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Porcine dermatitis and nephropathy syndrome: A new condition to include in the differential diagnosis list for skin discoloration in swine

C. Oliver Duran, DVM, PhD, MRCVS; José A. Ramos-Vara, DVM, PhD; James A. Render, DVM, PhD

Summary

A previously unreported condition in the United States, porcine dermatitis and nephropathy syndrome (PDNS), has been described in growing pigs in Michigan. PDNS has low morbidity—less than 1% of the pigs affected—but a high mortality rate. Clinically, most affected pigs develop multifocal erythematous lesions on the distal extremities and ventrum. Porcine dermatitis and nephropathy syndrome should be considered as a cause of skin discoloration in growing pigs from 5–100 kg (10–220 lb), to be distinguished from lesions related to septicemia, cyanosis of the extremities, erysipelas, or generalized erythema. The causes and pathogenesis of PDNS remain unclear.

Keywords: swine, diagnosis, dermatitis, nephropathy, skin

Recently, we have observed a condition new in the United States, porcine dermatitis and nephropathy syndrome (PDNS), diagnosed in pigs from two herds in Michigan. Both herds have different genetics and management, but were faced with outbreaks of respiratory disease coinciding with the diagnosis of PDNS.

Herd A is a 200-sow, farrow-to-finish operation that used total confinement and all-in–all-out (AIAO) by room. This herd was serologically positive to porcine reproductive and respiratory virus (PRRSV), but had no clinical manifestations of infection. Mortality in finishing pigs was due to pleuropneumonia (*Actinobacillus pleuropneumoniae*).

Herd B was a 1400-sow, farrow-to-feeder pig operation, with a separate onsite nursery. Pig flow was AIAO by room, and sows and piglets were housed in confinement on slatted floors. Herd B had been affected by an acute outbreak of PRRS, and had continued postweaning respiratory disease problems in the nursery caused by viral and bacterial agents (*Pasteurella multocida*, *Haemophilus parasuis*, *Streptococcus suis*).

The prevalence of PDNS in both herds was below 0.5%. Seven acute cases and two recovering cases were examined at the Animal Health Diagnostic Laboratory, Michigan State University.

COD: Department of Large Animal Clinical Sciences, A201 Veterinary Medical Center, Michigan State University, East Lansing, Michigan 48824–1314; e-mail: duranoli@msu.cvm.edu; JAR-V, JAR: Michigan State University, Animal Health Diagnostic Laboratory

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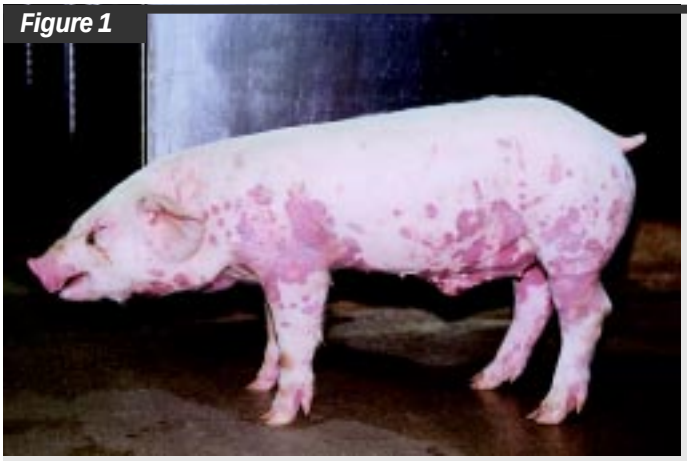
Clinical signs of PDNS

Pigs affected with PDNS are usually detected toward the end of the nursery phase or at the grower stage, but PDNS has also been described in finishing pigs and replacement gilts.[1,2] The first signs reported to us were skin lesions that were multifocal, well-circumscribed, slightly raised, dark red, circular to irregular, and 1 mm–2 cm in diameter. These lesions occurred over the ham, ventral abdomen, and groin (Figures 1 and 2). At this stage, the mildly affected pigs were afebrile and alert, and would continue to eat. In some pigs, the lesions coalesced and spread to other areas of the skin, such as the flank, ears, and thorax (Figure 3), and in some cases, skin lesions resolved spontaneously. Shortly after the appearance of the skin lesions, pigs that were pyretic, with rectal temperatures ≤ 41°C (106°F), displayed clinical signs of anorexia, severe weight loss, and depression and these pigs rapidly died. Although the producers administered antibiotics, it did not appear to prevent deaths or accelerate the resolution of the skin lesions in the Michigan herds, an observation consistent with the literature.[2,3] In the cases seen in Michigan, there was no swelling of the lower limbs or evidence of subcutaneous edema, but these have been described as typical findings in reports elsewhere.[1,2] In some "chronic" cases of PDNS, the lesions scabbed over during the resolution phase. Evidence of renal failure (elevated concentrations of blood urea nitrogen [BUN] and creatinine concentrations in blood) was observed in one case in Michigan.

Pathological and microbiological findings

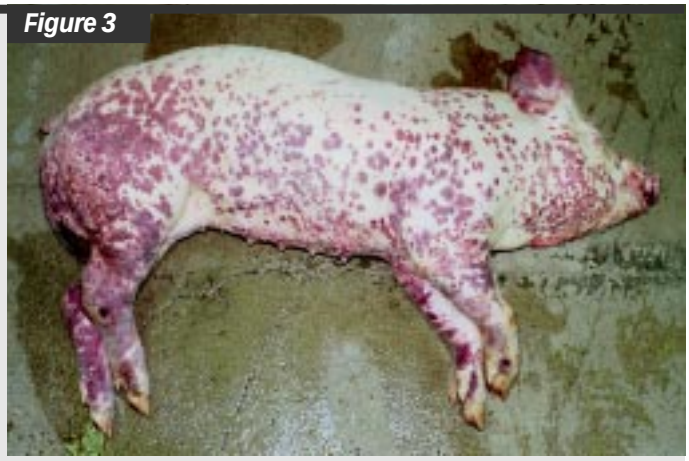
Gross findings in the seven cases of acute PDNS we examined were confined primarily to the skin, but upon careful examination of the surface of the kidney we observed red circular areas, 1 mm in diameter, in the renal pelvis. Other lesions were detected in the lungs (three pigs had suppurative bronchopneumonia, two had pleuritis, and two had pulmonary edema). Gastric ulceration was observed in two pigs and enlargement of superficial and renal lymph nodes in four pigs. Microscopically, the principal lesion in PDNS was the presence of a severe, fibrinoid, necrotizing vasculitis affecting the dermis, subcutis (Figures 4 and 5), and the renal pelvis and medulla (Figure 6). Curiously, eosinophils were not uncommon as part of the vascular inflammatory infiltrate, especially in the kidneys. Another lesion con-

Figure 1



Numerous multifocal to coalescing erythematous lesions are seen on the skin, mainly in the front and rear limbs and in the ventral abdomen.

Figure 3

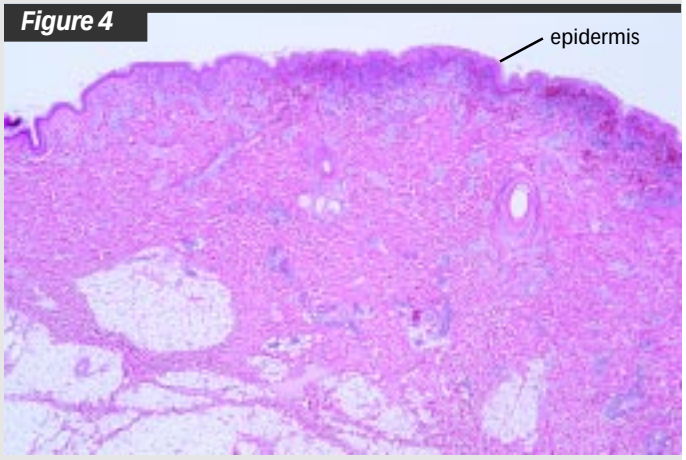


Cutaneous lesions in this pig were more widespread and also involved the flanks and pinnae. The most affected areas were found in the rear legs, where the lesions coalesced.

Figure 2

Detail of the cutaneous lesions (infarcts). Note that these lesions are raised, usually well-circumscribed, erythematous, and sometimes contain a red center surrounded by a clear halo (target lesions).

Figure 4



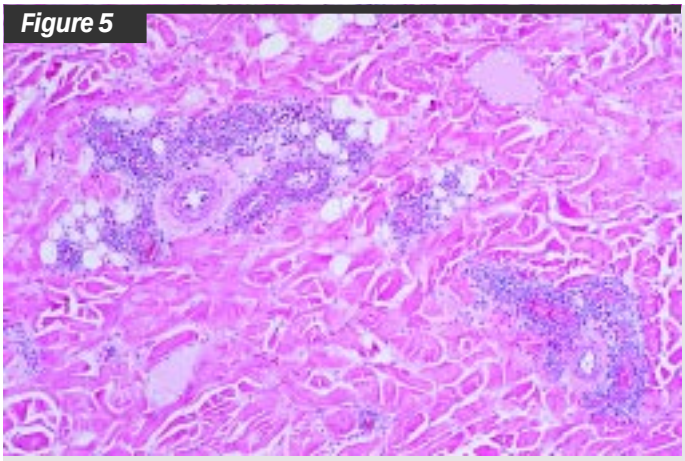
Low magnification of the microscopic appearance of the cutaneous infarcts. Note that two-thirds of the epidermis (right side) is necrotic and is associated with an underlying dermal perivascular leukocytic infiltrate and vascular collapse (necrotizing vasculitis). Affected vessels are also found in the deep dermis and subcutis. (Hematoxylin-eosin; 5×)

sistently found was an acute, diffuse, global exudative necrotizing glomerulonephritis (Figure 7).

Vasculitis of gastric and lymph node vessels was observed in two pigs. Cutaneous lesions in the chronic stage included exudative dermatitis and vasculitis with secondary bacterial infection. Chronic renal lesions were membranous glomerulonephritis and interstitial nephritis with fibrosis.

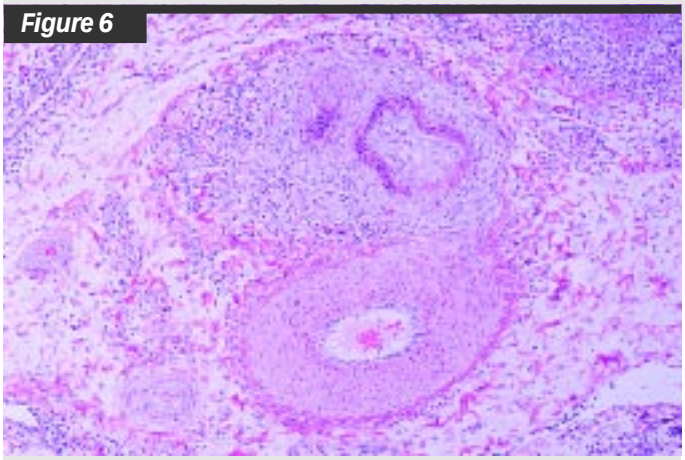
We did not find a common etiologic agent in this series of cases of PDNS. *Haemophilus parasuis* was isolated from the joint in two cases presenting polyserositis. Bacterial culture of lung tissue yielded *Actinomyces pyogenes*, *P. multocida*, *H. parasuis*, *Fusobacterium necrophorum*, and *Bordetella bronchiseptica*. *Erysipelothrix rhusiopathiae*, *Salmonella* spp., and *Leptospira* spp. were not detected from the skin or viscera. Tissues examined by fluorescent-antibody test, immunohistochemistry, and virus isolation were negative for PRRSV, although the serum of one live pig had a positive titer for PRRSV. All tissues examined were also negative for swine influenza virus, pseudorabies virus, and encephalomyocarditis virus.

Figure 5



Higher magnification of the necrotizing vasculitis in dermal vessels, mainly venules. Adjacent arterioles are not affected. (Hematoxylin-eosin; 25×)

Figure 6

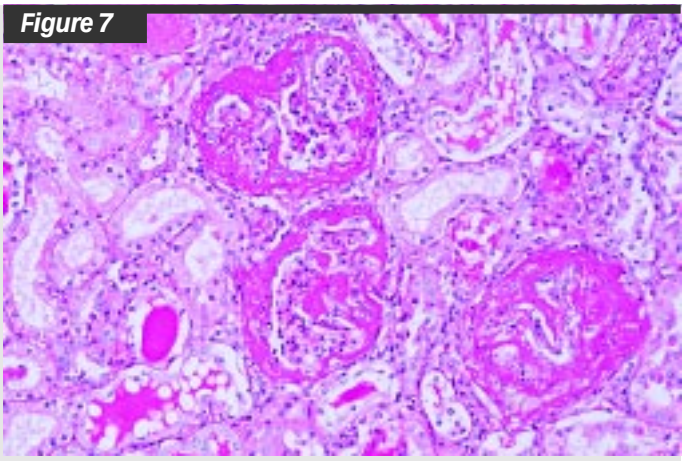


Renal pelvis. Two interlobular arteries (probably segments of the same vessel) are observed. One (bottom) has a severe necrotizing vasculitis characterized by a mixed inflammatory infiltrate, hyalinosis of the vessel wall, and obliteration of the vessel lumen. Compare this affected vessel with the adjacent normal one. (Hematoxylin-eosin; 10×)

Discussion

The pathogenesis and etiology of PDNS remains unexplained at present. The histological presentation suggests an immune-mediated insult to the vascular endothelium by immune complex deposit, or by direct damage to the vascular endothelium by an unknown agent. Nakajima, et al.,[4] suggested that damage to the vascular wall could be due to lipopolysaccharide of Gram-negative bacteria. Recently, a necrotizing vasculitis in the kidney has been described after experimental challenge with a strain of PRRSV in pigs[5] and the presence of PRRSV antigen has been detected in the myocardial endothelial cells.[6] Based on these facts and evidence of infection in affected herds, Segalés, et al., have postulated that PDNS is closely linked with PRRSV.[3] In human vasculitides, similar pathological damage may be caused by different infectious agents.[7]

Figure 7



All three glomeruli observed in this figure are enlarged due to the presence of abundant proteinaceous material and leukocytic exudate in the Bowman's space. There is mesangiolysis, mild interstitial inflammatory infiltrates, and protein casts within tubules. (PAS stain; 50×)

Differential diagnosis

There are several other diseases and conditions that can cause erythema (red discoloration of the skin) or skin discoloration secondary to clinical signs observed in other systems. The list for differential diagnosis includes:

- Erysipelothrix *rhusiopathiae*: Swine erysipelas presents in the acute form with the characteristic "diamond skin" lesions and can be differentiated from PDNS by different gross lesions as well as by the response to treatment with penicillin. It is typically confirmed by bacterial culture.
- Bacterial infections (*Actinobacillus suis*, *A. pleuropneumoniae*, and *S. suis*) and septicemia cause generalized reddish discoloration of the skin, but changes are usually transitory, diffuse, and not as multifocal as skin changes described in PDNS.
- Infection with *H. parasuis*, *A. suis*, swine erysipelas, and salmonellosis can cause cyanosis and congestion affecting extremities, with petechial hemorrhages and congestion of various organs. These diseases usually have a higher morbidity than that seen in PDNS and can be confirmed by bacterial culture and by the presence of bacterial emboli in superficial blood vessels.
- Transit erythema, related to urine-soaked floors or chemical burns.
- Porcine stress syndrome, where a generalized blotchy bluish or red discoloration of the skin is very apparent shortly after handling or other stressors.

Porcine dermatitis and nephropathy syndrome is a disease with a low prevalence in swine herds, and therefore has a low impact on the profitability of swine farms. This paper illustrates to swine practitio-

ners and diagnosticians the clinical and pathological presentation of this condition, previously unreported in the United States. Treatment with antimicrobial agents such as tetracycline and penicillin in this and other reported cases[1,3] has been unsuccessful in reducing mortality and accelerating the resolution of skin lesions; thus, antibiotic therapy in cases of PDNS is probably useless.

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