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SHORT COMMUNICATION

Pathology of concurrent pasteurellosis, fasciolopsiasis and sarcocystosis in a non-descript grower pig

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ABSTRACT

A three months old nondescript female pig carcass was submitted to postmortem facility, division of pathology, ICAR-IVRI, Izatnagar, Uttar Pradesh, India. Concurrent infections with *F. buski*, *Pasteurella* and *Sarcocystis* were detected based on cultural examination, Gram staining of bacteria and morphology of parasites. On necropsy examination, gross lesions observed were hydrothorax and hydroperitoneum, frothy exudate in trachea, severe congestion, consolidation and edema of lungs, lymphadenitis, hepatitis with whitish necrotic foci and prominent lobular pattern, congestion of kidneys, meningeal and mesenteric blood vessels and serosa of small intestine. Histologically, acute fibrino-suppurative bronchopneumonia, myocarditis with sarcocysts, enteritis, hepatic necrosis with thickened capsule and interlobular septa, perivascular cuffing, perivascular and perineuronal oedema in brain, lymphoid necrosis and depletion were observed. The case was diagnosed as acute fibrino-suppurative bronchopneumonia and septicaemia caused by *Pasteurella* spp. associated with cardiac sarcocystosis and intestinal fasciolopsiasis.

Keywords: *F. buski*, *Pasteurella*, pathology, *Sarcocystis*

Pig is one of the important domesticated livestock having significant role in food security and socioeconomic improvement. Pig industry is facing various challenges due to many infectious and non-infectious diseases. Concurrent infection of single host by different pathogens (macroparasites like helminths and microparasite like bacteria) is a very common phenomenon. They may guard or exacerbate infection risk and shifts disease severity[1]. Pasteurellosis is an important bacterial disease of pigs worldwide causing great economic losses. It is caused by gram negative coccobacillary bipolar organism, *Pasteurella multocida.* Based on antigenicity of its polysaccharide capsule, there are five different serogroups of *P. multocida* such as A, B, D, E or F[2]. *P. multocida type* A and *P. multocida* type D cause bronchopneumonia, pleuritis and atrophic rhinitis in pigs and *P. multocida* type B is associated with septicemic pasteurellosis in pigs in India[3-5]. It is the common inhabitant in the respiratory tract of healthy animals. It was considered as secondary pathogen associated with pig pneumonia. Recently, it is considered as primary agent associated with swine pneumonia and septicemia[6]. It is also mostly prevalent zoonotic pathogen associated with human infection. Fasciolopsiasis is an important food borne infection caused by giant intestinal digenetic fluke *Fasciolopsis buski* in pigs causing significant economic losses in pig farms. The pathology is due to traumatic, obstructive and toxic effect of adult parasite in duodenum and jejunum of pigs and humans. Hemorrhage, inflammation, small bowl stricture, ulceration, micro abscesses etc. occur at the attachment sites of the adult parasite in the intestine.

Pigs act as intermediate host for three different *Sarcocyst* species like *S. porcifelis, S. suihominis* and *S. miescheriana.* Later two parasitic species are pathogenic to pigs. Pigs get infected after ingesting sporocysts excreted in the definite host faeces. In the endothelial cells of capillaries and arterioles of internal organs, the parasites undergo merogonic reproduction and in the skeletal muscles, they produce tissue cysts containing banana shaped bradyzoites. Morphologically, the cyst wall of *S. miescheriana* has radial perpendicular papillomatous striations and *S. suihominis* has hair like villar protrusions[7].

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To the best of our knowledge, there are no published literature on pathology of concurrent infection of swine *with F. buski, Pasteurella* and *Sarcocystis*. Therefore, the aim of the present study is to describe pathological changes in a non-descript grower pig associated with natural concurrent infection with *F. buski, Pasteurella* and *Sarcocystis*.

A three months old non-descript female pig carcass was submitted to postmortem facility, division of pathology, ICAR-IVRI, Izatnagar, Uttar Pradesh, India. Detailed necropsy was conducted and gross pathological findings of different organs were recorded. Representative tissue specimens from different organs were collected and preserved in 10% percent buffered formalin

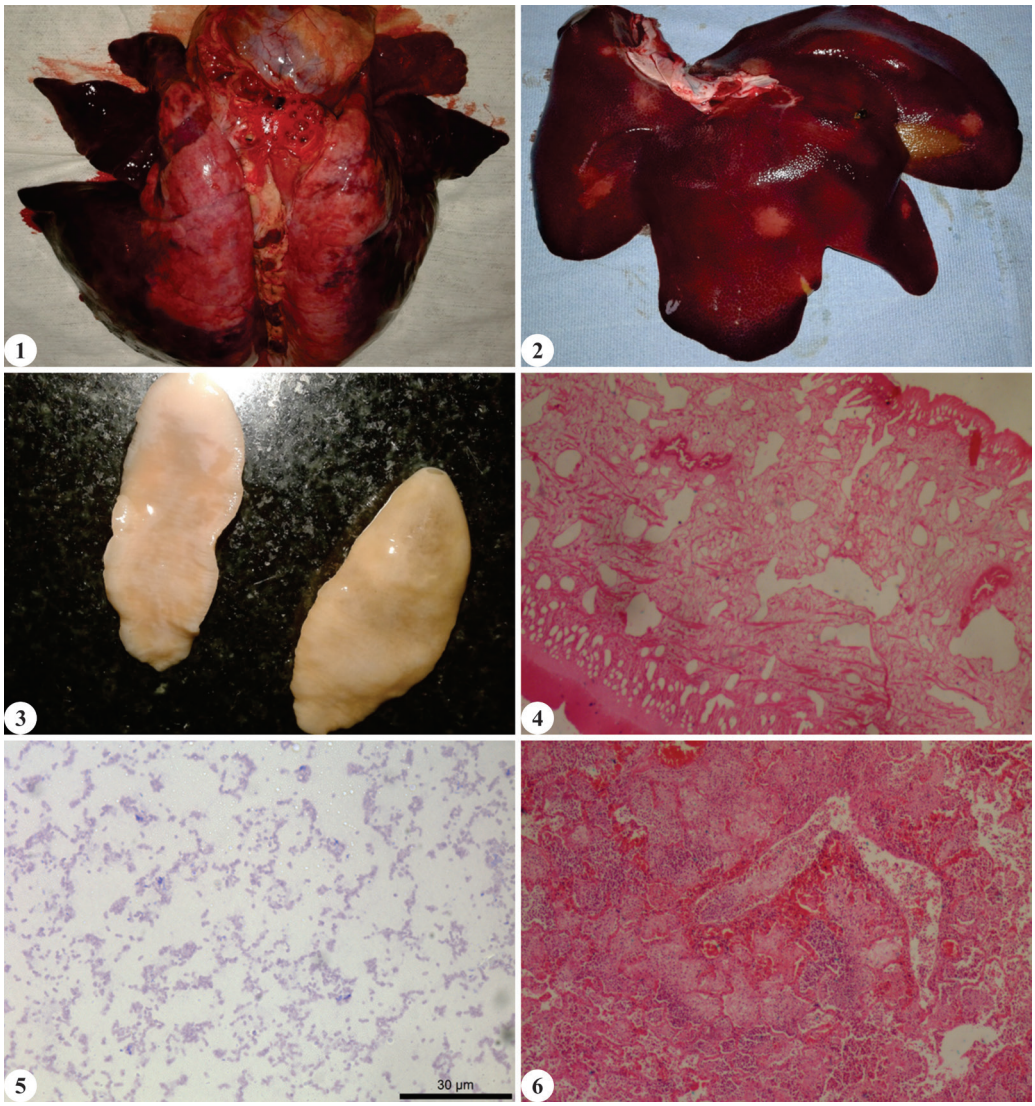

Fig. 1. Lung showing consolidations, haemorrhage and oedema; **Fig. 2.** Firm and enlarged liver with whitish necrotic foci and prominent lobular pattern; **Fig. 3.** Adult *Fasciolopsis buski* parasites; **Fig. 4.** Histological section of *F. Buski* parasite on H&E staining (x40); **Fig. 5.** Gram negative non-spore forming coccobacillary showing bipolarity on Gram stain (x1000); **Fig. 6.** Lung showing acute fibrino-suppurative bronchopneumonia on H&E staining (x40).

for histopathological processing. After fixation, tissue samples were dehydrated in serially ascending grades of alcohol, then cleared in xylene, embedded in paraffin wax and sectioned at 4 µm thickness and stained with hematoxylin and eosin routine stain. The slides were examined under light microscope. For bacteriological examination, heart blood sample was aseptically collected in a sterile syringe and stored at - 4^{O}C for further cultural examination. The blood sample was streaked on 5% sheep blood agar for bacterial isolation and incubated at 37^{O}C for 24-48 hours. The bacteria were identified based on colony morphology, hemolytic pattern on blood agar and microscopic examination of Gram-stained slide smear of culture. Adult *F. buski* and tissue cyst of *Sarcocystis* were identified based on their morphology[7,8].

The gross examination revealed moderate hydrothorax and hydroperitoneum, frothy exudate in trachea, severe congestion, consolidations and oedema of ventral parts of both left and right lung (Fig. 1), thickened pericardium of heart, congested and enlarged bronchial lymph nodes, firm and enlarged liver with whitish necrotic foci and prominent lobular pattern (Fig. 2), moderately enlarged and congested spleen, congested kidney and meningeal blood vessel congestion, congestion of mesenteric blood vessels and serosa of small intestine. Similar gross lesions were also reported earlier with natural outbreak of swine pasteurellosis[9]. Small intestine showed heavy infestation of adult *Fasciolopsis buski* parasites (Fig. 3 and Fig. 4). The parasites were of different sizes and dorsoventrally flattened, characterized by blunt anterior end with presence of large

ventral sucker and absence of cephalic cone[10]. Infection of human and pigs with zoonotic intestinal parasite *F. buski* has been reported in different parts of India[11-13]. In West Bengal, North-East India and Rajasthan 27.93%, 20.55% and 4.46% prevalence of *F. buski* were recorded in pigs[11-13]. Bacteriological culture of heart blood produced small greyish non hemolytic colonies in blood agar and gram staining of impression smear from lung revealed gram negative bipolarcocco bacillary organism indicating *Pasteurella* spp.[14-15] (Fig. 5). The characteristic colony morphology and Gram staining are useful methods for initial diagnosis of pasteurellosis.

Histopathological examination of lung revealed thickening of pleura with fibrin and sub pleural haemorrhages in lungs, thickening of interlobular septae with congested capillary, fibrin and inflammatory cells, severe hemorrhage, edema with fibrin strands and severe infiltration of PMNCs and MNCs in alveolar lumen, severe hemorrhage and cellular infiltration in and around the bronchi and bronchiolar wall and presence of RBCs, desquamated lining epithelial cells,

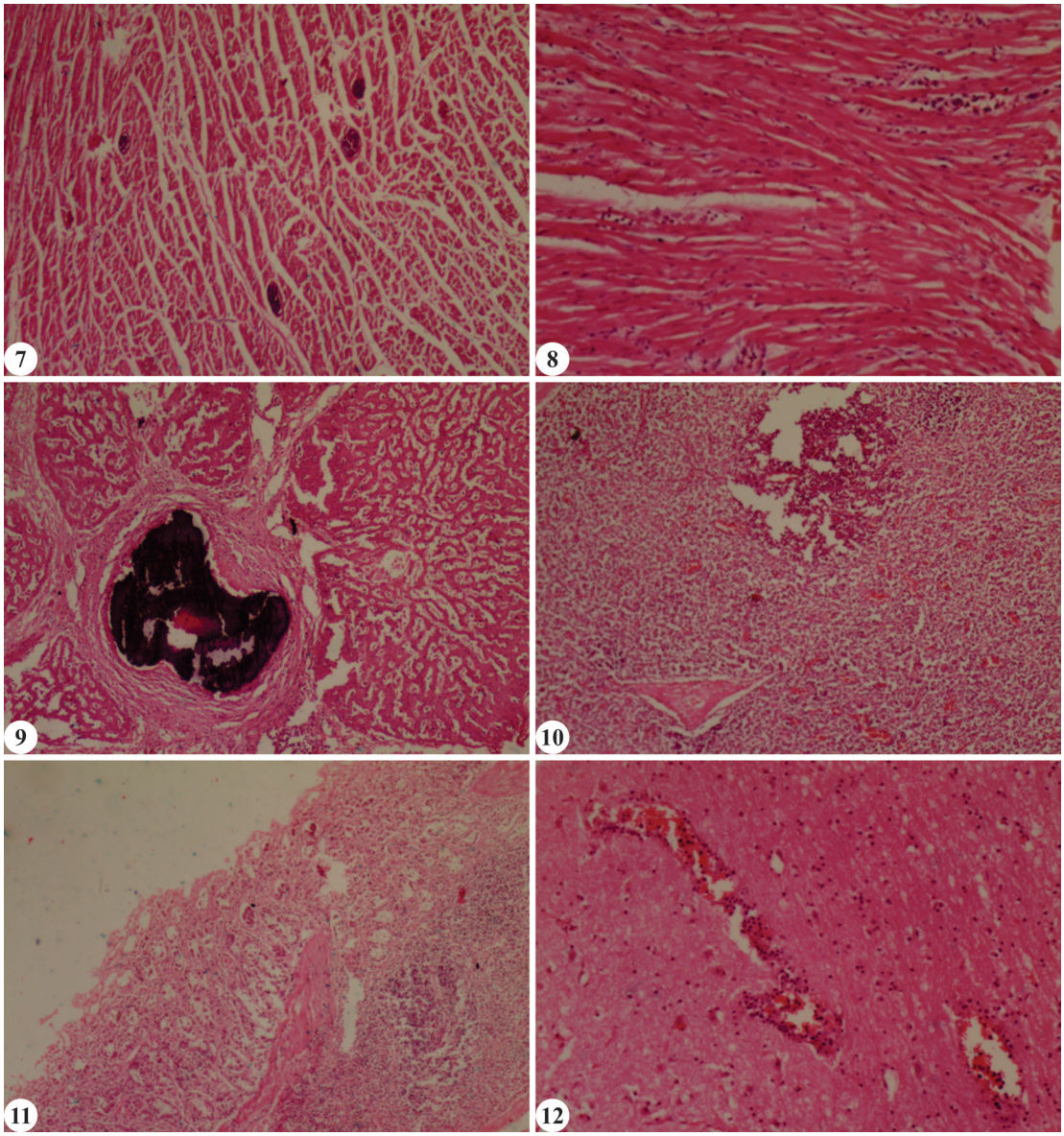

Fig. 7. Several sarcocysts with banana shaped bradyzoites in the myocardium of heart (X40); **Fig. 8.** Myocardium showing capillary congestion, haemorrhage, cellular infiltration and degeneration (X40); **Fig. 9.** Liver showing thickened interlobular septae, hepatocyte necrosis, atrophy of hepatic lobules, old granuloma with bluish stained calcified central mass surrounded by fibrous capsule and inflammatory cells in the periportal area on H&E staining (X40); **Fig. 10.** Lymphocyte necrosis and depletion, capillary congestion in lymphnode on H&E staining (X40); **Fig. 11.** Intestine showing necrosis of lining epithelial cells with infiltration of inflammatory cells in lamina propria on H&E staining (X40); **Fig. 12.** Brain showing perivascular cuffing, congestion, margination of leucocytes in blood vessels, perivascular and perineuronal oedema on H&E staining (X100).

inflammatory cells and fibrin strands in the lumen, presence of bacterial colonies in the lungs (Fig. 6). Lesions like thickening of pleura and septae with fibrin, fibrinous bronchopneumonia and filling of alveoli with fibrin, PMNCs and RBCs were described in previous studies[15, 16]. Heart revealed thickening of epicardium with fibrin and inflammatory cells; capillary congestion, neutrophil and eosinophil infiltration, hemorrhages and oedema with fibrin strands, degeneration and presence of many sarcocysts with banana shaped bradyzoites in the myocardium of heart (Fig. 7 and Fig. 8). The pathological changes in the myocardium might be due to combined effect of bacteria *Pasteurella* and parasite *Sarcocystis*. Severe non-suppurative myocarditis was described in a naturally infected sarcocystosis in a pig breeding stock[17]. However, natural Sarcocystis infections in pigs are unobvious due to development of immunity to usual low dose infections[17]. However, in highly organized farm where natural immunity to sarcocystis is diminishing due to high biosecurity measures, clinical signs may become severe after infection[17]. In a previous study, lesions like degenerative myocytolysis, inflammatory reactions, fibroblast proliferation and arterial hyperplasia were reported in naturally infected sheep with Sarcocyst species[18]. Liver revealed thickened Glisson's capsule and interlobular septae with fibrin and with accumulation of neutrophils, fibrin mass in the blood vessels with margination of neutrophils, sinusoidal congestion, haemosiderin laden macrophages, hepatocyte necrosis, atrophy of hepatic lobules, old calcified granuloma with bluish stained calcified central mass surrounded by fibrous capsule and inflammatory cells in the periportal area (Fig. 9). There are reports on severe hepatic pathology associated *Pasteurella* spp. in different animal species[19,20]. Lymphocyte necrosis and depletion, capillary congestion, haemorrhage and neutrophilic infiltration were observed in bronchial and mediastinal lymphnodes (Fig. 10) and spleen. Necrosis and desquamation of lining epithelial cells were noticed in stomach. Presence of inflammatory cells, RBCs, necrosed and desquamated lining epithelial cells in the lumen of intestine and capillary congestion and infiltration of inflammatory cells including eosinophils and plasma cells were observed in lamina propria of intestine (Fig. 11). Haemorrhage, ulceration and denudation of intestinal lining epithelial cells with infiltration of mononuclear cells and eosinophils in the intestinal tissue was reported in pigs infected with *F. buski*[11,21]. Helminths infection induces Th-2 cytokines and down regulates Th-1 cytokines inhibiting intracellular microparasite control and may cause exacerbation of concomitant infections those were controlled by Th-1 response[22]. The pathological changes in the intestine might be due to combined effect of bacteria and parasite. In kidney, thickened capsule, inflammatory cells in the interstitium, capillary congestion and tubular epithelial degeneration were observed microscopically. In our study, perivascular cuffing, congestion and margination of leucocytes in blood vessels, perivascular oedema, neuronal degeneration and perineuronal oedema were observed in the brain (Fig. 12). Cerebral lesions like perineuronal and perivascular oedema, encephalomalacia and fibrinous meningitis were reported in goat kid infected with *P. multocida*[23].

Concurrent infection alters disease progression in many instances. Concurrent parasitic infection may aggravate or ameliorate the pathological effect. The increased incidence of pneumonia due to increased gastro intestinal parasitism in Nigerian Goats was reported[24]. Impediment of pulmonary defense mechanism and aggravating pneumonia in Nigerian goats due to other bacterial and viral pathogens could be due to pulmonary oedema associated with parasitic protein loosing enteropathy[24]. In another study in different species, the direct and indirect effects of invasive parasite on disease outbreak were studied. It was demonstrated that infection of blue mussels with parasitic copepod *Mytiicolla intestinalis* increased the rate of secondary *Vibrio* infection as compared to mussels not exposed to parasitic copepod infection due to decreased efficiency of cellular immunity and not due to direct serious pathological effect[25]. Co-infection of cattle with *Fasciola gigantic* and tuberculosis was reported to increase the risk of developing TB lesions due to lower INF-γ Level[26]. In other instances, parasitic co-infection resulted in increased mortality of the host due to increased bacterial loads in different organs as a result of facilitation of bacterial invasion owing to more damage to the epithelium or by disturbing metabolic profile of GI tract or by increasing level of cortisol causing immunosuppression or by increasing blood β-hydroxybutyrate level and damaging mammary cellular immunity or by causing significant lymphopenia in co-infected host[27]. Therefore, in the present study, the parasitic infection (*F. buski* and *Sarcocystis* spp.) might have enhanced the pathological effect of *Pasteurella* infection directly or indirectly in pig resulting in fatal outcome. Further future studies are required at cellular and molecular level for better understanding of complex mechanism of interaction among above three pathogens and for future development of therapeutics and control measures.

CONCLUSION

Based on pathological, bacteriological and parasitological examination, the case was diagnosed as acute fibrino-suppurative bronchopneumonia and septicaemia caused by *Pasteurella* spp. associated with cardiac sarcocystosis and intestinal fasciolopsiasis. This is the first documented report on pathology of natural concurrent bacterial and parasitic infection in swine.

In field conditions, it is suggested to consider parasitic concurrent infection before initiating any treatment or vaccination against other pathogens. Due to one health significance of the above pathogens, coordinated multidisciplinary effort is essential to develop new strategies for effective diagnosis, treatment, prevention and control of the above pathogens.

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