

Original Article

Equine pastern dermatitis

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Summary

Bacteriological examination of pastern dermatitis in 12 cases resulted in a variable growth of bacterial species. *Dermatophilus congolensis* was found on direct smear in 2 cases, although only cultured in one of these. Eleven of the cases were treated with a new topical treatment, consisting of a combination of antibacterial agents and humectants - agents that lower water activity (Mud Stop). Ten of these cases showed a good response to treatment, the other showing partial improvement before the treatment was changed.

Introduction

Equine pastern dermatitis (EPD) is commonly known as mud fever, mud rash and cracked heels and doubtless has several other colloquial names. It is generally assumed to be a syndrome with multiple predisposing factors and causes (Yu 2003; Scott and Miller 2003b). *Dermatophilus congolensis* is often cited as a significant factor, although there are very few references to EPD in the scientific literature.

Dermatophilus congolensis is probably an opportunistic pathogen. Although not found free living in the environment, its zoospores can survive for long periods, possibly in the soil or on the horse's skin (Pilsworth and Knottenbelt 2007).

Yu (2003) and Scott and Miller (2003a,b) suggest there may be a complex syndrome involved in the development of EPD and list a number of factors that may predispose to development of the condition, precipitate its onset and perpetuate continuing problems. Currently, however, these appear to be largely speculative, and Yu (2003) specifically comments that "The lesions of EPD seem to progress in a similar fashion regardless of aetiology".

In general equine practice in the UK, EPD (or mud fever) is a commonly presented problem, generally but not always associated with wet or damp conditions. It would

be common for owners to have tried several forms of treatment themselves before seeking veterinary assistance. These treatments generally involve clipping hair from the area and scrubbing the skin with antibacterial agents such as chlorhexidine gluconate (Hibiscrub)¹. Frequently the areas have become very sore, and thorough examination and assessment of the area can be quite difficult. On presentation, cases show a range of severity of signs, varying from a few small dry scabs in the back of the heel of the foot, through multiple painful exudative lesions involving the pastern and fetlock, to oedematous weeping areas involving much of the distal limb.

The authors were in the position of having a potential new treatment for this condition, based on the assumption that *D. congolensis* was involved as the causative organism. Because of the paucity of information on this condition, we elected to carry out a limited trial on clinical cases, of EPD, including selective bacteriology procedures, but no further detailed examination of the condition or other possible predisposing factors. The trial was practice based, and of necessity investigations other than bacteriology were limited to the bare clinical requirement.

Materials and methods

Clinical material

The owners of 12 horses presented in the normal course of clinical practice, with a diagnosis of mud fever, agreed to join the trial. The severity of signs and previous treatments given by the owners were not considered in the case selection, all cases being treated as recently acquired EPD. The criteria for inclusion were purely symptomatic as given above. I.e. cases showed a range of severity of signs, varying from a few small dry scabs in the back of the heel of the foot, through multiple painful exudative lesions involving the pastern and fetlock, to oedematous weeping areas involving much of the distal limb.

In dry cases a quantity of scab material from the affected area was collected for analysis. In more exudative cases, scabs and/or swabs were collected from

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TABLE 1: Details of the 12 cases admitted to the trial

Case number	Age	Sex	Breed	Use	Comments
31308	9	f	7/8TB	Eventer	Good response
31595	10	f	TB	Brood mare	Good response
31647	Aged	f	Unknown	Hack	Good response
31653	3	g	TB	Racehorse	Good response
31687	11	g	Cob	Hack	Partial improvement
31688	13	f	Cob	Hack	Not treated
31720	11	g	TB	Hack	Good response
31928	10	g	Dutch Warmblood	Show hunter	Good response
31958	12	g	TB	Point-to-point	Good response
31967	13	g	Hunter	Hunting	Good response
32255	19	g	Unknown	Hack	Good response
32551	5	f	Hunter type	Potential eventer	Good response

the area. Details of the animals involved are given in **Table 1**.

Laboratory examinations

Swabs were transported in charcoal Amies medium and in most cases arrived at the practice laboratory on the same day.

Smears were made from wet preparation and emulsification of scab material (where available) and from swabs in the absence of scab. These were examined for the presence of inflammatory cells, *D. congolensis* and other bacteria. Scabs were also screened for the presence of chorioptic mites, and plated onto Sabouraud agar and dermatophyte test medium. No mites or dermatophytes were found.

Swabs were plated onto Columbia sheep blood agar and MacConkey agar for aerobic incubation at 37°C and Columbia sheep blood agar for anaerobic incubation using a mini incubation system that generates an atmosphere of approximately 18% carbon dioxide (Anaerocult P Merck)². For samples that consisted only of scab, a small amount of the sample was homogenised in sterile water and immediately plated using the same method as the swabs. Aerobically incubated plates were examined at 24 and 48 h. Anaerobically incubated plates were examined at 48 h for the presence of *D. congolensis* colonies. The presence of anaerobic bacteria was noted but no further attempt to separate and identify them was made due to cost restraints. The most common aerobic bacterial colony types were sub cultured for further identification using standard methods. Antibiotic susceptibility testing was carried out on staphylococcal isolates using a standard disc diffusion method. An oxacillin disc was included as a screen to detect MRSA strains.

New medication - Mud Stop

The primary action of the Mud Stop³ formulation is to exploit a physiochemical effect commonly known as water activity. To this end it contains 2 humectants

(monopropylene glycol and isopropanol) to reduce water activity.

In addition to the humectants, Mud Stop also contains triclosan, an antibacterial agent.

Treatment regime

Most of the cases involved had already received various topical treatments instituted by the owners, which had proved unrewarding (as discussed above). All treatments were ceased, and replaced with treatment using Mud Stop applied topically twice daily as the only treatment. There was one exception to this regimen: In Case 31687 there was a severe exudative discharge, with a very inflamed and swollen leg: in this case concurrent antibiotic treatment was given orally (trimethoprim sulphadiazine). There was a good initial response to treatment, but after 3 days the topical treatment was changed to silver sulphadiazine and treatment with Mud Stop ceased.

Owners applied the treatment themselves. They were advised initially to soak the affected area with Mud Stop spray. This was to be carried out twice daily. Typically a maximum of 8 squirts of the spray were sufficient for each application. The horse was then made to stand for about 10 min to allow the spray to soak into the affected areas, but they were not touched or bandaged in any way. Over a period of 4–8 days the scabs normally lifted and fell off, at which point, if the lesions were no longer painful, Mud Stop lotion was substituted for the spray.

Although it is recognised that there can be complicating or underlying factors associated with EPD, no attempt was made to exclude such factors before entry to the trial, other than checking for mange mites on appropriate samples.

Results

A wide variety of bacteria was isolated (see **Table 2**). Bacteria isolated included: β -haemolytic streptococci (4 cases), *Staphylococcus aureus* (3 cases) and *Corynebacterium pseudotuberculosis* (2 cases);

TABLE 2: Results of bacteriological examinations

Case No.	Cytology	Culture	Staph. sensitivity and notes	Sample type
31308	Mixed bacteria +++ No neutrophils recorded	Heavy mixed growth including anaerobes.		Swab
31595	Mixed bacteria +++ Neutrophils present	Heavy mixed growth including <i>C. pseudotuberculosis</i> , <i>E. coli</i> and β -haemolytic streptococci		Swab
31647	Hair roots and scale only. No neutrophils seen	<i>S. aureus</i>	Antibiotic sensitive	Swab
31653	Mixed bacteria + Neutrophils with bacterial inclusions	Heavy growth of β -haemolytic streptococci, <i>S. aureus</i> and <i>Corynebacterium jeikeium</i>	Antibiotic sensitive	Swab
31687	Few bacteria present No neutrophils seen	++ CN <i>Staphylococcus</i> with a few colonies of <i>S. aureus</i>	Antibiotic sensitive. Partial response to treatment	Swab
31688	Mixed bacteria +++ Neutrophils with Bacterial inclusions	<i>S. aureus</i> Few β -haemolytic streptococci	Antibiotic sensitive	Swab and scab
31720	Bacteria +++ No neutrophils seen	<i>Pseudomonas aeruginosa</i> with <i>S. aureus</i>	Resistant to neomycin and tetracycline	Swab
31928	Mixed bacteria +++ No neutrophils seen	Heavy mixed growth including <i>Proteus</i> sp., <i>E.coli</i> , α -haemolytic streps, CN <i>Staphylococcus</i> and <i>S. intermedius</i>	Antibiotic sensitive	Swab
31958	<i>D. congolensis</i> POSITIVE No neutrophils seen	Heavy mixed growth of CN <i>Staphylococcus</i> (<i>S. warneri</i>), β -haem <i>Streptococcus</i> and actinomycetes. <i>D. congolensis</i> not isolated.	<i>S. warneri</i> antibiotic sensitive	scab
31967	Mixed bacteria + No neutrophils seen	Mixed environmental bacteria and CN staphylococci.	Sensitivity not done	Swab
32255	Bacteria +++ No neutrophils seen	<i>Corynebacterium pseudotuberculosis</i> with β -haemolytic streptococci.		Swab and scab
32551	<i>D. congolensis</i> POSITIVE No neutrophils seen	<i>S. equorum</i> and <i>D.congolensis</i>	<i>S. equorum</i> antibiotic sensitive	Scab

CN = coagulase negative.

Staphylococcus intermedius, *Pseudomonas aeruginosa*, *Staphylococcus warneri*, *Staphylococcus equorum* and *Corynebacterium jeikeium* were each isolated on a single occasion only.

A mixed growth was cultured in all cases. All the staphylococci subjected to sensitivity testing were found to be completely sensitive to all antibiotics tested with the exception of one strain of *S. aureus*, which was resistant to neomycin and terramycin.

In Case 31688 treatment with Mud Stop was never carried out, the owner opting for more traditional treatment. Although culture results have been included in this paper for this horse, no clinical assessment has been made.

Case 31687 commenced treatment within the trial, and was showing a good response, when the owner opted to use an alternative topical treatment. This case has therefore been assessed as partial improvement, although the authors see no reason that a satisfactory outcome would not have resulted had treatment been continued.

Clinical assessment of results

Generally results were reported by the owners of the horses, although in a few cases the veterinary surgeon who initiated treatment did observe the response. This is a

common condition not generally associated with spontaneous recovery, without marked weather changes. The symptoms were initially reported by the owners, and they were able to report satisfactorily when the condition had resolved.

Clinically, all the cases that followed the treatment regime showed a good response, becoming clear of all signs. In the one case (31687) where concurrent systemic antibiotic treatment was given, and Mud Stop was ceased early, we have recorded this as a partial response. Although arguably the case could have been excluded, we preferred to record the result as a partial response, in acknowledgement that it may not be a successful treatment in all cases.

Discussion

Bacteriological considerations

Although *Dermatophilus congolensis* is often quoted as the cause of mud fever, the 2 major references to EPD (Scott and Miller 2003b; Yu 2003) accept that it is frequently not isolated from clinical cases. In this small series, *D. congolensis* was found on only 2 occasions. We suggest therefore that the linking of this organism to mud fever is equivocal, and should be reassessed.

The familiar presentation of dermatophilosis in horses is as 'rain scald' where there is a definite association with wet weather. *D. congolensis* is a member of the actinomycete family. It can exist as free motile zoospores which grow into germ tubes and eventually the characteristic branching parallel lines of zoospores, the so-called 'tram-track' appearance as seen in smears. As with many actinomycetes, it grows best in a microaerophilic atmosphere (5–10% CO₂) although it can be grown in both aerobic and anaerobic atmospheres. The small haemolytic colonies are visible after 48 h incubation. They are a yellow-grey colour and firmly adherent to the media (Quinn *et al.* 1994).

One of the biggest problems in isolating *D. congolensis* is contamination with other bacteria. The Haalstras method, where a portion of scab material is ground up with distilled water and left in a candle jar at room temperature for 15 min can be used to aid isolation. Motile spores are attracted to the surface and a loopful can then be cultured on blood agar in a microaerophilic atmosphere (Quinn *et al.* 1994). We have found that anaerobic culture also works well, presumably due to restricting the growth of some competitive bacteria. Experiments with sheep strains have shown that *D. congolensis* is inhibited *in vitro* when grown in the presence of many normal skin bacteria, notably *Bacillus* sp. (Kingali *et al.* 1990). Difficulties in recognition and culture might be one reason that *D. congolensis* has not been reported in more cases of pastern dermatitis.

Staphylococcus aureus is a normal inhabitant of the nasal mucous membranes in many species and has been cultured from the horse (Oeding *et al.* 2009). In this study *S. aureus* was found on 4 occasions (36%). Although *S. aureus* can produce many different toxins, dermatitis is generally considered to be secondary to a localised disturbance in the skin barrier. Various trigger factors have been described, such as photosensitivity and infection with chorioptic mange mites. In many cases, it may be that moisture due to weather conditions or sweat and friction may be sufficient to damage the skin barrier, leading to infection (Outerbridge and Ihrke 2003). Horses with staphylococcal pastern dermatitis can become very ill, developing a secondary lymphangitis. In this study, no MRSA strains were detected and all except one strain showed sensitivity to the complete antibiotic panel. This would suggest that antibiotic resistance is uncommon in pastern dermatitis although a much larger study would be needed to confirm this.

Although *D. congolensis* was only isolated in 2 cases, conclusions as to its involvement in EPD cannot be made on the evidence of this paper. It seems likely that some factor will cause a break in the skin barrier, which allows opportunist bacteria in the environment to infect the tissues. It may be that *D. congolensis* is the initiating factor in some EPD infections, breaching the skin, but subsequently being replaced by other bacteria. Alternatively it may simply be one of the opportunist

organisms that cause infections when other factors break the skin barrier. Further, more detailed investigations are needed to assess these 2 alternatives.

Considerations regarding treatment

The effects of modified water activity on reduction of bacterial and fungal growth have been well documented (McMeekin 2000) but have seldom been used in topical disinfection strategies. A contemporary example of this effect is the microbiological stability of honey, which is due to reduction of water activity by the carbohydrate content.

Chambers Dictionary of Science and Technology (Anon 2007) defines water activity as: An expression of the amount of water present in a food, raw material or product that is available to support microbial growth. As it is reduced, the rate of growth of microorganisms declines. The key principles are based on the reduction of water activity by removing water or by adding solutes such as sugar or salt. Water activity is commonly represented by the symbol a_w .

The simplest expression for water activity is $a_w = p/p_o$, where p = the partial vapour pressure of water in material being measured and p_o is vapour pressure of pure water at the same temperature. Thus, the a_w of pure water is 1, as $p = p_o$. Water activity is related to relative humidity by multiplying by 100: $\%RH = p/p_o \times 100 = a_w \times 100$.

Water content and water activity are not directly related. Materials with a low water content (e.g. sand with 5% water) may still have an activity close to pure water in which microorganisms may still thrive, as the water that is present is biologically available. Conversely honey may have a water content of up to 20% but a water activity below 0.7, as the water is not readily available for biological growth.

Of the bacteria identified in this study only *Staphylococcus* spp. show any growth when the water activity is below 0.9 but even then growth is much reduced, ceasing at a_w 0.86 (Lotter and Leistner 1978). Yeasts and fungi will often grow down to 0.80 (Leistner 2002) but this also depends on the nature of the humectants present. A few highly specialised microorganisms and moulds grow below a_w 0.8 (Leistner 2002) and then usually very slowly and are probably of no consequence in mud fever.

The contents of Mud Stop are designed to give a synergistic formulation reducing bacterial growth. This should effectively tip the balance in favour of the natural repair mechanisms. A further benefit is that the humectants have relatively high penetrability, meaning it is usually quite unnecessary to remove the scabs and encrustation seen with this condition. Typically the scabs will fall off unaided within 10 days of starting treatment, often rather quicker. The underlying tissue can initially seem a little sore but this rapidly resolves.

Mud Stop was administered as a spray at the start of treatment when the horses were sore, administering

approximately 10 ml of liquid spray. As no direct contact is needed this aids administration, as well as minimising any risk of cross infection. Once cases were no longer sore, the Mud Stop was applied as approximately 5 g of cream, which has a longer lasting contact time.

Whatever bacteria were isolated in these cases, the new medication appears to have been very effective in the treatment of pastern dermatitis. This could perhaps be anticipated as its primary action is to lift moisture away from the area of infection, thus stopping bacterial growth. Its ease of use, even in very sore and difficult patients, is also an important recommendation.

Conclusion

Whilst this survey is too small to draw any conclusions about the pathogenesis of equine pastern dermatitis, it does perhaps cast doubt upon the assumption that it is usually related to *Dermatophilus congolensis*.

The high percentage of response to treatment in this small trial is very encouraging, especially as several of the cases had already failed to respond to other treatments. This uncontrolled trial however can only be regarded as a preliminary investigation, and does not allow any statistically relevant conclusions to be drawn.

Manufacturers' addresses

¹Regent Medical Ltd, Irlam, Manchester, UK.

²Merck KGaA, Darmstadt, Germany.

³Equitech, Cookham, Berkshire, UK.

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