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A study of ten cases of focal peritarsal infection as a cause of severe lameness in the Thoroughbred racehorse: clinical signs, differential diagnosis, treatment and outcome

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Summary

The differential diagnoses of tarsal joint infection, fracture and peritarsal focal infection are of particularimportance in practice. The objective of the present report is to provide additional clinical data to assist in the understanding of one of these conditions. The haematological parameters, clinical signs, treatment and outcome of 10 cases of severe lameness associated with peritarsal infection are reviewed. All horses had a significant rise in rectal temperature. The majority of cases (6/10) had haematological changes consistent with acute bacterial infection on the day of first examination. In the remaining cases these changes were not apparent on a blood sample taken on the first examination, but became so after24 h. Nine out of 10 cases made a complete recovery on a regimen of i.v. antibiotic therapy, in conjunction with administration of nonsteroidal anti-inflammatory drugs and repeated forced exercise. One case developed osteomyelitis of the third and fourth metatarsal bones, but made a full recovery in due course. It is concluded that, in the majority of cases, prompt treatment with antibiotic therapy, analgesics and forced exercise results in complete resolution of the condition.

Introduction

Pain is frequently associated with bacterial infection. Joint sepsis is one of the most painful conditions encountered in equine practice (Schneider *et al.* 1992; Palmer and Bertone 1994). The diagnosis of joint infection is usually consequent upon a set of clinical signs involving heat, pain and swelling of the joint concerned; and changes in the constituent components of the synovial fluid indicating inflammation and infection. In the horse, there is a regularly encountered condition which mimics many of the features of a septic joint, but is associated with very localised, periarticular infection, around the tarsus. It is important that this condition is recognised and understood, as attempted needle entry of the tarsal joints, with the needle passing through the infected areas, could result in genuine joint sepsis.

The degree of pain shown by these horses, in conjunction with swelling of the tarsal region, often elicits a request by the owner or agent for radiographic examination. The differential diagnosis of severe lameness associated with swelling in this region would include fractures of the lateral malleolus, fractures of the trochlear of the talus, or slab fractures of the central or third tarsal bones (Stashak 1987). However, cases of fracture rarely show the degree of distress, pain on palpation and lameness seen in the currently described condition.

The prognosis for peritarsal infections is usually good with conservative treatment, but would be significantly worsened if inadvertent iatrogenic joint sepsis was produced. This study reviews the clinical signs, haematological findings, treatment and outcome of 10 of these cases, in the hope that this information may be of help in alerting colleagues, who may not be familiar with this syndrome, to recognise it on their first encounter and to differentiate it from traumatic injuries to the bones of the tarsus.

Materials and methods

Clinical case records of 10 horses under the care of the authors affected with acute onset lameness, associated with peritarsal infection, were collected over a 2 year period. In all cases, a blood sample was collected and submitted for haematology and biochemistry and, in *Cases 4*, *5*, *9* and *10*, follow-up blood samples were also collected 24–48 h after the onset of clinical signs. The population under study comprised approximately 600 Thoroughbred racehorses in training that were directly under the clinical care of the authors during the 2 year period of the study.

Haematology was performed using an ADVIA 120 haematology analyser[1] with equine software (Anon 1999). The plasma viscosity was measured according to the method of Harkness (1971) using a Coulter Viscometer II[2]. Fibrinogen was assayed using the nephelometric method on an ACL 200 coagulation analyser[3] according to the method of Rossi *et al.* (1988). The serum amyloid A was measured using a Hitachi 717 biochemistry analyser[4] using the immunoturbidimetric method (Wada *et al.* 1977; Yamada *et al.* 1993).

Rectal temperature on first examination was recorded. The treatment regimen of the affected horses was also recorded and the outcome noted.

Results

Clinical signs

All horses showed an acute onset severe hindlimb lameness (Table 1). In *Cases 3* and *6*, the horse appeared to be in a ‘shock-

TABLE 1: The clinical signs, treatment and outcome of the 10 cases reviewed

Horse	Age	Sex	Affected leg	Rectal temp. at onset (°C)	Lameness on Day 1 (1–5)	Antibiotic used	Duration of treatment	Outcome at 1 month
1	3	G	LH	39.3	4/5	Oxytetracycline (5 mg/kg bwt s.i.d.)	6 days	Sound and in full training
2	2	G	RH	39.4	4/5	Oxytetracycline (5 mg/kg bwt s.i.d.)	5 days	Sound and in full training
3	3	G	LH	39.6	4/5	Oxytetracycline (5 mg/kg bwt s.i.d.)	5 days	Sound and in full training
4	3	G	LH	39.4	5/5	Penicillin (9 mg/kg bwt b.i.d.) Gentamicin (6.6 mg/kg bwt b.i.d.)	5 days	Sound and in full training
5	3	G	RH	39.2	4/5	Oxytetracycline (5 mg/kg bwt s.i.d.)	7 days	Sound and in full training
6	3	E	LH	38.6	4/5	Ceftiofur (2.2 mg/kg bwt s.i.d.)	6 days	Sound and in full training
7	2	E	LH	39.7	4/5	Oxytetracycline (5 mg/kg bwt s.i.d.)	9 days	Sound and in full training
8	3	G	RH	39.2	5/5	Ceftiofur (2.1 mg/kg bwt s.i.d.)	15 days	Recurrent lameness
						Penicillin (9.0 mg/kg bwt b.i.d.), gentamicin (6.6 mg/kg bwt b.i.d.) and metronidazole (20 mg/kg bwt *per os* b.i.d.)	10 days	Recurrent lameness (osteomyelitis)
						Oxytetracyline (5 mg/kg bwt s.i.d.)	30 days	Sound
9	3	G	LH	39.3	4/5	Oxytetracyline (5 mg/kg bwt s.i.d.)	5 days	Sound
10	3	E	LH	38.8	3/5	Oxytetracyline (5 mg/kg bwt s.i.d.)	5 days	Sound

like'state, hyperventilating, sweating and profoundly depressed. In other cases, the horses simply showed a total unwillingness to bear weight on the affected limb. In each case there was obvious localised swelling either dorsolaterally or dorsomedially (Figs 1a,b). Manual examination of the tarsus was profoundly resented and the horse showed apprehensive behaviour when flexion of the limb was attempted. Many of these horses would stagger across the box on the remaining 3 legs to avoid the limb being touched. The site of the swelling was hot to the touch when compared to the remainder of the limb. Only light digital pressure elicited an extremely marked pain response. Rectal temperature was raised in every case between 38.6 and 39.7ºC.

When these horses were forced to walk, the first 3 or 4 steps were taken with the affected limb semiflexed and either bearing no weight or having the toe touched to the ground. If the horse was forced to walk further the lameness lessened with each step, and, in many cases, after a 20 m walk, the horse was bearing weight but still significantly lame at walk. In most cases, the heel would gradually sink lower to the ground with each step. In several cases there was a 'corrugated'appearance to the skin on either side of the tarsus, indicating engorged lymphatic vessels.

In most horses, there was a small wound or area of abraded skin on the distal limb, usually associated with 'cracked heels' or an interference 'brushing' injury on the medial aspect of the metatarsophalangeal joint. This separate wound appeared painfree on palpation in every case, and there was no oedema or swelling present in the limb between the wound and the tarsus.

Haematology and blood biochemistry

Haematology and biochemical results are shown in Table 2. The majority of cases (7/10) had a raised total white blood cell count with a profound neutrophilia and a raised fibrinogen. Serum amyloid A was sometimes normal if the sample was taken within 12 h of the onset of clinical signs (*Cases 2*, *4* and *10*) but became raised 24 h later, often reaching an extremely high level. In one horse, there was no haematological abnormality at the time the horse began treatment, but the raised white blood cell count, neutrophilia and raised plasma viscosity, fibrinogen and serum amyloid A occurred within 24 h.

Ultrasonography

In *Case 8*, an ultrasonographic examination of the swelling on the lateral aspect of the tarsus was carried out using a 7.5 mHz linear transducer and scanner (Concept 2000)[5]. This revealed floccular fluid to be present within disorganised fibrosis (Figs 2a,b). The fluid-filled 'locules' tapered towards the site of the retinacular ligaments and appeared to be associated closely with the long digital extensor tendon sheath. Ultrasonography of the remainder of the cases was either declined by the trainers responsible for the horses or was impractical due to the degree of pain experienced by these horses when attempts were made to apply pressure from the ultrasound transducer through the stand-off.

Treatment

A variety of antimicrobial agents were employed in the treatment of these cases, dependent on the preference of the clinicians concerned (Table 1). The majority of cases (*1*, *2*, *3*, *5*, *7*, *9* and *10*) received oxytetracycline i.v. at a dose of 5 mg/kg bwt, given once daily. Nonsteroidal anti-inflammatory drugs, usually phenylbutazone (4.4 mg/kg bwt), were administered at the first examination and this therapy was continued *per os* in all cases for 5 days and, in some, for longer. All of the horses were encouraged to walk repeatedly during the day, preferably on a horse-walker, and spend as little time as possible standing still. Usually, these horses would show severe lameness again after a period of box confinement, but this would quickly reduce to allow continued forced exercise.

In *Cases 4* and *8*, topical application of DMSO[6] or a

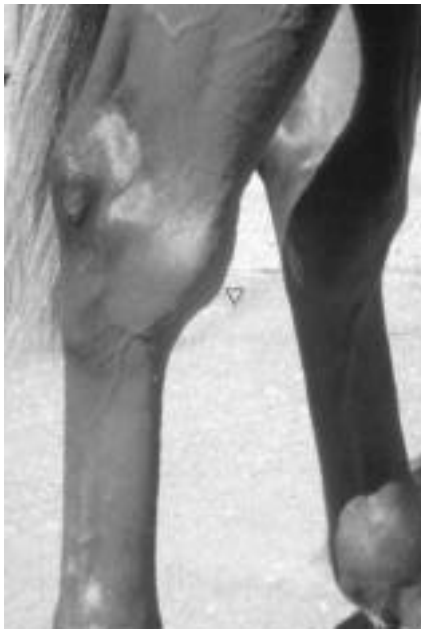

Fig 1a: Case 3*, showing focal swelling on the craniolateral aspect of the tarsus (arrow).*

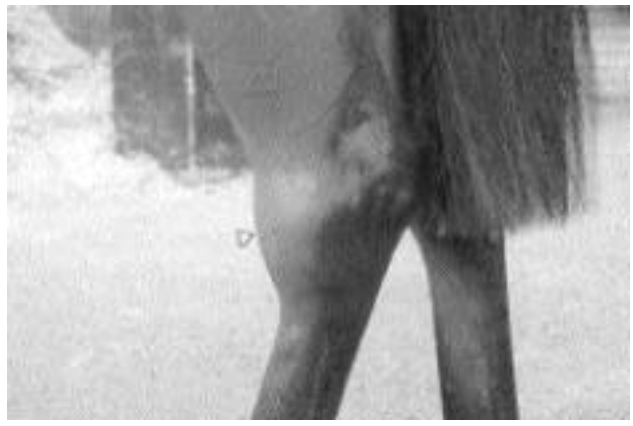

Fig 1b: Case 6*, illustrating how consistent the position of the swollen, painful area is from case to case (arrow).*

salicylic acid gel in *Cases 3*, *5* and *7* (Movelat)[7] was performed. When the pain level in these cases had reduced and the horses would allow manipulation, firm massage of the affected area was carried out in conjunction with application of these gels along with forced passive flexion of the limb. Antibiotic therapy was continued for 5–10 days. In *Case 8*, recurrent lameness resulted in 3 repeated courses of antibiotic therapy without complete resolution of the condition. This horse went on to develop osteomyelitis of the third and fourth metatarsal bones, which was treated by a one month course of oxytetracycline (5 mg/kg bwt i.v. daily) and metronidozole (20 mg/kg bwt b.i.d., *per os*). Although the horse appeared to develop multiple osteomyelitic cavities within the third metatarsus, it eventually made a full radiological and clinical recovery.

Most cases (8/10) were walking sound during exercise within 24 h. In all cases, trotting exercise was commenced on Day 3 of treatment and was repeated on many occasions for short periods (10–15 min) during the day. All cases made a full recovery to clinical soundness. Some cases showed residual swelling and periarticular fibrosis for many months. However, this appeared not to result in limited athletic function.

Discussion

Musculoskeletal sepsis is almost invariably associated with pain (Schneider *et al.* 1992). Sepsis inside the synovial cavity of joints usually results in marked lameness, a rise in rectal temperature, swelling and heat of the affected joint (Rooney 1962). The currently described condition is almost certainly a bacterial infection, based on the results of haematology and biochemistry analysis, in conjunction with the consistent rise in rectal temperature. In almost every case, there was a raised total white cell count with a neutrophilia and left shift. This is considered to be typical of bacterial infection (Archer and Jeffcott 1977). Plasma fibrinogen was raised above normal

TABLE 2: The haematological and biochemical parameters from the 10 cases of peritarsal infection

Case No.	RBC ($x10^{12}$/l)	WBC ($x\ 10^9$/l)	Segmented neutrophils (%)	Lymphocytes (%)	Monocytes (%)	Plasma viscosity (M Pas)	Fibrinogen (g/l)	Serum amyloid A (mg/l)
1	7.40	18.98	87	10	3	1.57	4.5	204.8
2								
(Day 1)	10.60	12.41	65	30	5	1.36	2.2	0
(Day 2)	10.97	18.63	83	14	3	1.57	4.3	368.3
3	10.21	15.00	84	14	2	1.51	3.2	399.6
4								
(Day 1)	10.09	8.47	61	36	2	1.42	1.7	0.6
(Day 2)	9.12	11.66	83	16	1	1.44	2.8	264.4
5								
(Day 1)	9.01	10.41	64	33	3	1.56	4.1	47.8
(Day 3)	8.11	10.31	62	35	3	1.65	6.2	599.9
6	11.66	18.48	84	15	1	1.51	3.1	32.7
7	9.39	14.43	78	19	3	1.45	3.2	168.4
8	9.15	11.12	67	32	1	1.63	4.1	N/A
9								
(Day 1)	9.25	15.66	74	24	2	1.42	2.1	3.8
(Day 2)	8.39	11.53	64	32	4	1.41	2.8	22.9
10								
(Day 1)	9.37	10.49	76	20	4	1.37	2.2	11.2
(Day 2)	8.91	8.51	59	36	4	1.38	2.9	49.9

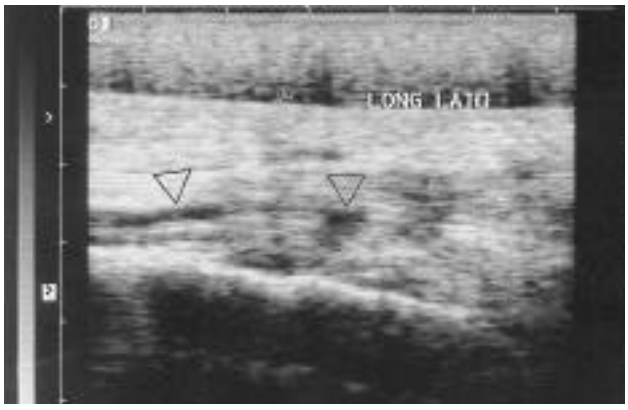

Fig 2a: Longitudinal ultrasound scan of the distal section of the peritarsal swelling. There is abundant fluid present in locules closely associated with the long digital extensor tendon (arrow).

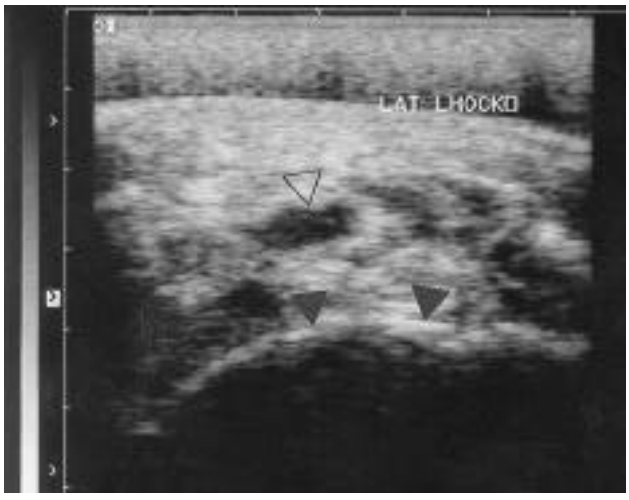

Fig 2b: Transverse scan at the same site. The surface of the third metatarsus is seen as a convex echogenic line (black arrows). Between the skin and this bone surface there is the appearance of disorganised fibrosis and fluid-filled -locules'(arrow).

values in every case although, on Day 1 of *Cases 2*, *4*, *9* and *10*, the level would have been considered normal. Fibrinogen is a well recognised indicator or inflammation and tends to rise relatively slowly and remain elevated for periods of 2 or 3 weeks following acute bacterial infection (Schalm 1987). Serum amyloid A is an acute phase protein which has been shown to rise rapidly and fall rapidly to baseline levels in the acute inflammatory response in the horse (Pepys *et al.* 1989). It is interesting to note that, in *Cases 2*, *4*, *9* and *10*, serum amyloid A was not raised on the first day of clinical signs, but in all 4 cases rapidly became elevated to levels usually associated with response to acute infection. However, the consistent rise in rectal temperature seen in all cases shows that some pyrogenic component of the infectious process must have been in the circulation at that time. In *Case 9*, the horse had been seen to be completely normal and sound in its box at 0630 h, only to be severely lame (4/5) on the affected limb by 1030 h. This shows how rapidly the swelling in the cranial aspect of the hock appears to occur, producing the marked pain response. Presumably, it is this rapidity of onset of swelling and pain which is sometimes in advance of the normal haematological changes seen with acute infection. However, in all cases, these changes were apparent 24 h from first sampling.

In this particular condition, although the infection is peri-articular, it appears to produce a pain response more severe than an intra-articular infection. The reasons for this are unclear. Ultrasonography of the one case, where this was possible, confirmed tightly distended fluid-filled 'locules'subcutaneously. In every case, the swelling was limited to the region of the dorsal aspect of the tarsus itself in the acute stage, and did not ascend or descend the limb, in contrast to lymphangitis, until much later in the course of the disease. It may be that this infection is contained within structures which are themselves constrained by the retinacular ligaments, above and below the tarsus. This might be the reason for the extremely localised nature of the swelling and the extreme degree of pain these cases appear to experience. Manual and digital palpation of the skin over the site results in a very exaggerated pain withdrawal response, and this condition is among the most painful encountered in practice.

The site of swelling is consistent from horse to horse and may reflect a lymphatic nidus of some sort in that location. However, examination of the lymphatic drainage of the hindlimb in the literature has failed to demonstrate such a recognised focus (Stashak 1987). Lymphoscintigraphy would be useful in determining the normal lymphatic drainage to the distal limb, to ascertain whether or not such a focus existed.

The majority of clinical cases had a small wound or an infected area of skin above the heels. It is postulated that the organisms which produce this infection may gain entry to the limb via these relatively innocuous wounds and ascend the limb, before localising in the site immediately medial or lateral to the tarsus. In no case was there any entry wound around the tarsus which could have been responsible for the lameness.

Forced exercise seemed to result in a very rapid attenuation of the degree of lameness and was effective in returning all but one of these cases to clinical soundness. Nonsteroidal anti-inflammatory drugs are helpful in allowing the horse to utilise the limb fully during forced exercise and should be included to promote movement.

An alternative line of treatment of these cases is surgical exploration and drainage of the abscess under general anaesthesia at the earliest signs. This is the treatment of choice in one North American clinic, with excellent results (L. R. Bramlage, personal communication). These clinicians report a lower incidence of periarticular swelling and fibrosis following the infection than in the cases they treated prior to surgical intervention. Certainly, some of the clinical cases described in this series retained a degree of peritarsal swelling for many months, following infection, but this did not appear to inhibit their performance abilities.

Although this condition is relatively frequent in equine practice, it appears not to have reached the literature on lameness (Stashak 1987). It is important that clinicians are made aware of it because of the temptation to attempt synoviocentesis when faced with a nonweightbearing lameness with obvious heat, pain and swelling around the tarsus. There would be a very high risk of iatrogenic introduction of infection into these joints should synoviocentesis be attempted.

While joint sepsis would have to be a differential diagnosis, in every case the tarsocrural joint capsule itself could be palpated as being either normal, or only mildly distended, when compared to the tension, degree of pain and heat detectable on examination of the subcutaneous swelling. Careful clinical examination in these cases is, therefore, probably the most

helpful indicator as to whether joint sepsis is involved or not. Synoviocentesis of the tarsocrural joint could be carried out via the plantar pouch if a degree of effusion is present. In the few cases where this was carried out (*Cases 2* and *4*), the cell count and protein levels of that fluid were only mildly elevated, typical of reactive synovitis, which may be triggered by toxins from the primary seat of infection and local inflammation.

Acute bone injury has to be included as a differential diagnosis in these cases, because the clinical signs of severe lameness, heat and swelling are associated with certain documented fractures of the tarsal region. Fractures of the lateral malleolus can occur during stable rest as a consequence of a fall, and these cases could conceivably be found nonweightbearing, in the absence of exercise (Butler *et al.* 2000). The important differential consideration in these cases is that the tarsocrural joint is often markedly distended following malleolar fracture, because of associated haemorrhage and synovial effusion. In the cases reported in this series, tarsocrural joint effusion was rarely present. Cases of bone fracture also rarely show the degree of heat, pain on palpation and lameness exhibited by all cases in this series.

Secondly, the pain response to palpation in the cases of sepsis described is far more profound than that experienced in digital palpation of the area affected following malleolar fracture. The rise in rectal temperature aids in the differentiation of these cases from cases of traumatic injury. This can be aided by histological and serum biochemistry. However, it should be noted that, in certain cases, the changes typical of acute infections are delayed in onset by 24 h, and a repeat sample may be necessary to confirm the condition.

One case, seen before the commencement of this study, was subjected to euthanasia because of laminitis with complete capsular detachment in the contralateral foot (R. Pilsworth, unpublished data). This horse had not received veterinary attention until 48 h after the onset of severe lameness. With antimicrobial treatment, analgesics and forced exercise it was sound at the walk in 24 h, but lame on the unaffected leg the next day, at which time the laminitis became apparent. It was assumed that the combination of pyrexia, toxaemia and nonweightbearing lameness for 48 h contributed to the demise of this case, emphasising the need for early intervention, analgesia and ambulation in these cases.

Postmortem examination was performed on this case. Grossly, the site of swelling was characterised by oedema, tissue necrosis and discolouration, with a coagulated necrotic centre. A swab taken aseptically from this necrotic centre produced a pure growth of haemolytic, coagulase-positive *Staphyloccus aureus* on culture (R. C. Pilsworth, unpublished data).

In contrast, in *Case 9*, intervention was made not more than 4 h from the onset of clinical signs. The serum amyloid in this horse peaked at 22.9 mg/l at 48 h and subsequently declined to normal. The horse went from 4/5 lame to clinically sound in only 24 h, indicative of the way in which this syndrome can be rapidly halted with early intervention.

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Manufacturers'addresses

[1]Bayer Plc Diagnostics Division, Newbury, Berkshire, UK.
[2]Beckman Coulter Ltd., Luton, Bedfordshire UK.
[3]Instrumentation Laboratories, Warrington, Cheshire, UK.
[4]Roche Diagnostics Ltd., Lewes, Sussex, UK.
[5]Dynamic Imaging Ltd., Livingston, UK.
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