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***Staphylococcus aureus* MASTITIS IN NURSING MINK AFFECTED WITH ALEUTIAN DISEASE[□]**

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Abstract: An outbreak of staphylococcal mastitis in nursing female ranch mink (*Mustela vison*) is described. Lesions were acute necrotizing mastitis, fatty infiltration of the liver and renal tubules, and adrenal cortical hyperplasia. The presence of Aleutian disease in the herd suggests a role of immunosuppression in the outbreak.

INTRODUCTION

Mixed streptococcal and coliform mastitis with septicemia in ranch mink (*Mustela vison*) has occurred in association with feeding of condemned beef.⁶ Staphylococcal dermatitis in female mink and their kits has been reported in Alaskan ranch mink.² This paper reports an outbreak of staphylococcal mastitis in nursing ranch mink in association with Aleutian disease (AD).

HISTORY

In June, 1978, 12 mink were submitted to the Northeastern Research Center for Wildlife Diseases from a ranch with a population of approximately 9,000. The ranch had experienced losses of nearly 300 nursing dams without an increase in kit mortality. The affected females showed anorexia and depression, but neither changes in mammary glands nor in nursing patterns were noted. The lactation period was nearly over, so little nursing activity was expected. Administration of tetracycline in the drinking water had no effect.

NECROPSY RESULTS

The mink submitted were severely dehydrated and debilitated. Nine had

gross or histologic evidence of necrotizing mastitis (Fig. 1). All animals had friable, fatty livers and pale yellow kidneys (Figs. 2 and 3). The adrenal glands were grossly enlarged and had microscopic cytomegaly and excess mitoses in the cortex.

Staphylococcus aureus was recovered in pure culture from mammary tissue of 11 animals and a *Salmonella* sp. was isolated from intestinal contents of 1 mink. One mink with mastitis also had lesions of AD: plasmacytic interstitial nephritis, glomerular atrophy, renal tubular protein casts and dilatation, biliary hyperplasia with lymphoplasmacytic pericholangitis and periportal fibrosis, and splenic plasmacytic proliferation.⁵

The high reproductive success of the breeding females in this and previous years convinced the rancher that he did not have a significant AD problem. He had not seen a need for serologic testing for AD and his practice of "pelting out" females with poor reproductive performance seemed to control the disease. Carcasses examined at fall pelting had histologic lesions of AD. Sera of 14 mink were examined at the Wisconsin Central Animal Health Laboratory by counter electrophoresis and all were positive.

[□] Scientific Contribution No. 759, Storrs Agricultural Experiment Station, University of Connecticut, Storrs, Connecticut 06268, USA.

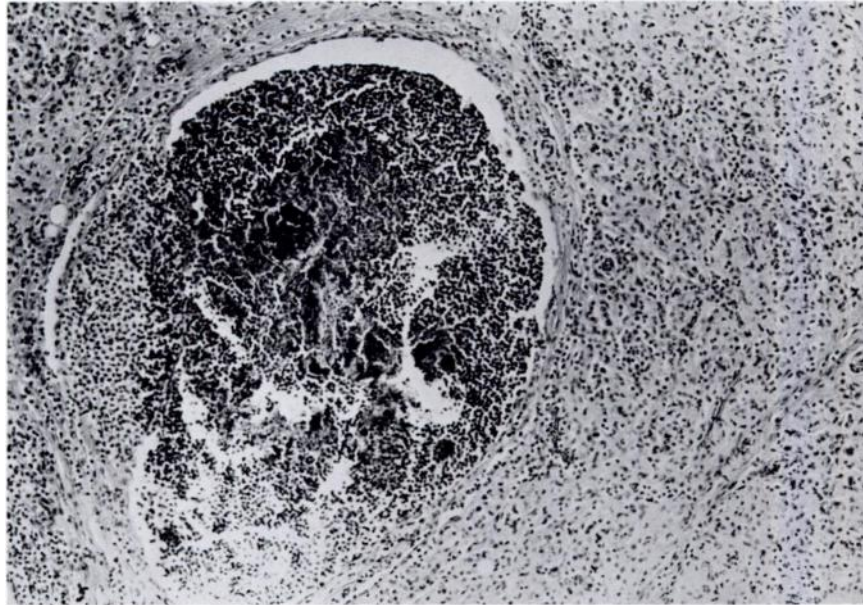


FIGURE 1. Suppuration and beginning encapsulation of abscess in regressing mammary gland. H & E; $\times 63$.

The staphylococcal isolate was grown for 24 h in tryptose broth. Washed cells and supernate both were non-pathogenic when injected intraperitoneally into 6-week-old BALB/c mice.

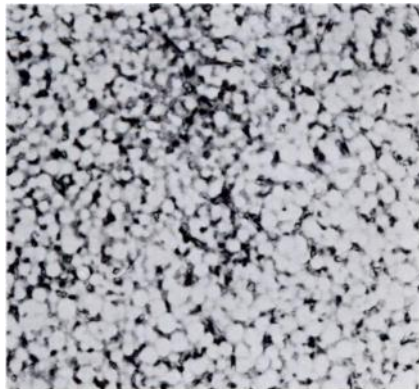


FIGURE 2. Fatty infiltration of liver. H & E; $\times 63$.

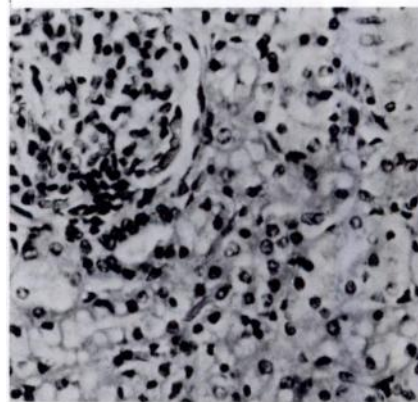


FIGURE 3. Fatty infiltration of renal tubular epithelial cells. H & E; $\times 160$.

DISCUSSION

The finding of severe necrotizing mastitis in nursing mink in the absence of disease in suckling kits and test mice

suggests that the female mink had a decreased resistance to infection. Earlier reports of similar bacterial disease of mammary glands and skin had shown involvement of both dams and kits.^{2,6} Considering the evidence of AD infection on the ranch, it seems likely that the dams had an increased susceptibility to

bacterial infection possibly due to immunosuppression related to AD.⁴

It was not possible to determine if the fatty change was secondary to anorexia caused by the staphylococcal infection, or due to a metabolic disease such as the incompletely understood "nursing anemia" or "fatty liver syndrome".^{1,3}

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