Postmortem and Histopathological Findings in a High-Mortality Outbreak at Rangamati Pig Farm: Assessing the Potential of "African Swine Fever''

A clinical report submitted in partial satisfaction of the requirements for the degree of Doctor of Veterinary Medicine

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CONTENTS

ABBREVIATIONS

Abbreviations	Elaborations
FVM	Faculty of Veterinary Medicine
CVASU	Chattogram Veterinary and Animal Sciences University
ASF	African Swine Fever
CSF	Classical Swine Fever

LIST OF FIGURES

ABSTRACT

In November 2023, an outbreak of high mortality among pigs was reported at the Pig Development Farm in Rangamati Sadar, Rangamati District, Bangladesh, housing approximately 400 pigs. The outbreak was characterized by sudden high mortality in young pigs following convulsions and the adults also presents with off-feed behavior, dehydration, weakness, and eventual mortality. A detailed postmortem and histopathological evaluation were conducted on an adult pig submitted to the Department of Pathology and Parasitology at Chattogram Veterinary and Animal Sciences University. Gross pathological findings included severe hemorrhagic lesion in skin and subcutaneous muscle, splenomegaly, extensive hemorrhagic lymphadenopathy, petechial hemorrhages in multiple organs, pulmonary edema, and hepatic congestion. Histopathological examination also revealed lymphoid depletion, thrombosis, necrosis, and diffuse hemorrhages in lymph nodes and spleen; alveolar edema and necrotizing bronchopneumonia in the lungs; necrosis of Kupffer cells in the liver; and renal hemorrhages with tubular necrosis in the kidneys. The observed pathological features, particularly the combination of severe hemorrhagic splenomegaly, lymphadenopathy, and disseminated intravascular coagulation-like changes, strongly align with African swine fever (ASF). Given Bangladesh's proximity to ASF-endemic regions such as India and Myanmar, the diagnosis raises serious concerns about the spread and management of ASF. While similarities in gross and histopathological findings with classical swine fever (CSF) may lead to diagnostic challenges, the presence of hallmark features such as severe hemorrhagic splenomegaly and lymph node necrosis is indicative of ASFV infection. This report underscores the necessity for robust surveillance, precise diagnostic tools, and stringent control strategies to address this emerging threat in Bangladesh.

Keywords: *high mortality, multisystemic hemorrhagic lesions, necropsy, histopathology, Pig, Rangamati pig farm, African swine fever.*

CHAPTER I: INTRODUCTION

African Swine Fever (ASF) is a highly contagious viral disease affecting both domestic pigs (*Sus scrofa domesticus*) and wild boars, causing severe economic losses and now a global threat for pig farming. The disease was first reported in East Africa during the early 20th century when European pig breeds were introduced to Kenya, and where developed high fever and hemorrhagic lesion with high morbidity and mortality (Penrith & Vosloo, 2009, Ruedas-Torres et al., 2024). ASF is caused by the African Swine Fever Virus (ASFV), a large, enveloped, double-stranded DNA virus belonging to the Asfarviridae family, replicate in hepatocytes, renal tubular epithelial cells, and endothelial cells and primarily targets monocytes and macrophages (Dixon et al., 2013). The virus can spread via direct contact, fomites, contaminated feed, or through soft ticks (Ornithodoros spp.), which play a significant role in its epidemiology (Gallardo et al., 2015).

ASF is characterized by hemorrhagic fever, like other hemorrhagic fevers seen in human, such as Ebola and Marburg filoviruses. In the acute phase of ASF, the most common lesions are petechial and ecchymotic hemorrhages in multiple organs, including the spleen, liver, kidneys, and lungs, which can progress to disseminated intravascular coagulation. These vascular changes lead to edema, ascites, and hydropericardium, particularly in the subacute phase of the disease (Sun et al., 2017). Although ASF was historically confined to Africa, it has spread to multiple continents, including Europe, Asia, and the Americas, causing significant outbreaks in countries like China, Vietnam, and the Philippines since 2007 (Penrith & Vosloo, 2009). In Europe, highly virulent strains of ASFV genotype II have been prevalent, leading to acute-lethal disease courses characterized by hemorrhagic fever (Gallardo et al., 2015).

Bangladesh is a Muslim dominated country with limited pig farming concentrated in its eastern and northern districts, considered to be free from ASF outbreaks to date. However, the proximity of Rangamati District, a significant pig-farming region, to ASF-endemic areas like India and Myanmar poses a high risk for disease introduction through cross-border movement of infected animals or contaminated products (Penrith & Vosloo, 2009).

However, ASF presents clinical and pathological similarities with Classical Swine Fever (CSF), which is complicating for differential diagnosis. Because, both diseases causes hemorrhage, high fevers, and lymphoid depletion, are necessitating laboratory confirmation for differentiation (Yoo et al., 2020). It's reported that wild pigs act as natural reservoirs without showing clinical signs, and domestic pigs often affected with acute and hemorrhagic disease (Dixon et al., 2020).

This report highlights an outbreak of suspected ASF in Rangamati District, Bangladesh, in November 2023, involving high mortality among pigs at a local farm. Pathological examination of affected animals revealed characteristic gross and histological lesions, underscoring the necessity of rapid surveillance, laboratory diagnostics, and stringent biosecurity measures to mitigate the risk of ASF introduction and spread in Bangladesh. Therefore, the aim of this study is to investigate and confirm the occurrence of African Swine Fever (ASF) in Rangamati District, Bangladesh, through clinical, pathological, and laboratory analyses.

Objectives of this study are:

1. Conduct pathological examinations of affected pigs to identify characteristic gross and microscopic lesions of ASF.
2. Provide recommendations for implementing biosecurity measures and surveillance systems to mitigate ASF risks in Bangladesh.

CHAPTER II: MATERIALS AND METHODS

2.1 Case History:

In November 2023, a mortality outbreak occurred at a Pig Development Farm in Rangamati Sadar, Rangamati District, housing 414 pigs. The farm reported 100% mortality in piglets and significant deaths among adults. Overall clinical signs included convulsions, weakness, dehydration, and sudden death. On November 13, the outbreak began, and on 15th November one adult pig carcass was sent to the Department of Pathology and Parasitology, Chattogram Veterinary and Animal Sciences University, for investigation of the mortality. Necropsy was conducted within 12 hours of death by Dr. Md. Shafiqul Islam, Associate Professor at the Department of Pathology and Parasitology, FVM, CVASU, maintaining standard protocols to ensure accurate and reliable findings. Gross pathological findings were meticulously documented, and tissue samples from vital organs, including the heart, lungs, liver, kidneys, brain, lymph nodes, and intestines, were collected for molecular and histopathological evaluations.

2.2 Postmortem examinations of the pig:

The necropsy procedure for the pig was conducted systematically to ensure thorough examination. The carcass was placed in dorsal recumbency, and an external examination was performed to assess skin and mucosal changes. The thoracic cavity was opened first, followed by the abdominal cavity, carefully avoiding contamination of internal organs. Major organs, including the heart, lungs, liver, spleen, kidneys, gastrointestinal tract, and lymph nodes, were inspected for gross abnormalities, hemorrhages, edema etc.

Figure 1: Physical examination before necropsy and dissection of the carcass

2.3 Collection, preservation processing of samples for histopathology:

Samples of visibly affected tissue were carefully collected, labeled, and placed in plastic containers filled with 10% buffered formalin and Bouin's solution using a volume ten times larger than the size of the tissue. The tissue pieces, around 3-5 cm thick, were cut and preserved for slide preparations

2.4 Processing of tissue for histopathology:

Tissue processing involved a series of steps to prepare samples for sectioning. Preserved tissues were labeled, rinsed under running tap water overnight to remove the fixative, and then dehydrated gradually with increasing concentrations of ethanol (80% for 2 hours, 95% for two 1-hour changes, and 100% for three 1-hour changes) to prevent shrinkage. Clearing was performed with xylene in two 1-hour changes, followed by an additional 2-hour clearing step to ensure thorough removal of ethanol. The tissues were then impregnated with liquid paraffin at 56-58°C, with three 2-hour changes, and left overnight. Finally, the tissues were embedded in melted paraffin and allowed to harden into blocks for sectioning.

2.5 Preparation of section block:

The paraffin-embedded tissue block was placed in a rotary microtome, and sections were cut into thin slices of 3-5 μm thickness until a suitable ribbon was obtained. The ribbon of tissue sections was transferred to a warm water bath (55-58°C) to allow the sections to spread evenly. A small amount of gelatin was added to the water bath to improve the adhesion of the sections to the slides. The sections were carefully placed onto clean, grease-free slides. The slides were air-dried and arranged on a rack for further use.

2.6 Staining of tissue slides:

The tissue slides were carefully stained using Hematoxylin and Eosin (H&E) through a regressive staining process to ensure clear and precise results.

CHAPTER III: RESULTS

3.1 Necropsy findings and gross pathological lesions in various organs:

At necropsy, the affected pig was in moderate body condition, with pale mucous membranes suggestive of anemia and systemic illness. The animal show cyanosis on the ears, abdomen, and limb. Hemorrhagic lesions were observed on subcutaneous region in neck, ventral abdomen, and legs, indicating vascular damage which is consistent with African Swine Fever (ASF). No external injuries, wounds unrelated to hemorrhage, or ectoparasitic infestations were noted. Necropsy, was conducted following standard operating protocols, revealed additional gross lesions across multiple organs:

Trachea: Frothy exudates were observed within the tracheal lumen.

Esophagus: The mucosal lining of the esophagus exhibited prominent hemorrhages.

Lungs*:* The lungs were markedly edematous and congested. Pleural surfaces showed petechial and ecchymotic hemorrhages, and the bronchi were filled with blood-stained exudate. Multifocal areas of consolidation and dark hemorrhagic areas also observed in both lungs. Frothy fluid in the bronchi also appeared due to edema.

Heart*:* Severe hydropericardium is observed, characterized by an accumulation of straw-colored fluid in the pericardial sac. Ecchymotic hemorrhages were noted on the epicardium and myocardium. The myocardium appeared pale and mottled, with areas of subendocardial hemorrhage.

Liver*:* After opening the abdominal cavity, red colored fluid appeared due to hemorrhage. The liver showed significant congestion and was grossly enlarged with a darkened appearance. The surface was smooth but mottled with areas of dark red discoloration, indicating severe parenchymal hemorrhages. The cut surface revealed extensive blood-filled sinusoids and multifocal areas of necrosis.

Spleen*:* The spleens of affected pigs demonstrated severe hemorrhagic splenomegaly, characterized by rounded edges and black coloration due to extensive blood pooling a significant portion of the abdominal cavity. On cross-section, the red pulp appeared hyperemic, filled with red blood cells, and interspersed with fibrin thrombi, disrupting the normal architecture.

Kidney*:* Petechial hemorrhages were observed on the cortical surfaces of the kidneys, along with severe perirenal edema that caused tissue expansion and distortion of the organ's normal structure. The kidneys also displayed multifocal infarcts, and upon sectioning, the medulla appeared dark and congested.

Lymph Nodes*:* A multifocal hemorrhagic lymphadenitis is noticed during necropsy. Due to extensive and multifocal hemorrhages it becomes marbled appearance with prominent congestion and edema. The most affected lymph nodes were the gastrohepatic, renal, and mesenteric nodes which exhibited severe hemorrhagic lymphadenopathy. The cut surface revealed dark red discoloration and extensive areas of necrosis, obliterating normal lymphoid architecture.

Adrenal Gland: Marked hemorrhage and necrosis found in both gland.

Intestines*:* The intestinal serosa displayed multifocal petechial hemorrhages, and hemorrhagic colitis was prominent in sections of the small and large intestines.

Urinary Bladder: The bladder mucosa showed extensive petechial hemorrhages and was thickened due to submucosal edema.

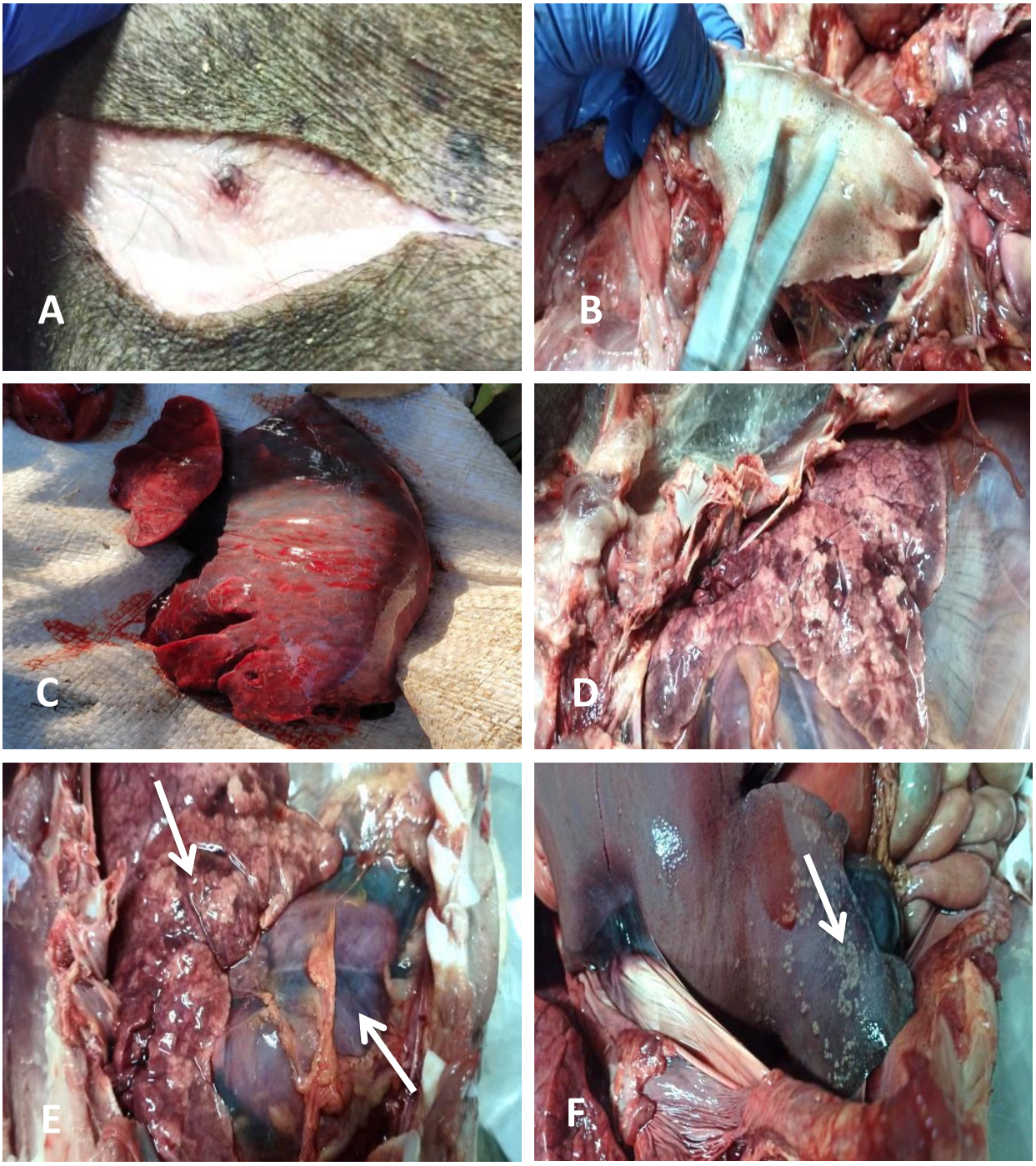

Figure 2. (A) Subcutaneous hemorrhage **(B)** Frothy exudates in trachea (**C, D)** Edema congestion severe hemorrhages in lung **(E)** Consolidation in lung with hydropericardium **(F)** Cogestion and pyogranulomatous lesion in liver.

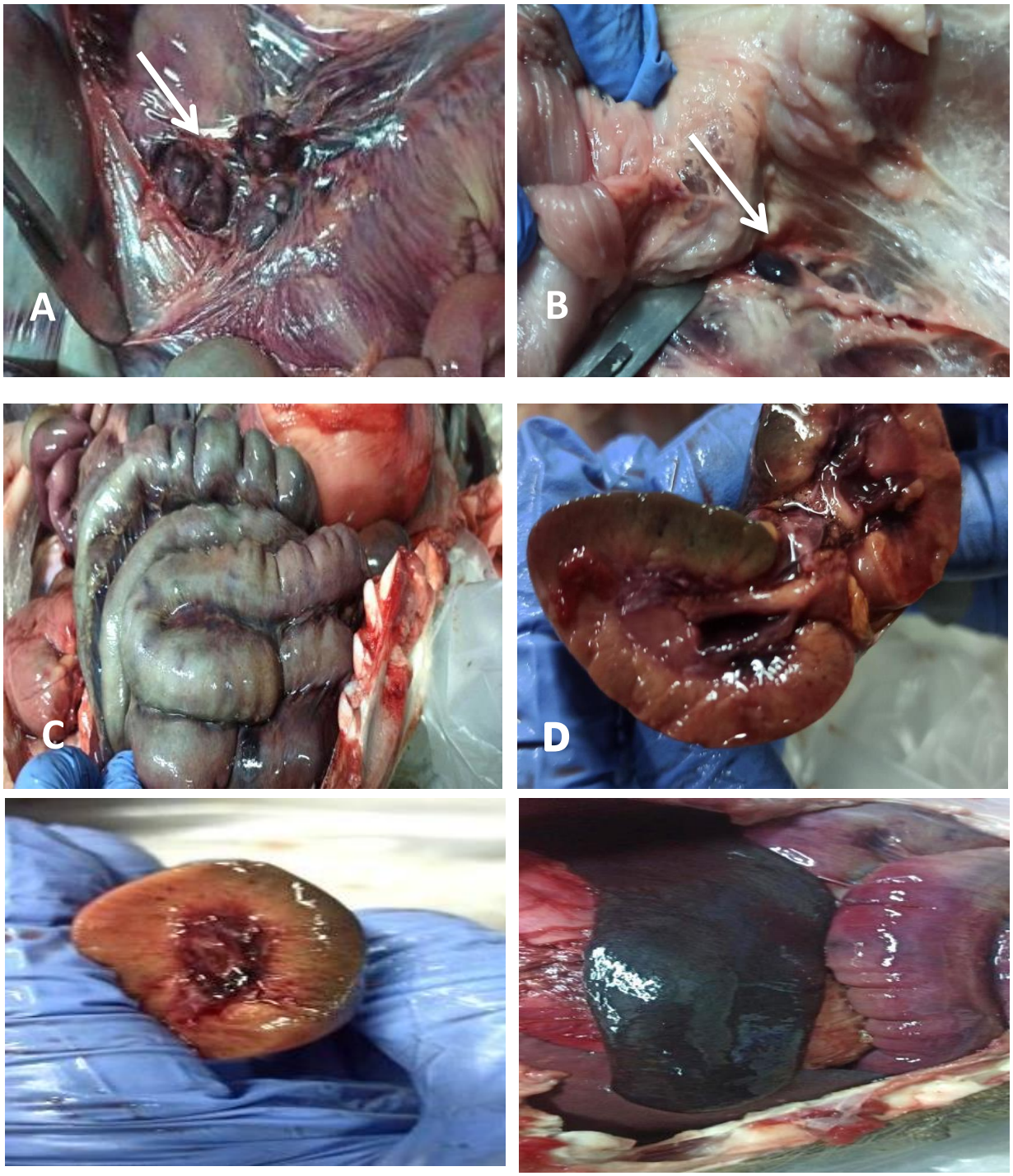

Figure 3. (A, B) Hemorrhagic lymphadenopathy in the mesenteric lymph nodes (arrow) **(C)** Severe hemorrhage in intestine **(D, E)** Petechial hemorrhage in kidney **(F)** **S**plenomegaly observed at the opening of the abdominal cavity with dark colored and rounded edges (hemorrhagic splenomegaly).

3.2 Histopathological findings in various organs:

***Lymph Nodes**:* Histopathology revealed extensive lymphoid depletion and necrosis in cortex and medullary cords. These changes were accompanied by diffuse hemorrhages, leading to the effacement of the normal lymph node architecture. Fibrin deposition and mixed cellular infiltrates of macrophages and neutrophils were evident, indicating the destruction of the lymphoid tissue.

***Spleen**:* The spleen exhibited severe lymphoid depletion, with a loss of lymphocytes in the white pulp and widespread necrosis of myelomonocytic cells. The red pulp showed marked congestion, with fibrin in the sinusoids. Marked hemorrhages is found which disrupting the normal splenic architecture. Reticular fibers in the red pulp were also fragmented, and necrotic debris was prominently found in many foci.

***Lungs**:* The lung tissue displayed alveolar edema, with proteinaceous fluid filling the alveolar spaces. Congestion of pulmonary vessels was evident, with severe hemorrhages expanding the interalveolar septa. Necrosis of the alveolar epithelium, infiltration of inflammatory cells, and formation of hyaline membranes along the alveolar walls indicating necrotizing interstitial pneumonia. Presence of neutrophilic infiltration in bronchi and alveoli indicating fibrino-purulent bronchopneumonia.

***Liver**:* The liver showed extensive necrosis of hepatocytes and Kupffer cells. Mixed cellular infiltration, primarily of lymphocytes and macrophages, was present around blood vessels and bile ducts. The hepatic sinusoids were dilated and filled with blood, and multifocal areas of parenchymal necrosis were prominent. Some focus also shows degenerative changes including fatty change.

***Kidney**:* The kidneys exhibited diffuse cortical and medullary hemorrhages, disrupting the normal structures. Congestion and fibrin deposition were observed in the glomeruli, contributing to ischemic damage. degeneration and necrosis of tubular epithelial cells accompanied by mononuclear cell infiltration indicating tubulointerstitial nephritis.

***Adrenal Gland**:* The adrenal glands showed extensive cortical and medullary hemorrhages, with focal necrosis of adrenal cells. Mononuclear cell infiltration surrounded necrotic foci, and the medullary region exhibited structural disorganization.

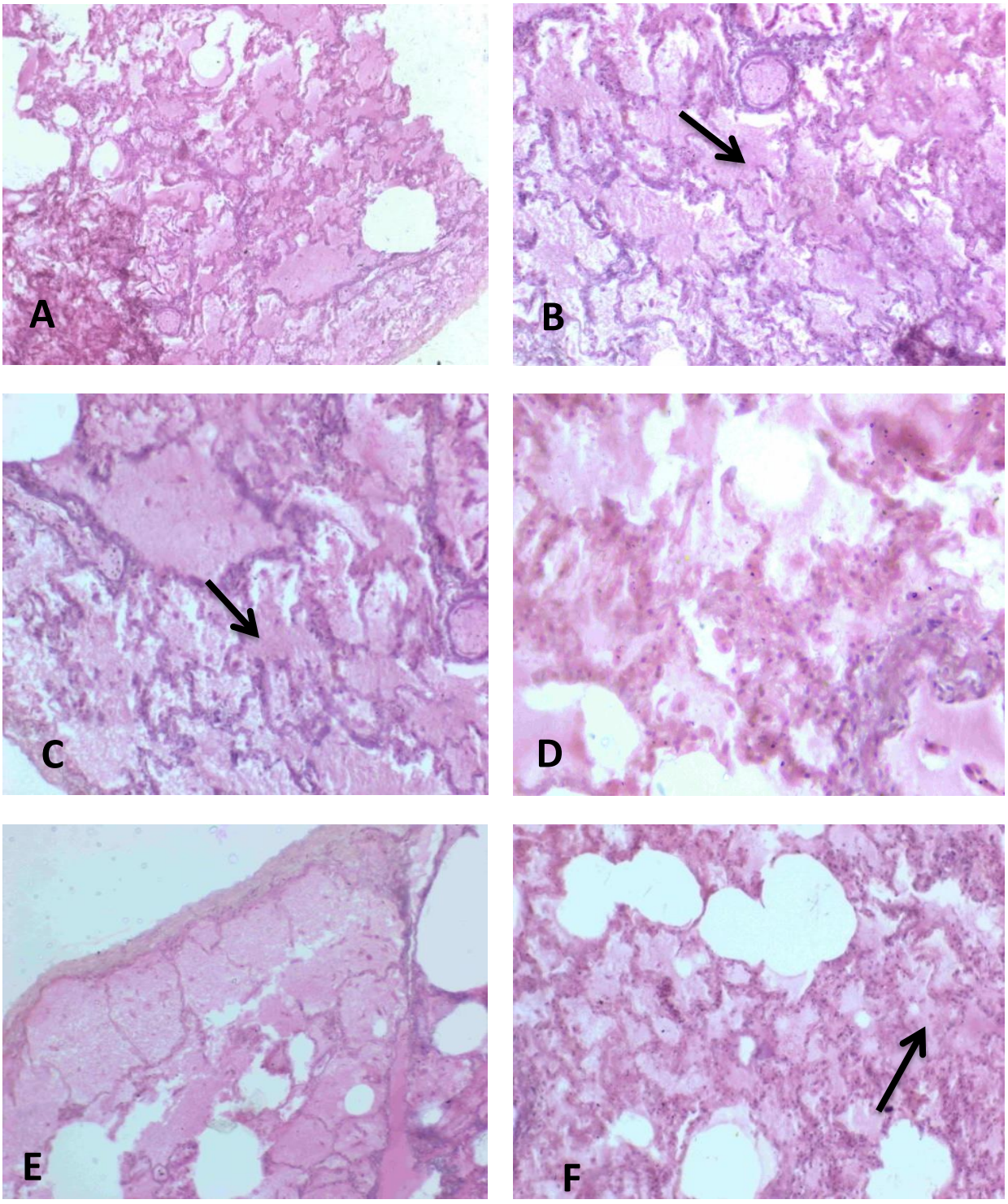

Figure 4: Histopathological examination in lung reveals: (A-C) Congestion of pulmonary vessels with severe hemorrhages and pink colored protienacious fluid in alveoli (D, E) Infiltration of neutrophils, macrophages and fibrin along the alveolar walls indicating fibrino-purulent bronchopneumonia (F) neutrophilic infiltration in bronchi and alveoli

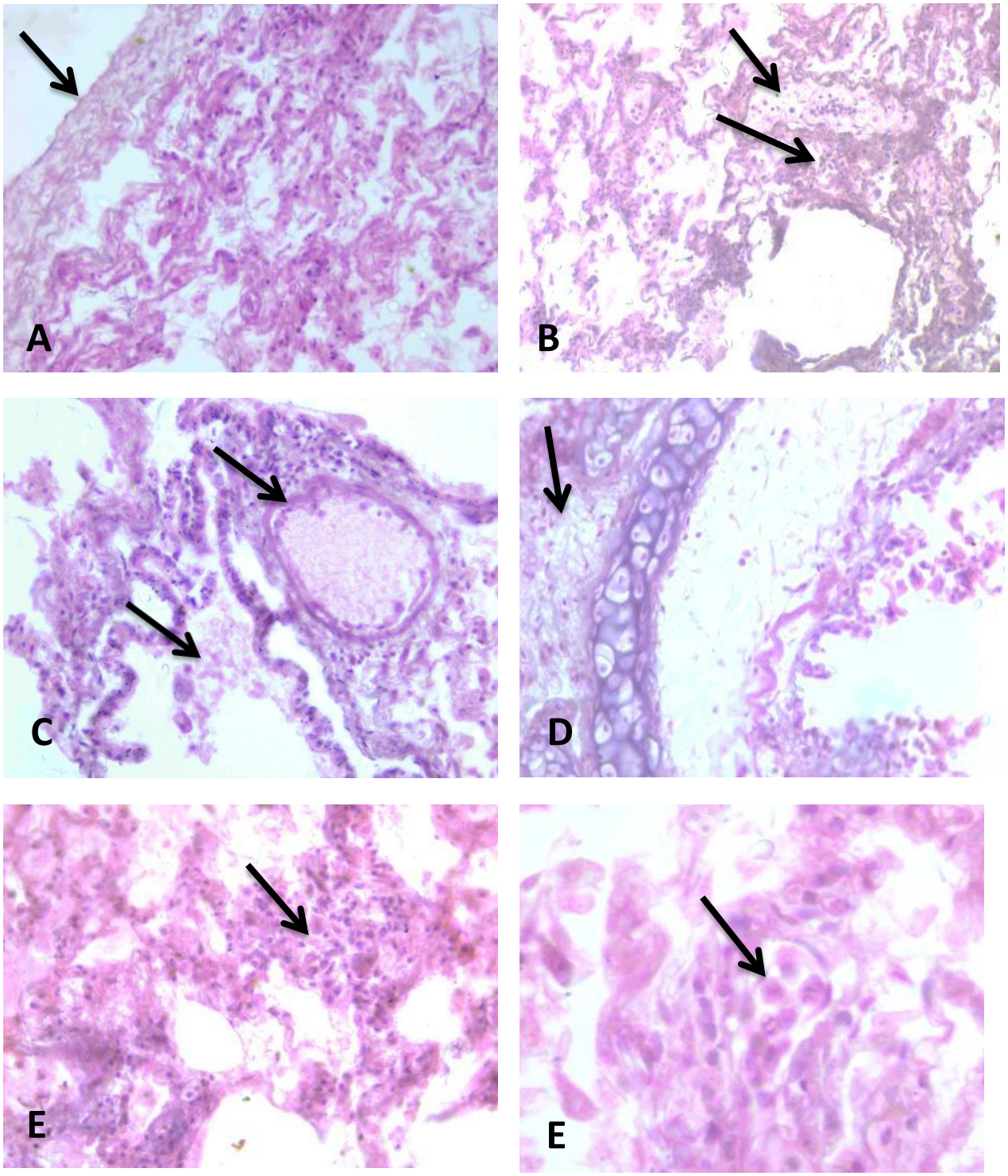

Figure 5: Histopathological examination in lung reveals: (A) Pleural effusion and thickening with infiltration of nutrophil (B) Hemorrhage with inflammatory cells in alveolar septa (C-E) Mononuclear cell infiltrates around the blood vess, alveolar septa and bronchi with fibrinous infitrates (F) Plasma cells accumulstion within the alveoli

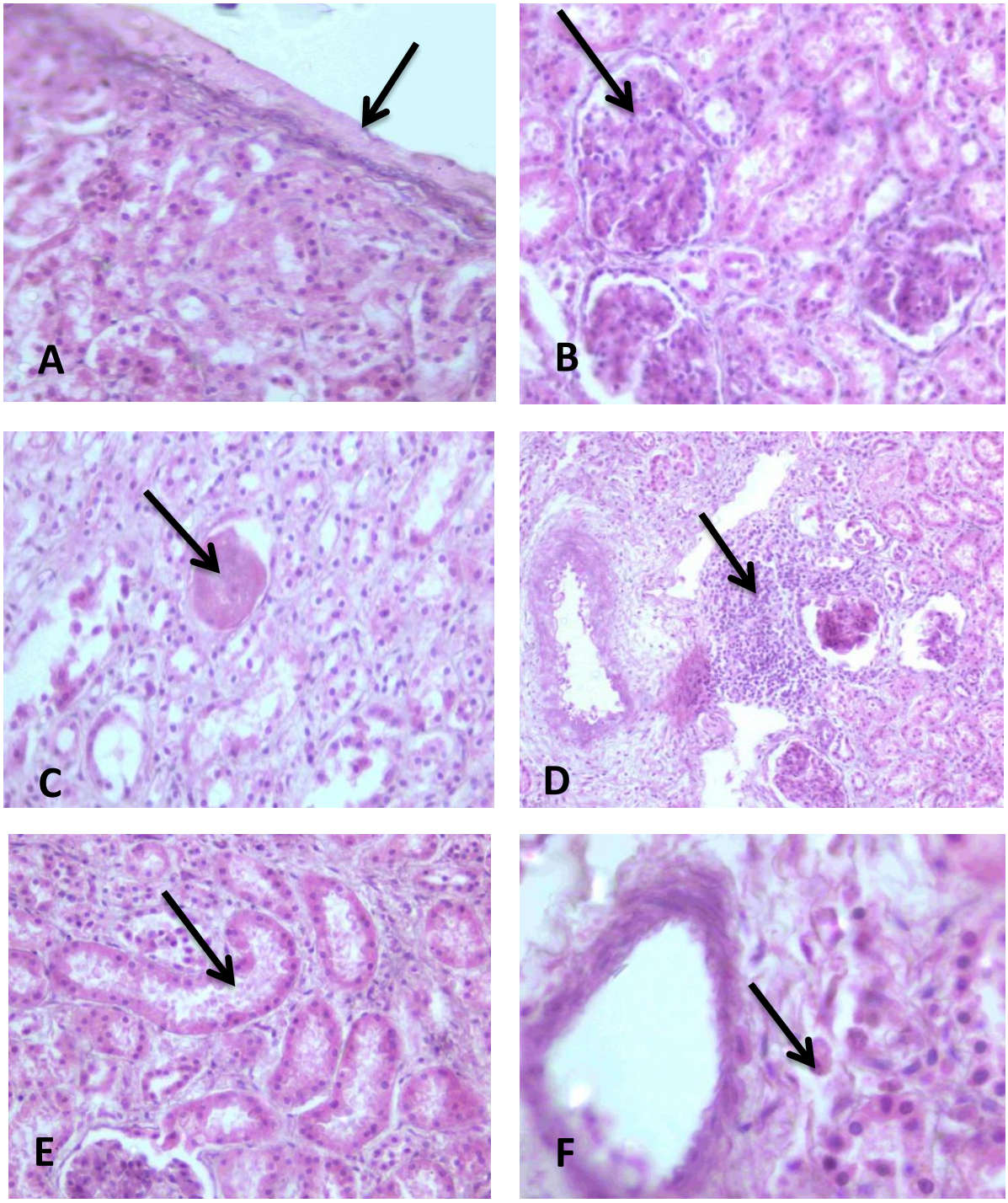

Figure 6: Histopathological examination in kidney reveals: (A) Capsular thickening with inflammatory cells (B) Congestion in glomeruli (C) Congestion, hemorrhage and tubular necosis (D) Mononuclear cell infiltration (E) Tubular degeneration in kidney (F) plasma cell infiltration

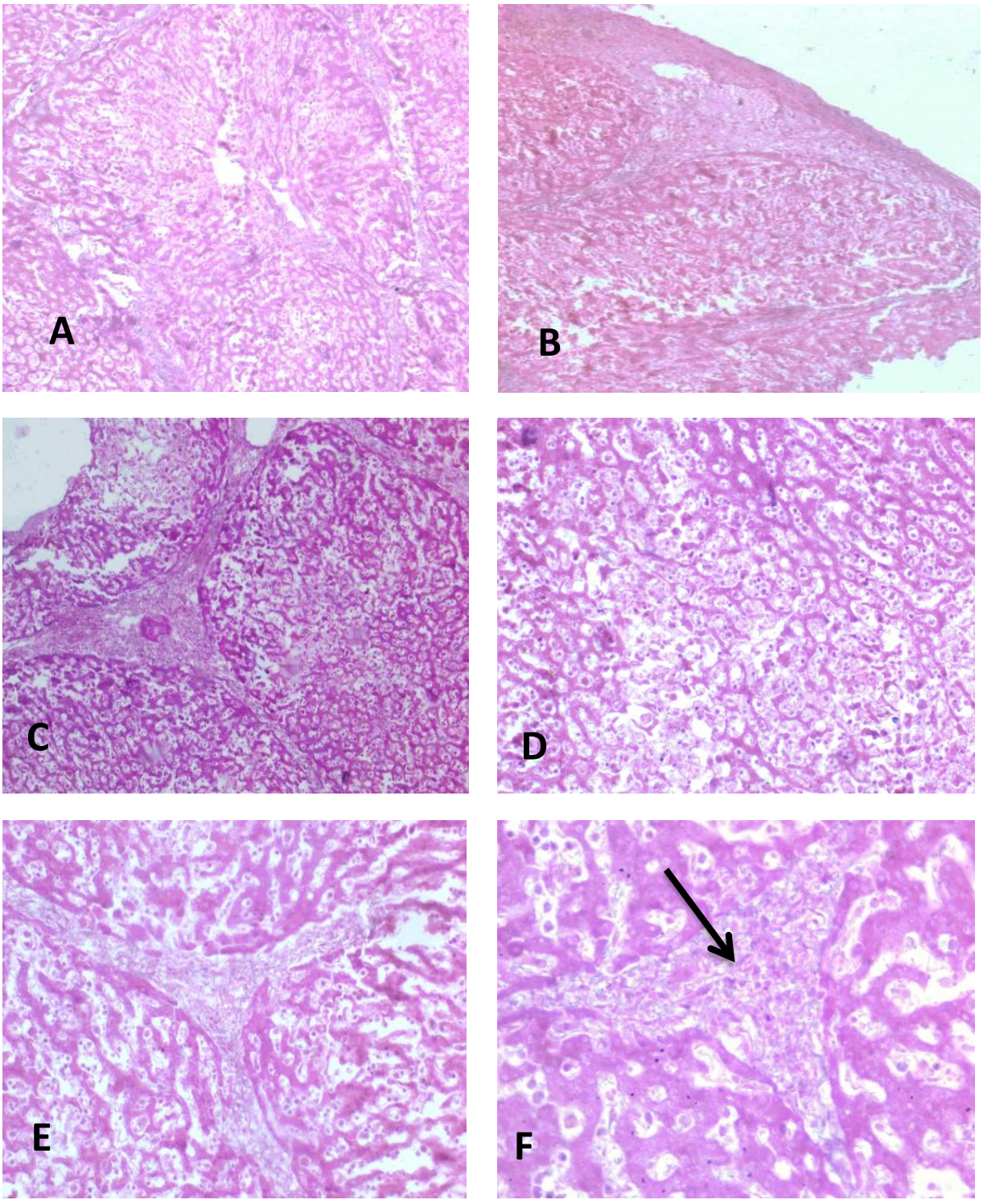

Figure 7: Histopathological examination in liver reveals (A) Hepatic cord necrosis and hemorrhage (B) Sub-capsular hemorrhage (C) Centrilobular necrosis in liver with fatty change (D-F) Hemorrhage and periportal infiltration of reactive cells

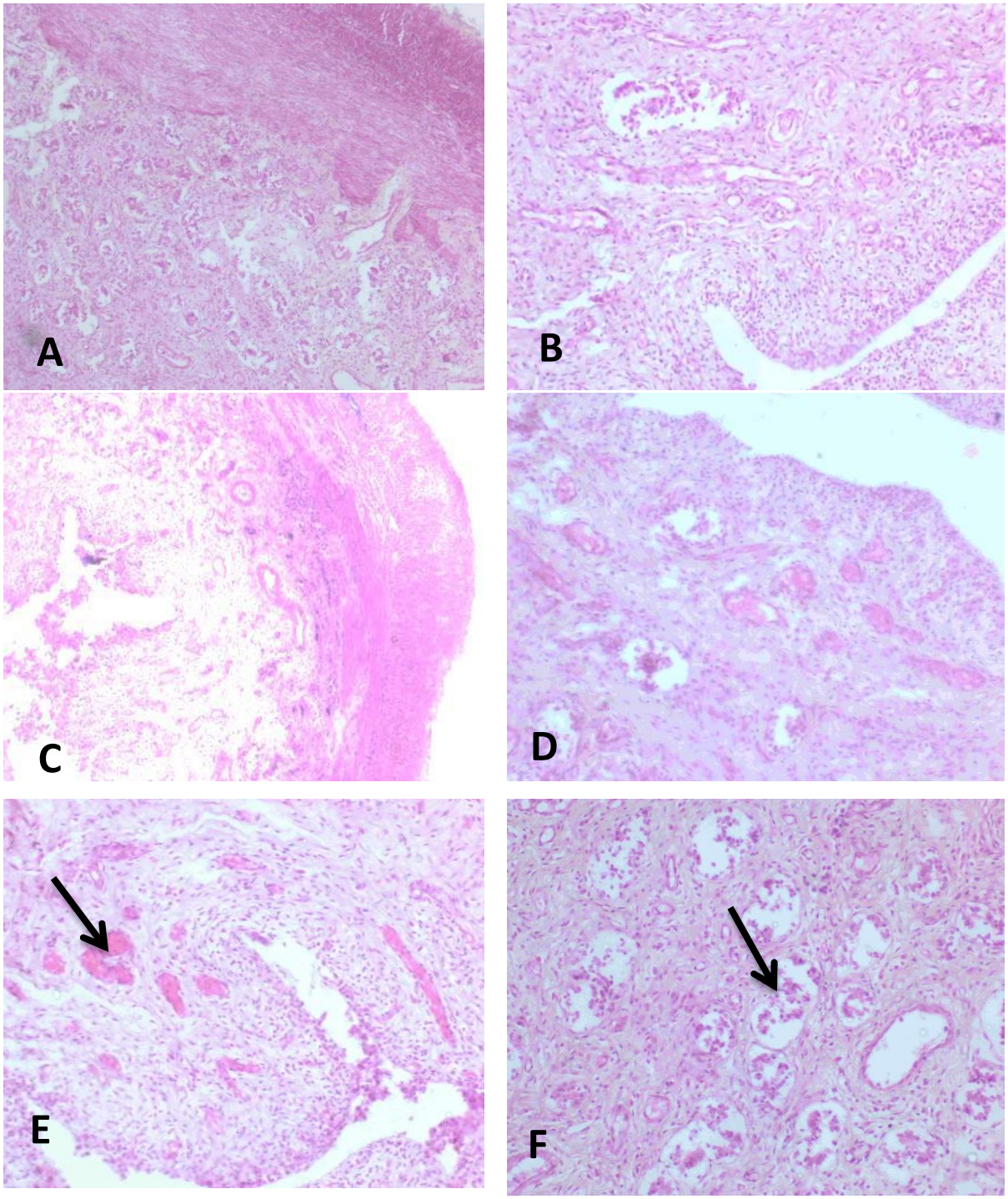

Figure 8: Histopathological examination in intestine reveals (A,B) Extensive hemorrhage in mucosal lining and lamina propria with infiltration of mononuclear cells (C,D) Blunting of villi with hemorrhagic destruction in lining epithelium and gland (F,F) Severe congestion in mucosal crypts with necrosis.

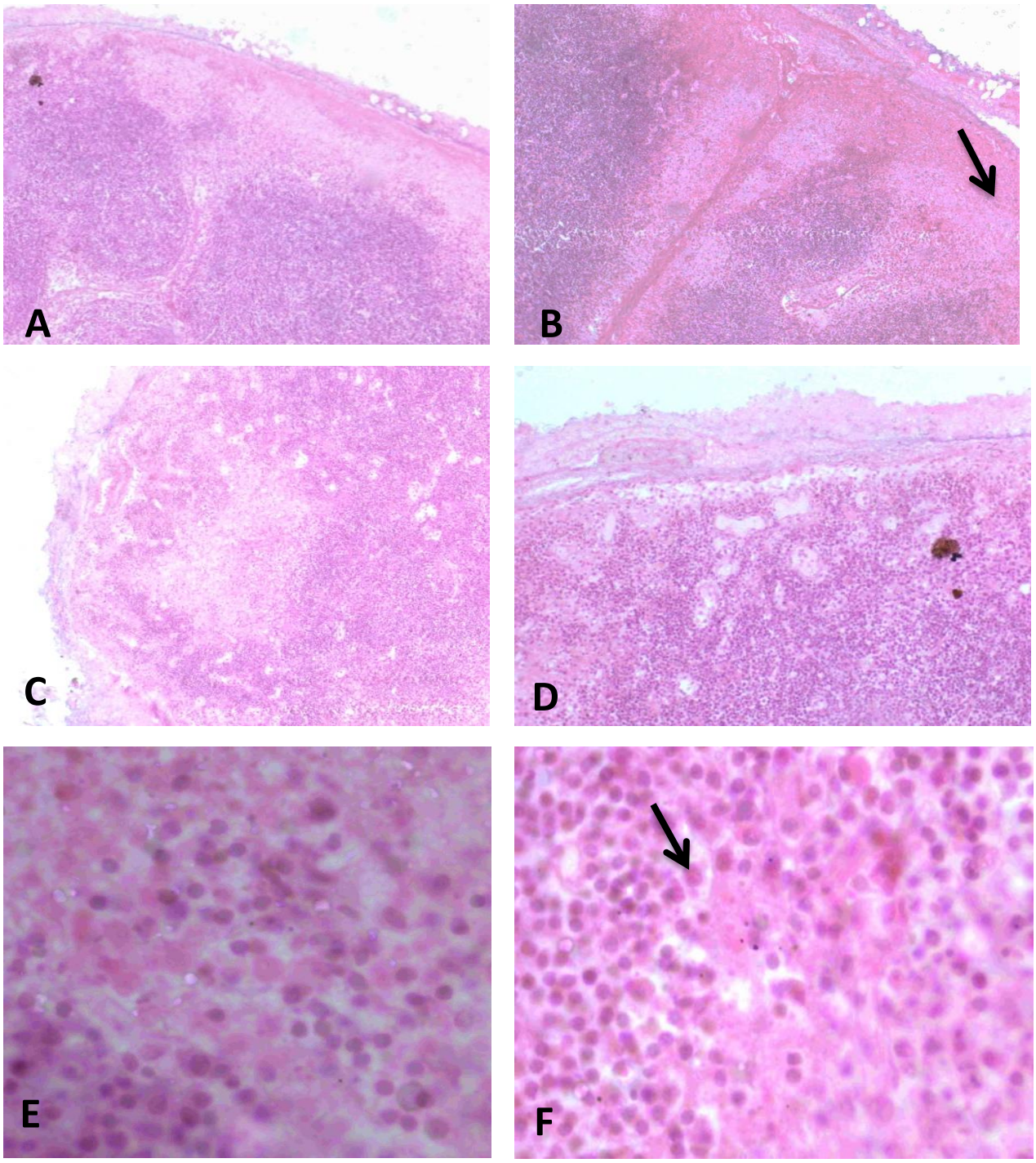

Figure 9: Histopathological examination in LN reveals: (A,B) Extensive lymphoid depletion and necrosis in cortex and medullary cords (C,D) Diffuse hemorrhages, leading to the effacement of the normal lymph node architecture (E,F) Fibrin deposition and mixed cellular infiltrates of macrophages and neutrophils were evident.

This comprehensive gross and histopathological analysis highlight the systemic nature of ASFV infection, with profound vascular changes and necrotizing lesions affecting multiple organ systems. These findings underscore the virulence of ASFV and its devastating impact on affected animals.

CHAPTER IV: DISCUSSION

African Swine Fever (ASF) is a highly contagious viral disease that poses a significant threat to both domestic and wild pig populations worldwide. In this study, we aimed to identify and characterize ASFV outbreaks in Rangamati District, Bangladesh, through clinical, pathological, and laboratory analyses. Our findings highlight the devastating impact of ASF on local pig populations and provide valuable insights into the disease's pathogenesis, which is essential for the development of effective control measures. The observed clinical and pathological features of ASF are consistent with previous reports from other countries, confirming the pathological conditions of the disease and its potential to cause widespread devastation if not properly managed.

ASF is characterized by hemorrhagic fever with the most common lesions are petechial and ecchymotic hemorrhages in multiple organs, including the spleen, liver, kidneys, and lungs (Salguero, 2020). These vascular changes lead to edema, ascites, and hydropericardium, particularly in the subacute phase of the disease (Sun et al., 2017; Sehl-Ewert et al., 2022). Our study also confirmed these findings, as gross and histopathological examinations of affected pigs which revealed hemorrhagic and hyperaemic splenomegaly, hemorrhagic lymphadenitis, severe congestion and hemorrhage in lung congestion with widespread edema.

Hemorrhages in ASF are particularly pronounced in the later stages of the disease, with affected organs including the kidneys, lymph nodes, and gastrointestinal tract. Although ASFV can replicate in endothelial cells, this is typically observed in the later stages of the disease, and hemorrhages can occur even before viral replication in endothelial cells is evident (Yoo et al., 2020). Phagocytic activation of capillary endothelial cells may lead to endothelial cell hypertrophy and the subsequent occlusion of capillary lumens resulting in hemorrhage. This increase in intravascular pressure promotes the activation of the coagulation system, platelet aggregation, and fibrin deposition, contributing to the formation of hemorrhages and disseminated intravascular coagulation (Ruedas-Torres et al., 2024). In our study, we also observed hemorrhages in the Lungs, kidneys and lymph nodes, which were consistent with this proposed mechanism.

The spleen and lymphnodes, are key affected organ by ASF, and its histopathological changes have been well documented in previous studies. Hyperaemic splenomegaly, a hallmark of ASF, results from red pulp engorgement with red blood cells, thrombi, and cell debris, driven by macrophage necrosis and coagulation cascade activation (Sehl-Ewert et al., 2022; Ruedas-Torres et al., 2024). The severity of this lesion varies depending on the virulence of the virus strain, with more virulent isolates causing more extensive tissue damage (Sehl-Ewert et al., 2022). In our study, we observed the same hemorrhagic splenomegaly in infected pigs, which was accompanied by significant structural damage to the spleen.

Clinically and pathologically, ASF and CSF share overlapping features, particularly hemorrhagic lesions in the spleen and lymph nodes. However, the severe hemorrhagic splenomegaly and extensive lymphadenopathy observed here are hallmark features of ASF, distinguishing it from CSF (Penrith & Vosloo, 2009). Thrombocytopenia, is commonly occuerd in ASF, also occurs in CSF, and may contribute to hemorrhage development in mid-stage infections (Zapata et al., 2014; Blome et al., 2012).

Histopathological findings such as lymphoid depletion, thrombosis, and interstitial pneumonia provide additional diagnostic value, supporting the gross findings. The presence of fibrin thrombi in multiple organs highlights the vascular damage caused by ASFV, contributing to its hemorrhagic manifestations (Gallardo et al., 2015).

Pulmonary edema is another common feature of ASF, which is thought to result from the infection of pulmonary intravascular macrophages (PIMs), the main target cells for ASFV in the lungs. Infected PIMs show signs of secretory activation, leading to the release of proinflammatory cytokines, such as interleukin-1 alpha (IL-1α) and tumor necrosis factor-alpha (TNF-α). These cytokines increase endothelial permeability, allowing fluid to leak into the alveolar spaces and interalveolar septa, resulting in pulmonary edema (Ruedas-Torres et al., 2024). In our study, pigs with ASF showed significant pulmonary edema, which was consistent with the described pathogenesis. Pulmonary edema is often accompanied by pleural effusion and hydrothorax, particularly in subacute cases, as observed in our study.

Hepatic malfunction is thought to play a significant role in the development of the edema, particularly in the later stages of the disease (Sun et al., 2017). Histopathological examination of the liver in ASF-affected pigs reveals congestion, periportal inflammation, and Kupffer cell

activation, all of which contribute to the development of multifocal edema. In our study, the liver showed similar signs of congestion and inflammation, which further supports the notion that liver dysfunction, contributes to the pathogenesis of ASF.

Our study also highlights the ongoing threat of ASF in the surrounding regions, including neighboring countries such as Myanmar, Nepal, Bhutan, and India, where ASF outbreaks have been reported in recent years (Yoo et al., 2020). These outbreaks further emphasize the need for robust surveillance systems and biosecurity measures to prevent the introduction and spread of ASF in Bangladesh. ASF has the potential to devastate the pig farming industry in Bangladesh, which is an important part of the country's agricultural economy. Therefore, it is crucial to implement early detection and containment strategies to minimize the impact of ASF outbreaks.

CHAPTER V: CONCLUSION

In conclusion, our study provides a comprehensive analysis of the pathological features of ASF in Rangamati District, Bangladesh. The observed lesions, including hemorrhagic splenomegaly, pulmonary edema, thrombocytopenia, and multifocal edema, are consistent with previous reports on ASF pathogenesis. The findings from this study underscore the importance of implementing biosecurity measures, improving diagnostic capabilities, and conducting further research to better understand the virus's pathogenic mechanisms. With the continued threat of ASF in neighboring countries, it is imperative that Bangladesh enhances its preparedness for future ASF outbreaks to protect its pig population and ensure food security.

LIMITATION

The study is limited by the small sample size, which may not fully represent the diversity of ASF presentations in the region. Additionally, molecular confirmation of ASFV strains was not included in this study which could have provided deeper insights into virus evolution and transmission dynamics. However this will be reported later.

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All praise goes to the Almighty Allah, who enabled me to complete the clinical report on Postmortem and Histopathological Findings in a High-Mortality Outbreak at Rangamati Pig Farm: Assessing the Potential of "African Swine Fever''

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Biography

Sanjida Karim Chowdhury Lipe, daughter of Mahbubul Karim Md. Yeahia and Hosne Ara Begum, is currently enrolled in the Doctor of Veterinary Medicine (DVM) program at Chattogram Veterinary and Animal Sciences University, within the Faculty of Veterinary Medicine. She completed her Secondary School Certificate (SSC) in 2016 with a perfect GPA of 5.00 from Satkania Govt. Girl's High School, Chattogram. In 2018, she earned a GPA of 4.58 in her Higher Secondary Certificate (HSC) from Kapasgula City Corporation Mohila College, Chattogram. Aspiring to become a veterinary surgeon, she also aims to lead a life filled with goodness and integrity.