An outbreak of urticarial form of swine erysipelas in a medium-scale piggery in Kiambu District, Kenya

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ABSTRACT

This report concerns an outbreak that occurred during July/August 1997. Ten pigs from a herd of 181 pigs in a medium-scale, semi-closed piggery in Kiambu District, Kenya, contracted the clinical disease. The main clinical findings in affected pigs included: fever (40.5–41.8 °C), prostration, inappetence, dog-sitting posture, abortion, erythema and raised, firm to the touch and easily palpated light pink to dark purple diamond-shaped to square/rectangular spots on the skin around the belly and the back. Based on the pathognomonic skin lesions, a clinical diagnosis of swine erysipelas was made. The diagnosis was confirmed by the isolation of *Erysipelothrix rhusiopathiae* organisms from the blood and skin biopsies taken from the affected pigs. Response to treatment with a combination of procaine penicillin and dihydrostreptomycin at the dosage rate of 20 000 IU/kg body weight (based on procaine penicillin) for 3 days was good and all the affected pigs recovered fully. The farm was placed under quarantine to prevent spread of the disease.

Key words: erysipelas, Kenya, pigs, urticaria.

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INTRODUCTION

Swine erysipelas was first recognised as an important disease of pigs in North America in 1928[10]. It has since been reported to occur in most countries of the world and is of economic importance owing to mortality in pigs, costs incurred in the control and eradication of the disease, reduced growth in pigs, abortion and the devaluation of the carcasses at slaughter (especially those with arthritis – the chronic form)[5,10].

The disease is caused by the bacterium *Erysipelothrix rhusiopathiae*[1,5]. Currently, at least 22 serotypes of the bacterium are known to occur[3,4,8]. The virulence and the serotype antigens across the serotypes vary considerably and only a few of them are associated with the clinical disease[3–5,7]. The organism can persist in the soil for long periods as it is resistant to most environmental influences such as dry weather conditions, and is not readily destroyed by chemical disinfection. It can persist in frozen or chilled meat and decaying carcasses, and has been reported to withstand salting, pickling and smoking[10].

The most important natural reservoir of *E. rhusiopathiae* is probably the domestic pig[10]. Other animals from which the organism has been isolated and that may act as natural reservoirs include horses, cattle, sheep, dogs, reindeer, kangaroo, wild boars, turkeys, pigeons, mice and cats[4,10]. Other natural reservoirs such as wildlife may also be present since very little is known about the epidemiology of swine erysipelas in Kenya. Man is also susceptible to *E. rhusiopathiae*, and erysipeloid, as the disease is known in man, is characterised by a painful swelling at the point of inoculation in the skin.

Under natural conditions, skin abrasions and alimentary tract mucosa are considered to be the probable ports of entry, while transmission occurs through ingestion of contaminated feed or water[10]. Feed, water and soil are contaminated through faeces or urine from sick or carrier pigs or other animals[4,10].

Clinical diagnosis of swine erysipelas is facilitated when the pathognomonic skin lesions are present. However, acute and subacute septicaemia without the characteristic skin lesions may be mistaken for other diseases such as African swine fever, hog cholera and salmonellosis, which also manifest as septicaemia. Laboratory diagnosis that involves bacteriological and virological isolations, and serological tests assist in differentiating these diseases.

In Kenya, swine erysipelas is a notifiable disease (Animal Disease Act, CAP 364) and it is mandatory for all veterinary surgeons having reason to suspect the existence of the disease on any farm or in any area to report to the veterinary department. This case report describes an outbreak of swine erysipelas in a medium-scale piggery in Kenya and is the 1st documented outbreak of swine erysipelas in Kenya.

MATERIALS AND METHODS

The disease outbreak occurred between the end of July and beginning of August 1997, in a medium-scale, semi-closed piggery in Kiambu District, approximately 20 km west of Nairobi in Kenya. There were 181 pigs on the farm during the outbreak: 17 sows, 1 boar, 12 gilts, 85 piglets, 10 weaners and 56 growing/finishing pigs. The farm manager reported an unusual disease that was spreading rapidly in the piggery, with skin lesions as the major presenting clinical sign. Within a period of 1 week, 10 pigs had acquired this unusual disease, including 4 sows, 5 gilts and 1 growing/finishing pig.

The history of each sick pig was taken, followed by a thorough general and physical examination, and the findings were recorded separately. Blood was collected from each of the sick pigs via the ear vein while skin biopsy samples were taken from the pigs that had well-developed skin lesions. The blood and skin biopsy samples were transported to the laboratory in thioglycolate semi-solid medium. Bacterial isolation procedures were carried out as described by Buchanan and Gibbons[1]. Samples were placed on blood-agar plates that were later transferred to a gas jar filled with 5 % oxygen, 10 % CO_2 and 85 % nitrogen (microaerophilic conditions) and incubated at 37 °C for 48 h. Primary inocula

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were made by spreading the samples over the surface of the blood-agar using a sterile wire loop to produce discrete colonies that were incubated for a further 48 h as before. Smears were then prepared from the colonies and stained with Gram's stain. Carbohydrate tests were carried out on the culture in 1 % tryptone water, 1 % carbohydrate, 5 % bovine serum in deionised water, and the fermentable compounds used were glucose, lactose, sucrose, mannitol, raffinose, inulin and fructose. Owing to technical problems, histopathology was not performed.

All the sick pigs were treated with a procaine penicillin/dihydrostreptomycin combination at the dosage rate of 20 000 IU/kg body weight (based on procaine penicillin) administered intramuscularly for 3 days. Appropriate control measures were instituted immediately and included placing the farm under quarantine and the disinfection of all the pens where the sick pigs were found using Kerol, 38 % v/v tar acids (Agrevo East Africa Limited, Kenya).

RESULTS

The main clinical findings in the sick pigs during the outbreak were: fever (40.5–41.8 °C), prostration, inappetence, dog-sitting posture, abortion, and light pink to dark purple areas on the belly and the back that were raised, firm and easily palpated. Details of age, history and body temperature are shown on Table 1. The skin lesions were about 1–7 cm in diameter, some having the classic diamond-shaped appearance. The number of skin lesions varied greatly among the affected pigs and the most numerous lesions were found on the suckling sows with some spreading and forming continuous deep purple areas over the greater part of the skin surface. In some pigs, skin disquamation occurred in the affected areas.

A clinical diagnosis of swine erysipelas was initially made on the basis of the pathognomonic skin lesions. The diagnosis was confirmed by isolation and identification of *E. rhusiopathiae* organisms from the blood and skin biopsy samples from the sick pigs. The resulting colonies were round, tiny, translucent, and greyish and 0.5–1.0 mm in diameter. Slender Gram-positive rods appearing singly, in pairs or chains and either straight, curved or hooked were observed microscopically. The organism produced acid but no gas from glucose, lactose and fructose. The *in vitro* bacterial culture and drug-sensitivity pattern showed that the organisms were highly sensitive to penicillin.

Table 1: **Characteristics of the 10 pigs that acquired clinical swine erysipelas during an outbreak on a medium-scale pig farm in July/August 1997 in Kiambu District, Kenya.**

Age class	Last farrowing	Litter size	Parity	Skin lesions[1]	Body temperature[2]
Sow	18 July 1997	8	3	2	41.5
Sow	4 June 1997	10	3	1	41.7
Sow[3]	23 Feb 1997	10	1	2	41.6
Sow	29 March 1997	11	2	2	41.8
Gilt	—	—	—	3	41.2
Gilt	—	—	—	3	41.7
Gilt	—	—	—	3	40.5
Gilt	—	—	—	3	41.0
Gilt	—	—	—	3	40.5
Grower	—	—	—	3	41.0

[1]Distribution of skin lesions: 1 = very extensive (belly and back); 2 = extensive; 3 = less extensive.
[2]Mean body temperature (°C) during the height of clinical manifestation and before treatment commenced.
[3]The affected sow aborted on 3 August 1997 during the course of the disease outbreak.

DISCUSSION

In Kenya, no documented record of an outbreak of swine erysipelas is available. The disease has been reported in other parts of the world such as the United States of America, Europe, Asia, Australia and South Africa[2–5,9,10].

In the outbreak reported in this paper, only subadult and young adult pigs appeared to manifest the clinical disease. These included young sows, gilts and growing/finishing pigs. The 'middle-aged' pigs appeared to be the high-risk age group as has been reported elsewhere[5,10]. This age group seems more vulnerable since the maternally-acquired immunity has waned by this age and active immunity from the environmental exposure has not yet developed substantially[4,10].

No piglet was reported to have acquired clinical disease on the farm and this was probably attributed to maternally-acquired passive immunity from their already immune mothers, as has been observed in the past[4]. This is an important finding, since local farmers who may contemplate using swine erysipelas vaccines may be advised to delay the vaccination till the piglets are weaned. Radostitis *et al.*[4] have suggested that the ideal age at which vaccination should be carried out is 10–12 weeks of age, since this achieves an effective active immunity.

The apparent resistance to clinical disease in older pigs may have been due to the development of active immunity following prolonged or repeated exposure to a less virulent strain of the infectious agents[4]. However, swine erysipelas has been reported to be severe in lactating sows[4], and this finding was apparent in the outbreak reported in this paper. Since all age groups did not acquire clinical disease, it appears that the *E. rhusiopathiae* strain associated with this outbreak was not very virulent, as very virulent strains have been associated with clinical disease across all age groups, including piglets a few weeks old[4]. Another possible reason for apparent resistance to clinical disease may be the type of piggeries in Kenya. In Kenya the piggeries are small, secluded and about 68 % have fewer than 20 sows[6]. The less dense population may result in a build-up of immunity from the low level of challenge, and this type of scenario may be a great asset to Kenyan farmers who raise pigs.

Penicillin was very effective in the treatment of the disease, confirming earlier observations that penicillin is the most effective drug in the treatment of swine erysipelas[10]. Given the nature of the outbreak on this farm, we believe that the pigs were previously exposed to a less virulent strain of *E. rhusiopathiae* that produced no clinical disease but boosted their immune status. It is difficult to explain the circumstances that led to the current outbreak of swine erysipelas on this farm, as no apparent change in environmental factors such as weather or managerial factors such as change of feeds or presence of other diseases were observed on the farm.

This paper reports and describes the 1st documented outbreak of swine erysipelas in Kenya. It further sheds light on the potential for outbreaks of the disease in Kenya and other countries with small isolated piggeries, especially in the developing world, where few records of the disease exist.

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