An Outbreak of Pseudomonas Infection in Poults

By H. L. CHUTE*

THIS CONDITION involved 87 day-old White Holland poults. The mortality was about 80 per cent. A bacteriological examination revealed that the cause of death was *Pseudomonas aeruginosa*. This organism was isolated in pure culture.

Review of the Literature

All textbooks of bacteriology discuss this organism, but in no instances do they state that it is a known cause of infection in turkeys. However, there are numerous references in journals to the organism as a cause of disease in poultry. Stafseth, Mack, Walter and Ryff (1) reported an outbreak of a disease in approximately 19,000 turkeys, with a morbidity rate of 50 per cent and a very low mortality, and that a hemolytic pseudomonas was isolated in pure culture from the heart and liver of a turkey which had recently died. Kaupp and Dearstyne (3) mentioned that they had isolated *Pseudomonas aeruginosa* from four of 300 autopsies on adult fowls. They did not refer to turkeys. Pomeroy and Fenstermacher (4) recovered Pseudomonas frequently from poults in connection with studies on paratyphoid infection in turkeys. Hinshaw and Lloyd (5) reported they isolated *Ps. aeruginosa* from the bone marrow of one chick from the heart and liver of 11 chicks, and from one poult in connection with studies on Vitamin A deficiency in turkeys. Essex, McKenney and Mann (6) reported an epizootic in a flock of 400 chicks from five to nine weeks old. The only organism isolated was *Ps. aeruginosa*. Peterson (10) reported an outbreak of Pseudomonas infection in 300 poults. The organism was isolated and day-old chicks were infected. Jones and Anderson (9) experienced an outbreak in 3000 poults. Glover (7) reports that many bacterial species may be implicated in omphalitis in chicks and poults. He states that in the dry form of the disease many different non-spore formers are found, and among these organisms he lists Pseudomonas. In 1946-47 O.V.C. report, Glover (8) listed among cases which came to the laboratory, two cases of *Pseudomonas aeruginosa*, four cases of *Ps. aeruginosa* with coryza, and one case of *Ps. aeruginosa* with ascariasis.

There is ample evidence that this organism does infect turkey poults.

Field Conditions

This outbreak occurred on a farm where no poultry had been kept for years. A new brooder house 14′ x 24′ had been built and all the equipment — drinking fountains, feed hoppers, brooder — was new. The inside of the building had been painted with creosote. A temperature of 95°F. was maintained under the brooder. Baled shavings were used as litter for the first two weeks but was then changed to a mixture of gravel and sand. Spring water was used and a sample of this water was examined and found to contain no colon organisms. One teaspoonful of Diversol in two gallons of water was used. A standard brand of turkey starter was fed to

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the birds. Fresh whole milk was given once a day. One could state from these facts that the brooding conditions were ideal.

Numbers Involved and Death Rate

Eighty-seven poults were involved, and from the very first a few died each day. The poults were hatched on June 10th, and by July 1st 68 had died. Not many died after this date. The peak period of the mortality was from the eighth to 13th day after hatching. In this case, the birds died suddenly without showing symptoms longer than one day.

Avenue of Infection

Failure to infect healthy poults with the organism has been reported by many workers. Stafseth, Mack, Walter and Ryff (1) did manage to artificially infect two-week poults and also newly hatched chicks per orum when the organism was placed in the drinking water for two successive days. Jones and Anderson (9) transferred the organism successfully to poults by using an atomizing spray, and all poults that died were between 14 and 21 days of age. Green (2) observed spontaneous *Ps. aeruginosa* infection in rabbits suffering from lack of Vitamin A. From these facts and those reported by Hinshaw and Lloyd (5) which have been previously mentioned, one might suppose that a lack of Vitamin A could cause a change in the normal epithelium of the intestinal tract and that infection could enter by that route. In all cases observed and those reported by other workers an enteritis ranging from an acute catarrhal type to a hemorrhagic enteritis is always present.

Symptoms and Post Mortems

The poults in this case showed a staggering gait and drooping wings. A brown, foamy, soft diarrhoea was manifested. From the time the infection had first appeared, the owner had noted a brownish, slimy discharge from some of the birds. Death was rapid. The usual post mortem appearance was fluid in the abdominal cavity, gelatinous exudate under the skin, pale liver, and inflammation of the intestines with some petechial hemorrhage. We did isolate the organism from one poult which showed no enteritis, but only pale watery bile and swollen kidneys.

Stafseth, Mack, Walter and Ryff (1) state in their cases that the incubation was short, the poults showed a droopiness, depression, weakness, torticollis, a thin watery yellow diarrhoea, a high temperature, and that there was sudden death. The post mortem on these birds showed blood which was dark, syrupy and incompletely coagulated even after several hours; mucous or hemorrhagic enteritis; petechial hemorrhage on serous membranes; small gray to green spots on the liver and a slightly enlarged spleen. Jones and Anderson (9) reported symptoms ranging from complete listlessness with a watery diarrhoea to only a slight morbidity.

Characteristics and Pathogenicity of the Organism

On two occasions, with an interval of ten days, the organism *Pseudomonas aerug nosa* was isolated from these poults. The organism corresponded in all its characteristics to those given in Bergey's Manual for *Pseudomonas aeruginosa.*

In the main, these characteristics were the production of intense greenish blue pigment in agar, broth, and peptone water; non-fermentation of "sugars" except glucose and xylose and peptonization of milk; and a gram negative motile rod.

The organism was isolated from the intestine, liver, and fluid in the abdominal cavity.

One-half c.c. of a 24-hour broth culture given intra-peritoneally killed a guinea pig in twenty-four hours. The organism was isolated from the guinea pig.

Serological reactions showed no agglutination with homologous, variant and regular positive pullorum serum. As a matter of interest, good agglutination with several different sera from positive Brucella abortus blood samples was obtained.

Stafseth, Mack, Walter and Ryff (1) in their work with the organism stated that the one they isolated was slightly different from *Ps. aeruginosa*. Generally their organism fermented dextrose without gas, indole was not produced and the culture after a time produced a brownish tint. It would not cross agglutinate with proteus. Smears from heart, blood and liver showed the organism. It was very pathogenic for mice, turkeys, chickens, pigeons, rabbits, guinea pigs, and rats.

Peterson (10) isolated the organism from the blood stream, viscera, and intestinal contents of infected poults. He also infected day-old chicks by feeding a broth culture in the drinking water for two days. He found the incubation period to be seven to ten days.

Treatment

The peak period of mortality was over before the cause of death was determined, and therefore no specific treatment was given. The usual sanitary precautions — particularly cleansing of utensils — was advised.

Jones and Anderson (9) reported they found that sulfamerazine 0.5 per cent in the mash was not effective in controlling the condition, but that sulfamerazine 0.75 per cent was effective in an out-break when administered before the morbidity had exceeded five per cent in any affected lots.

This treatment may be satisfactory, but from our experience the morbidity rate rises very rapidly and it is several days before a bacteriological report is available in order to treat the specific organism.

Discussion

Ps. aeruginosa is found frequently as a saprophyte in water, or organic matter, and occasionally is found as a parasite. It is also encountered in some pathologic conditions as a secondary invader, and often is not the true cause of disease. Due to its prolific growth, the organism may obscure other organisms which are the true cause of the disease. In spite of this, *Ps. aeruginosa,* at least some strains, has proven to be very highly pathogenic. The case reported here and the review of the literature support this statement.

Summary

1. An outbreak of infection with *Pseudomonas aeruginosa* is described in 87 White Holland poults.

2. Infection with *Ps. aeruginosa* in poults is more common than is generally believed.

3. Cause of disease must be determined by a bacteriological examination because the infection resembles pullorum and paratyphoid infections.

4. The infective route is not known, but reports indicate that it is through the intestinal tract by ingestion of organism. Vitamin A deficiency may be a predisposing cause of infection.

5. Incubation period is seven to ten days.

6. Sulfamerazine given soon after the outbreak of infection may be a satisfactory treatment. Due to the saprophytic nature of the organism, strict sanitation is recommended.

Acknowledgements

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Dr. Harold C. Collins

DR. HAROLD C. COLLINS, V.S., B.V.Sc. (Tor.), 1941, died at Winnipeg General Hospital after a few days' illness, April 16, 1949. He was born at Kincardine, Ont., 1906, and went west to McAuley, Man. with his parents (in 1927) and there received his high school education. Before attending the Ontario Veterinary College, from which he graduated in 1941, Dr. Collins served with the R.C.M.P. Before graduation he saw small animal practice both in Detroit and Los Angeles.

In 1941, he was appointed Field Biologist to the Manitoba Game and Fisheries Branch (Department of Natural Resources) and was posted to The Pas where he made a first hand study of muskrats on the marshes in that area. Later he joined Dr. R. J. Kirk at the Manitoba Government Experimental Fur Farm on the University Campus at Winnipeg. He was appointed Superintendent of that station in 1946.

Dr. Collins was widely and favorably known to all members of the fur industry in Western Canada, particularly for his practical work in the control of distemper in ranch animals. He is survived by his widow, his parents and one brother.

The burial at Brookside Cemetery was attended by a large number of veterinary surgeons from the Winnipeg area.