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CONCURRENT FOWL CHOLERA, NODULAR TAENAESIS (*Railletina* sp.) AND NECROTIC GLOSSITIS IN DESI CHICKEN

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Abstract: In a farm comprising 100 desi chicken, 5 per cent morbidity and 3 per cent mortality were reported. Out of which two carcasses (One 72 weeks-old male and another 48 weeks-old female) was presented for necropsy. Gross lesions revealed circumscribed greyish white necrotic plaques of 1 mm in the tongue, ulcers and nodular foci in the gizzard, greyish white pin point foci in the liver and raised nodular lesions in the intestine clogged by numerous tape worms. Impression smears from the liver and spleen revealed teaming bipolar organisms. Histopathological examination revealed necrotic mucosa and sublingual glands in the tongue, interstitial fibrosis and multifocal mononuclear infiltration in the gizzard, congestion of the liver with bacterial colonies, cut section of worms embedded in the intestine with necrotic areas infiltrated with eosinophils and mononuclear cell infiltration. The worms were identified as *Railletina*sp. Heart blood swab subjected for biological test, biochemical characterization studies and pathogenicity test revealed *Pasteurella multocida.* The PCR assay with species specific primers for *Pasteurella multocida* (KMT1T7 & KMT1SP6) yielded a product of 460 bp size. The case was diagnosed as concurrent occurrence of fowl cholera nodular taenaesis (*Railletina*sp.) and necrotic glossitis.
Keywords: Fowl cholera, nodular taeniasis, necrotic glossitis, poultry.

INTRODUCTION

Indigenous chickens, otherwise known as traditional or backyard chickens, are local breeds of chickens (*Gallus gallusdomesticus*) reared in rural areas of most parts of the world (Say, 1987). In India, desi chickenare a large number of fowls of different size, shapes and colours and for the most part resembling the jungle fowls and are found all over India, bred by local farmers on a small-scale basis or as backyard holdings. Furthermore, poultry reared under this system face high mortality due to cross diseases, infection transmission, predators, poor management and nutrition (Conroy *et al*., 2005).

Fowl cholera (avian pasteurellosis) is a commonly occurring avian disease that can affect all types of birds and is distributed world-wide (OIE 2015). Fowl cholera is a contagious septicaemia (caused by *Pasteurellamultocida*) often transmitted by wild birds or other domestic birds and spreads by contamination of the feed or water, by oral or nasal discharges from infected birds. The incubation period is four to nine days. In some cases, birds die within a few hours of showing the first signs, which vary depending on the form of the disease. The respiratory form is characterized by gasping, coughing and sneezing, while in

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the septicaemic form there is diarrhoea with wet grey, yellow, or green droppings. In the localized form, the signs are lameness and swelling of legs or wing joints. Fowl Cholera is common everywhere among free-range village flocks, because they are comprised of different species and are in continuous contact with wild birds as summarised by FAO. In addition, free ranging backyard chicken still remain highly susceptible to parasitic infection via litter droppings and due to their scavenging habits. (Puttalakshmamma *et al*., 2008).

The present report documents an outbreak in a small farm comprising of 100 desi chicken with 5 per cent morbidity and 3 per cent mortality, out of which two (One 72 weeks-old male and another 48 weeks-old female) carcasses were presented for necropsy examination to the department of Veterinary Pathology, Madras Veterinary College.

MATERIALS AND METHODS

A detailed post mortem examination was conducted on the birds and gross lesions were recorded. Representative tissue samples of liver, tongue, gizzard, intestine and ovaries were collected in 10% formalin for histopathological examination. Sections of four micrometers thickness were made and stained by haematoxylin and eosin staining technique. Impression smears were collected from liver and spleen. Heart blood swab was collected for cultural examination. Liver and splenic impression smears were stained by Leishman-Giemsa stain. The worms were collected in a petridish for identification. Heart blood swab was inoculated in Brain Heart Infusion broth and incubated at 37°C and injected into Swiss albino mice. Aspirated heart blood from dead mice was streaked on 10 percent sheep blood agar and incubated at 37°C. Biochemical tests were carried out using Enterobacteriaceae-25 identification test kit (Hi Media, India). DNA was extracted from the culture by high salt method as described by Fisher and Lerman 1979. The PCR assay was performed to amplify KMTIT7 and KMTISP6 genes in *P. multocida* using the method of Townsend *et al*. 1998

RESULTS

Impression smears from the liver and spleen revealed teaming bipolar organisms (fig-4). Biochemical characterisation and biological tests were positive for *Pasteurellamultocida*. PCR assay using species specific primers for *Pasteurellamultocida* genes KMTIT7 and KMTISP6 yielded a product size of 460 bp.

Grossly gizzard revealed ulcers (fig-1) and nodular foci, while microscopic changes suchas ventricular myositis, interstitial fibrosis and multifocal mononuclear cell infiltration were observed. A circumscribed greyish white necrotic plaque of 1-2 mm diameter was observed in the dorsal and ventral surface of the tongue (fig-2). Microscopically, tongue revealed

multifocal areas of necrotic eosinophilic exudate with mononuclear cell infiltration in the submucosa and sublingual glands. The liver revealed greyish white pin point foci dispersed over the surface and on incision the foci extended about 0.5mm into the parenchyma. Histopathological examination revealed multi focal areas of congestion in the liver with bacterial colonies (fig–5). Intestines revealed raised granulomatous nodular lesions in the mucosa (fig -3) which were evident from the serosa and the lumen was clogged by numerous tape worms. The worms were identified as *Railletina* sp. Histologically, cut section of worms were seen embedded in the intestine (fig -6) with necrotic areas infiltrated with eosinophils and mononuclear cells. Epicardium revealed diffuse petechial haemorrhages over the epicardial fat. Ovaries were congested.

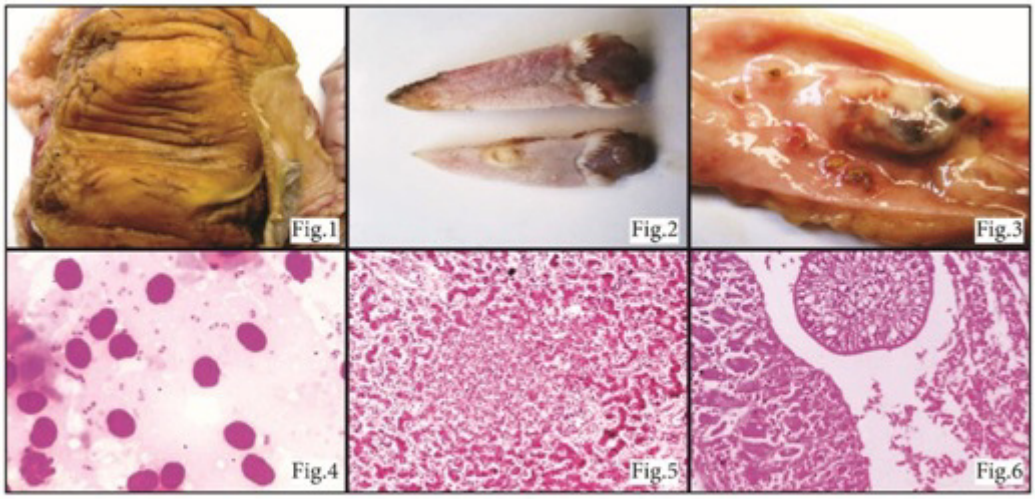

Fig.1Ulcers in the gizzard. Fig.2 circumscribed grayish white necrotic plaques of 1 -2mm in the dorsal and ventral surface tongue. Fig.3 Nodular lesions in the mucosa Fig.4 Teaming bipolar - liver imprint liver Fig. 5 Congestion with bacterial colonies Fig. 6 Nodular taeniasis cut section of worm

DISCUSSION

Gross lesions pathognomonic for fowl cholera recorded were in accordance with the observations of Rhodes, 1964. Researchers have observed swollen liver containing multiple, small focal areas of necrosis in the acutely affected birds. Large amounts of viscid mucus in the digestive tract particularly in the pharynx, crop and intestine. Catarrhal exudates in oesophagus and respiratory tract and caseous material in air sacs are also common. Petechial and ecchymotic haemorrhages are reported in 90% birds (Prantner*et al*., 1990; Hungerford, 1968; Rhoades, 1964). Surviving birds from diseased flocks appear to represent a risk, but more recent investigations indicate that carriers of *P. multocida*may exist within poultry flocks with no history of previous outbreaks of fowl cholera (Christensen and Bisgaard, 2000).

Nodular lesions observed were in accordance with Nadakal *et al.* (1973) reporting parasitic granulomas approximately 1-6mm diameter at the sites of worm attachment infected with 200 cysticercoids of *Railletinaechinobothrida* after experimental infection. The condition was associated with catarrhal hyperplastic enteritis as well as lymphocytic, polymorphonuclear and eosinophilic infiltration.

The presence of cut section of worms seen embedded in the intestine with necrotic areas infiltrated with eosinophils and mononuclear cells corroborates with nodular taeniasis. The histopathological observations of degeneration and necrosis of the intestinal mucosa and granuloma formation in the intestine have been reported earlier (Ramesh Kumar *et al*., 2007). In conditions of heavy infestation, *R. echinobothrida* is listed as one of the most pathogenic tapeworms, causing striking intestinal nodules in chicken, with characteristic hyperplastic enteritis associated with the formation of granuloma (McDougald. 2003).

Pathology of necrotic glossitis suggestive of a toxin entity most probably T-2 toxicosis is suspected but feed from the farmer could not be retrieved. Similar observation was recorded by Wyatt *et al.* 1972 suggesting these oral lesions appear to be characteristic enough to aid in the diagnosis of field cases of consumption of grain infested with *Fusarium* sps.

Pramod *et al.* (2011) reported that in a study on experimental infection of *Pasteurella multocida* inoculated in one-month old ducklings via two different routes – subcutaneous and intranasal found pasteurellosisto be immunosuppressive as there was depletion, necrosis and cystic changes in the bursa, caecal tonsils and spleen which accounted for the increased morbidity due to pasteurellosis, as these immune suppressed birds become highly susceptible to other infections. Similar pathogenesis may have played a role where in subclinical infection of pasteurellosis and possibly a toxin entity with a heavy load of parasitic infestation could have caused peracute manifestation of fowl cholera leading to sudden death of birds.

REFERENCES

[1] Christensen, J.P. and M. Bisgaard.2000. Fowl cholera - A Review. Rev. Sci. Tech. Dept Veterinary Microbiology, Royal Veterinary and Agricultural University, Frederiksberg C, Denmark, 19(2):626.

[2] Conroy, C., N. Sparks, D. Chandrasekaran, A. Sharma and D. Shindey*et al*., 2005. The significance of predation as a constraint in scavenging poultry systems: Some findings from India. Livestock Res. Rural Dev., Vol. 17.

[3] Fisher SG, Lerman LS. 1979. Length-independent separation of DNA restriction fragments in two-dimensional gel electrophoresis. Cell 16: 191-200.

[4] FAO - www.fao.org/3/al875e/al875e00.pdf

[5] Hungerford, T.G. (1968). A clinical note on avian cholera. The effect of age on susceptibility of fowls. Aust. vet. J. 44: 31-32.

[6] McDougald LR. 2003. Cestodes and trematodes. In: Saif YM, Barnes HJ, Glisson JR, Fadly AM, McDougald LR, Swayne DE. Diseases of poultry.11th edn. Iowa State Press, Ames, IA: 961-972.

[7] Nadakal AM, Mohandas A, John KO, Muraleedharan K (1973) Contribution to the biology of the fowl cestode *Raillietinaechinobothrida* with a note on its pathogenicity. Trans Am Microsc Soc 92(2):273–276

[8] OIE 2015- www.oie.int/animal-health-in-the-world/oie-listed-diseases-2015

[9] Pramod. S, N.D. Nair, M. Litty, V.R. Ambily1, N.Vijayan and C.R. Lalithakunjamma (2011) Indian J. Vet. Pathol., 35(2): 189-192,

[10] Prantner, M.M., B.G. Harmonj., J.R. Glissona, E.A. Mahaffey.1990. Septicemia in Vaccinated and Nonvaccinated Turkeys Inoculated with *Pasteurella multocida* Serotype A:3,4. Vet Pathol. **27**: 254-260.

[11] Puttalakshmamma, G.C., K.J. Ananda, P.R. Prathiush, G.S. Mamatha and S. Rao, 2008. Prevalence of gastrointestinal parasites of poultry in and around Banglore. Vet. World, 1: 201-202.

[12] Ramesh Kumar P, Reghu Ravindran, Bindu Lakshmanan, Senthamil Selvan P, Subramanian H and Sreekumaran T. 2007. Pathology of nodular tapeworm in backyard poultry. J. Parasitic Dis., 31: 54-55.

[13] Rhoades, K.R. (1964). The microscopic lesions of acute fowl cholera in mature chickens. Avian Dis. 8: 658-665.

[14] Say, R.R. (1987). Manual of poultry production in the tropics- CABI, Wallingford.

[15] Townsend KM, Frost AJ, Lee CW. 1998. Development of PCR assays for species- and type-specific identification of *Pasteurella multocida* isolates. *J Clin Microbiol* 36: 1096-1100.

[16] Wyatt, R.D., B.A. Weeks, P.B. Hamilton and H.R. Burmeister. 1972. Severe oral lesion in chickens caused by ingestion of dietary fusariotoxin T-2. *Appl. Microbiol.*, **24**: 251-257.