Isolation of Edwardsiella tarda from Swine

DWIGHT R. OWENS, STUART L. NELSON, AND J. BRUCE ADDISON

Veterinary Medical Diagnostic Laboratory and Departments of Veterinary Microbiology and Veterinary Pathology, School of Veterinary Medicine, University of Missouri, Columbia, Missouri 65201

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Edwardsiella tarda was isolated from the intestinal tract of a 2-month-old pig. This is the first reported isolation of Edwardsiella tarda from swine in the United States. Swine have been reported as potential carriers of Edwardsiella tarda, but pathogenicity of this organism for swine has not been determined. Although the pig had access to several farm ponds, the exact source of infection was not determined.

Edwardsiella tarda was recognized in the United States as early as 1959, but it was not until 1965 that it was proposed as a new genus and species in the family Enterobacteriaceae (5). At that time, 37 E. tarda isolants had been reported. These isolants were from the United States and several other countries. Two of these isolants were from animal sources, 34 were from humans, and 1 source was unidentified. One of the animal isolants was from a bovine with diarrhea, but the source of the other was not indicated (4).

E. tarda was first reported in Japan in 1962 and was designated the "Asakusa Group" (9). It had been recognized in Japan since 1959. In Japan, of the 256 cultures which were originally studied, 248 were isolants from snakes, 2 were from seals, and 5 originated from the feces of humans affected with symptoms of gastroenteritis. Isolation of the organism in man has been reported from feces, wounds, blood, and urine (2, 5, 7, 9). It has also been incriminated as the etiological agent of a case of fatal meningitis (10). In the United States, E. tarda has been reported from animals in zoological surroundings and in aquatic environments—E. tarda has been recovered from a sea lion, two alligators, and an Australian skink (11, 12). It has also been reported to cause diarrhea and severe enteritis in an ostrich (12). It has been isolated from several species of turtles in the Southeastern and Southern United States (6). It has also been recovered from the common snapping turtle (Chelydra serpentina) in central Missouri. Most recently, E. tarda has been reported as a pathogen of channel catfish (Ictalurus punctatus) (8).

There have been two reports of E. tarda isolation from swine. The first isolation was from pig bile which was collected from apparently healthy animals in Philippine abattoirs (1). The second is reported from an animal in Vietnam. Our isolation of E. tarda from a pig in Missouri apparently constitutes the first finding of E. tarda in swine in the United States.

MATERIALS AND METHODS

On 31 July 1970, a 2-month-old pig was presented to the Veterinary Medical Diagnostic Laboratory, School of Veterinary Medicine, University of Missouri, Columbia, Mo., from a swine herd producer in Williamsburg, Mo.

A necropsy was performed on the animal immediately after it was killed. Tissue specimens were removed aseptically from several organs and submitted for bacteriological examination. Their surfaces were seared with a hot spatula, and a sample was removed with sterile scissors and forceps and inoculated onto several agars. Trypticase soy agar enriched with 5% sheep blood and MacConkey agar were utilized for culturing all tissue. In addition, brilliant green agar was utilized when the small and large intestines were cultured. All media were products of BBL, Cockeysville, Md.

At the time the bacteriological sample was taken, a duplicate tissue specimen was collected from select organs and placed into 10% buffered Formalin for histopathological examination. Histopathological examination was performed on slides stained with hematoxylin and eosin.

RESULTS

The pig was one of eight in a litter and had been weak from birth. Its litter mates were healthy and showed no signs of disease. Rectal temperature prior to death was 101 F. Before death, the animal went through a period of diarrhea and depression and did not respond to medication. Postmortem examination failed to reveal remarkable lesions in the abdominal and
thoracic cavities. The stomach and small intestine were empty, the large intestine contained a semi-formed stool, and the spleen was pulpy. The heart was very flabby and had serious atrophy of fat deposits. There were no remarkable changes in the lungs and endocrine glands.

Histopathological examination of the kidney revealed petechiae and congestion in the area of collecting tubules, exhaustion of lymphoid nodules in the spleen, infiltration of leukocytes along portal triads of the liver, and diffuse pneumonitis. No other remarkable histological changes were observed.

Microbiological examination of the lung and liver failed to reveal bacterial growth after 48 h. Culture of the small intestine resulted in a scant growth of Escherichia coli within 24 h. E. tarda and E. coli were isolated from the large intestine in heavy concentration within 24 h. The organisms were identified by the methods of Edwards and Ewing (3). Identification of our isolate was confirmed by the Center for Disease Control, Atlanta, Ga.

**DISCUSSION**

At the present time, little is known about the pathogenicity of E. tarda for animals. It has been associated with disease in the ostrich (12), in channel catfish (8), and found in association with bovine diarrhea in one case (5).

Although E. tarda has been isolated from swine in two countries, no specific lesions have been reported. The only lesion observed in this case was that of infiltrating leukocytes along the portal triads and diffuse pneumonitis. Both of these lesions are common in infectious diseases and septicemias and cannot be considered as being specific lesions associated with E. tarda.

Since the necropsy was performed shortly after death, it was felt that bacterial transmigration had not occurred and that the organisms recovered were a true picture of the intestinal bacterial flora at the time of death.

The E. coli which was recovered from the small intestine was not considered to be a pathogen because of the small number present. Experience has shown that if E. coli is a significant etiological factor, large numbers of the organisms will be present in the upper intestine which was not the case here.

Biochemically, the organism is similar to Salmonella spp. and, perhaps in the past, the organism might have been falsely identified as a Salmonella spp. It is easily differentiated from salmonellae in that it is indole positive and does not ferment mannitol. Its serological and biochemical properties have previously been reported (4).

Retrospectively, we learned that the swine herd from which the pig came had access to several farm ponds. E. tarda is present in many amphibians and snakes (6, 9) but has not been associated with disease. It is interesting to speculate that reptiles or amphibians may act as reservoirs of infection for domestic animals. One report indicated swine may act as carriers of E. tarda as the organism localizes in the gall bladder (1). If this is true, swine may then shed the organism which would return to the ponds and contaminate other reptiles and amphibians.

To determine the importance of this organism for swine and other animals, it is essential that diagnostic laboratories be alerted to the possibility that E. tarda may be carried by swine. It is also important to recognize this species and not to confuse it with other enterobacteriaceae.

At the present time, it remains for the organism to be incriminated as an enteric pathogen of swine.

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