Case Report

Severe bilateral physitis with instability and Salter-Harris type 1 fractures in two foals

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Summary

This Case Report describes the clinical, radiographic, computed tomographic and post mortem findings in 2 foals with severe bilateral physitis of the distal third metatarsal bones (MtIII). The foals were admitted because of bilateral hindlimb lameness of several weeks' duration. Initial examination revealed that the physeal regions of the distal MtIII were enlarged, warmer than normal and painful on palpation. Case 1 was treated conservatively and discharged but was readmitted 8 weeks later because of clinical deterioration. In both foals, radiographs showed plantar dislocation of both epiphyses through the physis and a Salter-Harris type 1 fracture. The foals were subjected to euthanasia and a post mortem examination.

Introduction

Physitis is a disturbance of enchondral ossification in the growing horse characterised by enlargement of the physeal region of the long bones and often accompanied by heat, pain and swelling (Baxter and Turner 2002). “Physial dysplasia” is probably a more appropriate term because initially there is no active inflammation (Firth 1990). There are few published studies on physitis. Some authors consider it to be part of the developmental orthopaedic disease (DOD) complex, characterised by alterations in bone growth or development (Baxter and Turner 2002). Although the exact cause of physeal dysplasia has not been identified, the aetiology is most likely multifactorial and includes rapid growth, trauma to the ossification centres, genetic predisposition and nutritional imbalances. There is also speculation that physitis is a manifestation of osteochondrosis (White 1980). The disease manifests mainly between 4–8 months and one and 2 years of age (Baxter and Turner 2002). The distal third metacarpal/metatarsal bones (McIII/MtIII) and the distal radius are most commonly affected clinically, followed by the distal tibia. Usually both front or both hindlimbs and, occasionally, all 4 limbs are affected. Clinical signs include lameness, stiffness, swelling and pain on deep palpation. The most common radiographic finding is an irregular and widened physis (Firth 1990). Histologically, the disease is characterised by arrested ossification, an increase in height of the physeal cartilage and fissures between the physeal cartilage and adjacent metaphyseal region. These findings may indicate impaired metaphysseal blood supply (Firth 1990). Treatment of physitis consists of stall rest, a nonsteroidal anti-inflammatory drug (NSAID) and reduction of the energy content of the diet, adequate mineral supplementation in the feed including a correct balance of calcium and phosphorous. The prognosis is usually good, although cases of angular limb deformities following premature closure of the affected growth plates have been reported (Firth 1990; Ellis 2003).

The present report describes the clinical, radiographic, computed tomographic, post mortem and histological findings in 2 foals with severe bilateral physitis in the distal MtIII with secondary instability and Salter-Harris type 1 fractures.

Case details

Case 1

History, clinical findings and treatment

A 3-month-old Warmblood filly was admitted to our clinic because of bilateral hindlimb lameness of one month duration and difficulty in rising. The lameness was worse early in the morning and after periods of rest. There was no history of trauma. The clinical examination revealed moderate bilateral hindlimb lameness (grade 2/5, American Association of Equine Practitioners lameness scale, grades 0–5) and stiffness in the hindlimbs. Lameness was worse when the foal was turned in a circle on a hard
surface. Both physeal regions of the distal MtIII were enlarged and warmer than normal and deep palpation was painful. The forelimbs were normal. The rectal temperature and results of a complete blood cell count and biochemical profile were within normal limits. Radiographs showed an irregular growth plate of the distal MtIII in both hind legs and sclerosis of the surrounding bone (Fig 1a), which confirmed the diagnosis of physitis. On lateral views, mild plantar dislocation of both epiphyses was evident (Fig 1b). Radiographs of the distal MtIII and the distal radius of both forelimbs showed normal growth plates. The foal was treated with an NSAID (phenylbutazone, 2.2 mg/kg bwt per os q. 12 h for one week and q. 24 h for 2 weeks), mineral supplementation and stall rest for 4 weeks, followed by turnout in a small paddock for another 4 weeks. The dam’s grain intake was reduced.

Follow-up

The foal was discharged and the lameness improved initially, but 2 months later, she was referred to our clinic again because of sudden onset of severe lameness. Both hind pasterns were upright and the physeal regions of both distal MtIIIs were larger than at the time of discharge 2 months previously (Fig 2a). The foal was severely lame in both hindlimbs at walk. The growth plates were warmer than normal and painful on palpation and flexion of both fetlock joints also elicited pain. During manipulation of the fetlocks, there was no mediolateral or dorsoplantar instability. Radiographs of both hind fetlock joints showed extensive new bone formation and deformation of both distal MtIIIs with large radiolucent areas in the growth plates (Fig 2b). Furthermore, both epiphyses were displaced plantarly through the physis (Fig 2c). Computed tomography (CT) revealed a Salter-Harris type 1 fracture of both distal metatarsal bones (Figs 3a and b). The foal was subjected to euthanasia because of a poor prognosis.

Fig 1: a) Dorsoplantar view of the left hind fetlock of Case 1 at initial examination, with irregular growth plate thickness and sclerosis of the surrounding bone. b) Lateral view of the left hind fetlock of Case 1 at initial examination. Mild caudal subluxation of the epiphysis along the growth plate is evident.

Fig 2: a) Enlargement of the distal metaphysis of both MtIII of Case 1 at 5 months of age. b) Dorsoplantar view of the left hind fetlock of Case 1 at 5 months of age. Severe bone proliferation and extensive areas of radiolucency in the growth plates are present. c) Lateral view of the left hind fetlock of Case 1 at 5 months of age. A caudal subluxation of the epiphysis with deformation of the distal MtIII is evident.
Post mortem and histopathological examination

Both distal MtIIIs were collected at necropsy and cut longitudinally in the sagittal plane. Tissue samples were fixed in neutral buffered formalin, demineralised in 5% nitric acid for 2–3 weeks, sectioned and stained with haematoxylin and eosin. As expected from the CT images, the distal epiphyses were separated from the metaphyses and displaced plantarly (Fig 4a). Examination of histological sections showed islands of disorientated cartilage along the fracture line (Fig 4b). There were remnants of necrotic bone, osteoclastic resorption,
the physis remains open and potentially active until foals are about 10 months old (Fretz et al. 1983). Physitis and physeal dysplasia are terms used to describe a disturbance of enchondral ossification in the growth plates (Baxter and Turner 2002). The incidence of physitis involving the distal McII and MtIII in foals is highest between 4 and 8 months of age (Coffman 1973), which coincides with the beginning of physeal closure. Possible causes of physitis include mechanical stress of the physis attributable to excessive bodyweight or strenuous exercise and conformational abnormalities (Bramlage 1993). Both fillies described in this report underwent rapid growth; however, several studies have shown that overfeeding and rapid growth alone are not sufficient to cause physitis (Williams et al. 1982). A genetic predisposition and several other factors are thought to sensitise the physis to trauma or compression (Williams et al. 1982). Another possible cause of physitis is structurally deficient bone, which results in cartilage retention and poor trabecular bone formation and eventually inflammation of the physis when the deficient bone collapses (Bramlage 1993). In our 2 cases, it is conceivable that instability of an abnormal growth plate led to a shear fracture through the physeal cartilage (Salter-Harris type I fracture). In Case 1, histological examination revealed massive new bone formation and fibrous tissue in the space between the separated metaphysis and epiphysis. This was most likely the result of continuous shear forces that acted in the physis secondary to cranial displacement of the metaphysis, in combination with resorption and further deterioration of the abnormal growth plates.

A similar condition that involves separation of the proximal femoral epiphysis has been reported in dogs, swine and humans. This condition is not associated with trauma and in adolescent human beings is recognised as a distinct clinical syndrome referred to as slipped capital femoral epiphysis (SCFE; Moores et al. 2004). This syndrome occurs most commonly during periods of rapid growth, when the stability of the physis is relatively low because of a mismatch between a high volume of hypertrophic cells and a comparatively low volume of interstitial matrix (Moores et al. 2004). It is bilateral in 20–30% of cases (Dupuis et al. 1997). Slipped capital femoral epiphysis may be classified as preslip, acute or chronic and stable or unstable. The displacement of the epiphysis is gradual but the epiphysis does not become separated from the femoral neck. This condition is considered stable when the capital epiphysis remains firmly attached to the femoral neck (Moores et al. 2004). Both foals described in this report had a gradual displacement of the epiphyses with increase in lameness and no history of trauma. Furthermore, although the occurrence of microinstability could not be assessed clinically, no instability was detected on gross manipulation of the distal metatarsal bones and fetlock joints. Therefore, the Salter-Harris type I fractures diagnosed in the 2 foals appear to resemble chronic bilaterally stable SCFE.
Epiphysiolysis, which is considered a traumatic lesion associated with osteochondrosis in the growth cartilage, is a similar condition that occurs in swine (Grondalen 1974). Both foals had undergone rapid growth and it is conceivable that osteochondrosis may have played a role in their condition, especially in view of the bilateral occurrence of physitis. However, the growth plates of Case 1 were examined histologically at an advanced stage of the disease and it was not possible to determine whether a pre-existing cartilaginous defect had been present. Therefore, the primary cause of epiphyseal displacement could not be determined. Histological examination at an earlier stage of the disease would be more revealing, although this is not feasible in a clinical situation.

In adolescent people with SCFE, the outcome of conservative treatment is good in most cases (Ordeberg et al. 1984), although continued displacement is seen in some patients. Therefore, surgical treatment involving stable fixation of the epiphysis and femoral neck using pins and/or screws is preferred (Loder et al. 2000). In the present report, in Case 1 we used conservative treatment with an NSAID and mineral supplementation. In retrospect, early weaning of the foal and more severe restriction of exercise should have been instituted in this foal. Although the onset of displacement of the distal epiphyses had been diagnosed in Case 1, surgical fixation was not considered. The radiographic and CT examination 2 months later showed worsening of the displacement, lack of evidence of bone healing and separation along the physis, which was confirmed at post-mortem examination. It is questionable whether stabilisation by internal fixation in the early stage of the disease, which is done in man with SCFE, should be considered in foals. In our opinion, a DCS plate or 2 transphyseal screws and a cast could stabilise the physis and prevent further displacement and fracturing. However, the growth plates of both foals had undergone rapid growth and it is questionable whether stabilisation by internal fixation in the early stage of the disease would be more revealing, although this is not feasible in a clinical situation.

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Conclusions

This report describes gradual displacement of the distal epiphyses of MtIII in 2 foals. We propose that severe physitis led to instability of the growth plate and subsequently to a pathological fracture. The clinical manifestation of physitis in our 2 cases is similar to that of slipped capital femoral epiphysis in people, characterised by chronic displacement of the epiphysis without actual separation (Morscher 1968). Computed tomography yielded a definitive diagnosis of a Salter-Harris type I fracture of the distal metatarsal bones. To ensure an early diagnosis of such fractures, foals with physitis that do not respond to conservative treatment should be monitored closely using serial radiographic examination. When early signs of plantar dislocation of the distal epiphysis are detected, surgical stabilisation of the bone using internal fixation should be considered to prevent further displacement and fracturing.

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References


