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Original Article
Livestock Diseases

An outbreak of fatal Pullorum disease (*Salmonella* Pullorum) in Guinea fowl keets (*Numida meleagris*)[1]

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ABSTRACT.- Pinto P.N., Torres A.C.D., Rodrigues M.P., Oliveira L.B., Costa C.S., Ecco R., Freitas Neto O.C. & Martins N.R.S. 2023. **An outbreak of fatal Pullorum disease (*Salmonella* Pullorum) in Guinea fowl keets (*Numida meleagris*)**. *Pesquisa Veterinária Brasileira 43:e07088, 2023*. Escola de Veterinária, Universidade Federal de Minas Gerais, Av. Presidente Antônio Carlos 6627, Bairro São Francisco, Belo Horizonte, MG 31270-901, Brazil. E-mail: priscilanatalia24@gmail.com, nelsonrodrigo@vetufmg.edu.br

Pullorum disease is described worldwide and is caused by *Salmonella enterica* subspecies *enterica* serovar Gallinarum biovar Pullorum (*S.* Pullorum). *S*. Pullorum infection is important in commercial poultry, provoking a systemic disease with high mortality rates. Its occurrence requires notification, and when it is diagnosed in commercial breeding flocks, its eradication is demanded. The aim of this study was to report a severe outbreak of Pullorum disease in young Guinea fowl (*Numida meleagris*), resulting in 100% mortality of keets (n=290) within the first two weeks of age. All examined keets had enlarged liver, kidneys and spleen (5/5), and the affected tissues were submitted to histological and bacteriological examination. On histopathology, random paratyphoid nodules characterized by areas of necrosis with fibrin and a moderate infiltrate of macrophages and heterophils were observed in the liver. In kidneys, discrete areas of necrosis associated with moderate multifocal infiltrates of lymphocytes, and plasma cells were observed. In the spleen, a moderate infiltrate of macrophages was noticed. Isolation of colonies suggestive of *S*. Pullorum from liver and spleen was performed in selective agars and, after biochemical tests, confirmed by specific duplex-PCR. The antimicrobial susceptibility test of the isolated strain revealed resistance to only sulfamethoxazole + trimethoprim among the tested antimicrobials. The *S.* Pullorum isolate recovered in the present study was highly pathogenic to *N. meleagris* and may represent a risk to other avian species, including industrial poultry.

INDEX TERMS: Chick, Galliformes, Numididae, Pullorum disease, salmonellosis, Guinea fowl, *Numida meleagris*.

RESUMO.- [**Surto fatal de pulorose (*Salmonella* Pullorum) em pintinhos de galinhas-d'Angola (*Numida meleagris*).**] A pulorose é descrita mundialmente e é causada por *Salmonella enterica* subespécie *enterica* sorovar Gallinarum biovar Pullorum (*S.* Pullorum). A infecção por *S*. Pullorum é importante em aves comerciais, provocando doença sistêmica com altas taxas de mortalidade. Sua ocorrência requer notificação e quando diagnosticada em aves de criação comercial resulta na erradicação do plantel. O objetivo deste estudo foi relatar um surto grave de pulorose em filhotes de galinhas-d'Angola (*Numida meleagris*), resultando em 100% de mortalidade das aves (n=290) nas primeiras duas semanas de idade. Os pintinhos recebidos tinham hepato, espleno e nefromegalia (5/5). Os tecidos dos cinco indivíduos recebidos foram submetidos a exame histológico e bacteriológico. Na histopatologia, foram observados nódulos paratifoides aleatórios caracterizados por áreas de necrose com fibrina e infiltrado moderado de macrófagos e heterófilos no fígado. Nos rins, foram observadas áreas discretas de necrose associadas a infiltrados multifocais moderados de linfócitos e plasmócitos. No baço, foi observado infiltrado moderado de macrófagos. O isolamento de colônias

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sugestivas de *S*. Pullorum de fígados e baços foi realizado em ágares seletivos e, após testes bioquímicos, confirmado por duplex-PCR específico. A susceptibilidade antimicrobiana da cepa isolada revelou resistência apenas ao sulfametoxazol + trimetoprim entre os antimicrobianos testados. O isolado de *S.* Pullorum recuperado no presente estudo foi altamente patogênico para *N. meleagris* e pode representar um risco para outras espécies de aves, incluindo aves industriais.

TERMOS DE INDEXAÇÃO: Galinhas, Galliformes, Numididae, pulorose, salmonelose, galinhas-d'Angola, *Numida meleagris*.

INTRODUCTION

Pullorum disease (PD) was first described by Dr. Leo F. Rettger, in 1899, in the state of Indiana (USA), initially named as fatal septicemia of chicks, subsequently as white bacillary diarrhea and finally, the present denomination was adopted (Bullis 1977). PD occurs worldwide and it is caused by *Salmonella enterica* subspecies *enterica* serovar Gallinarum biovar Pullorum, being generally fatal in chicks and keets of less than three weeks of age, while adult chickens are more resistant and rarely clinically affected. Morbidity and mortality may vary according to the strain of *S.* Pullorum, and with management, nutritional condition, route and dose of exposure, age, immune status, and concomitant infections (CFSPH 2019). Chickens are considered natural hosts for *S.* Pullorum and outbreaks are rarely described in other species, although reported in turkey (McBride et al. 1991), pheasant (Pennycott & Duncan 1999), duck (Chute & Gershman 1962), quail (Buchholz & Fairbrother 1992), mallard, pigeon, sparrow, canary and parrot (Hofer et al. 1997, Shivaprasad & Barrow 2013). PD is extremely rare in humans (Mitchell et al. 1946, McCullough & Eisele 1951, Shivaprasad & Barrow 2013, CFSPH 2019). Fowl typhoid, a related disease, was described in Japanese quail (Casagrande et al. 2014).

PD causes major economic losses in the poultry industry due to the high mortality and morbidity rates in chicks, reaching up to 100% in some lines. PD provokes reductions in general growth and egg production performances in addition to a decrease in hatchability and an increase in medication costs. The disease poses marketing restrictions on international trade (Shivaprasad & Barrow 2013, CFSPH 2019). In Brazil, the "Secretaria de *Defesa Agropecuária*" (Department of Agricultural Defense), through the "*Departamento de Saúde Animal*" (Department of Animal Health), of the "Ministério da Agricultura, Pecuária e Abastecimento" (Ministry of Agriculture, Livestock and Supply – MAPA), conducts constant monitoring of the epidemiological situation and the health risks imposed on poultry, as based on the "*Programa Nacional de Sanidade Avícola*" (National Poultry Health Program – PNSA) (Brasil 2003). The PNSA establishes the permanent monitoring of commercial breeding flocks in order to certify the free of infection status (Brasil 1994, 2003).

However, the health status of small breeding farms, ornamental or subsistence flocks, and losses for nonconventional species is unknown (Bessin et al. 1998, Boko et al. 2011). The present work aimed to report a severe outbreak of PD in Guinea fowl keets.

MATERIALS AND METHODS

Guinea fowl. The owner of a small commercial rural enterprise located in the metropolitan region of Belo Horizonte (Minas Gerais, Brazil) reported severe disease and mortality in Guinea fowl (*Numida meleagris*) (n=290) chicks (keets) within the first two weeks of age, reaching 100% mortality at the end of the episode. Five severely sick chicks were brought to the Laboratory of Avian Diseases (Universidade Federal de Minas Gerais, Brazil) for examination. Chicks had apathy, prostration, white diarrhea, ruffled feathers and gravely impaired mobility. All birds were humanely killed by cervical dislocation and submitted to necropsy and laboratory tests.

Anatomopathological examination. The complete necropsy was performed. For histopathology, fragments of the trachea, lung, thymus, heart, liver, spleen and kidneys were collected and fixed in neutral-buffered 10% formalin. The fixed tissues were processed for histology, embedded in paraffin, cut at 5µm thick sections, stained with hematoxylin and eosin (HE) following routine procedures, and analyzed under a light microscope (Luna 1968).

Bacteriological examination. The bacteriology was performed according to the official technical recommendations (Brasil 1995) and elsewhere (Andrews et al. 2011). Fragments of liver and spleen (2g) were placed in sterile tubes with 18mL of brain infusion broth (BHI) (OXOID), which were incubated at 37°C for 24 hours. After that, samples were plated on MacConkey (OXOID) and brilliant green agar plates (OXOID), which were incubated overnight at 37°C. Five typical colonies of *Salmonella enterica* subsp. *enterica* serovar Gallinarum biovars Pullorum or Gallinarum (*S*. Gallinarum) were randomly selected and then submitted to preliminary biochemical screaming on triple sugar iron agar (TSI), lysine iron agar (LIA), sulfide-indole-motility medium (SIM) and urea. They all produced suggestive biochemical profiles and were streaked on lysogen agar and then tested with somatic and flagellar antisera (Probac).

Duplex PCR for identification and differentiation. Chromosomal DNA was extracted from the isolated colonies, as described previously (Marmur 1961), and then submitted to a duplex-PCR reaction previously described by Batista et al. (2016). This PCR allows the identification of *Salmonella enterica* subspecies *enterica* serovar Gallinarum by the identifier region (*SIR*) and differentiation between biovars Gallinarum and Pullorum by polymorphisms of the *ratA* gene. *S*. Pullorum strain ATCC 9120, *S.* Gallinarum strain ATCC 9184 and *Escherichia coli* strain ATCC 25922 were used as control.

Antimicrobial susceptibility test. After the identification, the isolate was submitted to an antimicrobial susceptibility test by disc diffusion (Bauer et al. 1966) according to the methodology described by the Clinical and Laboratory Standards Institute (CLSI 2017). The antimicrobial discs (OXOID) tested included amikacin, amoxicillin with clavulanic acid, ampicillin, cephalothin, ceftiofur, enrofloxacin, erythromycin, fosfomycin, neomycin, sulfamethoxazole plus trimethoprim and tetracycline. *Escherichia coli* (strain ATCC 25922) was used as the quality control for the Kirby-Bauer disk diffusion test.

RESULTS AND DISCUSSION

All clinical descriptions made by the farmer were confirmed at the laboratory, including paralysis, severe apathy, low body score and inactivity. At necropsy, the celomatic cavity was distended (Fig.1), and the liver was enlarged (hepatomegaly) (Fig.2), with mottled red areas at the cut surface. The kidneys (Fig.3) and spleen (Fig.4) were enlarged and pale with petechiae, the lungs and intestines were congested, and the intestines

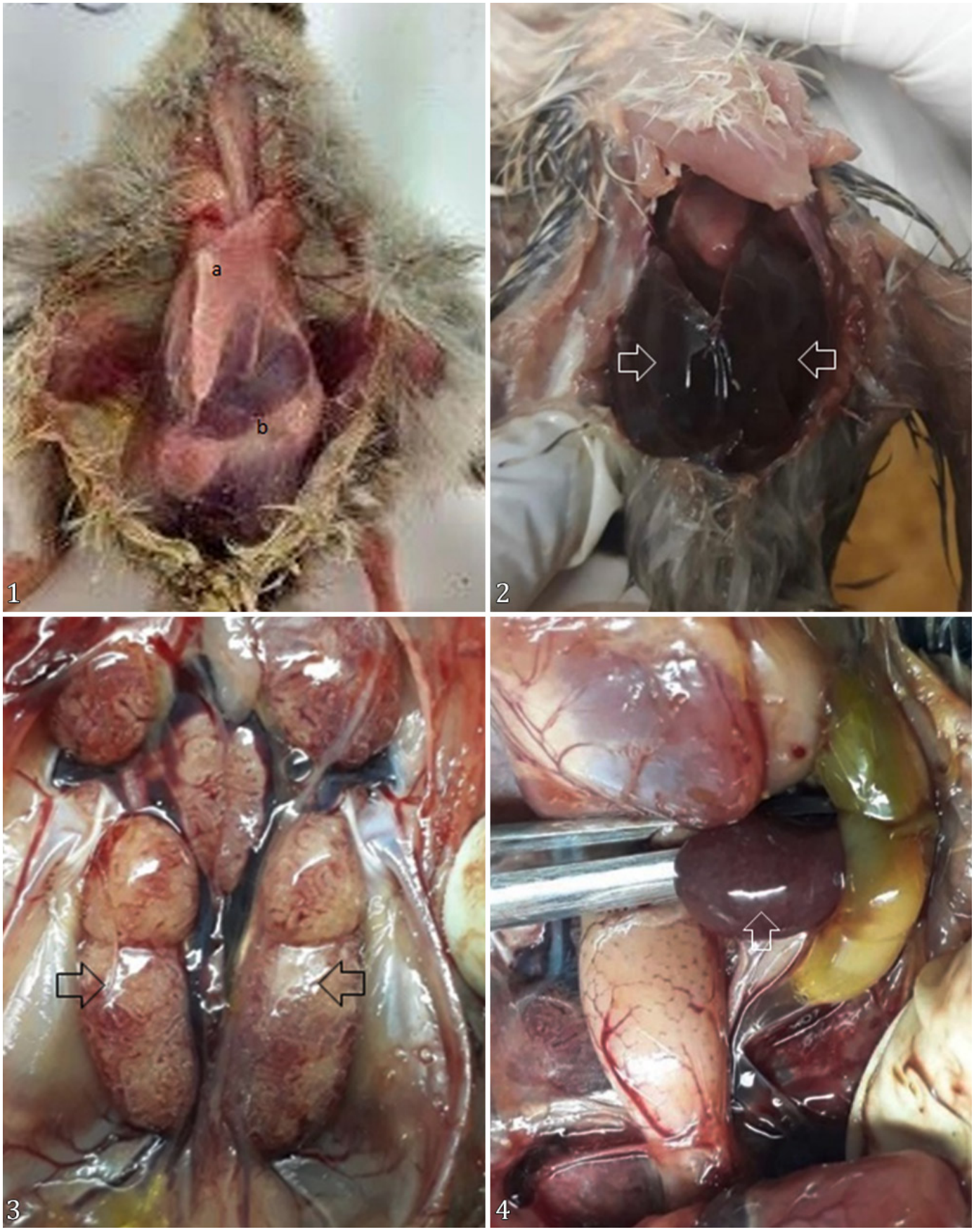

Fig.1-4. (**1**) Atrophic pectoral muscles (a) and distended posterior celomatic cavity (b). (**2**) Hepatomegaly (arrows). (**3**) Nephromegaly (arrows). (**4**) Splenomegaly (arrow).

ballooned in areas of gas retention (Fig.4). The affected age, the super-acute emergence and the macroscopic lesions especially in liver and spleen were considered suggestive of pullorosis.

The histopathology of the liver revealed random paratyphoid nodules characterized by areas of architecture loss (necrosis) with fibrin and moderate infiltration of macrophages and heterophils (Fig.5-6). In the portal space, the lymphoid tissue

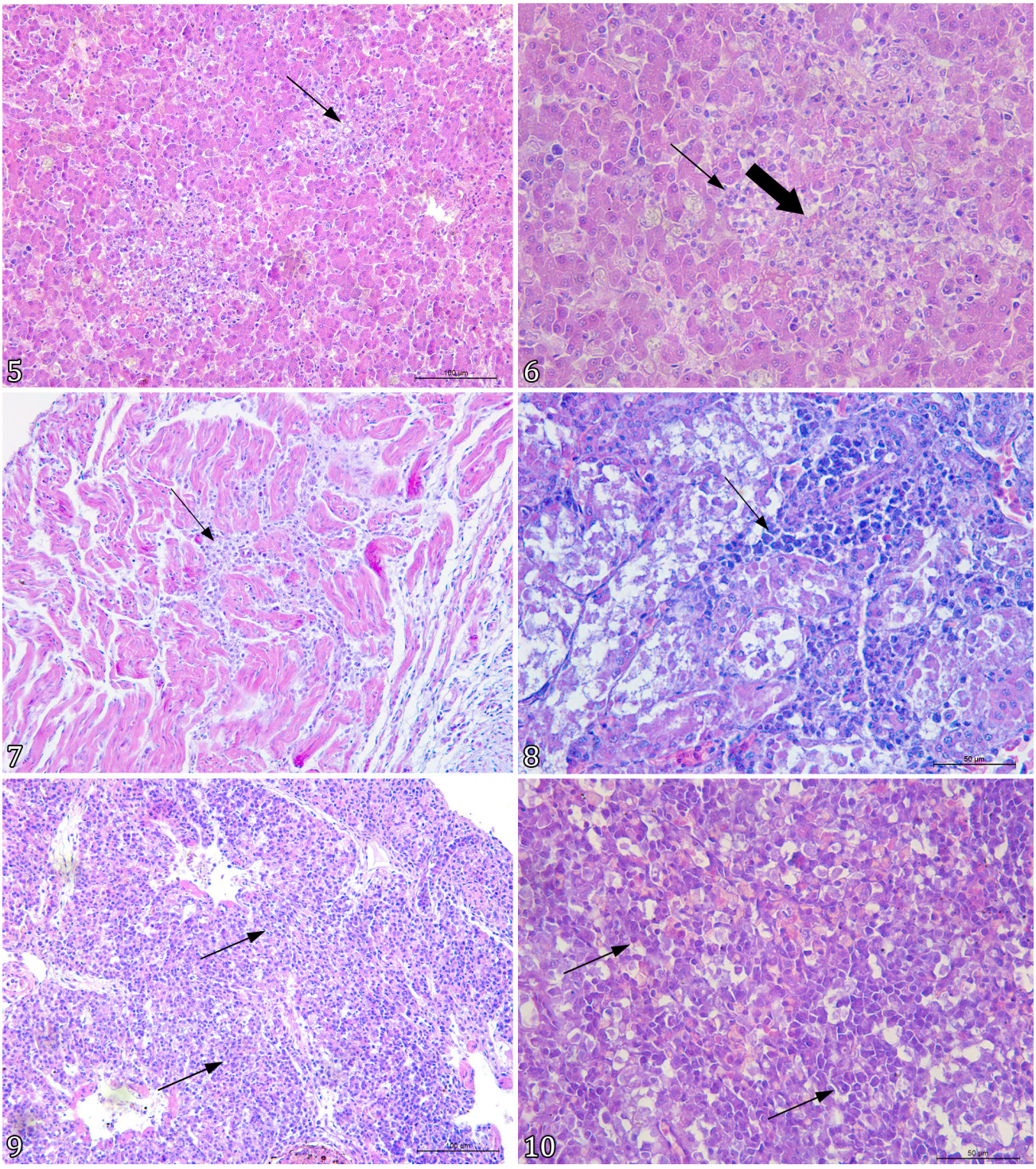

Fig.5-10. (**5**) Liver. Multiple paratyphoid nodules (arrow). HE, obj.10x. (**6**) Liver. Areas of necrosis with fibrin (thick arrow) and macrophages and heterophils infiltrate (thin arrow). HE, obj.40x. (**7**) Heart. Multifocal and moderate infiltration of lymphocytes, plasma cells and macrophages, extending from the epicardium into the myocardium (arrow). HE, obj.10x. (**8**) Kidney. Multifocal moderate infiltrate of mononuclear cells (fine arrow). Note intense tubular necrosis with necrotic tubular epithelial cell debris. HE, obj.40x. (**9**) Lung. Inflammatory infiltration (arrows) expanding the pulmonary parenchyma. HE, obj.10x. (**10**) Spleen. Moderate infiltration of macrophages and plasmacytosis (arrows). HE, obj.40x.

was hyperplastic. The heart had multifocal and moderate infiltration of lymphocytes, plasma cells and macrophages, extending from the epicardium to the myocardium (Fig.7). The kidneys had discrete areas of necrosis associated with moderate lymphocytes and plasma cells infiltrate (Fig.8) with rare tubules containing uric acid in the lumen. In the pulmonary parenchyma, numerous macrophages, lymphocytes and plasma cells were observed multifocal to coalescing distribution (Fig.9). Moderate plasmacytosis with moderate infiltration of macrophages were detected in the spleen (Fig.10). No changes were observed in the trachea and thymus.

Typical *Salmonella* spp. colonies were isolated in MacConkey and brilliant green agars. They were non-motile and produced only small amounts of H_2S in TSI and LIA. The duplex PCR reaction for amplification of *SIR* and *ratA* chromosomal regions (Batista et al. 2016) confirmed the isolate as *S*. Pullorum (Fig.7).

To the best of our knowledge, no previous descriptions of *S*. Pullorum causing mortality in keets were found of *S.* Pullorum causing mortality in Guinea fowl keets. Similar occurrences have been previously described in *Colinus virginianus* (Buchholz & Fairbrother 1992) and pheasant chicks (Pennycott & Duncan 1999). *Salmonella* serovars previously reported infecting and/or causing disease in Guinea fowl include *S.* Adelaide, *S.* Farakan, *S.* Kingston, *S.* Legon, *S.* Luke, *S.* Oakland, *S.* Sangalkam and *S.* Teshie, isolated from the cecum and liver of dead keets and serotypes Farakan, Kingston, Legon, Oakland and Sangalkam recovered from feces of laying Guinea fowl and surrogate chicken mothers (Boko et al. 2013). Up to 70% of mortality in keets was also previously associated with infection with *Salmonella* sp. (Boko et al. 2011), *S.* Gallinarum (Johnson & Anderson 1936) and *S.* Typhimurium (McCrea et al. 2006). However, *Salmonella* spp. has also been isolated from Guinea fowl without clinical signs (Kilonzo-Nthenge et al. 2008).

Pullorum disease is currently eradicated in the grandparent and parent stocks of all commercial breeds of chickens for egg or meat production (Brasil 2003). However, the risk of infection from free-range chickens persists, as the infection is considered endemic in these (Anderson et al. 2006). In addition, other species may be infected and represent epidemiological risk, and, according to the host, different degrees of susceptibility are described for the expression of clinical disease (Shivaprasad 2000).

The antimicrobial susceptibility test revealed that the isolate was sensitive to amoxicillin with clavulanic acid, amikacin, ampicillin, neomycin, enrofloxacin, erythromycin, cephalothin, fosfomycin, tetracycline and ceftiofur, with resistance only for the sulfamethoxazole and trimethoprim association. Pan et al. (2009) have found resistance to ampicillin and trimethoprim-sulfamethoxazole, and resistance to erythromycin was also reported (Anderson et al. 2006). Apparently, the isolate recovered in the present study was not under conditions of selection pressure for the majority of the tested antimicrobials. On the other hand, the chicken isolates of *S.* Pullorum tested by Penha Filho et al. (2016), were sensitive to amoxicillin with clavulanic acid and sulfamethoxazole with trimethoprim, while 94% were sensitive to enrofloxacin.

CONCLUSIONS

Salmonella enterica subspecies *enterica* serovar Gallinarum biovar Pullorum was associated with 100% morbidity and mortality rates in Guinea fowl keets (*Numida meleagris*)

The relevance of our findings associates with the relative scarcity of information on Pullorum disease in Guinea fowl. Further studies are planned to elucidate the role of the Guinea fowl in the epidemiology of this relevant disease.

Authors' contributions.- Authors declare the contribution of each author. P.N.P., O.C.F.N. and N.R.S.M. conceived and designed the study. A.C.D.T., M.P.R., A.A.F., L.B.O., C.S.C. and R.E. executed the histological, and molecular and bacteriological analyses. N.R.S.M. and S.Y.M.G. edited and revised the manuscript and English language. All authors interpreted the data, critically revised the manuscript for important intellectual contents and approved the final version.

Conflict of interest statement.- The authors declare the absence of conflicting interests.

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