Aujeszky’s Disease

Outbreak on an Iowa farm

Gravers K. L. Underbjerg, Ph. D., fall ’43

Because of their rapid development and rare occurrence, certain diseases are difficult to diagnose. Often the anamnesis of such cases is lacking because the owner does not observe anything before he finds that one or more animals in the herd has succumbed. Such cases of rare diseases in a community are puzzling to the owner as well as to the veterinarian. Furthermore, it is probable that a rare enzootic may easily escape attention because of failure of a diagnosis. Aujeszky’s disease falls into this category since it is sporadic in occurrence, concealed in its development, and of short duration before death ensues in the affected animals. In species such as the pig, the course of the disease may be insidious and the recovery uneventful.

Following Aujeszky’s report of the existence of the disease in cattle, dogs, and cats in Hungary in 1902,1 numerous records of the disease in that country and reports of the occurrence in other parts of the world have appeared. Most outbreaks, however, have been sporadic with only a few animals apparently being affected. For a comprehensive review on the subject, the reader is referred to Galloway,4 and Hutyra and Marek.5

Occurrence

The occurrence of the virus of Aujeszky’s disease has been reported under various names, and the common synonyms given are: “pseudorabies,” “infectious bulbar paralysis,” and “mad itch,” the latter under which it was first reported by Shope11 in this country. Most authorities agree that the infectious agent reported under the various synonyms is identical with the virus of Aujeszky. Another outbreak of Aujeszky’s disease in America was reported by Morrill and Graham.7 At the Veterinary Research Institute, Iowa State College, the virus has been recovered from cattle and swine by McNutt6 in several cases.

The virus of Aujeszky’s disease has been found to infect dogs, cats, cattle, swine, sheep, horses, and rats under natural conditions. Experimentally, the disease has been produced by inoculation or feeding in rabbits, cats, dogs, guinea pigs, rats, mice, pigs, sheep, cattle, goats, horses, asses, foxes, hedgehogs, monkeys, porcupines, opossums, pigeons, geese, ducks, sparrowhawks, and domestic fowl.

Outbreak Investigated

Recently, an outbreak which was finally diagnosed as Aujeszky’s disease occurred on a farm near Sheffield, Iowa. The owner had lost seven head of cattle from an unknown cause within a week. Five weeks previously, the owner had lost several head of hogs, and three weeks later, three more pigs had died. The herd of cattle in which the deaths occurred consisted of 30 head of milk cows and heifers and one steer. These cattle were yarded in a lot (Lot 1) with the hogs. In an adjacent lot (Lot 2) there were 27 head of feeder steers but no hogs. Both groups of cattle were housed in the same cattle shed, but separated by a hayrack which divided the shed. Both groups had access to drinking water from the same watering tank. All deaths occurred in Lot 1.

On the morning of December 28, 1942, the owner noticed that one of the cows acted strangely and a veterinarian was
called. The cow was treated for hypocalcemia but died during the treatment. The owner was advised that the dead cow could be safely fed to his hogs and this was done. On the morning of December 30, another cow was found dead. It had been milked the night previously and appeared then in good health. Because of the blood scattered about the place, the owner thought that the cow had cut herself in some way and bled to death. The morning of December 31, a third cow was sick. The attending veterinarian diagnosed the case as the “skin form of hemorrhagic septicemia.” This animal died during the following night. On the morning of January 1, 1943, one of the cows was noticed to be off feed. In the early afternoon it became very violent and died at 7:30 p.m. There were no sick or dead animals on January 2, but on January 3, one cow was found dead and two others were off feed and had an unsteady gait. One was showing extreme irritation about the head and neck, but the other went down showing no pruritus and went into a coma at 3:30 p.m. Both cows were dead at 8:30 p.m. The heads of the latter three cows were taken to the Iowa State College Veterinary Research Institute for a laboratory diagnosis on January 4 by the veterinarian in attendance since January 1. The owner was immediately advised to remove the hogs from the cattle pen.

The length of time the animals showed symptoms before they succumbed ranged approximately from 14 to 20 hours. The temperatures of two of the animals were 101° F. and 103° F. Other animals were too violent to approach. It is worthy of note that the owner had been medicating the hogs in Lot 1, indicating an illness.

Brain Cultures Made

Cultures made from the brains of the affected cattle were found to be sterile of bacteria. Physiological salt solution suspensions of the brain were injected into rabbits by the subcutaneous and intravenous routes. The subcutaneous injections reproduced the clinical picture of “mad itch” as it is seen in cattle. After an incubation period of 48 hours, the rabbits appeared uneasy and began biting and scratching at the site of inoculation. The condition became progressively worse and the biting and scratching more persistent and savage. The skin was soon abraded and denuded of hair. As the animals became weaker, the attempts at scratching became fewer. The animals fell on their sides and were unable to rise. Their respirations became rapid and noisy, and death ensued in about 12 to 20 hours after the first evidence of pruritus.

Injected intracerebrally, the brain suspensions produced the uniformly fatal disease in rabbits. The rabbits died in 24 to 48 hours following inoculation. After an incubation period of 20 to 44 hours, the animals appeared nervous and excited. They would run about wildly in their cage, striking their heads violently against the sides of the cage. Death was preceded by coma and rapid, noisy respiration.

Diagnosis

The history of the case and the clinical symptoms of the inoculated rabbits leave but little doubt of a diagnosis of Aujeszky’s disease or “mad itch.” The disease has been shown to be present in Iowa, occurring sporadically in cattle and swine by Shope, and McNutt. It is apparently contagious in rats and swine, but little is known about its transmission under natural conditions. The disease apparently is not contagious in cattle. The means by which cattle become infected, and the method of spread in these animals is not clear. Patto cited by Shope noted that in all the outbreaks which came under his observation in Brazil, the infected cattle had been kept in intimate contact with swine. Burggraaf and Lourens have stated that in an outbreak of pseudorabies in Holland in 1932, in which the disease was recognized as a relatively mild illness in swine but highly fatal in cattle, the swine became ill before the cattle on the farms. These authors further noted that, with the exception of four cows on two farms, all cattle which became infected had been housed in the same building with sick hogs. Shope

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been confirmed, but suggests the possibility of inadequate vitamin B synthesis in the rumen under certain conditions.

**Vitamin C**

Vitamin C (ascorbic acid) is synthesized in the bovine, but is sometimes deficient, producing sterility in both the male and the female. No other symptoms from vitamin C deficiency have been reported. Recent work indicates that the deficiency in some cases, at least, is associated with inadequate amounts of vitamin A. In such cases the vitamin C content of the blood is restored to normal levels with the oral administration of vitamin A. As ascorbic acid is destroyed in the rumen, oral administration is contra-indicated, and it should be administered parenterally.

**Vitamin D**

The bovine needs vitamin D which is normally obtained from sun-cured roughage during the winter and from direct exposure to the sun's rays during the pasture season. It is known that grains are devoid of vitamin D and deficiencies of sufficient magnitude to produce marked symptoms are found when large amounts of grain are fed with little or no sun-cured roughage, and also when the amount is not exposed to sunlight. This condition has been observed in steers during the winter when allowed all the grain they wanted and a low quality hay, of which they ate little or none. The first symptom is swollen and painful joints. The soreness rapidly becomes so great that the animal refuses to get up; simultaneously, there is a complete loss of appetite. Erosion of the articular surfaces of the bone is accompanied by infection with pus formation and irreparable damage. Other symptoms are quickly relieved by the administration of vitamin D, including restoration of the appetite.

Recent reports indicate that massive doses of vitamin D (4,000,000 units per day) for 30 days prior to parturition prevents the onset of parturient paresis in susceptible cows. Since milk fever is caused by a hypocalcemia and vitamin D is known to mobilize calcium in the blood, the prophylactic effect of vitamin D seems plausible.

**Vitamin E**

A great deal has been said about the value of vitamin E (alpha-tocopherol) in relation to sterility. There is not as yet a good, well controlled experiment to suggest the value of vitamin E in the treatment of sterility. Experiments now in progress indicate that cattle do not need vitamin E either for their physical well-being or for reproduction.

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has presented experimental evidence that swine may serve as the source of infection for cattle. He showed also that fatal infection can be induced in rabbits by bringing their abraded skin in contact with the snouts of infected pigs, and he thought it possible that cattle may become infected in a similar manner. Shope visualized a cycle of infection from rats to pigs (he showed that carcasses of infected rats were eaten voraciously by pigs which as a result became infected), to cattle, and back to rats (the rats and/or pigs feeding on carcasses of dead cattle).

If this mode of transmission is accepted, an explanation of the infection of the cattle in Lot 1 in the case reported is made possible. It is not likely, however, that the deaths of the pigs previous to the infection of the cattle were due to Aujeszky's disease, since it is rarely fatal to this species except in baby pigs, and animals affected exhibit only a transient depression and inappetence. On the other hand, the pigs may have been affected with the disease and since the virus is present in the nose of a hog it is likely that the cattle lying about the barn lot may have become infected by coming in contact with the noses of the pigs. Cattle lying about a barn lot in which hogs are also kept frequently come in contact with the noses of pigs. Swine under such conditions can be observed to approach a cow and probe...
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It in the flank or side with their snouts. In support of the supposition that the swine in Lot 1 may have been carriers of the infection and transmitted it to the cattle is the nonexistence of any fatalities occurring among the cattle in Lot 2, to which the pigs did not have access. In addition, following the removal of the pigs from the cattle pen, no more fatalities have been reported on the farm.

(The writer is indebted to Dr. S. H. McNutt of the Veterinary Research Institute, Iowa State College, who diagnosed the case and furnished valuable material.)

BIBLIOGRAPHY

2. Burggraaf, A. and Lourens, L. F. D. E. Infec-

Virus

Briefly, simultaneous virus as it is used in conjunction with anti-hog-cholera serum is phenolized, defibrinated whole blood (i.e., blood with fibrin removed) obtained from inoculated cholera sick pigs which weigh between 40 to 110 pounds. Before bleeding, these animals are observed clinically for symptoms of hog-cholera. Rectal temperatures must reveal a pronounced elevation. Bleeding is done by way of the throat using an aseptic surgical technic. The carcass of the virus pig is then observed officially for lesions indicative of hog-cholera. Bleedings from pigs which pass official inspection are defibrinated to remove the fibrin which has formed in the sterile collecting vessel. After sufficient blood has been pooled to make a representative mix or serial, 5 percent phenol is added, quantity sufficient to make a final dilution of .5 percent. After the product is bottled it is held under government lock and key until it has passed all official tests. Not until it is proved that the product is pure and potent is it released for marketing.

Serum

Anti-hog-cholera serum, as it is known in commerce, is the defibrinated, clarified (i.e., red cells removed), pasteurized and phenolized blood from hogs hyperimmunized against hog-cholera. Before a hog can be brought into the production of anti-hog-cholera serum, it must first be immunized against hog-cholera by the simultaneous method for at least 90 days. After this time the animal is admitted to the plant and observed to make sure that it is free from all other swine diseases. The hyper-immunization process, which has already been referred to, consists of an intravenous injection through an ear