

AN OUTBREAK OF *TRYPANOSOMA EVANSI* INFECTION IN PIGS

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SUMMARY

An outbreak of pig trypanosomiasis due to *Trypanosoma evansi* characterised by fever, anorexia, emaciation, abortion and death among breeding stock is described. Prior to the investigation 11 sows and one boar were dead and more than 20 sows aborted. *Trypanosoma evansi* was detected in six pigs at the time of investigation. The affected pigs were successfully treated with suramin at the rate of 2.5 g (10%) per sow. The clinical, economic and epidemiological importance of the outbreak are discussed.

Keywords: Pig, *Trypanosoma evansi*, Malaysia.

Pigs like any other domestic animal have been infected by several species of trypanosomes around the world (Soltys and Woo, 1977; Onah, 1991). Many of the species of trypanosomes found in pigs, namely, *Trypanosoma congolense*, *T. vivax*, *T. brucei brucei* are non pathogenic and of little economic importance (Stewart, 1947; Ilemobade and Balogun 1981; Onah, 1991). However, *T. semiae* infection causes a serious and fatal disease in pigs (Stewart, 1947; Unsworth, 1952; Losos, 1986) while another species, *T. suis* causes acute and chronic infections in domestic pigs, and wild pigs are the reservoir host in Africa (Hoare, 1972). Although *T. brucei brucei* is considered non-pathogenic to domestic pigs there have been several reports of natural fatal outbreaks of pig trypanosomiasis in Africa (Agu Bajeh, 1986; Onah and Uzoukwu, 1991).

Like any other trypanosome, *T. evansi* infects a wide range of domestic animals all over the world including South east Asia (Mahmoud and Gray, 1980; Dieleman, 1986) but in pigs it is generally considered less pathogenic (Woo, 1977; Verma, 1980). The present report discusses an outbreak of *T. evansi* infection in pigs in Malaysia.

In December 1994, a farm with 180 sows was experiencing an outbreak of an unknown disease with clinical symptoms of pyrexia, anorexia, dullness, chronic emaciation, abortion and a few deaths among the breeding stock and did not respond to antibiotic treatment. During this episode 11 sows and a boar died, while more than 20 sows aborted. A number of sows showing chronic emaciation had been sold for slaughter. Three sows and a boar with chronic emaciation exhibited nervous signs prior to death. Some of the sows that had recovered were in poor body condition.

In January 1995, an investigation was carried out. On the first visit, two sows were found to be anorexic

and dull. The farmer was advised to treat the sows with antibiotics in combination with an anti-inflammatory drug to rule out bacterial infection, if any, as the causative factor was unknown.

During the second visit, (after four days of the first visit) 4 sows were showing signs of anorexia, dullness and had pyrexia. Five mL of blood sample was collected from each of the four sows from the jugular vein using EDTA and heparin as anticoagulant and submitted for haematological, parasitological, bacteriological and virological investigations. Post-mortem was not carried out as there was no death during the investigation period. On one occasion an aborted foetus was submitted for laboratory investigation.

A wet blood film was made on a glass slide with a coverslip and examined under dark ground microscopy. On examination, active motile organisms resembling trypanosomes were seen. Thick and thin blood smears as well as thin smears from the buffy coats from haematocrit tubes were also made, fixed in methanol and stained by 8% Giemsa.

One mL of well mixed EDTA blood was inoculated intraperitoneally into each of two mice (Swiss Albino strain) for each blood sample. Wet films were made from the blood collected from the tail and examined daily for trypanosome using the microscope.

Three of the four blood samples examined by wet films, haematocrit technique and thick and thin smears were positive for trypanosomes. In two of the blood samples numerous parasites were seen in the wet smears. The blood of all mice were positive for *T. evansi* 3-4 days post inoculation.

Haematological parameters of the 4 sows showed only slight variation. The PCV and the erythrocyte counts were slightly below the normal range. Three of the 4 sows showed eosinophilia and also slight

leucopenia. On bacteriological examination no aerobic bacteria was isolated and under dark ground microscopical examination no leptospira-like organism was seen. No swine fever virus was isolated on virological examination. There was no significant finding from the aborted materials.

There are several reports on the successful use of suramin (Naganol, Bayer, Germany) in the treatment of trypanosomiasis of domestic animals (Stephen, 1963; Mohmoud and Gray, 1980; Higgins, 1983; Dieleman, 1986). Treatment with suramin drug was therefore carried out in the farm. Four sows with clinical signs were treated initially with suramin at the rate of 2.5 g (10%) per sow intravenously. All the four sows recovered after the treatment. Suramin treatment was continued for one month on those sows as the clinical signs appeared. Treated sows recovered within a day and there were no deaths recorded in the farm after initiating the treatment.

The disease was diagnosed as trypanosomiasis based on clinical signs, presence of trypanosomes in the blood and the quick recovery to suramin. *T. evansi* has been considered less pathogenic to pigs (Woo, 1977; Verma, 1980). Srivastava and Ahluwalia (1972) observed only mild intermittent fever with no other visible signs of disease in indigenous pigs experimentally infected with *T. evansi*. Similar observations were also made by Cabrera and Lui (1956). Gill and others (1987) suspected a natural outbreak of pig trypanosomiasis due to *T. evansi* in India. They were of the opinion that the mange infestation might have precipitated the condition with the possible cause being the immunosuppressive effect of mange.

This investigation suggests that *T. evansi* could be pathogenic to pigs. During clinical trypanosomiasis especially at the peak of parasitaemia the pigs go off-feed which may lead to chronic emaciation, abortion and death. Similar findings were also observed by Ilemobade and Balogun (1981) and Agu and Bajeh (1986) in other types of trypanosomiasis in pigs.

The trypanosome detected in the pigs appeared to be an opportune parasite that could have been transmitted from the buffaloes found freely roaming around the farm. These buffaloes are semi-wild in nature and as a result could not be examined for the parasite. Buffaloes are said to harbour the parasite for life without symptoms of illness but under certain stressful conditions the parasitaemic stage increases thus act as reservoir host to other susceptible animals (Woo, 1977; Mahmoud and Gray 1980; Dieleman, 1986). A buffalo herd about 3 km away from this farm was diagnosed as having trypanosomiasis with the detection of trypanosome in the blood smears. Thus, there is a possibility that the trypanosome may have been introduced into the pig herd through the parasitaemic (carrier) buffaloes by haematophagous flies.

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RINGKASAN

SATU WABAK JANGKITAN TRYPANOSOME EVANSI DALAM BABI

Satu wabak tripanosomiasis babi disebabkan Trypanosome evansi yang dicirikan oleh demam, anoreksia, kurus kering, keguguran dan maut dikalangan stok pembiakbaka telah dihuraikan. Sebelum penyiasatan ini dilakukan 11 ekor babi betina dewasa dan seekor babi jantan dewasa telah mati dan lebih daripada 20 ekor babi bunting mengalami keguguran. Trypanosome evansi telah dikesan dalam enam ekor babi pada masa penyiasatan dilakukan. Babi yang terlibat telah berjaya dirawat dengan suramin pada kadar 2.5 g (10%) per babi betina dewasa. Kepentingan klinikal, ekonomi dan epidemiologi wabak ini telah dibincang.