

Clinicopathological and bacteriological investigation of chronic fowl cholera in commercial laying hens



Atria Destriana Fath¹, Armanda Dwi Prayugo^{2,*}, Popy Christin Ziliwu², Rocky Silvester², Sarasati Windria², Tyagita², Ita Krissanti², Faisal Amri Satrio²

¹ Veterinary Professional Education Program, Faculty of Medicine, Universitas Padjadjaran, Sumedang, Indonesia

² Department of Biomedical Sciences, Faculty of Medicine, Universitas Padjadjaran, Sumedang, Indonesia

ABSTRACT: Fowl cholera, caused by *Pasteurella multocida*, remains a major threat to poultry health and productivity. This case study identified the etiological agent associated with clinical disease and gross lesions in laying hens from a commercial farm in Sumedang, West Java, Indonesia. The investigations included necropsy, bacterial culture, Gram staining, and biochemical characterization. Affected birds showed torticollis, conjunctivitis, mild wattle and comb swelling, respiratory rales, reduced feed intake, decreased egg production, greenish-white diarrhea, and lethargy. Necropsy revealed hemorrhagic lesions in the trachea, lungs, and intestinal mucosa, with absent developing ovarian follicles. Culture on blood and chocolate agar produced whitish-gray translucent colonies. Gram staining showed Gram-negative coccobacilli singly or in pairs. Biochemical findings supported the identification of *P. multocida*. Overall findings were consistent with chronic fowl cholera and underscored the importance of vaccination, biosecurity, and early surveillance to limit economic losses in commercial layer farms.

Keywords:

bacteria, fowl cholera, gross pathology, laboratory diagnosis, laying hens, *Pasteurella multocida*

INTRODUCTION

Fowl cholera is a bacterial disease in domestic and wild birds caused by *Pasteurella multocida*. It occurs as peracute septicemia, acute systemic infection, or chronic disease, depending on susceptibility, virulence, and flock-level risk factors (Rhoades & Rimler 1991). In commercial poultry, it causes mortality, reduces productivity, and decreases egg production in layers. Transmission occurs through infected or carrier birds, contaminated water and feed, fomites, vectors, and reservoirs (Zainuddin 2014). Peracute disease may cause death without any signs. Acute infection causes depression, anorexia, respiratory distress, diarrhea, cyanosis, and hemorrhagic lesions. Indonesian cases include visceral hemorrhage and hepatic lesion.

Poultry diseases overlap; therefore, differential diagnosis is essential. Newcastle disease causes torticollis and hemorrhagic lesions (Napriila & Prasetyo 2022) but shows tracheal, proventricular, and cecal lesions. Infectious coryza causes facial, comb, and wattle swelling (Rawendra & Waluyo 2022); edema and thick nasal exudate are more characteristic than fowl cholera. Thus, diagnosis requires postmortem findings and laboratory identification. This case describes fowl cholera in laying hens in Sumedang, West Java, Indonesia.

CASE

This case occurred in a layer farm in Sumedang Regency, West Java Indonesia, with 4,500 birds aged 15 weeks to 2

years. None were vaccinated against fowl cholera. The hens showed torticollis, conjunctival redness, wattle and comb swelling, respiratory rales, reduced appetite, decreased egg production, diarrhea, lethargy, and activity.

Two affected hens were subjected to postmortem examination. Necropsy evaluated the pathological changes. Ancillary microbiological examination was performed using aseptically collected swab samples from the predilection sites, which were inoculated onto blood and chocolate agar for culture. Biochemical tests characterized the isolate and supported its identification.

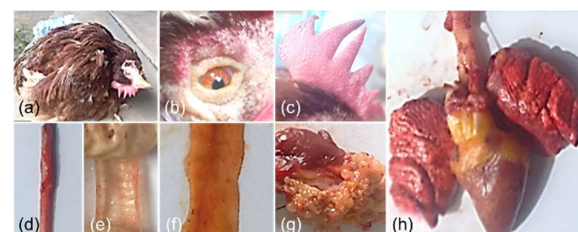


Figure 1. Gross lesions in laying hens with suspected chronic fowl cholera. Clinically observed were torticollis (a), conjunctivitis (b), and wattle swelling (c). Necropsy revealed tracheal hemorrhage (d-e), small intestinal hemorrhage (f), absent ovarian follicles (g), and pulmonary hemorrhage (h).

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■ RESULTS AND DISCUSSION

Gross necropsy revealed lesions in the affected laying hens: torticollis, conjunctivitis, wattle swelling, and hemorrhage in the trachea, lungs, and small intestinal mucosa (Figure 1). These findings were consistent with chronic fowl cholera, as chronic *P. multocida* infection may cause lesions in the respiratory tract, conjunctivae, wattles, and nervous system-associated structures (Rhoades & Rimler 1991). No developing ovarian follicles were observed, suggesting impaired reproductive activity and systemic disease. Hemorrhagic lesions reflected the septicemic and vascular nature of pasteurellosis; vascular injury and hemorrhage are key features of avian pasteurellosis (Rhoades & Rimler 1991). Similar hemorrhage in broiler chickens with fowl cholera in Banda Aceh, Indonesia (Zainuddin 2014) supports systemic *P. multocida* involvement in this case.

Newcastle disease was considered because torticollis and intestinal hemorrhage may occur in different layers (Napriila & Prasetyo 2022). However, cases often show hemorrhagic lesions in the trachea, proventriculus, and cecum of the infected birds. In this case, hemorrhage was mainly observed in the trachea, lungs, and small intestinal mucosa, without predominant proventricular or cecal tonsil involvement. Infectious coryza was suspected because of wattle swelling. However, it typically causes facial, comb, and wattle swelling and nasal exudates (Rawendra & Waluyo 2022). This case showed mild wattle swelling and lacked facial edema, which supports the diagnosis of Fowl cholera.

Microbiological examination supported the diagnosis. Swabs from predilection organs produced colonies on blood and chocolate agar, consistent with *P. multocida* in avian pasteurellosis (Belal 2013). Gram staining showed gram-negative coccobacilli singly or in pairs, consistent with *P. multocida* (Belal 2013). Kligler Iron Agar showed glucose fermentation without gas and no acid from lactose or sucrose. Motility, Indol, and Urea showed indole positivity, urease negativity, and non-motility, and citrate testing was negative (Figure 2). These findings matched biochemical profile of *P. multocida* (Belal 2013). Although phenotypic tests alone cannot confirm molecular identification, morphology, biochemical results, and lesions supported *P. multocida* as the etiologic agent.

Virulence mechanisms of *P. multocida* explain the lesions. The capsule aids survival and defense evasion (Wilkie *et al.* 2012). Lipopolysaccharide causes inflammatory and vascular injury (Wilkie *et al.* 2012). Outer membrane proteins mediate adhesion, immune evasion, and persistence (Hatfaludi *et al.* 2010). These structures explain vascular damage, hemorrhage, and respiratory lesions. Tracheal and lung involvement was consistent with tropism and systemic spread in avian pasteurellosis (Rhoades & Rimler 1991). Based on signs, necropsy findings, morphology, and biochemical profile, case was interpreted as chronic fowl cholera caused by phenotypically identified *P. multocida*.

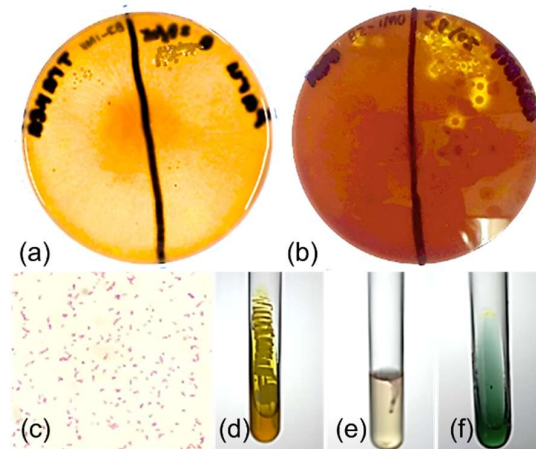


Figure 2. Microbiological examination of bacterial isolate from laying hens with suspected chronic fowl cholera. Culture growth on blood agar (a) and chocolate agar (b), Gram-negative coccobacilli on Gram staining (c), and biochemical profiles supported phenotypic identification of *Pasteurella multocida*.

■ CONCLUSION

Hens had lesions consistent with chronic fowl cholera. Diagnosis was supported by torticollis, conjunctivitis, wattle swelling, gram-negative coccobacilli, and *Pasteurella multocida*-compatible biochemistry. Vaccination, biosecurity, carrier removal, and diagnosis are recommended.

■ AUTHOR INFORMATION

Corresponding Author

*ADP: armanda@unpad.ac.id

Department of Biomedical Sciences, Faculty of Medicine, Universitas Padjadjaran, Jl. Ir. Soekarno Km. 21, Hegarmanah, Jatinangor, Sumedang, West Java, 45363 INDONESIA.

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