activity. It will take less time in a yearling, but the time required to heal and reorganise inflammation caused by a disturbance in bone growth will depend upon the amount of disruption and inflammation that is present. Rarely is complete elimination of exercise desirable unless instability is present.

In an unstable physis that has been damaged from traumatic causes it might be necessary to stabilise the physis. It is detrimental to protect the physis totally from weightbearing, so cast immobilisation or total stall rest is not elected unless there is no other choice. Implants can be used in some instances depending upon the cause. It certainly should be used in the instance of fractures that structurally destabilise the physis (Fig 4).

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In the rapidly developing yearling with relative physseal immaturity, anabolic steroids can sometimes be used effectively to advance the maturation of the physis. This approach encourages the process that results in closure of the physis during normal growth. The onset of sexual hormone production is part of the symphony of events that takes place near the end of growth and converts the adolescent skeleton, which contains physes, to the mature skeleton, which does not. Mimicking this process with a short course of anabolic steroids is quite useful in strengthening the physis. Prolonged treatment, which encourages cessation of growth, is not advisable, but limited treatment that matures an immature physis is useful. The anabolic steroids are used in 3 weekly doses at half of the maximum dose recommended. Once a week for 3 weeks mimics approximately how long it takes for a physis to respond to exercise stimulus and produce structurally stronger bone, but is not long enough to close the physes.

Correction of any secondary angular deformities that occur as a result of physitis is important, because angulation will persist after growth ceases. Thus correction of angular limb deformities is a critical component of the treatment of physitis. Elimination of the angular deformity that predisposes to physeal inflammation caused by conformation is also an important treatment consideration.

The prognosis for physitis depends upon the degree of inflammation and the amount of secondary damage that has occurred (Ellis 2003). Serious damage to a physis, especially from traumatic causes, will result in closure of the physis. Focal inflammation as a result of the surgical correction of angular limb deformities using a transphyseal screw can cause physitis and must be assessed and treated to prevent the collapse of the metaphysis and inadvertent creation of another angular limb deformity opposite to the one that is being treated.

The physis is a temporary structure. It disappears at the conversion of the adolescent skeleton to the mature, so any amount of inflammation directly related to the physis is a temporary condition. If secondary problems related to physitis or its associated pain can be avoided, the long-term prognosis is favourable because once skeletal maturity is reached, the physitis disappears with the physis.

References


