

## Incidence of Fowl Cholera with Anomalous Lesions in Laying Hens: A Case Study

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**Abstract:** In a commercial layer farm of 1500 flock capacity, daily mortality of 2 to 3 birds was noticed for a period of 7 days. The birds exhibit the signs of dullness, paleness in comb along with blood tinged exudates from the nostril. On postmortem examination, severe congestion vascular disturbance in various visceral organs were noticed in all birds. Tissue samples were collected aseptically and subjected to bacterial isolation, it was revealed as *Pasteurella multocida*. However, the lesions recorded were atypical of fowl cholera. Antibiotic sensitivity test revealed that the isolate was sensitive to sulphadiazine, co-trimoxazole, ceftriaxone, chloramphenicol, gentamicin, Amikacin and Amoxyclav. As per the Antibiotic sensitivity test results the flock was treated with sulphadiazine + trimethoprim combination and the recovery was observed after 5 days. The vascular disturbance in visceral organs may be due to the endotoxin produced by the organism.

**Key words:** Layer • Pasteurella • Anomalous Lesions • Sensitivity

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### INTRODUCTION

Fowl Cholera is a serious, highly contagious disease caused by the bacterium *Pasteurella multocida* (*P. multocida*), which is a small gram-negative, non-motile, non-spore-forming, facultative aerobic bacillus that grows best at 37°C. Virulence of *P. multocida* is highly variable, depending upon whether the strains are encapsulated [1]. Fowl cholera is an enzootic disease with a remarkable trend to spread. All domestic and wild species of birds are susceptible to fowl cholera [2]. *P. multocida* usually enters the host through the mucous membranes of the upper respiratory tract and probably the digestive tract also. The ability of Pasteurella to resist phagocytosis [3, 4] after invading tissues allows these bacteria to multiply very quickly, causing septicemia and severe endotoxemia; death often ensues within 24 hours. Toxins other than endotoxin may also be a possible virulence factor in some of the lesions observed in avian infections with *P. multocida*. The commonly observed signs are anorexia, ruffled feathers, oral and nasal mucus discharge, cyanosis and white or greenish watery mucoid diarrhoea. The post-mortem findings [5] are dominated by general septicaemic lesions including vascular disturbances, as reflected by general passive hyperemia and congestion throughout the carcass. Petechial and

ecchymotic haemorrhages are often present in the abdominal and coronary fat and haemorrhages may be observed in the intestinal mucosae and on subserosal surfaces in the thoracic and abdominal cavities. The liver and spleen are often swollen and may contain multiple small focal areas of coagulative necrosis or the organs may undergo more generalized necrosis. However, some unusual lesions are also noticed. In the present paper, incidence of fowl cholera in commercial layer with atypical lesions is detailed.

**History:** Sudden mortality was noticed in a commercial layer farm having 1500 layers of 17 weeks age. The daily mortality was around 0.1% and morbidity was around 5% for the period of 7 days. The farmer called the local veterinarians (ourselves) to the farm for treatment. The birds were reared in grower cages and previously vaccinated against New-castle disease, Infectious bronchitis, Infectious bursal disease, Fowl pox and Infectious coryza.

### Diagnosis of a Case Study and Discussion

**Clinical Signs and Postmortem:** Initially, the clinical signs exhibited in the birds were dullness, depression, ruffled feathers, paleness and petechial haemorrhages in comb (Fig. 1), blood tinged mucoid exudates in nostril.

The lesions recorded in the necropsy findings were blood tinged mucoid exudate in oral cavity and trachea (Fig. 2a and 2b), petechial haemorrhages and focal necrosis in breast muscle (Fig. 3a and 3b), generalized visceral congestion, multiple dark patchy necrotic areas in liver (Fig. 4a and 4b), focal necrosis and nodular growth over pancreas and epicardium ( Fig. 5a and 5b), reddened intestinal mucosa with greenish slimy content, vascular disturbance in kidney with marked congestion (Fig. 6a, 6b and 6c), lungs (Fig. 7) and gizzard (Fig. 8) showing hemorrhage and congestion. The various endotoxins [6] produced by the organism acting on the vascular endothelium may be the cause of haemorrhages in various organs and effusions into the serous cavities.



Fig. 1: Paleness and petechial haemorrhages in comb



Fig. 2a & 2b: Blood tinged mucoid exudate in oral cavity and trachea



Fig. 3a & 3b: Petechial haemorrhages and focal necrosis in breast muscle



Fig. 4a & 4b: Multiple dark patchy necrotic areas in liver,



Fig. 5a, 5b & 5c: Focal necrosis and nodular growth over epicardium and pancreas,

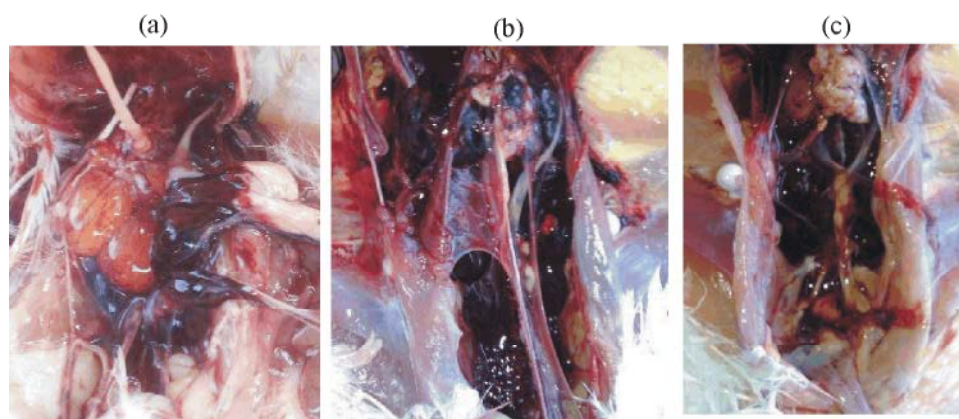


Fig. 6a, 6b & 6c: Vascular disturbance in kidney with marked congestion

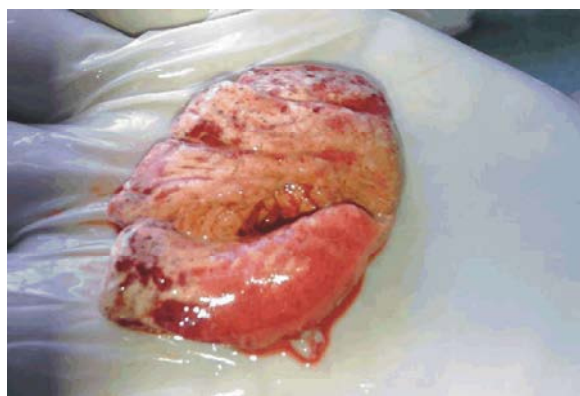


Fig. 7: Lungs showing haemorrhages and congestion



Fig. 8: Congestion over Gizzard

**Isolation and Identification of the Organism:** The vital organs such as heart, liver, spleen, kidney and bone marrow were collected aseptically in glycerol saline transport medium in individual containers. Impression smear from liver, kidney and heart were prepared and stained with gram's, methylene blue and Leishman's stains after fixing with ethanol. Bipolar organisms were well distinguished with Leishman stain. Swabs from the above samples were inoculated in Nutrient, XLD and Mac-Conkey's agar plates. The plates were incubated at 37°C for 24 hours. In the bacterial isolation, it was revealed as *Pasteurella multocida*. Identification of the organism was based on the staining reactions, colony morphology and absence of growth on Mac-Conkey's and XLD agar and biochemical tests [7].

**The Antibiotic Sensitivity Test (ABST):** The isolated colonies were subjected to antibiotic sensitivity test by disc diffusion on Muller – Hinton agar [8].

The Antibiotic sensitivity test (ABST) revealed that the isolate is sensitive to sulphadiazine, cotrimoxazole, ceftriaxone, chloramphenicol, gentamicin, amikacin and Amoxy clav. Antibiotics such as levofloxacin, neomycin, norfloxacin, ciprofloxacin and azithromycin showed intermediate results whereas Penicillin G, Ampicillin,

Furazolidone and Tetracycline were resistant. The results are in accordance with Shiva Chandra *et al.* [9] whereas sensitive patterns of chloramphenicol, gentamicin and tetracycline are against the results observed by Srinivasan *et al.* [10].

**Treatment:** As per the ABST results, the birds were treated with sulphadiazine with trimethoprim at the dose rate of 30mg/kg body weight through drinking water for 5 days along with water acidifier. The clinical signs in the morbid birds subside after 3 days of antibiotic treatment whereas mortality reduced after 5 days of therapeutic course. The farmer was advised to continue water acidifier through drinking water for next 7 days. No relapse of infection was noticed after the completion of antibiotic course.

### CONCLUSION

From this case study it was revealed that there are chances of fowl cholera incidence with atypical lesions which can be identified by isolation procedures and further screening by PCR techniques can help to isolate different strains.

### REFERENCES

1. Snipes K.P., G.Y. Ghazikhanian and D.C. Hirsh, 1987. Fate of *Pasteurella multocida* in the blood vascular system of turkeys following intravenous inoculation: comparison of an encapsulated, virulent strain with its avirulent, acapsular variant. *Avian Dis.*, 31: 254-259.
2. Bisgaard, M., W. Frederiksen, W. Mannheim and R. Mutters, 1994. Zoonoses caused by organisms classified with Pasteurellaceae. In *Handbook of zoonoses*, 2<sup>nd</sup> Ed. CRC Press, Boca Raton, pp: 203 - 208.
3. Harmon, B.G., J.R. Glisson, K.S. Latimer, W.L. Steffens and J.C. Nunnally, 1991. Resistance of *Pasteurella multocida* A: 3, 4 to phagocytosis by turkey macrophages and heterophils. *Am. Vet. Res.*, 52: 1507-1511.
4. Harmon, B.G., J.R. Glisson and J.C. Nunnally, 1992. Turkey macrophage and heterophil bactericidal activity against *Pasteurella multocida*. *Avian Dis.*, 36: 986-991.
5. Glisson, J.R., C.L. Hofacre and J.P. Christensen, 2008. Fowl cholera. In: *Diseases of poultry*, Saif YM, Barenes HJ, Glisson JR, Fadly AM, McDougald LR and Swayne DE (Editors). Blackwell Publishing, Ames, Iowa, USA, pp: 739-758.
6. Christensen J.P. and M. Bisgaard, 1997. Avian pasteurellosis: taxonomy of the organisms involved and aspects of pathogenesis. *Avian Pathol.*, 26: 461-483.
7. Rimler, R.B., T.S. Sandhu and J.R. Glisson, 1998. Pasteurellosis, Infectious Serositis and Pseudotuberculosis. In: *A laboratory Manual for the Isolation and Identification of Avian Pathogens*, 4<sup>th</sup> Edition, Swayne, D.E., J.R. Glasson and J.E. Pearson and W.M. Reed (Eds). Amer. Assoc. Avian Pathol., Pennsylvania, USA, pp: 17-28.
8. Quinn, P.J., M.E. Carter, B.K. Markey and G.R. Carter, 1994. *Pasteurella* sp. In *Clinical Veterinary Microbiology*. Wolfe Publishing, London, pp: 258.
9. Shiva Chandra S.B., A.A. Kumar, A. Biswas, M.A. Ramakrishnan, V.P. Singh and S.K. Srivastava, 2004. Antibiotic Sensitivity Patterns among Indian Strains of Avian *Pasteurella multocida*. *Trop Anim Health Prod*, 36: 743-750.
10. Srinivasan, P., T.R. Gopalakrishnamurthy, B. Mohan and S. Saravanan, 2011. Occurrence of sub acute fowl cholera in a broiler flock. *Tamilnadu J. Veterinary & Animal Sciences*, 7: 45-47.