SKIN AND OTHER MISCELLANEOUS DISEASES

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General Discussion

Most skin abnormalities encountered in the United States have little economic significance. Even so, certain systemic diseases have skin manifestations that are very important clinical signs and must be carefully evaluated. Many Foreign Animal Diseases (FAD’s) have typical skin lesions requiring astute investigation if observed. Any time a vesicular syndrome is discovered immediate action should follow to rule out a FAD. In this case aggressive action to prevent spread and to arrive at a diagnosis is essential.

There are also acquired injury related lesions that may be associated with modern housing and management practices. Significant presences of these lesions are indicators of husbandry and welfare issues. Pigs also have a few congenital skin issues that are frequently encountered but in a very small percentage of growing pigs.

The North American pig industry is the healthiest in the world. Perhaps that is why we take interest in these lesser known anomalies. However, it is important that we recognize some of these as a part of the case evaluation and diagnostic differential process that all veterinarians go through in problem solving and converting observations into knowledge.

These conditions are listed in my order of significance based on economics and clinical frequency.

Greasy Pig Disease – Exudative Epidermitis (EE)
Introduction
• Descriptions of this disease go back more than 150 years.
• The causative (etiologic) agent is Staphylococcus hyicus which was first identified in 1965. It was separated from other non-pathogenic staphylococci and identified in 1978 as the etiologic agent in Greasy Pig Disease.
• The etiology of this disease is not completely understood and is difficult to reproduce by researchers but like many neonatal diseases of pigs it is likely associated with the dynamics of colostral immunity, infectious dose, timing of exposure, and the development of a functional adaptive immune response.
• It is an acute exudative dermatitis that starts acutely but usually develops over a course of days to a few weeks.
• It is most often observed in modern start up herds. Cases usually occur continuously but at a very low incidence once the herd matures (normal parity distribution).

Etiology
• Staphylococcus hyicus is a Gram-positive cocci which appears to be a ubiquitous part of the bacterial flora on the skin and other tissues of adult swine.
It can be easily recovered from the farrowing house environment and from the skin of normal neonatal piglets.

There are 6 or more serotypes but not all are pathogenic and colonization with these strains may provide some protection against toxin producing strains.

Epidemiology

- The epidemiology is confusing and likely complex.
- The bacterium has a global distribution.
- Many herds that are positive never experience the disease while others suffer serious epidemics.
- It was this disease that led to the customary clipping of needle teeth in neonatal piglets. This was long believed to be a necessary husbandry procedure.
- Many environmental and management factors are associated with this disease but most are based on speculation and common sense. Most interventions appear to work with “tincture of time”. Likewise most interventions are slow to solve the issue.

Pathogenesis/Lesions

- The disease can only be reproduced when virulent strains are applied to aggressively scarified skin or by subcutaneous injection in CDCD pigs. This is not reproducible in pigs that have apparent immunity.
- Initial skin lesions typically develop on the nostril in skin lacerations caused by fighting.
- Lesions quickly spread with micro-colonies developing on the epithelial surface and stratum corneum. This spreads to hair follicles leading to a suppurative folliculitis. Inflammation follows with erosion and ulceration.
- The sebaceous glands respond by secreting copious quantities forming a black greasy exudate.
- These lesions quickly enlarge and coalesce engulfing the skin surface of the pig.
- Death occurs due to starvation, dehydration, and loss of serum proteins and electrolytes.
- Virulent strains produce an exfoliative toxin (ET) which can locally reproduce the disease when injected subcutaneously into study pigs.

Clinical Signs

- The first lesions appear while pigs are still suckling. These appear around the nostril and face of the pig. Typically giving the appearance that the pigs found a bucket of tar to play in. This is the end point in most pigs but others develop fulminating disease to which they eventually succumb. This usually occurs immediately after weaning.
- Pigs can become generalized within 48 hours after the initial lesions are noted.
- Pigs become greasy and have a bad odor that can be easily detected in outbreaks even before approaching the pigs.
- Sows housed in hot humid climates frequently develop exudative epidermitis on their necks just behind the ear in the summer months. Problems in pigs are not associated with this condition in adults.
- Morbidity and mortality can be significant. I have observed morbidities greater than 25% and case mortalities greater than 50%. Outbreaks in startup herds may experience up
to a 10% mortality rate for several weeks before control measures and herd immunity intervene.

**Diagnosis**
- Clinical signs and history are definitive.
- Histopathology and culture of *S. hyicus* from the lesions can confirm the diagnosis.

**Control**
- Control measures are usually implemented in a crisis situation rather than as a preventative plan.
- When stocking a new farm a history of other same source startups should be obtained from the genetic company.
- If there is a history of greasy pig then needle teeth should be clipped at birth for the first 6-12 months of operation.
- Sanitation and disinfection may be of some value. Only hot water/detergent cleaning with targeted disinfectant use will alter the incidence.
- Avoid mixing pigs in the nursery. Litter integrity is difficult in modern systems but if it can be done and will significantly reduce spread in the nursery.
- Autogenous vaccines may or may not be of value. It is difficult to evaluate this in the field. They always are added after the outbreak starts and since this is a self limiting condition over time as the breeding herd matures, any strategy will look effective. These vaccines are routinely given to the sow just before farrowing in an attempt to improve colostral immunity. I have used them in select situations but no longer than 6 months. There is little doubt that immunity is important in control and a well made autogenous is likely of some value.
- Early treatment with parenteral antibiotics will save many pigs. *B* lactams are most frequently used but resistant strains of *S. hyicus* are common.
- There are many “favorite” treatments used by individuals in the industry. All seem to be equally effective or non-effective depending on observer perception. Dipping in disinfectants, tamed iodine, rubbing with vegetable oil with or without added antimicrobials, and many others are popular.
- Hospital pens, aggressive search and treatment of early cases, electrolytes, added warmth, and a high quality protein diet may be most effective. Generalized cases require prolonged therapy and care.

**Sarcoptic Mange**

**Introduction**
- Mange in pigs is caused by *Sarcoptes scabiei* var. *suis* and is the most common ectoparasite of swine around the world.
- There are two syndromes, chronic and acute. Both are immune mediated but differ significantly.
- The disease is characterized by pruritis and papules in growing pigs and hyperkeratosis in adults.
History
- This parasitic disease was first reported over 60 years ago although no economic importance was attributed to it.
- As pigs moved indoors in an attempt to improve productivity and welfare the economic importance of the disease became more apparent.
- Over the past 15 years the introduction of effective acaricides (Ivermectin) has significantly reduced the incidence in the field.
- All modern breeding stock companies in the US and Canada are mange free.
- Most of the industry expansion done from 1980 to 1995 was with mange free genetics. Multi-site all-in, all-out production eliminates the Sarcoptes mite.
- Many hobby and small continuous flow operations are still positive as well as many Pot Belly Pigs.

Etiology
- The Sarcoptes scabiei var. suis mite causes all significant mange in pigs. There is a demodectic mange mite but it is not important in North America.
- All stages of the Sarcoptic mite develop in the epidermis. Ova and adults will contaminate bedding and other locations in the pigs’ environment but only survive in the best of conditions for 15 days. This is typically less than 24 hours in cleaned facilities.

Epidemiology
- Asymptomatic carriers are the main route of introduction into naïve herds.
- Commercial pig trucks/trailers are also guilty of tracking mange to many farms in the past.
- Functional Biosecurity is an effective deterrent.
- In the past this has been a purchased agent coming with the new genetics.
- Spread within a herd is from direct body contact.
- Piglets usually pick up mites while suckling from their mother.
- Farm staff may move mites on clothing under ideal conditions. If they move from site to site without biosecurity precautions they may facilitate area spread. This was common in earlier times.

Pathogenesis/Clinical Signs/Lesions
- The Sarcoptes mite burrow through the epidermis laying eggs as they go. This sets off a profound allergic response. The pruritis leading to constant rubbing of penning, gaits, flooring and each other.
- This chronic skin damage and allergic response leads to hyperkeratosis over time.
- When pigs are first exposed to the mite the ear canal is usually an area of first colonization. This process sets off an allergic hypersensitivity reaction and papules develop over the rump and along the underline but no mites will be present in the lesions.
- Outdoor pigs frequently rub most during the heat of the day which is assumed to be due to mite activation and activity.
• Heavy and lifelong infestations lead to decreased growth rates, lowered feed efficiency. Reproductive performance may also be affected in severely and chronically infested herds.
• Morbidity will be near 100% in non-treated continuous flow herds but mortality low if not zero. Mortality in these herds are typically multi-factorial with mange a minor contributor. Only a few adults will develop severe hyperkeratosis but all will have some lesions.

Diagnosis
• History and clinical signs should place mange at the top of the differential list.
• Scraping the external auditory canal with a spoon or melon scoop should reveal larvae, nymphs, and adults.
• Postmortem examination of adults is a good way to make a diagnosis. Skin for histopathology from the ear epidermis is ideal.
• Mites are very difficult to find in growing pigs.
• There is an ELISA test typically done on serum or meat juice collected at slaughter that when used on a herd/flock basis may help differentiate between mange and insect bites. This is a costly problem at the abattoir leading to delays in the process and skinning of carcasses.
• Mange is usually most active in growing pigs during the winter months and insect bites will occur mostly in the Spring and Fall.

Control
• Eradication is has been relatively successful in modern confinement operations.
• Avermectins (Ivomec) have been successful in controlling and eradication from breeding herds and growing pigs.
• Pour-on and spray miticides (acaricides) provide good control but eradication has been more difficult, have more withdrawal issues, and more are more toxic to the pig and the environment compared to the Avermectins.
• Depopulation with the standard 30 days down is highly effectively but costly.

Erysipelas and A. suis – Diamond Skin Disease

Discussion
• Both of these septicemic diseases have been discussed in other sections but the skin lesions are significant signs of serious infection and should be recognizable by the practicing veterinarian.
• *Erysipelothrix rhusiopathiae* or *Actinobacillus suis* are the bacterial agents that can cause “Diamond Skin Disease”
• These lesions typically appear early after infection often by the second day and often when the animals go off feed and appear lethargic.
• In severe cases the skin will become necrotic and slough.
• Aggressive therapy with penicillin or other β-lactams is usually effective.
Many times these lesions occur in gilts in late gestation. Erysipelas vaccination does not always prevent this.
Diagnosis may be accomplished by blood culture but is rarely done since both agents are typically sensitive to antimicrobials.

**Sunburn**

**Discussion**
- In older production systems females were frequently bred in confinement, housed for 30 to 60 days and then moved to out door lots. White skinned females especially gilts would occasionally abort within a few days after the move.
- Photosensitization due to prolonged tetracycline exposure may have been the real culprit in these cases. In these systems feed antimicrobials were under control of the farmer who often took advice from the ingredient sales person. I have diagnosed this on two occasions early in my career.

**Dermatophytosis – Ring worm**

**Discussion**
- This is common in sows housed outside but rarely seen today.
- *Trichophyton mentagrophytes* is the agent most often involved.
- Infections are usually prolonged but recovery is spontaneous.
- There are no practical treatment or interventions available or needed.

**Pityriasis Rosea - Pseudoringworm**

**Discussion**
- This condition is common but only a very few pigs are affected in any group.
- The cause is not known but congenital and perhaps heritable. The Landrace has been incriminated but the relationship is not clear.
- The syndrome is not contagious and there is no therapy of value nor is it needed.
- The lesions appear to be very similar to true ringworm with raised erythematous borders and apparent healing in the centers of the rings. They gradually spread and coalesce.
- Cases resolve spontaneously.

**Swine Pox**

**Discussion**
- This was first reported in 1842 and is still occasionally observed in small operations, purchased commingled pigs, and in poor sanitary conditions.
- Swine Pox Virus is in the family *Poxiviridae* along with other mammalian pox viruses.
- This virus is host specific and only infects pigs.
- It is spread by the pig louse *Haematopinus suis* by mechanical means but may also be spread by fomites and bedding. Oat straw has been incriminated because it inflicts many superficial skin nicks when fresh.
• Morbidity is usually very high in affected groups. Mortality is low but may occasionally occur with other concurrent diseases.
• The lesion is typical of most pox viruses and easily identified.
• This disease was very common in the days of feeder pig markets where pigs were grouped by size and condition with no regard to farm source.
• Typical lesions progress through the classic stages of macule (reddening), papule (reddening with edema), vesicle (fluid exuding from the pox lesion), and pustule or crust formation.

Melanomas

Discussion
• These are not uncommon in Duroc or Hampshire breeds. The Duroc is most susceptible and this is often encountered in boar production multipliers which utilize this or other colored breeds.
• They are usually benign but may become malignant.
• They may be present at birth but are usually first observed in late nursery or finishing ages.
• Pig melanomas will frequently undergo spontaneous resolution.

Epitheliogenesis Imperfecta

Discussion
• This is a condition where piglets are born with patches of missing skin.
• It is a developmental defect caused by a single autosomal recessive allele.
• These lesions are raw and have distinct skin edges that often appear to “roll under” at the margin.
• They will eventually heal with scaring.
• Pigs with large areas of missing skin should be euthanized for humane reasons. This lesions is not uncommon but not considered economically important.