

Sudden Deaths in Pigs Associated with *Stephanurus dentatus*

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The swine kidneyworm (*Stephanurus dentatus*) has wide geographical distribution in tropical and subtropical regions of the world. In the United States the parasite is most prevalent in the South Atlantic and South Central States; however, it has been reported that the parasite has spread to other sections of the country. Kidneyworm larvae migration and resultant damage is responsible for a large percentage of liver and carcass condemnation in abattoirs. A survey showed that at least 95% of condemned swine livers in the Southeastern United States had lesions associated with infections by *Ascaris suum* and/or swine kidneyworms.

Pigs become infected with kidneyworms by: (1) ingestion of infective larvae with contaminated food or water, (2) penetration of skin by infective larvae, (3) ingestion of earthworms serving as transport host or (4) prenatal infection by histotrophic larvae migrating to the uterus and infecting the developing fetus. Sows harboring a patent infection of kidneyworms void urine containing large numbers of ova which serve as the source of infection for their piglets. Infection of piglets at an early age by kidneyworm larvae results in condemnation of livers at slaughter.

The life cycle may be summarized as follows: (1) production of large numbers of ova by female parasites located in the urinary tract of the pig, (2) voiding of the ova with the urine, (3) embryonation and subsequent hatching of the ova on soil, followed by growth of the free living larvae to the infective or third stage, (4) entrance of the larvae into the pig's body, (5) migration within the pig, (6) a period of development in the liver and (7) migration of immature parasites to the urinary tract where maturation is achieved.

From one to nine days after oral infection, large numbers of larvae are found in the mesenteric lymph nodes and none in the liver. A migration of larvae from the mesenteric lymph nodes occur 10 to 32 days post infection (P.I.). The fourth molt occurs in the liver 24 to 32 days P.I.

Experimental exposure of specific-pathogen-free piglets to the larvae resulted in an extremely widespread fibroblastic reaction in the liver. Cross sections of the liver were characterized by massive and widespread increase in fibrous tissue associated with marked loss of liver parenchyma. Where there were numerous parasitic infiltration cellular reaction was seen throughout the hepatic lobules. In more advanced cases there were atrophy of the liver lobules which was replaced by fibrous tissue. Larvae not encapsulated migrated to regions of the ureters between the 4th and 9th month P.I. Cysts were formed in the walls of the ureter and ova were found in the urine 9 to 16 months P.I.

Experimental infection of parasite-free piglets with infective larvae consistently resulted in death of some infected animals. The deaths occurred 20 to 30 days P.I. This correlated with the migration of larvae from the mesenteric lymph nodes. The piglets were apparently normal in the afternoon but some died overnight.

At necropsy severe peritonitis, intussusception, adhesions and necrosis were observed. Histological examination of amorphous reactionary tissue masses showed kidneyworm larvae surrounded by lymphoid and reticuloendothelial cells and a few eosinophils. There was extensive fibrosis surrounding the inflammatory foci in the liver, pancreas, lymph nodes, lungs and heart.

Kidneyworm larvae were obtained from the lesions by Baermanization and were observed in histological sections. These techniques could be useful in differential diagnosis.

Control of kidneyworms in the breeding animals would aid in the prevention of the sudden deaths of piglets. It would decrease the high rate of liver condemnation experienced by producers in endemic areas. In some areas of the Southeastern United States liver condemnation has reached 85%.

There are two drugs effective against kidneyworms. Treatment of sows with levamisole kills kidneyworms in the urinary tract and in cysts contiguous to the urinary tract. Sows may start voiding ova 5 to 6 weeks after treatment due to migration of the parasites.

Fenbendazole was the first anthelmintic to demonstrate efficacy against the immature and mature kidneyworms in the urinary tract and throughout the body. No live mature or immature kidneyworm were found in sows treated with single or multiple doses of Fenbendazole. Removal of immature kidneyworm in sites away from the urinary tract prevented migration of the parasites to establish patent infection around in ureters.

Selected references: Batte, E. G., McLamb, R. C., and T. J. Vestal; *Vet Med/SAC* 70(7) 809-812, 1975.
Batte, E. G.; *Vet Med/SAC* 73(9) 1183-1186, 1978.
Tromba, F. G.; *Jour Par* 41(2) 157-161, 1955.
Lichtenfels, J. R. and F. G. Tromba; *Jour Para* 38(4) 757-766, 1972.