Porcine ear necrosis syndrome: A preliminary investigation of putative infectious agents in piglets and mycotoxins in feed

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A R T I C L E   I N F O

Article history:
Accepted 29 May 2012
Available online xxxx

Keywords:
Porcine ear necrosis
Mycoplasma
Histopathology
Bacteriology
Virology

A B S T R A C T

The aim of this study was to identify the causative factors of porcine ear necrosis syndrome (PENS) in 72 pigs, 5.5–10 weeks in age housed on nine farms. Biopsy samples of ear pinnae were collected from all piglets for bacteriology, histopathology and in situ hybridization for porcine circovirus type 2 (PCV2). At the same time, serum samples were taken for serological analysis and viral PCR, and feed was sampled for mycotoxin analysis.

The initial lesion of PENS seemed to be a focal epidermal necrosis. Streptococci were isolated from 44 and staphylococci from 36 pinnae. PCV2 could not be detected by in situ hybridization or qPCR. Seven piglets were positive for porcine reproductive and respiratory syndrome virus, and one for Mycoplasma suis. One piglet had antibodies against Sarcoptes scabiei var. suis. No infectious agents were found in 15 samples. Positive virology and parasitology were often found alongside positive bacteriology. Deoxynivalenol, zearalenone and ergot alkaloids were detected in feed. The findings suggest that PENS is multifactorial in origin and that although infectious agents can be involved in the development of the syndrome they are not the exclusive triggering factor.

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Introduction

Porcine ear necrosis syndrome (PENS) is characterized by large erosive lesions at the margin or the tip of the pinna (Richardson et al., 1984). The syndrome is usually seen in weaning piglets during the summer months (Penny and Hill, 1974) and presents with two distinct stages: a mild form characterized by the appearance of easily ruptured vesicles followed by a more severe form with deeper lesions caused by bacteria (Richardson et al., 1984). Many causative agents for the development of PENS have been suggested and although potential triggering factors can be divided into infectious (Richardson et al., 1984; Thibault et al., 1998; Molnar et al., 2002; Thacker, 2006) and non-infectious agents (Osweiler, 2006; Schroeder-Petersen and Simonsen, 2001) no definitive aetiology has been identified.

The aim of the present study was to evaluate the role of infectious agents in the initial stage of PENS, using histopathology and bacteriology. In addition, the role of mycotoxins in the syndrome was investigated by analysing the feed that the affected pigs were being fed for mycotoxins.

Materials and methods

Animals and husbandry

Seventy-two Large White × Landrace × Pietrain crossbred piglets aged 5.5–10 weeks from nine farms were included in the study. The farms were selected on the basis that all had had ear necrosis consistently in previous years. On these farms pig density, temperature, and air velocity as well as carbon dioxide and ammonia load were all within industry recommendations. Only one farm did not use enrichment strategies. Piglets were housed on perforated slatted plastic floor with 0.3 m² floor space per pig. The compartments were cleaned in all farms between pigs, but only two of the farms additionally carried out disinfection. All farms

Please cite this article in press as: Weissenbacher-Lang, C., et al. Porcine ear necrosis syndrome: A preliminary investigation of putative infectious agents in piglets and mycotoxins in feed. The Veterinary Journal (2012), http://dx.doi.org/10.1016/j.tvjl.2012.05.026
had under pressure ventilation systems. Feed was provided by Biomin (Biomin F4 pro aktiv, Biomin Prof 570 Gold), WfMa Mirakel Spezialfutter (Mirakel 1, Mirakel Topzym), Sano – Moderne Tierernährung (Sano Suggi) and Schaumann (Schaumalac F80/M, Schaumalac Protect FR0). The composition of feed corresponded to standard diets for this age class.

All piglets had been vaccinated against Mycoplasma hyopneumoniae in the first week of life and at weaning. On one farm the sows had been vaccinated against porcine circovirus 2 (PCV2) 2 weeks before farrowing; on all other farms the piglets had been vaccinated against PCV2 at weaning. Two of the farms had previously diagnosed porcine reproductive and respiratory syndrome (PRRS), porcine respiratory disease complex and infections with Streptococcus spp. or Staphylococcus hyicus. These diseases had not been recorded on the other seven farms.

Blood and tissue samples were collected contemporaneously on two occasions, 1 year apart, from two different batches of 36 piglets (Table 1). Piglets were selected for the study on the basis of showing early clinical signs of PENS, i.e. oedematous plaques of 5–10 mm size with thin, removable crusts (Figs. 1 and 2), irrespective of age. Samples were not taken from animals with more severe disease. Five millimetre diameter tissue samples, containing all tissue layers of the ear, were collected with a mechanical punching tool. For bacteriological analysis a swab was taken from the biopsy site after tissue collection. Feed was collected three times: once during the summer, when the symptoms occurred for the first time; once in the winter after storage, and once again the following summer.

Biopsy samples were fixed in formalin, embedded in paraffin and sectioned. These sections were stained with haematoxylin and eosin (H&E). Samples where thrombosed blood vessels were suspected were stained using Weigert’s elastic stain. All samples were subjected to in situ hybridization (ISH) for PCV2 nucleic acid (Bukovsky et al., 2007). In order to evaluate the association between mycotoxin concentrations and morphological features the microscopic lesions were classified as: (1) Focal epidermal necrosis; (2) bacterial growth in the superficial cell debris; (3) infiltration with granulocytes; (4) infiltration with histiocytes; (5) lysis of collagen; (6) vasculitis; (7) generation of granulation tissue, and (8) hyperkeratosis. Lesions 1 and 2 were the initial stages of ear necrosis, 3–6 were progressive disease, while 7 and 8 were healing lesions.

Swabs were applied to blood agar plates immediately after collection; these were incubated overnight at 37 °C. Staphylococci and streptococci were identified macroscopically based on colony form and size and Gram’s stain. Bacterial quantities on solid media were classified based on evaluation of the three serial loop smears as no growth (score −, negative), doubtful (score (+), <10 colony forming units (CFUs) in first loop smear), sparse growth (score +, <5 CFUs in second loop smear), moderate growth (score ++, <5 CFUs in third loop smear) and pronounced growth (score ++++, ≥5 CFUs in third loop smear). The presence of PRRS virus, PCV2 and Mycoplasma suis in serum were investigated by using previously described PCR procedures (Balka et al., 2008; Hoelzle et al., 2007; Lang et al., 2011). Detection of specific serum antibodies against Sarcopotes scabiei var. suis was undertaken using a commercial Sarcopotes ELISA (AFOSA) according to the manufacturer’s instructions.

All feed samples were analysed by high performance liquid chromatography (HPLC) for the mycotoxins deoxynivalenol, zearalenone, T2 toxin, HT2 toxin, diacetoxyscirpenol, acetyldeoxynivalenol and nivalenol as well as the ergot alkaloids ergosine, ergotamine, ergocornine, ergocryptine and ergocristine (Griesler et al., 2010; Schiessl et al., 2010).

All statistical analyses were undertaken using PASW 17 (SPSS). The association between histopathology and infectious agents was analysed using Pearson’s χ² test, while that between histopathology and mycotoxin results was analysed using the Student’s t-test.

**Results**

The prevalence of ear necrosis on the farms ranged from 10%–100% of piglets. The incidence and distribution pattern of affected

<table>
<thead>
<tr>
<th>Sample type</th>
<th>Number of samples at different time points</th>
<th>Method</th>
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<tbody>
<tr>
<td></td>
<td>First summer</td>
<td>Winter</td>
</tr>
<tr>
<td>Ear tissue</td>
<td>36 piglets</td>
<td>36 piglets</td>
</tr>
<tr>
<td>Swab of lesion</td>
<td>36 piglets</td>
<td></td>
</tr>
<tr>
<td>Serum</td>
<td>36 piglets</td>
<td></td>
</tr>
<tr>
<td>Feed</td>
<td>9 samples</td>
<td>9 samples</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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Fig. 1. Early stage of PENS. Macroscopic visible alterations of the ear.

Fig. 2. More advanced stage of PENS. Macroscopic visible alterations of the ear.

piglets showed a high degree of variation and could not be associated with any other observations or the obtained results. Microscopically, a focal epidermal necrosis was found to be the initial lesion. In 67% of the cases at this stage of disease, bacteria that morphologically resembled cocci were present in the necrotic epithelium (Fig. 3). These primary necrotic foci tended to expand to larger areas of epidermal necrosis, which were consistently accompanied by a moderate to severe infiltration with mixed, partly...
degenerate inflammatory cells. In addition, there was superficial cell debris with considerable bacterial growth. Particles of plant material, most likely originating from faeces or litter, adhered to the lesions.

The necrotic areas were demarcated from the dermis by neutrophilic granulocytes (Fig. 4). Granulation tissue was generated subepidermally as well as in deeper areas of the dermis. In three cases, re-epithelialization of the lesions was observed presenting as growth of regenerating epithelium beneath the necrotic tissue (Fig. 5). Fully re-epithelialized areas were characterized by severely hyperplastic epithelium and hyperkeratosis. Occasionally, new necroses occurred in these re-epithelialized areas. In cases of acute lesions, a marked emigration of neutrophilic granulocytes from numerous medium-sized to small blood vessels could be seen. In many cases inflammatory cells were also present in the vascular wall, periadnexally and diffusely distributed in the dermis. In more advanced lesions, granulation tissue and a perivascular and periadnexal pyogranulomatous inflammation was seen in the deep dermis. In 32% of the cases large blood vessels showed subintimal proliferations suggestive of endarteritis or endophlebitis obliterans, sometimes with thrombus formation (Fig. 6). In the majority of these cases, lesions indicative of arteriosclerosis of small and medium-sized blood vessels, predominantly characterized by hyperplasia of the intima and hyaline degeneration of the blood vessel wall, were present.

The results of testing for infectious agents are summarised in Table 2. Streptococci were found in 44 and staphylococci in 36 of the swabs. PCV2 could not be detected by ISH or by qPCR. Seven piglets were PRRSV positive, but only one piglet showed a positive result for Mycoplasma suis. One piglet had antibodies against Sarcopotes scabiei var. suis. At the farm level, on one farm neither streptococci nor staphylococci were isolated from lesions, on another, only staphylococci were isolated and on the remaining two only streptococci were isolated. During the first sample collection, 5/9 farms were PRRSV negative. On the second occasion 7/9 farms were PRRSV negative. Four of the five farms which were negative at the first sample collection were negative 1 year later. The piglet with the positive Sarcopotes scabiei var. suis diagnosis came from a PRRSV positive farm, while the Mycoplasma suis positive one originated from a PRRSV negative farm. The different combinations of diagnostic findings are presented in Table 3.

T2 toxin, HT2 toxin, diacetoxyscirpenol, acetyldeoxynivalenol and nivalenol were not detected in any feed sample. The results for the other toxins are summarised in Table 4. Eight samples

![Fig. 3. Early stage of PENS. Multiple colonies of cocci are present within the necrotic epithelium. In addition, particles of plant matter adhere to the surface. Haematoxylin and eosin staining, bar = 40 μm.](image3)

![Fig. 4. More advanced stage of PENS. The necrotic tissue is demarcated by neutrophilic granulocytes. Abundant plant material adheres to the surface. Haematoxylin and eosin staining, bar = 150 μm.](image4)

![Fig. 5. Advanced stage of PENS. Re-epithelialization occurs by hyperplastic epithelium (hyp) moving under the necrotic tissue (necr). Haematoxylin and eosin staining, bar = 150 μm.](image5)

![Fig. 6. Several cases of PENS showed prominent lesions of dermal blood vessels. An artery shows marked subintimal proliferations leading to partial occlusion of the lumen. Haematoxylin and eosin staining, bar = 80 μm.](image6)
(29.6%) exceeded a deoxynivalenol concentration of 0.200 mg/kg, the minimum emetic dose of this mycotoxin (Forsyth et al., 1977), and 9 (33.3%) had a zearalenone concentration of >0.050 mg/kg, the critical dose for the establishment of symptoms in prepubertal pigs (Bauer et al., 1987).

In Table 5 the different ergot alkaloid combinations and patterns are presented, and Table 6 shows the correlation between mycotoxin and alkaloid concentrations and the progressive development of microscopic lesions. Deoxynivalenol was found only in the initial phase of PENS. Ergotamine was negatively correlated with collagen lysis, but positively associated with vasculitis in the initial phase of PENS. Ergocryptine and ergocristine were negatively correlated with histiocyte infiltration during the acute phase. Ergocytoine concentrations were not associated with any effect during the healing phase.

The microscopic alterations were divided with regard to their occurrence in superficial and deeper tissue layers. There were also significant correlations between increased ergosine (P = 0.017) and ergotamine (P = 0.033) concentrations and the microscopic alterations in the superficial tissue layers. There was no significant difference between the three different sample collections in the summer time, when the symptoms occurred for the first time, in the winter after storage and once again in the following summer. The deoxynivalenol concentrations of the three consecutive sample collections were 362, 219 and 173 mg/kg, and the zearalenone values were 42, 36 and 43 mg/kg.

Table 5

<table>
<thead>
<tr>
<th>Combination</th>
<th>No.</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>All samples (n = 72)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>First collection (n = 36)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Second collection (n = 36)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No infected agent</td>
<td>15</td>
<td>20.8</td>
</tr>
<tr>
<td>STREPтокocci/staphylococci</td>
<td>2</td>
<td>2.8</td>
</tr>
<tr>
<td>Streptococci/staphylococci/Sarcoptes antibodies</td>
<td>50</td>
<td>69.4</td>
</tr>
<tr>
<td>STREPтокocci/staphylococci/PRRSV</td>
<td>1</td>
<td>1.4</td>
</tr>
<tr>
<td>PRRSV</td>
<td></td>
<td></td>
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</tbody>
</table>

**Discussion**

In the present study, alterations in piglet ear tissue in the initial stage of PENS were analysed histopathologically in an attempt to define the time course of the disease. As this was a preliminary study, only piglets with ear necrosis were investigated to obtain as much information as possible about this syndrome. Focal epidermal necrosis was seen as the initial lesion. Such necrosis can either be the consequence of a vascular alteration resulting in local ischaemia as leading to epidermal necrosis and subsequent ulceration or of an outside event such as trauma or bacterial toxins.

Based on the data, a definitive answer about the causative factor cannot be given. The selection of piglets for sample collection was made when the first macroscopic lesions were visible, and the very early stage of vesiculation as documented by Richardson et al. (1984) could not be observed in the present field study although the histological pattern of more severe cases was comparable. Nevertheless, the lack of cases at the early stage of disease in the present study is due to the timing of sample collection and does not suggest different aetiological causes.

The most frequent ear lesions found by Richardson et al. (1984) were hyperkeratosis, acanthosis and intraepidermal abscesses as well as a hyperplastic perivascular dermatitis (Mirt, 1999). Depending on the duration of the lesions, the necrotic dermis was replaced by granulation tissue, as in the present cases. Vascular changes could also be observed in both studies. The occlusion of the lumen of affected vessels by thrombi has already been described by Richardson et al. (1984).
In the present study, staphylococci and streptococci could be seen in various disease stages. Staphylococci were found in higher concentrations, as well as in a higher percentage of cases. Richardson et al. (1984) isolated Staphylococcus hyicus from both early and advanced lesions, whereas they only isolated streptococci from ulcerated lesions. The colonization of the lesions by bacteria is viewed as important in the breakdown of the epidermis, the extension of lesions into deeper tissues and the development of vascular thrombosis, ischaemia and necrosis (Richardson et al., 1984). Staphylococci are adapted for residence on normal pig skin and can be considered an aetiologic agent of early skin lesions (L'Ecu- yer, 1967), whereas the invasion of streptococci must be preceded by damage of the skin due to infection or trauma (Wannamaker, 1970). As seen in the present study, both kinds of bacteria can appear at different stages of disease independent of their pathogene- sis, thus underlining a multifactorial aetiology.

Particles of plant material originating from faeces or litter stuck to the lesions emphasized the assumption of local trauma as a starting point, with secondary colonization of those wounds by bacteria underlying the development of severe necrotizing lesions. A significant association between ear scratches and an increased risk of mild ear necrosis was also shown by Busch et al. (2008). Mirt (1999) isolated staphylococci twice as frequently from lesions as from healthy skin and assumed that infection with staphylococci began at sites of slight trauma, but definitely ruled out an exclusively traumatic origin. Maddox et al. (1973) mainly detected streptococci in samples from necrotic ear margins, which pre- sented as thromboarteritis. The lesions healed after treatment with ampicillin.

Since trials with antibiotic treatment of ear necrosis against staphylococci (Hansen and Busch, 2008) resulted in no significant clinical effect, it seems unlikely that bacteria alone can cause this clinical pattern, which further supports other infectious or non- infectious agents being involved in this apparently multifactorial disease. Comparable microscopic lesions were also observed in association with a cutaneous necrotizing vasculitis in weaning pigs even when the lesions did not remain limited to the margins of the ears (Thibault et al., 1998). Aside from the distribution of necrotic lesions, the only difference from the present study was the pres- ence of eosinophils, which could have resulted from an immune- mediated inflammation. The initial causative agent of this cutane- ous necrotizing vasculitis could not be determined, but the authors suggested that these lesions probably developed spontaneously when a specific combination of predisposing events, including PRRSV infection, occurred. In the present study PRRSV was de- tected in some cases, but there were no histopathological differ- ences compared with other samples.

The association between PRRSV and the microscopic findings was not significant, but a contribution to the development of the lesions cannot be completely ruled out, particularly in the cases with bacterial and PRRSV co-infections. The detection of PCV2 in various tissues of pigs with symptoms of postweaning multisys- temic wasting syndrome or porcine dermatitis and nephropathy syndrome is generally possible (Molnar et al., 2002; Zlotowski et al., 2008), but PCV2 has not been proven to be a causative agent for necrotizing vasculitis in the context of PENS. Pejsak et al. (2011) showed reductions in both prevalence and severity of ear necrosis in weaners after sow vaccination against PCV2, even though factors such as the herd immune status or co-infections were not consid- ered in this study.

All of our piglets were vaccinated either actively or passively against PCV2 and the virus could not be detected by ISH or PCR. Nevertheless, a high prevalence of ear necrosis was recorded on some farms. So, in contrast to the findings of Papatsiros (2011), a direct association between PCV2 and the development of PENS could not be demonstrated. Because vaccination of piglets and sows against PCV2 is widely used, the relevance of this pathogen in relation to the development of ear necrosis has to be questioned. A similar conclusion could also be made for Mycoplasma suis and Sarcoptes scabiei var. suis.

Mycotoxins do not necessarily and exclusively cause ear necro- sis, but because they have dermonecrotic potential (Osweiler, 2006), their concentration was determined in feed. T2 toxin, HT2 toxin, diacetoxyscirpenol, acetyldeoxynivalenol and nivalenol were of no importance. Only deoxynivalenol and zearalenone could be detected in biologically significant concentrations (Forreth et al., 1977; Bauer et al., 1987). Combinations of different mycotoxins may potentiate the action of each other or at least

| Table 6 Correlation of mycotoxin and ergot alkaloid concentrations with the different morphological features of microscopic lesions. |
|---------------------|-----------------|----------------|----------------|----------------|----------------|----------------|----------------|----------------|
| Deoxynivalenol | p | 0.298 | 0.409 | 0.216 | 0.235 | 0.194 | 0.131 | 0.176 | 0.203 |
| | P | 0.014 | <0.001 | 0.073 | 0.047 | 0.105 | 0.324 | 0.140 | 0.090 |
| Zearalenone | p | 0.140 | 0.098 | 0.052 | −0.100 | −0.035 | 0.232 | 0.047 | 0.032 |
| | P | 0.255 | 0.414 | 0.672 | 0.403 | 0.770 | 0.077 | 0.696 | 0.794 |
| Ergosine | p <0.030 | −0.190 | −0.130 | −0.106 | −0.169 | 0.083 | 0.117 | 0.058 |
| | P | 0.807 | 0.109 | 0.285 | 0.375 | 0.158 | 0.530 | 0.326 | 0.634 |
| Ergotamine | p =0.079 | −0.227 | −0.148 | −0.193 | −0.248 | 0.721 | 0.016 | 0.035 |
| | P | 0.521 | 0.056 | 0.221 | 0.104 | 0.037 | 0.038 | 0.894 | 0.774 |
| Ergocryptine | p | 0.008 | 0.162 | −0.025 | −0.259 | 0.013 | 0.381 | −0.263 | −0.316 |
| | P | 0.948 | 0.173 | 0.839 | 0.28 | 0.914 | 0.003 | 0.026 | 0.007 |
| Ergocristine | p | 0.010 | −0.042 | −0.064 | −0.326 | −0.172 | 0.124 | −0.075 | −0.140 |
| | P | 0.936 | 0.727 | 0.598 | 0.005 | 0.150 | 0.350 | 0.530 | 0.245 |

ρ, Spearman’s coefficient of variation; P, level of significance 0.05 (bold = significant P-values). As only one sample was positive for ergocornine, the calculation of correlation was not possible.

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exert an additive effect (Huff et al., 1988). The co-existence of zearalenone with other mycotoxins such as nivalenol or deoxynivalenol has already been described by some authors (IARC, 1993) due to some compounds being produced by the same Fusarium species. On the other hand, different biological and climate conditions are necessary for the production of the common mycotoxins and these rarely appear in the same grain at a specific time point. Therefore, concurrent production of mycotoxins could be relatively uncommon, and there is presently little evidence that common mycotoxins act synergistically (Osweiler, 2006).

In the present study, higher deoxynivalenol concentrations were correlated with microscopic alterations in the initial stage of the disease. A synergistic effect with infectious agents or an impact as a precursor has not been documented, but cannot be ruled out. Ergot alkaloids cause gangrenous ergotism as a result of combined vasoconstriction and endothelial damage leading to prolonged ischaemia of appendages and eventually dry gangrene (Osweiler, 2006). The contractile response of veins depends not only on the concentration of alkaloids, but also on their types due to their chemical diversity as well as on interactions between different alkaloid types (Klotz et al., 2008). As described by other European authors (Uhlig et al., 2007), ergocristine dominated the alkaloid spectrum of most extracts in the present study, followed by ergotamine, ergosine and ergocryptine. Ergocornine was of secondary importance. Ear and tail necrosis have rarely been described in the context of alkaloid concentrations and have been observed after intake of a combination of ergotamine, ergocristine, and ergonovine (10 mg/kg grain) (Lopez et al., 1997). For this reason, their relevance as well as the required dose for the development of ear necrosis still need to be investigated.

Conclusions

Our study clearly showed that multiple infectious agents could be involved in the development of PENS, but also that none of the agents we investigated were the exclusive triggering factor. This does not mean that there is not one factor – indeed other infectious agents that we did not investigate here could be of primary importance in the aetiology of PENS. Exclusion of more bacterial, viral and parasitic diseases as well as non-infectious factors is necessary. Further investigation should include challenge trials with different infectious agents, with a focus on the resulting histopathology. In addition, the potential synergistic effect of ergot alkaloids needs to be considered as do other non-infectious agents, such as endotoxin. Finally, a comparison between diseased and healthy piglets is required.

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriate influence or bias the content of the paper.

Acknowledgments

This study was supported by the Dres. Jutta und Georg-Bruns-Stiftung. The bacteriological analysis of the feed samples was carried out at the Futtermittel labour Rosennau (Chamber of Agriculture Lower Austria, Petzenkirchen, Austria), the mycotoxin analysis at ROMER Labs Diagnostic GmbH (Tulln, Austria).

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