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Co-occurrence of infectious bursal disease and multidrug-resistant *Salmonella* infection in a vaccinated flock of three-week old pullets: A case report

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Abstract

This report presents a case of co-occurrence of infectious bursal disease (IBD) and infection with multidrug-resistant (MDR) *Salmonella enterica* subsp. *enterica* in a flock of 487 three-week old ISA Brown pullets in Maiduguri, Nigeria. The flock had a history of having been previously vaccinated against IBD using a foreign-sourced vaccine. The outbreak was marked by acute lethargy, anorexia, 100% morbidity and 63.9% mortality. Post-mortem examination of carcasses of the dead chicks and histopathological examination of tissues obtained at necropsy revealed enlarged, hemorrhagic bursae with severe lymphoid depletion, renal pathology and widespread vascular lesions. Virological assays confirmed IBD virus antigen and high IBD virus antibody titre, while bacteriological culture identified MDR *Salmonella* strains resistant to tetracycline, sulfonamides, ampicillin and enrofloxacin, but which were susceptible only to ciprofloxacin and gentamicin. Clinical recovery of the surviving chicks was achieved following ciprofloxacin therapy. This case portrays the complex interplay between a viral disease and opportunistic bacterial infections in poultry, particularly under inadequate biosecurity and questionable vaccination history. It further highlights the escalating threat of antimicrobial resistance and its implications for poultry health, food safety, and zoonotic diseases transmission. Based on the findings in this case report, we advocate for strengthened vaccine quality control, improved farm-level biosecurity and integrated One Health approach to disease surveillance and antimicrobial stewardship.

Keywords: Infectious bursal disease; Multidrug-resistant *Salmonella* infection; Vaccination failure; Biosecurity; One Health.

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Introduction

Infectious bursal disease (IBD), also known as Gumboro disease, is a highly contagious and immunosuppressive viral disease of poultry that primarily affects young chickens, especially those between three to six weeks of age (Dey *et al*., 2019; Franciosini and Davidson, 2022). The disease is caused by the infectious bursal disease virus (IBDV), a double-stranded RNA virus of the *Birnaviridae* family. IBD is a significant cause for concern to the poultry industry globally, due to its profound economic impact, which includes high mortality, growth retardation and predisposition to secondary bacterial infections (Zhang *et al*., 2022; Aliyu *et al*., 2022; Nasser *et al*., 2024). Despite the widespread implementation of vaccination programmes against the disease, outbreaks continue to occur, often with atypical or subclinical presentations that compromise the bird's immune competence, particularly affecting the B-lymphocyte-rich bursa of Fabricius (Dey *et al*., 2019; Aliyu *et al*., 2021; Audu *et al*., 2023).

The immunosuppression associated with IBD is well-documented in poultry, particularly due to its role in increasing susceptibility to secondary and opportunistic bacterial infections with organisms such as *Escherichia coli*, *Staphylococcus* spp. and *Salmonella* spp. (Fancher *et al*., 2020; Aliyu *et al*., 2024; Kovács *et al*., 2025). Such co-infections not only exacerbate clinical outcomes but also complicate diagnosis and therapeutic response. In particular, IBD-induced lymphoid tissue atrophy reduces the host's ability to mount effective humoral and cellular immune responses, facilitating the colonization and systemic dissemination of bacterial pathogens. This immune compromise also potentially accelerates the selection pressure for antimicrobial resistance due to prolonged and repeated antibiotic interventions (Lawal *et al*., 2017; Li *et al*., 2018; Dey *et al*., 2019).

Salmonella infections, particularly those caused by non-typhoidal serovars such as *Salmonella enterica* subspecies *enterica*, remain a critical zoonotic threat and a source of considerable morbidity and mortality in poultry (Lamichhane *et al*., 2024; Kumar *et al*., 2025). These infections, especially when due to multidrug-resistant (MDR) strains, pose serious food safety and public health challenges, complicating therapeutic management in both veterinary and human medicine (Teklemariam *et al*., 2023; Sahu *et al*., 2025). The increasing prevalence of MDR *Salmonella* in poultry has been associated with indiscriminate antibiotic usage, poor biosecurity, and the immunosuppressive consequences of concurrent infections, particularly viral diseases (Castro-Vargas *et al*., 2020; Shaji *et al*., 2023; Aliyu *et al*., 2024).

Despite extensive literature on IBD and *Salmonella* infections as individual entities, reports on their co-occurrence in vaccinated pullet flocks are scarce, particularly within the critical early stages of pullet growth. Even rarer are detailed case studies that explore the interaction between IBD-induced immunosuppression and multidrug-resistant bacterial infections in the same clinical setting. The identification of such dual infections in young, vaccinated pullets highlights important gaps in vaccine efficacy, disease monitoring, and antimicrobial stewardship.

This case report describes a unique and clinically significant co-occurrence of infectious bursal disease and multidrug-resistant *Salmonella* infection in a flock of three-week old pullets in Maiduguri, Northeastern Nigeria. The chicks, although reportedly vaccinated against IBD, manifested classic signs of the disease, coupled with systemic salmonellosis that was not responsive to treatment with commonly used antimicrobials. This dual-pathogen scenario emphasizes the complex interplay between viral immunosuppression and antimicrobial resistance (AMR) in poultry, emphasizing the

need for integrative diagnostic approaches, enhanced surveillance and more judicious antimicrobial use. Additionally, it raises critical questions about the field efficacy of commercially available IBD vaccines, especially in regions burdened with suboptimal farm management practices, biosecurity lapses and high level of misuse of antibiotics.

Case Presentation

On July 20, 2024, a team of poultry clinicians and veterinary consultants from the University of Maiduguri Veterinary Teaching Hospital (UMVTH) conducted a routine inspection at a backyard poultry farm located within the University Staff Quarters, Maiduguri, Borno State, Nigeria. The farm visit aimed to evaluate flock health and certify readiness for an upcoming oral vaccination exercise scheduled in the subsequent week. Upon arrival, the team observed alarming clinical signs in a flock of 491 birds comprising 487 ISA Brown pullets and 4 Cornish Cross broilers – all aged three weeks. Affected birds presented with marked dullness, inappetence, prostration, lethargy, huddling and severe weakness.

The vaccination history revealed that the flock had received a single dose of a foreign-sourced infectious bursal disease (IBD) vaccine five days prior to the visit (i.e., at two weeks of age). The vaccine was reportedly a 500-dose vial, but specifics regarding its manufacturer, strain type and method of administration were not provided.

Environmental assessment of the farm revealed multiple concerning risk factors. The poultry farm operated a mixed-species, semi-intensive management system, housing other avian species including 55 indigenous/local chickens, 25 adult Noiler chickens, 42 multi-aged mixed-breed domestic pigeons, 180 seven-week-old broilers, and several waterfowls (number not definitive), all within the farm in close proximity, but in separate pens. The specific pen housing the affected pullets was structurally suboptimal; it was constructed from galvanized roofing sheets with wire mesh fronting, providing inadequate cross-ventilation. Despite the presence of footbaths at the entrance, they were evidently not in use, and other critical biosecurity practices were grossly inadequate or entirely absent.

Within 96 hours of the inspection, the flock experienced a morbidity rate of 100% and a cumulative mortality rate of 63.9% (311 deaths). The rapid onset of clinical signs following recent IBD vaccination in conjunction with significant mortality prompted further investigation.

Freshly dead and moribund birds were promptly subjected to detailed necropsy under aseptic conditions, with gross lesions carefully documented and lesion scoring applied to assess the severity and distribution of organ involvement for epidemiological analysis. An on-site farm-level epidemiological and biosecurity assessment was conducted using a standardized checklist, capturing data on chick source and history, vaccination records, particularly the strain and method of infectious bursal disease (IBD) vaccine administration, biosecurity protocols, feeding and watering systems, litter management, cohabitation with other species. Possible environmental stressors such as ventilation adequacy, ambient temperature fluctuations, and stocking density were also considered.

Tissue samples including the bursa of Fabricius, spleen, liver, and kidneys were aseptically collected; portions were fixed in 10% buffered formalin for histopathological examination, while parallel samples were processed for bacteriological culture. Blood samples were obtained via the jugular vein, and serum was separated and stored at -20 °C for subsequent serological testing. Histopathological analysis focused on lymphoid tissues, particularly the bursa, while the spleen and liver were examined for lesions

indicative of systemic bacterial infection. The liver, spleen and intestinal contents were subjected to bacteriological culture, and bacterial isolates were subjected to antimicrobial susceptibility testing.

Clinical Findings

The clinical course of the disease outbreak was characterized by a sudden onset and rapid progression within 48 to 96 hours. Affected birds exhibited marked morbidity and mortality, with clinical signs highly suggestive of infectious bursal disease (IBD). Notable among these were somnolence and pronounced huddling behavior (Figure 1), consistent with systemic malaise and a possible febrile response. Feathers appeared diffusely ruffled, and most birds demonstrated a visibly poor body condition. A prominent feature in over 70% of symptomatic birds was severe pasting of the vent, with peri-cloacal feathers stained by profuse creamy to whitish-brown watery droppings, indicative of enteric disturbance. Additional clinical signs included marked lethargy, depression, and reluctance to move, frequently terminating in sudden death.

Figure 1. Clinical signs of huddling together of pullets with co-occurring infectious bursal disease and salmonellosis.

The cumulative mortality reached up to 63.9% (311 out of 487) within four days (Figure 2). The pattern of clinical signs and the mortality profile strongly implicated an immunosuppressive viral etiology, with infectious bursal disease virus (IBDV) being the principal suspect.

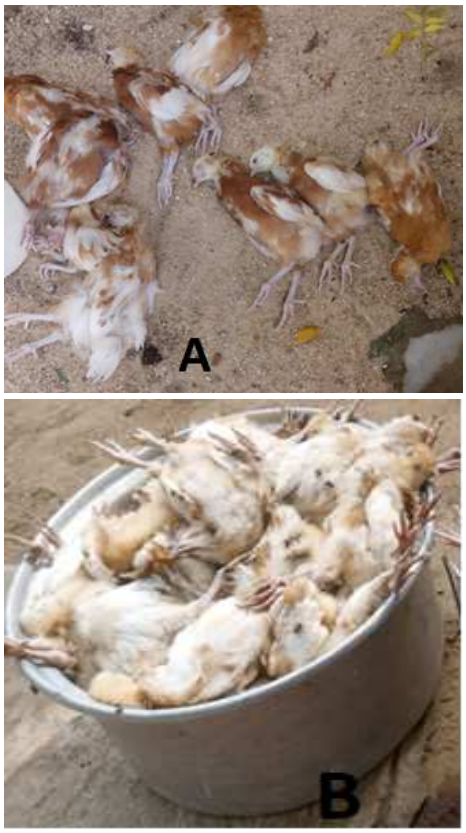

Figure 2. High mortality of pullets with a co-occurring infectious bursal disease and salmonellosis. Top (A) – dead pullets on the floor (litter) of the farm, and below it (B) – a metal container full of dead carcasses of the pullets.

Postmortem Findings

Necropsy of representative carcasses revealed multifocal and systemic gross lesions suggestive of both viral and bacterial etiologies. One of the most conspicuous findings was marked enlargement of the thymic lobes, an atypical observation indicative of early reactive lymphoid hyperplasia, possibly associated with secondary bacterial infection such as salmonellosis.

Striking petechial to ecchymotic hemorrhages were evident on the pectoral and thigh musculature (Figures 3a and 3b), lesions commonly associated with acute vasculitis induced by very virulent strains of infectious bursal disease virus. At the proventriculus-ventriculus junction, pronounced petechiae were observed (Figure 4a), a lesion often attributed to vascular compromise linked to severe IBD or systemic bacterial dissemination. The lungs appeared diffusely congested with focal areas of oedema and a marbled appearance of some lobes, suggesting concurrent bacterial septicemia and pneumonia (Figure 4b).

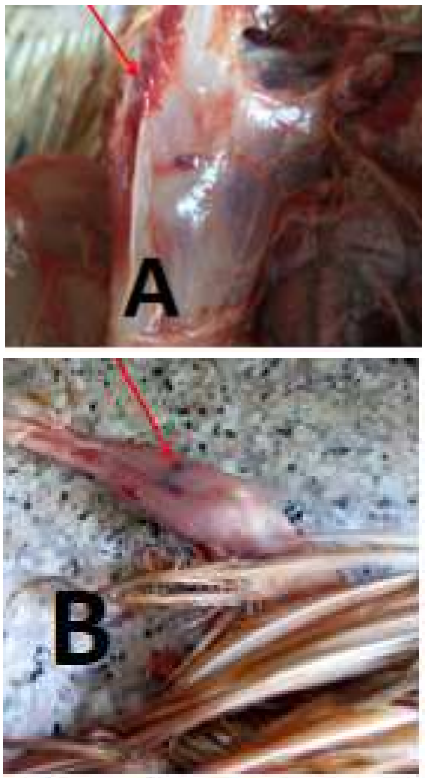

Figure 3. Haemorrhages on the pectoral (A) and thigh (B) muscles of pullets with co-occurring infectious bursal disease and salmonellosis.

The bursa of Fabricius was grossly enlarged, oedematous and hemorrhagic, with yellowish exudates exuding from the lumen upon incision (Figures 5a and 5b), features considered pathognomonic for IBD. Additionally, some bursae exhibited widespread hemorrhages involving the entire bursal wall (Figure 6). Histopathological examination confirmed significant lymphoid depletion, follicular necrosis and cystic cavitation within the medullary zones of bursal follicles (Figure 7).

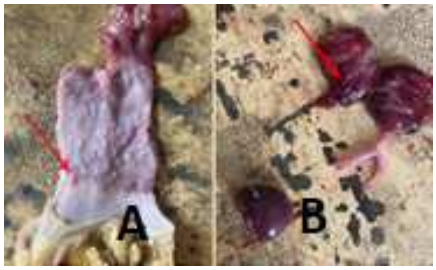

Figure 4. Haemorrhages at the proventriculus-ventriculus junction (red arrow in A) and congestion of the lungs (red arrow in B) and spleen (blue arrow in B) of pullets with co-occurring infectious bursal disease and salmonellosis.

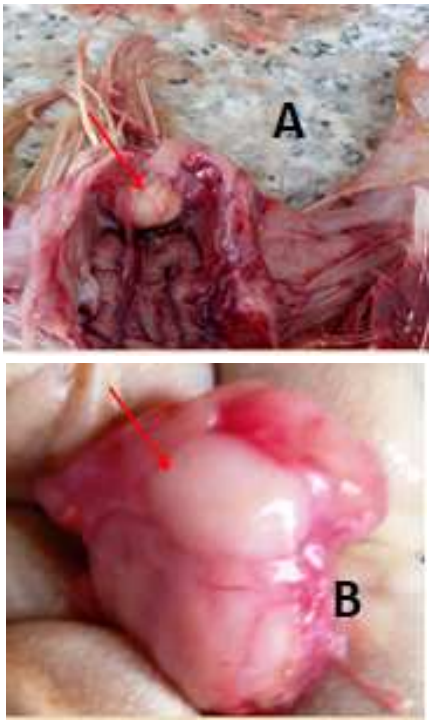

Figure 5. Grossly enlarged bursa of Fabricius (A) with creamy exudate exuding from the lumen of the bursa (B) of pullets with co-occurring infectious bursal disease and salmonellosis.

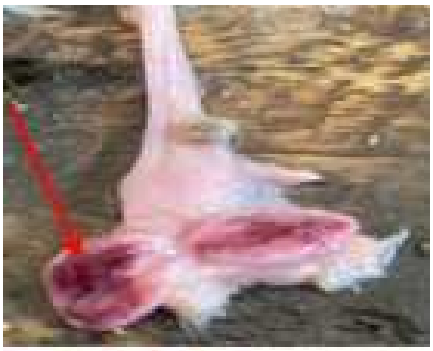

Figure 6. Widespread haemorrhages involving the inner wall of the bursa of Fabricius of pullets with co-occurring infectious bursal disease and salmonellosis.

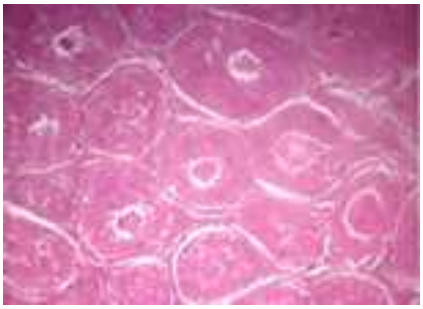

Figure 7. Cystic cavitation in the medullary region of the bursal follicles of pullets with co-occurring infectious bursal disease and salmonellosis [H & E, ×10].

Figure 8. Congested kidneys with urate deposits on the kidney in pullets with co-occurring infectious bursal disease and salmonellosis.

Renal lesions included generalized pallor, congestion and marked urate deposition within the renal tubules (Figure 8), indicative of nephrosis and impaired renal excretory function. Histological sections of the kidney further revealed dilated/expanded Bowman's space and necrosis of renal tubular epithelium (Figure 9), suggestive of either endotoxaemic nephropathy due to salmonellosis or uric acid nephropathy linked to dehydration.

Figure 9. Dilated/expanded Bowman's space and necrosis of renal tubular epithelium of pullets with co-occurring infectious bursal disease and salmonellosis [H & E, ×10].

Differential Diagnosis

The differential diagnosis was developed based on clinical signs, post-mortem lesions, epidemiology and vaccination history. The high mortality in three-week old pullets previously vaccinated against infectious bursal disease virus (IBDV) prompted careful evaluation of possible causes. Infectious bursal disease (IBD) was strongly suspected when the age of the affected birds was considered, and also when clinical signs such as acute depression, ruffled feathers, prostration and rapid death were taken into consideration. Lesions such as enlarged, oedematous and hemorrhagic bursae, splenic congestion and nephrosis suggested a virulent IBDV strain aetiology, despite the reported vaccination of the birds against the IBDV. Newcastle Disease (ND) was ruled out despite being endemic, as typical respiratory signs (gasping, rales) and neurological symptoms (tremors, torticollis, paralysis) were absent. Colibacillosis was

excluded due to the absence of hallmark lesions such as pericarditis, perihepatitis, air sacculitis and fibrinous organ exudates. Histopathology did not support *E. coli* involvement. Chicken infectious anemia (CIA) was considered but dismissed, because of its chronic, immunosuppressive nature, which differs from the acute course observed. Also, CIA typically causes pancytopenia, subclinical immunosuppression and aplastic marrow in younger chicks, not severe bursal lesions. This process of exclusion supported a tentative diagnosis of IBD, pending laboratory confirmation.

Tentative Diagnosis

Taking into account the clinical presentation, sudden onset of high flock mortality, characteristic gross lesions of the bursa of Fabricius, renal pathology, and the flock's recent IBD vaccination history, a tentative diagnosis of infectious bursal disease (IBD) was established. However, the severity of lesions despite prior vaccination raised suspicion for possible co-infection with enteric bacterial agents, warranting further laboratory investigations.

To validate the clinical suspicion of infectious bursal disease (IBD) and investigate the possibility of concurrent bacterial infections, a comprehensive diagnostic work-up was initiated. A suite of clinical and post-mortem samples was systematically collected from affected birds at peak morbidity. These included aseptically obtained whole blood, cloacal swabs and freshly excised tissues – specifically the bursa of Fabricius, intestine, spleen, liver, and kidneys. Each sample was strategically selected to represent both systemic and organ-specific pathogen reservoirs. The specimens were processed and distributed to relevant laboratories for microbiological, serological and histopathological analyses. This multi-pronged approach was aimed at delineating the aetiologic complexity of the disease outbreak.

Microbiology Laboratory Analysis and Findings

Bacterial Isolation and Characterization: Microbiological analysis was conducted to identify systemic bacterial pathogens potentially contributing to morbidity and mortality. Samples comprising whole blood, cloacal swabs, and tissue homogenates were cultured using standard protocols. Primary inoculations were made on MacConkey agar and Xylose lysine deoxycholate (XLD) agar and incubated aerobically at 37°C for 24 – 48 hours. Non-lactose fermenting colonies with black centres indicative of hydrogen sulfide production were presumptively identified as *Salmonella* spp.

Stepwise Isolation and Confirmation of *Salmonella* spp.: A classical stepwise protocol was employed for the isolation and confirmation of *Salmonella* spp. Initially, samples underwent pre-enrichment in buffered peptone water (BPW) at 37°C for 18 – 24 hours. This was followed by selective enrichment in Rappaport-Vassiliadis (RV) broth and Selenite F broth, incubated at 42°C and 37°C, respectively, for 24 hours. Post-enrichment cultures were streaked onto xylose lysine deoxycholate (XLD) agar, brilliant green agar (BGA), and Hektoen enteric (HE) agar. Plates were incubated at 37°C for 18 – 24 hours. Colonies with morphology typical of *Salmonella* were identified: red colonies with black centres (XLD), pink-white colonies on a red background (BGA), and blue-green colonies with black centres (HE).

Presumptive *Salmonella* isolates were confirmed by Gram staining. Gram-negative rods were observed, and the isolates were subjected to a comprehensive panel of biochemical tests. On triple sugar iron (TSI) agar, isolates exhibited an alkaline slant with an acid butt (red/yellow), hydrogen sulfide

production (black precipitate) and gas formation. Additional biochemical reactions and their results included citrate utilization (positive), urease (negative), indole (negative), methyl red (positive), Voges-Proskauer (negative), lysine decarboxylase (positive) and motility (positive). These results were consistent with *Salmonella enterica* subsp. *enterica*, a zoonotic pathogen of significant public health relevance in poultry

Antimicrobial Susceptibility Profiling: To assess therapeutic options and potential public health risk, antimicrobial susceptibility testing was conducted on the *Salmonella* isolates using the Kirby-Bauer disk diffusion method, following Clinical and Laboratory Standards Institute (CLSI) guidelines. The isolate demonstrated multi-drug resistance (MDR) to several commonly used antibiotics, including tetracycline, ampicillin, sulfonamides and enrofloxacin, but was susceptible to ciprofloxacin and gentamicin.

Serological Investigations: To evaluate the immune status of the flock and assess exposure to IBDV, blood samples were collected from 30 clinically affected pullets and 15 asymptomatic counterparts. In severely moribund birds with collapsed veins, blood was pooled from 2 – 4 birds to obtain sufficient volume. Blood was gently drawn into plain vacutainer tubes, allowed to clot, and centrifuged to harvest clear serum. Serological screening was performed using a commercial enzyme-linked immunosorbent assay (ELISA) test kit specific for IBDV antibodies (IDEXX IBD Ab Test Kit, IDEXX Laboratories, Inc., Westbrook, Maine, USA). Symptomatic birds demonstrated markedly elevated antibody titres (mean: 6,200), while control birds showed significantly lower levels (mean: 1,200), indicating seroconversion consistent with a recent or ongoing infection. This immunoprofile suggested a possible vaccination failure, potentially due to infection with a very virulent IBDV strain, which may have circumvented the protective threshold conferred by the administered vaccine.

Virological Confirmation: Complementary virological assays were carried out on bursal tissue samples to confirm the presence of active IBDV antigen. The agar gel immunodiffusion (AGID) test was employed using known IBDV-specific antiserum. Strong white precipitin lines (Figure 10) in the agar gel matrix confirmed the presence of viral antigen in the affected tissues, reinforcing the diagnosis of IBD. Furthermore, the passive haemagglutination assay (PHA) conducted on the serum samples also revealed high titres of antibodies against IBDV, further substantiating recent infection and active immune response in the affected birds.

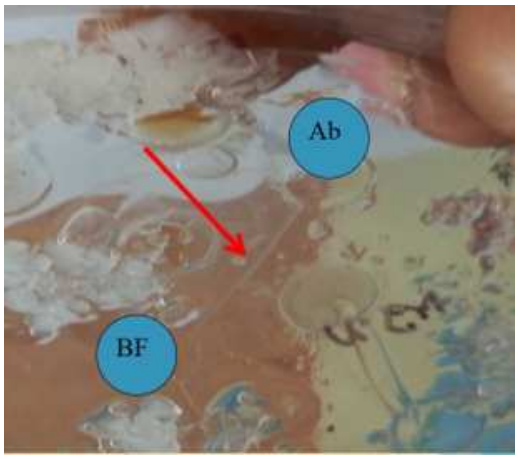

Figure 10. Strong white precipitin line (red arrow) on the agar gel matrix, which confirmed the presence of IBD viral antigen in the affected tissues, during the agar gel immunodiffusion (AGID) test.

Confirmatory Diagnosis

Diagnosis was confirmed based on serological, microbiological and pathological findings. The convergence of clinical signs and laboratory findings established a definitive diagnosis of concurrent infectious bursal disease and multidrug-resistant *Salmonella* infection in the pullet flock.

Management

Immediate Stabilization and Supportive Care: Following the tentative clinical diagnosis of infectious bursal disease (IBD), and in anticipation of laboratory confirmation, an immediate multi-faceted intervention was deployed to stabilize the flock, reduce mortality, and mitigate the adverse systemic effects of the suspected immunosuppressive viral infection. Supportive therapy was initiated with a dual objective of addressing the dehydration and energy deficits associated with acute viral enteropathy, and to modulate stress-induced immunosuppression pending aetiological confirmation. An oral rehydration-electrolyte-energy-amino acid supplement combination therapy was administered (Vitaflash®, MegaVet, Tbilisi, Georgia) at 5 g per 4 litres of drinking water and Dextrose-D at 50 g per 10 litres were provided ad libitum for five consecutive days. This regimen was intended to correct fluid and electrolyte imbalances, prevent hypoglycemia, and provide readily metabolizable energy substrates to sustain metabolic function during peak morbidity.

Reinforcement of Farm-Level Biosecurity Protocols: Strict biosecurity measures were escalated to stop/contain horizontal transmission of both the viral and potential bacterial pathogens. Movement of personnel and materials into and out of the infected poultry unit was restricted, and designated entry protocols were enforced. All equipment were disinfected daily using quaternary ammonium-based disinfectants, while footbaths were refreshed every six hours. Carcasses of dead birds were promptly retrieved, double-bagged, and incinerated at a controlled temperature to reduce environmental contamination with infectious pathogens. Litter management protocols were enhanced by applying hydrated lime to suppress microbial load and ammonia volatilization. In addition, strict inter-species segregation was implemented to prevent cross-species transmission of pathogens amongst the different species of birds reared on the farm premises. The use of physical barriers and separate equipment for each species was implemented.

Targeted antimicrobial therapy and stewardship (post-laboratory confirmation of diagnosis): Following microbiological confirmation of *Salmonella enterica* subsp. *enterica* co-infection, and guided by antibiogram results revealing multidrug resistance (MDR) to tetracycline, ampicillin, sulfonamides and enrofloxacin, a rational antimicrobial therapy was instituted, as the isolates were only susceptible to ciprofloxacin and gentamicin. Considering the practical limitations of administering injectable gentamicin in a flock-level outbreak and the lack of water-soluble gentamicin monotherapy in the local veterinary pharmacopeia, ciprofloxacin was chosen as the therapeutic agent of choice.

A commercially available veterinary-grade oral ciprofloxacin preparation (CIPROCIN ASPCO®, Kepro B.V., Netherlands) was administered at 100 ml per 200 litres of drinking water, continuously for 3 to 5 days. This broad-spectrum fluoroquinolone was selected not only based on sensitivity results but also due to its favorable pharmacokinetics, including high tissue penetration and efficacy in systemic salmonellosis.

Monitoring and Follow-Up: Daily farm visits were conducted to monitor clinical response to therapy, and further morbidity and mortality recorded to evaluate treatment efficacy. Notable improvements, including normalized behavior, increased feed and water intake, and cessation of new mortalities were recorded within 72 hours to seven days post-intervention. Survivors continued to show normal growth and demeanor (Figure 10).

Figure 11. Pullets that survived co-occurring infectious bursal disease and salmonellosis after treatment, showing normal activity and demeanor.

Discussion

The clinical signs observed in this study, with regards to IBD and salmonellosis, align with findings from previous reports (Zannah *et al*., 2020; Ekiri *et al*., 2021; Omer and Khalafalla, 2022). Infected chickens typically show signs such as lethargy, whitish diarrhoea, soiled vent feathers and body tremors. The observed mortality pattern in the affected flock exhibited a classical bell-shaped distribution, which is characteristic of an outbreak of infectious bursal disease (IBD). This trend commenced with a gradual rise in mortality shortly after the emergence of clinical signs, reaching a peak between days 3 and 5 post-onset, followed by a progressive decline in mortality rates, typically tapering off by days 7 to 8.

The gross and histopathological lesions recorded in this case, including bursal enlargement, edema, lymphoid depletion and follicular necrosis, are consistent with classical IBD-induced lymphocytolysis (Dey *et al*., 2019; Damairia *et al*., 2023). The bursa of Fabricius, a central lymphoid organ in young birds, was profoundly affected, compromising both humoral and cell-mediated immunity. The occasional thymic enlargement observed in this case/outbreak may represent a compensatory response or secondary systemic involvement, reflecting lymphoid cross-talk under immunologic stress (Szõcs *et al*., 2024). The observed gross lesions of haemorrhages on pectoral and thigh muscles, band haemorrhages at the proventriculus-ventriculus junction, pulmonary and splenic congestion and a swollen bursa of Fabricius exuding creamy material, are classical pathological features of the virulent form of IBD. Haemorrhages on skeletal muscles, particularly in the thigh and breast (pectoral) muscles, have long been recognized as hallmark lesions of acute IBD, associated with endothelial damage and disseminated intravascular coagulation caused by the virus (Ingrao *et al*., 2013). Likewise, the prominent hemorrhagic band at the proventricular-ventricular junction is considered pathognomonic of highly virulent IBD virus infection (Singh *et al*., 2015; Mahajan *et al*., 2022), reflecting severe vascular injury within gastrointestinal lymphoid aggregates. The pulmonary and splenic congestion observed in this case may have resulted from systemic inflammatory responses triggered by the viral infection or by concurrent *Salmonella* bacteremia. The spleen, being a primary lymphoid organ, is often congested or enlarged due to lymphoid depletion or secondary bacterial colonization during IBD (Lewis *et al*., 2019). Additionally, the lungs are susceptible to congestion and edema due to circulatory compromise, which may be exacerbated by endotoxins produced by *Salmonella* species.

The most notable lesion (swelling of the bursa of Fabricius with accumulation of creamy exudate) strongly suggests an acute IBDV infection. The bursa, as the primary site of viral replication, undergoes marked inflammation, oedema and lymphoid necrosis during the early stages of IBD, especially in chicks of three to six weeks of age, which are the most susceptible age group (Sharma *et al*. 2000). The presence of creamy discharge suggests secondary bacterial invasion, most

likely by *Salmonella* spp., facilitated by bursal epithelial damage.

Despite prior IBD vaccination, the manifestation of classic and severe lesions in this flock points to either vaccine failure or infection with a highly virulent field strain. Vaccine failure may arise from improper administration, suboptimal vaccine storage, interference by maternal-derived antibodies, or mismatch between vaccine and circulating field strains (Jakka *et al*., 2014).

The concurrent isolation of multidrug-resistant *Salmonella* underscores the probable role of IBD-induced immunosuppression in predisposing birds to secondary bacterial infections. *Salmonella enterica*, especially serovars such as *Typhimurium* and *Enteritidis*, are known to act opportunistically in immunocompromised hosts (Mahajan *et al*., 2002; Lamichhane *et al*., 2024). The co-infection likely worsened the clinical course, increasing morbidity and mortality.

The isolation of *Salmonella* spp., along with histological evidence of septicemia, renal tubular necrosis and hepatic involvement, points to systemic bacterial dissemination. IBDV-induced immunosuppression reportedly creates a permissive environment for secondary bacterial colonization and haematogenous spread, which intensifies clinical outcomes (Worley, 2023; Lamichhane *et al*., 2024). Notably, *Salmonella* enterica was isolated from blood, liver, spleen, and intestinal contents, illustrating systemic invasion in immune-compromised birds (Worley 2023). These findings align with reports of previous studies where immune compromise by viral agents enhances susceptibility to opportunistic bacteria (Sharma *et al*., 2000). Clinically, the presence of signs such as diarrhoea, lethargy and soiled vents, accompanied by post-mortem findings including petechial haemorrhages, renal degeneration and urate accumulation, are characteristic of acute salmonellosis. These lesions are likely exacerbated by systemic endotoxemia and dehydration, in agreement with the observations of Shaji *et al*. (2023).

Antimicrobial susceptibility profiling of the *Salmonella* isolate in this report revealed multidrug resistance, against tetracycline, ampicillin and sulfonamides, and susceptibility only to ciprofloxacin and gentamicin. This resistance pattern brings to the fore the therapeutic limitations posed by multidrug-resistant (MDR) strains and reflects a trend to be concerned about in poultry pathogens, as similarly reported by Mthembu *et al*. (2019), Dagnew *et al*. (2020), Peruzy *et al*. (2020), Shang *et al*. (2023), Korkoil *et al*. (2024), and Moraes *et al*. (2024)

In the present case report, a central concern in the outbreak was the apparent failure of the infectious bursal disease (IBD) vaccination given to the flock. Despite the documented administration of the IBD vaccine, the pullets exhibited clinical signs and bursal lesions that were pathognomonic for infection with very virulent IBD virus. The presence of elevated antibody titres, together with severe bursal pathology, strongly indicates a vaccination failure. Such failures may possibly be attributed to several factors, including antigenic mismatch between the vaccine and circulating field strains, inadequate vaccine potency, compromised cold chain integrity, or suboptimal administration practices (Aliyu *et al*., 2016). In addition, the timing of IBD vaccination is also critical, as these challenges are often exacerbated by improper vaccination scheduling. Administering to birds IBD vaccine at around two weeks of age often coincides with the decline of maternally derived antibodies (MDAs), which can interfere with immune priming. If vaccine administration occurs when MDAs are still at inhibitory levels, they may neutralize the vaccine virus, thereby preventing effective seroconversion and long-term protection. Consequently, precise scheduling of IBD vaccination is essential to avoid both

immunization failure and/or potential vaccine-induced immunosuppression (Aliyu *et al*., 2016).

The high mortality recorded in this outbreak, despite vaccination, may possibly also reflect a failure of the vaccine to confer adequate protection against the circulating field strain of IBDV. It is possible that the intermediate-type vaccine used at two weeks was neutralized by residual MDAs. Furthermore, the possibility of using a poorly attenuated vaccine strain, or one that was not adequately matched to the prevalent field strain, cannot be ruled out. Such a scenario may result in insufficient protection or even pathogenicity, thereby predisposing the birds to secondary infections such as multidrug-resistant *Salmonella*, as observed in this case.

Previous studies have demonstrated that high levels of MDAs at the time of IBD vaccination can impair the host's immune response by neutralizing the vaccine virus, thus delaying or completely preventing the development of humoral immunity (Bhuiyan *et al*., 2021; Lawal and Bello, 2021; Opoku *et al*., 2022; Ramon *et al*., 2022). Additionally, virulent field strains of IBDV that belong to the same serotype as the vaccine strain but differ antigenically have been shown to overcome high MDA levels in commercial flocks, resulting in significant mortalities ranging from 60% to 70% (Dey *et al*., 2019; Shegu *et al*., 2020; Lawal and Bello, 2021; Nasser *et al*., 2024). While vaccination remains the cornerstone of IBD prevention and control in young poultry (Lawal and Bello, 2021; Aliyu *et al*., 2024; Śmiałek *et al*., 2024), its success hinges largely on the antigenic relatedness between vaccine virus and field strains.

Notably, some studies have documented that IBDV strains may undergo antigenic drift as they disseminate from their original geographic locations and co-evolve with local field strains (Mahajan *et al*., 2002; Asfor *et al*., 2022; Nour *et al*., 2023). These antigenic differences may compromise vaccine efficacy and lead to immunization failure. Consequently, the use of locally adapted vaccines, developed from IBDV strains circulating within the locality is recommended, to enhance the protective outcomes of vaccination campaigns and prevent similar outbreaks in future. This scenario emphasizes the need for molecular surveillance of field strains to ensure alignment with vaccine antigens. Effective control requires integrated vaccination programs, reliable cold chain logistics, strategic immunization timing and routine post-vaccination monitoring. Without these safeguards, flocks remain vulnerable to vaccine preventable infections and their sequelae.

From a management perspective, the poultry unit's semi-intensive, multi-species system that houses broilers, layers, pigeons, and waterfowl created an ecosystem conducive to pathogen exchange. Certain avian species, particularly pigeons and waterfowl, may serve as asymptomatic carriers of enteric and respiratory pathogens, including *Salmonella* spp. (Alders *et al*., 2018; Grace *et al*., 2024). Spatial overlaps and interspecies interactions promote horizontal transmission, especially in the absence of strict biosecurity. Observations in the present study also revealed critical lapses in preventive infrastructure, including disused footbaths, unrestricted personnel movement, poor ventilation and inadequate disinfection. These conditions facilitate pathogen persistence and dissemination, particularly in flocks of varying ages and immune status (Neelawala *et al*., 2024; Ayebare *et al*., 2025). Robust biosecurity frameworks must therefore encompass environmental hygiene, validated immunization protocols, personnel discipline and strategic flock management. These pillars are essential to prevent the introduction, amplification and spread of pathogens within poultry systems.

The detection of MDR *Salmonella* strain in this outbreak carries serious public health implications. Inadequate biosecurity and close proximity of poultry units to residential areas pose a significant food safety threat due to limited therapeutic options and possible human morbidity (Alam *et al*., 2020). This emphasizes the need for coordinated One Health approach that integrates veterinary, environmental, and public health disciplines (Igbinosa *et al*., 2023; Lamichhane *et al*., 2024; Aliyu *et al*., 2024). To mitigate these risks, antimicrobial stewardship, surveillance of resistance trends, targeted therapy through routine susceptibility testing, and public education on poultry handling must be prioritized. Evidence-based therapeutic protocols, in combination with improved farm hygiene, vaccination coverage, and structural biosecurity, are vital to curbing AMR emergence and protecting both animal and human health.

Conclusion: This case report reveals the complex interaction between infectious bursal disease virus (IBDV) and multidrug-resistant (MDR) *Salmonella enterica* subsp. *enterica* in three-week old flock of vaccinated ISA Brown pullets, highlighting significant gaps in flock immunity and biosecurity within multiple-species semi-intensive poultry systems. The outbreak was characterized by severe lymphoid depletion and overwhelming morbidity and mortality, suggesting infection with a very virulent IBDV strain. The IBD associated immunosuppression possibly facilitated systemic dissemination of MDR *Salmonella enterica*, evidenced by septicaemic lesions. The integration of clinical, microbiological, histopathological, and serological investigations proved essential in untangling the etiological complexity and guiding successful therapeutic intervention with ciprofloxacin, based on antibiogram data.

Recommendations: To enhance flock resilience, reduce economic losses, and safeguard public health, an integrated evidence-based approach is necessary. It is recommended that vaccination strategies should prioritize strain-matched infectious bursal disease (IBD) vaccines sourced from accredited suppliers and administered by trained personnel, with regular serological monitoring, molecular strain typing and routine cold chain audits to ensure vaccine efficacy. Standardized biosecurity measures, including perimeter control, species segregation, litter management and disinfection, should be institutionalized, while surveillance systems combining virology, bacteriology and field observations must be established, particularly in high-density and smallholder farms, for timely detection of co-infections. Rational antimicrobial use should be promoted within a One Health framework, guided by culture and sensitivity testing, routine microbial screening, and antibiogram data, alongside stricter regulation of sales of antibiotics and increased farmer awareness of antimicrobial resistance and zoonotic threats. Post-outbreak monitoring should involve cloacal swab screening of survivors to detect asymptomatic carriers, with follow-up measures such as selective culling, environmental decontamination, and enforcement of farm-to-fork safety protocols to prevent recrudescence and zoonotic spillover. Lastly, strengthening farmers capacity through targeted training in vaccination, disease recognition, and reporting, alongside enhanced veterinary outreach and regulatory support for vaccine surveillance and pathogen research, is critical for sustainable disease control.

Conflict of interest

The authors declare that they have no competing interests.

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