

A NOTE ON THE OCCURRENCE OF PULLET DISEASE: AVIAN MONOCYTOSIS IN EAST PAKISTAN

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Jungherr (1952), citing a poultry disease investigation work of Beaudette, described a condition that affected highly productive adult fowls. Sudden death with cyanosis of the comb and wattles and comparatively low flock mortality were the spectacular clinical features. On post-mortem examination the internal organs presented congestion with echymotic haemorrhage on the heart and abdominal fat with evidence of necrosis in the liver. Similar conditions were studied in various breeds of poultry by Jungherr and Levine (1940), Hurt (1941) and Gordon & Blaxland (1945), who also observed the lesions mostly in laying pullets.

Occurrence of the Disease on the Farm.—

The first carcass of a cross-bred pullet, autopsied on August 21, 1958 at the Sitakundo poultry multiplication centre, presented fairly good physical conditions with apparent dehydration of muscular tissue and perceptible damage to the liver and kidney.

Again on October 18, 1958, another carcass of Jungli pullet of about 6 months of age, was sent in this laboratory for inquiry into its cause of death. The bird was fed with whole paddy, broken wheat, ksheshari dal, meat offal, oilcake, local vegetables, etc., with usual drink and pasture. The post-mortem examination revealed marked darkening of the comb and wattle with fair general condition. The skeletal muscle showed extreme dehydration and patches of degeneration with crop & gizzard distended with sour smelling food material.

The proventriculus and intestine were flabby and showed catarrhal enterities with viscous mucoid contents in the lumen of the gut. No parasite was present. The liver was necrotic and presented multiple petechial haemorrhage. Kidney was enlarged. The physical change in the blood seemed to be increased viscosity and coagulability. The monocytes appeared larger with round nuclei, which indicated immaturity of the blood cells when examined under microscope. Usual bacteriological and transmission experiments of

suspected material, carried out on suitable nutrient media and susceptible animals, failed to produce any positive result.

The available data on post-mortem, bacteriological and pathogenesis tests clearly ruled out the possibilities of any transmissible oetiological agent bacterial or otherwise, which supports the findings of Jungherr (1952) and other earlier workers in Avian monocytosis. Besides, they observed the ailment in young pullets on lay. It has been indicated by Morrison (1956) and Maynard & Loosli (1956) that wheat and barley may contain enough nitrate to cause poisoning to animals fed. It may, however, be stated that nitrates might have been transformed into nitrites (NO²) in the digestive tract. These nitrites when absorbed into the blood would change the haemoglobin into methemoglobin, which could not transport oxygen through the blood stream. Paranchymatus cells of the liver, renal tubuli and other neighbouring glandular tissues were thus affected with deposit of CO₂ for want of supply of oxygen in the system. Consequently the skeletal muscle became dehydrated and the hepatic and renal tissues necrotic. Sudden death of the bird was, therefore, caused by the detrimental effect of nitrites on the involuntary cardiac muscle.

Summary.—Pullet disease or avian monocytosis was diagnosed in East Pakistan. The ailment seems to exist mostly in imported strains of fowls and is limited to certain age group. No biological factor, bacterial or otherwise, could be assigned as the direct cause of death, but the visible evidence tend to lay blame on food supplied.

References

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