P-ISSN: 2304-3075; E-ISSN: 2305-4360
International Journal of Veterinary Science
www.ijvets.com; editor@ijvets.com
Short Communication https://doi.org/10.47278/journal.ijvs/2022.157

Histopathological Changes in Some Vital Organs of Nursery Pigs in Mortalities Associated with African Swine Fever Outbreak

Davinson Chuka Anyogu*, Onyinyechukwu Ada Agina and Remigius Ibe Onoja

Department of Veterinary Pathology and Microbiology, University of Nigeria, Nsukka
***Corresponding author**: E-mail: davinson.anyogu@unn.edu.ng

Article History: 22-508 Received: 15-Jan-22 Revised: 17-Mar-22 Accepted: 19-Mar-22

ABSTRACT

African swine fever (ASF) is a contagious viral disease that affects domestic and wild pigs. The disease is of serious economic importance as outbreaks lead to the culling of millions of pigs in Africa, Eastern Europe and some parts of Asia. The present study evaluated the histopathological changes in some visceral organs in nursery pigs infected with ASF virus. The pigs were euthanized in carbon dioxide chamber and necropsied. Tissue samples were fixed in 10% neutral buffered formalin, routinely processed, sectioned at 5µm thickness and stained with hematoxylin and eosin (H&E). Results showed petechial and ecchymotic hemorrhages grossly on the skin and in the lungs, heart, spleen, lymph nodes, and kidneys. In the heart, there was hydropericardium and the pericardial sac was cloudy. The liver was also enlarged and mottled. Microscopically, hemorrhages were present in the lungs, heart, lymph nodes, and kidneys. There was interstitial pneumonia and necrosis of renal tubules. Severe lymphoid necrosis occurred in the spleen. It was concluded that the disease involved a moderately pathogenic isolate of ASF virus and that hemorrhages in visceral organs and destruction of lymphoid tissues remained the major findings in sub-acute ASF infection.

Key words: African swine fever, Haemorrhages, Mortality, Histopathology, Visceral organs

INTRODUCTION

African swine fever (ASF) is a moderately contagious, febrile disease of pigs associated with high mortality (Urbano et al. 2021). It is caused by African swine fever virus (ASFV), a large double stranded DNA (dsDNA) virus and the sole member of the Asfaviridae family, Genus Asfavirus (Urbano et al. 2021). The disease outbreak occurs regularly in the pig industry in Africa and Eastern Europe, with severe economic losses in pig production. In China, ASFV was first detected in August 2018 and by March 2020, 1.2 million pigs have been culled due to the disease further stressing the economic importance of ASF (Wang et al. 2018; FAO 2020).

In natural infection, the incubation period of ASF ranges from 4-19 days (Gallardo et al. 2015). The clinical signs are nonspecific and may vary depending on the virulence of the ASFV strain involved. Highly virulent strains of ASFV are responsible for the per-acute form of the disease, in which mortality may be up to 100% within 4-10 days post infection, and there may not be obvious clinical signs of the disease or gross pathologies (Sánchez-Cordón et al. 2018). Virulent strains cause acute form of the disease in which mortality may be 90 – 100% within 4-7 days after the first clinical signs (Salguero 2020). The disease course in infections with the moderately virulent strain is usually 11-22 days and mortality ranges from 30-70%, with adult pigs having the lowest level of mortality (Gallardo et al. 2019). Low pathogenic isolates give rise to the chronic form of the disease, which may also occur in pigs that survive the sub-acute form of the disease due to innate or acquired immunity (Zani et al. 2018).

The similarities between the reported lesions of ASF and those of porcine leptospirosis, erysipelas infections, classical swine fever, porcine dermatitis and nephropathy syndrome, porcine reproductive and respiratory syndrome and Aujeszky's disease (Beltrán-Alcrudo et al. 2017) have made it relevant to constantly review the pathological changes in ASF for consistency or lack of it with previous reports. Hence, this study evaluated the histopathological changes in the bronchial lymph node, heart, kidney, liver, spleen and lungs in mortalities in nursery pigs associated with African swine fever outbreak.

MATERIALS AND METHODS

Diagnosis of ASF was made in an outbreak in a pig farm that restocked with nursery pigs after attempt to stamp out the cause of inexplicable mortality in the farm – veterinarian's attention was not sought. As a result of the renewed waves of mortality, serum samples were sent to the National Veterinary Research Institute, Jos, Nigeria, where antibodies against ASF virus were detected in all the samples by indirect ELISA.

Cite This Article as: Anyogu DC, Agina OA and Onoja RI, 2023. Histopathological changes in some vital organs of nursery pigs in mortalities associated with African swine fever outbreak. International Journal of Veterinary Science 12(1): 132-135. https://doi.org/10.47278/journal.ijvs/2022.157

The farm is located in a suburban community, south of University of Nigeria, Nsukka, Enugu State, South-eastern Nigeria, with GPS coordinates 6°51′24″N and 7°23′45″E. There had not been previous report of ASF in the community. The total pig mortality was 16/21 (76.2%). Mortality was 15/16 (93.8%) and 1/5 (20%) in the nursery and adult pigs, respectively. Most of the pigs died within 14 days after clinical signs of anorexia, cyanosis and recumbence were observed. At the time of visitation for necropsy services, most of the carcasses were no longer fresh. However, two moribund nursery pigs, aged 5.5 weeks and weighing 20-22.6lbs were euthanized in carbon dioxide chamber. The carotid arteries were severed afterwards to ensure complete euthanasia. Necropsy was done in the farm for biosecurity reasons. Tissue samples from the lungs, bronchial lymph node, heart, kidney, liver, and spleen were fixed in 10% neutral buffered formalin, routinely processed, sectioned at 5µm thickness and stained with haematoxylin and eosin.

The farmer was advised on safe culling and disposal of the remaining adult pigs, fumigation and resting of the farm premises before restocking from reputable breeders. Routine biosecurity measures were also emphasized in the course of reintroduction of naïve pigs to the farm.

RESULTS

Grossly, the carcasses were pale. Cyanosis and haemorrhage were observed in the skin of the dorsum, flank, snout, ears and limbs. Severe widespread ecchymotic and petechial haemorrhages were present in the lungs (Fig. 1). The interlobular connective tissues of the lungs were dilated with oedema fluid. The pericardial sac was cloudy and contained about 20mL of straw-coloured fluid (hydropericardium). Haemorrhages were also seen in the epicardium and endocardium, and at focal areas in the spleen. The liver was congested, mottled and had multifocal pale patches.

Microscopically, the lymphoid follicles of the bronchial lymph nodes were normal. However, severe haemorrhage was observed in the medullary sinuses and mild depletion of lymphoid cells occurred in the medullary cords (Fig. 2). Multiple haemorrhagic foci were found in the heart especially in the perivascular spaces, which also contained few mononuclear inflammatory cells, made up of lymphocytes, plasma cells and macrophages (Fig. 3). Severe coagulative necrosis of the renal tubules and glomerular podocytes were observed along with severe haemorrhage in the interstitial spaces and infiltration of mononuclear inflammatory cells in the blood vessels and interstitium (Fig. 4). In the liver tissue, the central veins were severely congested and contained mononuclear inflammatory cells. The sinusoids were congested and the Kupffer cells were hyperplastic. The interlobular connective tissues were also infiltrated by mononuclear inflammatory cells. Random, focal hepatocellular degeneration and coagulative necrosis were also observed (Fig. 5). Severe atrophy of the splenic white pulp and massive sequestration of red blood cells in the red pulp were observed. The splenic tissue was also oedematous (Fig. 6). The lung tissue had severely thickened interalveolar connective tissue with severe haemorrhage, oedema and infiltrated mononuclear inflammatory cells (Fig. 7).

DISCUSSION

In the present report, the total pig mortality (76.2%) and the relatively less severe gross and histological changes in tissues suggest a sub-acute form of ASF caused by a moderately virulent ASFV isolate. This isolate causes less mortality in adult pigs (Gallardo et al. 2019), which is in agreement with the present report. Variations in the lesions of ASF are dependent on the virulence of the ASF virus isolate, the route and dose of the infection, as well as on the host characteristics (Sanchez-Vizcaino et al. 2015). Gross and histopathological changes are rarely demonstrated in the per-acute cases of ASF (Salguero 2020). However, in the clinical forms (acute, sub-acute or chronic), typical ASF lesions, characterized by fatal haemorrhages are seen in various organs. Typical lesions such as spleens that were extremely swollen and severely necrotic, hemorrhages in tonsils and lungs, marbled lesions in mandibular and mesenteric lymph nodes, and diffuse hemorrhages in a large part of gastric serosa have been reported even in regions where the disease was not endemic (Ge 2018). Petechial haemorrhages, mucoid diarrhoea and reddening of the skin around the ears, stomach and limbs are common in the less severe form of the disease (Pikalo et al. 2019).

Microscopically, infections with highly virulent ASFV isolate result in extensive destruction of lymphoid tissues with severe necrosis, while infections involving moderately virulent isolates display less lymphoid destruction, although they produce more severe histopathological vascular changes and haemorrhages affecting a higher number of lymphoid organs (Sánchez-Cordón et al. 2021). Majority of the lymph nodes in sub-acute ASF infection resemble a blot clot (Sanchez-Vizcaino et al. 2015) and this was reflected histologically in the present case as large haemorrhages were observed, especially in the medullary sinuses of the bronchial lymph nodes, while lymphoid depletion was mild and was only seen in the medullary cords. In the spleen, however, both red blood cells sequestration and lymphoid depletion were marked. Lesions of sub-acute form of ASF are more severe in the lungs and include poor deflation of the lungs at necropsy, marked rib imprint, severe diffuse congestion, severe alveolar oedema, severe distension of the interlobular septa (interstitial oedema) and multifocal to coalescent petechial haemorrhages, ecchymosis and purpura in the pulmonary interstitium and pleura (Sánchez-Cordón et al. 2021). These lung lesions were observed in the present case, but were relatively less severe grossly, and were characterised, microscopically, by severe interstitial pneumonia, oedema and haemorrhage.

Lesions in the heart, kidney and liver compared favourably with previous reports of gross and histo-pathological changes in subacute forms of ASF (Sánchez-Cordón et al. 2021). Differences were mainly in terms of the severity of the lesions; the present case showed milder lesions compared to previous reports (Sánchez-Cordón et al. 2021). In the heart, hydropericardium was also reported in wild boars with sub-acute form of ASF, along with ascites and hydrothorax (Rodríguez-Bertos et al. 2020).

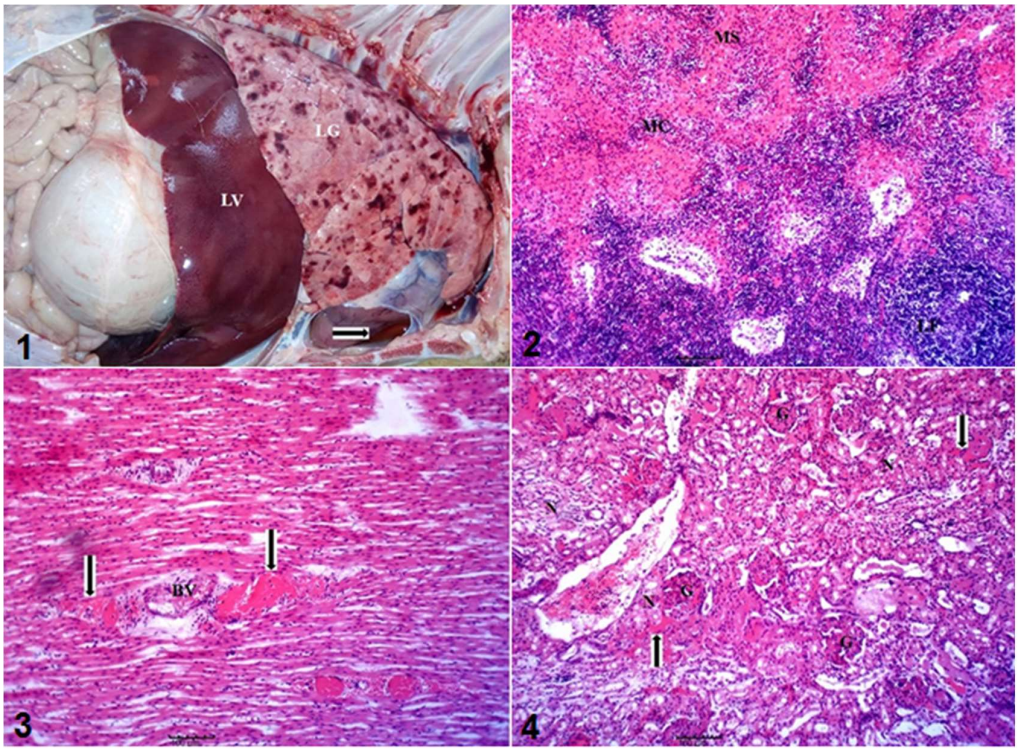
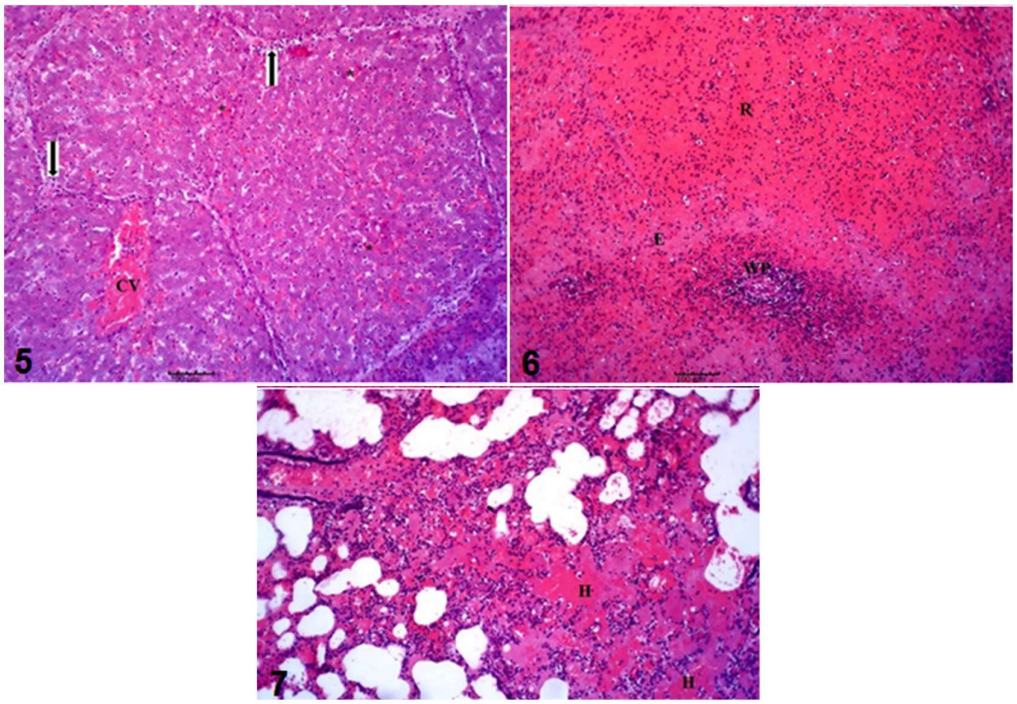


Fig. 1: Picture showing some visceral lesions of African swine fever (ASF) in nursery pig. Note the poorly deflated lungs (LG) with severe petechial and ecchymotic hemorrhages and prominent interalveolar connective tissue, slightly enlarged and mottled liver (LV) and hydropericardium (*arrow*). **Figs**. **2-4:** Histologic lesions in visceral organs of ASF in nursery pigs. **2)** Bronchial lymph node showing severe hemorrhage in the medullary sinuses (MS) and lymphoid depletion in the medullary cords (MC). Note normal lymphoid follicle (LF) in the cortex. **3)** Heart showing hemorrhages (*arrows*) in the myocardium. **4)** Kidney showing massive coagulative necrosis of renal tubules (N), interstitial hemorrhage (*arrows*) and infiltration of mononuclear inflammatory cells. G=glomerulus. H & E stain, x100.



Figs. 5-7: Histologic lesions in visceral organs of nursery pigs with African swine fever. **5)** Liver section showing random hepatocellular degeneration and necrosis, congested central vein (CV), dilated sinusoids (*asterisks*) and hyperplasia of Kupffer cells and infiltration of mononuclear inflammatory cells in the interstices (*arrows*). **6)** Section of the spleen showing massive sequestration of red blood cells (R) in the red pulp with oedema (E) and severe lymphoid depletion and atrophy of the white pulp (WP). **7)** Section of the lung showing severely thickened interalveolar connective tissue with infiltration of mononuclear inflammatory cells. Note interstitial haemorrhage (H), surrounded by oedema. H & E stain, x100.

It was thought that host factor, such as presence of maternal derived antibodies, may have doused the severity of the lesions in the nursery pig. The duration of the maternally derived antibodies post weaning is unknown (Blome et al. 2020) and may require further studies to substantiate this presumption. However, it is posited that maternally-derived antibodies as well as acquired immunity from recovery from ASF play a significant role in the epidemiology of the disease. In a study in Vietnam, both surviving sows and their off-springs were monitored serologically for more than 14 months and they were found to have high serum antibody levels post recovery (Taehwan et al. 2021).

In conclusion, the study has shown that moderately virulent isolate of ASFV was likely responsible for the outbreak of ASF in the farm. Haemorrhages and lymphoid depletion remain the major pathological changes in visceral organs of infected pigs. Lymphoid depletion was, however, marked in the spleen compared to lymph nodes in this report.

Author's Contribution

DCA conceptualized, designed, carried out the study and produced the initial draft of the manuscript. OAA participated in necropsy and contributed reagents. RIO participated in histopathological studies. All the authors read, edited and approved the final copy of the manuscript for publication.

REFERENCES

Beltrán-Alcrudo D, Arias M, Gallardo C, Kramer S and Penrith ML, 2017. African swine fever: detection and diagnosis – A manual for veterinarians. FAO Animal Production and Health Manual No. 19. Rome. Food and Agriculture Organization of the United Nations (FAO).

Blome S, Franzke K and Beer M, 2020. African swine fever: A review of current knowledge. Virus Research 287: 198099. https://doi.org/10.1016/j.virusres.2020. 198099

Food and Agriculture Organization of the United Nations (FAO), Situation in Asia update, March 5, 2020.

Gallardo MC, Reoyo AT, Fernández-Pinero J, Iglesias I, Muñoz MJ and Arias ML, 2015. African swine fever: a global view of the current challenge. Porcine Health Management 1: 21. https://doi:10.1186/s40813-015-0013-y

Gallardo C, Soler A, Rodze I, Nieto R, Cano-Gómez C, Fernandez-Pinero J, Arias M, 2019. Attenuated and non-haemadsorbing (non-HAD) genotype II African swine fever virus (ASFV) isolated in Europe, Latvia 2017. Transboundary and Emerging Diseases 66: 1399-1404. https://doi.org/10.1111/tbed.13132

Ge S, Li J, Fan X, Liu F, Li L, Wang Q, Ren W, Bao J, Liu C, Wang H, Liu Y, Zhang Y, Xu T, Wu X and Wang Z, 2018. Molecular characterization of African swine fever virus, China, 2018. Emerging Infectious Diseases 24: 2131–2133. https://doi.org/10.3201/ eid2411.181274

Pikalo J, Zani L, Hühr J, Beer M and Blome S, 2019. Pathogenesis of African swine fever in domestic pigs and European wild boar-lessons learned from recent animal trials. Virus Research 271: 197614. https://doi:10.1016/j.virusres.2019. 04.001

Rodríguez-Bertos A, Cadenas-Fernández E, Rebollada-Merino A, Porras-González N, Mayoral-Alegre FJ, Barreno L, Kosowska A, Tomé-Sánchez I, Barasona J A and Sánchez-Vizcaíno JM, 2020. Clinical course and gross pathological findings in wild boar infected with a highly virulent strain of African Swine fever virus genotype II. Pathogens (Basel, Switzerland) 9: 688. https://doi.org/10.3390/pathogens 9090688

Salguero FJ, 2020. Comparative pathology and pathogenesis of African swine fever infection in swine. Frontiers in Veterinary Science 7: 282. https://doi.org/10.3389/ fvets. 2020.00282

Sánchez-Cordón PJ, Vidana B, Neimanis A, Nunez A, Wikstrom E and Gavier-Widen D, 2021. Pathology of African Swine Fever. In: Understanding and Combatting African Swine Fever, pp: 87-139. Lacolina L, et al. (eds). https://doi.org/10.3920/978-90-8686-910-7_4.

Sánchez-Cordón PJ, Montoya M, Reis AL and Dixon LK, 2018. African swine fever: A re-emerging viral disease threatening the global pig industry. The Veterinary Journal 233: 41-48. https://doi.org/10.1016/j.tvjl. 2017. 12.025

Sanchez-Vizcaino JM, Mur L, Gomez-Villamandos JC and Carrasco L, 2015. Anupdate on the epidemiology and pathology of African swine fever. Journal of Comparative Pathology 152: 9-21. https://doi.org/ 10.1016/j.jcpa.2014. 09.003

Urbano A, Forth JH, Olesen AS, Dixon L, Rasmussen TB, Cackett G, Werner F, Karger A, Andres G, Wang X, Perez-Nunez D, Galindo I, Malogolovkin A, Revilla Y, Alonso C, Gallardo C, Blome S, Arabyan E, Zakaryan H and Ferreira F, 2021. African swine fever virus: cellular and molecular aspects. In: Lacolina L, et al. (eds). Understanding and Combatting African Swine Fever, pp: 25-61. https://doi.org/10.3920/978-90-8686-910-7_2.

Wang T, Sun Y and Qiu HJ, 2018. African swine fever: an unprecedented disaster and challenge to China. Infectious Diseases of Poverty 7: 111. https://doi.org/ 10.1186/s40249-018-0495-3

Zani L, Forth JH, Forth L, Nurmoja I, Leidenberger S, Henke J, Carlson J, Breidenstein C, Viltrop A, Hoper D, Sauter-Louis C, Beer M and Blome S, 2018. Deletion at the 5'-end of Estonian ASFV strains associated with an attenuated phenotype. Scientific Reports 8: 6510. https://doi.org/ 10.1038/s41598-018-24740-1