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Pathology of Porcine Circovirus 2, Porcine Parvovirus 1 and *Streptococcus suis* Co-infections in Postweaning Multisystemic Wasting Syndrome in Piglets

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ABSTRACT

This study investigated an outbreak of PMWS during April–June, 2025 in an organized pig farm in Bareilly, Uttar Pradesh, India to elucidate the pathological interplay among porcine circovirus 2, porcine parvovirus 1 and *Streptococcus suis* in piglets. Postweaned piglets (n=10) showing progressive wasting, respiratory distress and followed by death were examined by detailed necropsy, histopathology, PCR and immunohistochemistry. Grossly, piglets were emaciated and showed hydrothorax, fibrinous pleuritis and pericarditis, severe pulmonary lesions, lymphadenopathy, hepatic necrosis, enteritis, nephritis and cerebral congestion, indicating multisystemic involvement. PCR detected PCV2 and PPV1 in all 10 piglets and *S. suis* in six, while all were negative for PCV3. Microscopically, interstitial and bronchointerstitial pneumonia, marked lymphoid depletion with giant cells formation, myocarditis with chronic fibrinous pericarditis, granulomatous enteritis, interstitial nephritis and hepatic degeneration were prominent. Masson's trichrome and Brown and Brenn stains demonstrated fibrinous pericarditis and bacterial colonies, respectively. IHC demonstrated PCV2 antigen in bronchial epithelium and lymphoid tissues and PPV1 antigen in intestinal mononuclear cells and myocardium, supporting active viral association in lesion development. The concurrent detection of *S. suis* in pigs with severe serositis and cardiac lesions exacerbated disease severity. In conclusion, the findings confirmed a multifactorial pathogenic association of PCV2, PPV1 and *S. suis* in PMWS affected piglets, with coinfections intensifying lesion severity and broadening organ tropism.

KEYWORDS: Pathology, PMWS, PCV2, PPV1, *Streptococcus suis*

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1. INTRODUCTION

Postweaning multisystem wasting syndrome (PMWS) is important clinical manifestations of porcine circovirus associated disease (PCVAD) which causes considerable economic losses globally despite vaccination against primary pathogens. The disease is challenging problem in pig health management due to viral mutations, diverse clinical manifestations, immunosuppression and co-infections. PMWS is characterized by generalized wasting, dyspnoea, skin lesions swelling of superficial inguinal lymph nodes and frequently jaundice (Kim et al., 2002). The Porcine circovirus 2 (PCV2), single stranded circular DNA virus belongs to the family *Circoviridae* is the principal etiology of PMWS. PCV2 associated with PMWS has been reported from northern India and southern states (Rajkhowa and Saikumar, 2012; Keerthana et al., 2019). Early studies on PMWS revealed that the tissues of affected pigs contained porcine circovirus antigens and nucleic acid. Though PCV2, is not adequate to produce clinical manifestations but concurrent infections with other viral and bacterial pathogens causes severity of clinical syndrome. Multiple pathogens are involved and it varies herd to herd. The PCV2 and porcine parvovirus 1 (PPV1) are two important viral pathogens in domestic pigs, frequently noticed as coinfections in many swine producing countries including India (Sharma and Saikumar, 2010). The occurrence of PPV1 infection in pigs was first reported in early nineteen sixty and currently, it is considered to be endemic in major swine producing countries. The PPV1 infection primarily affects gilts or sows. It leads to significant production loss in herd due to abortion, stillbirth and poor litter size. The PPV1 is a well recognized abortigenic pathogens that causes stillbirth, mummification, embryonic death and infertility (SMEDI). Apart from, porcine reproductive failure, PPV1 causes mortality in post-natal live in unvaccinated herd specially associated with PMWS (Das et al., 2025). PCV2 also associated with reproductive disorder (Vidyarani et al., 2024) and skin lesions as coinfections with swine poxvirus (Sangavi et al., 2026). The immune system's involvement lies at the core of PMWS pathogenesis. The syndrome usually occurs in grower pigs between 8 and 16 weeks of age. Piglets with PMWS show different degrees of cachexia, generalized lymphadenopathy, pneumonia, ulceration of stomach. Microscopically, there are characteristic histological lesions in lymphoid tissues consisting of a variable degree of lymphocyte depletion, often intracytoplasmic inclusion bodies and giant cell infiltration. It causes lymphoid depletion in lymph nodes, tonsil, Peyer's patches, thymus and spleen (Darwich et al., 2004; Krakowka et al., 2004). There are lymphohistiocytic to granulomatous inflammation in vital organs, such as intestine, liver, kidney, lymphoid tissue and lung. Lungs show interstitial to broncho-interstitial pneumonia. Several farms at Uttar Pradesh experienced high mortality in post weaning piglets due to PMWS (Rajkhowa and Saikumar, 2012) In this context, a spontaneous surveillance is necessary for better understanding of etiological association, epidemiology and pathology in endemic areas. The study aimed to investigate and understand the pathological interplay between PCV2 and PPV1 in piglets in Bareilly, Uttar Pradesh.

2. MATERIALS AND METHODS

2.1. Outbreak and sampling

Outbreak was investigated in April–June, 2025 at organized pig farm in Bareilly, Uttar Pradesh, India. Detailed herd history was taken from pig farm. Pigs of the herd were vaccinated against CSFV but no vaccination against PCV2 and PPV1 were being practiced in herd. Farm hygiene and biosecurity measures were generally adequate. The reproductive performance was fair having history of abortion and stillbirths. The piglets were routinely weaned at 21–25-day post farrowing. After 7–14 days post weaning piglets exhibited progressive wasting and poor growth despite maintaining appetite, along with pallor, rough hair coat, generalized weakness, enlarged lymph nodes, and respiratory distress. Some animals showed diarrhea, coughing and in severe cases, sudden death with high case fatality and mortality within the group. Piglet carcasses (n=10) after mortality were submitted to the post mortem facility at Division of Pathology, ICAR-IVRI, Bareilly, Uttar Pradesh. A detailed necropsy was carried out and gross pathological findings of various vital organs like liver, lung, heart, lymph node, intestine, spleen were recorded and these organs were further collected in 10% NBF for histopathological study and some portion of above organs were taken for molecular assay without any preservative.

2.2. Molecular detection

Total DNA was extracted from the collected samples by using DNeasy® Blood and Tissue Kit (Qiagen). The DNA in all the samples was quantified by NanoDropND-1000 spectrophotometer (Nanotechnologies, USA). The extracted DNA was subjected to PCR for diagnosis of PPV1, PCV2, PCV3 and *Streptococcus suis* using diagnostic primers as given in Table 1.

2.3. Histopathological examination

Formalin fixed tissue samples were processed routinely through graded alcohols for dehydration and cleared in xylene. The tissues after all the processes were paraffin wax embedded and 4–5 µ thickness was cut and taken for Hematoxylin and Eosin (H&E) staining as described by Bancroft and Gamble (2008). Histopathological lesions were examined in all the tissues. Special staining like Masson's trichrome and Brown and Brenn staining were performed

Table 1: Primer details of all primer for PPV1, PCV2, PCV3 and *Streptococcus suis*

Sl. No.	Primer sets	Sequence 5' - 3'	Target Gene	Amplicon size (bp)	Reference
1.	PPV F	agttagaataggatgcgaggaa	NS1 of PPV1	265	Xu et al., 2013
	PPV R	gagtctgttggtgtatttattgg			
2.	PCV2 F	cggatattgtagtcctggtcg	ORF2 of PCV2	481	Ellis et al., 1999
	PCV2R	actgtcaaggctaccacagtca			
3.	PCV3F	ccacagaaggcgctatgtc	ORF2 of PCV3	330	Palinski et al., 2017
	PCV3R	ccgcataagggtcgtcttg			
4.	S. suis F	gcagcgtattctgtcaaacg	gdh of *Streptococcus suis*	688	Okwumabua et al., 2003
	S. suis R	ccatggacagataaagatgg			

to demonstrate collagens and bacterial colonies, respectively in paraffin sections.

2.4. Immunohistochemistry

For immunohistochemistry, 4–5 µm paraffin sections mounted on APES (3-Aminopropyltriethoxysilane, Sigma)-coated slides were deparaffinized, rehydrated, and subjected to heat-induced antigen retrieval in citrate buffer containing 0.1 M citric acid and 0.1 M trisodium citrate. Endogenous peroxidase and nonspecific binding were blocked by 3 % hydrogen peroxide and 5% goat serum, and the sections were incubated overnight with primary antibody (1:100) for PPV1 (Anti-PPV Capsid protein VP2 Polyclonal antibody, (Antibody System) and PCV2 (1:200 GeneTex) both raised in rabbit, followed by HRP-conjugated secondary anti-rabbit antibody (Sigma). The reaction was developed with DAB, counterstained with hematoxylin, mounted, and examined under a light microscope.

3. RESULTS AND DISCUSSION

On external examination, all carcasses were markedly emaciated, with prominent bony structures and pale mucous membrane. Gross examination of the carcasses revealed severe systemic involvement with lesions in all vital organs like lung, mediastinal lymph node, liver, heart, spleen, intestine, kidney, mesenteric lymph node, brain and intestine. After opening carcass showed hydrothorax with accumulation of straw-coloured fluid in the thoracic cavity and yellowish-white fibrinous deposition over the surface of the right lung (Figure 1). The lungs were enlarged, edematous, and severely haemorrhagic, with fibrinous adhesion of the left lobes to the pericardium and fibrinous thickening on the posterior surface of the left diaphragmatic lobe (Figure 2). The heart showed fibrinous pericarditis with yellowish fibro-adhesive tissue between pericardium and epicardium (Figure 3). The mediastinal lymph nodes were enlarged and exhibited edema and haemorrhages (Figure 4). Liver showed multiple focal areas of necrosis and spleen

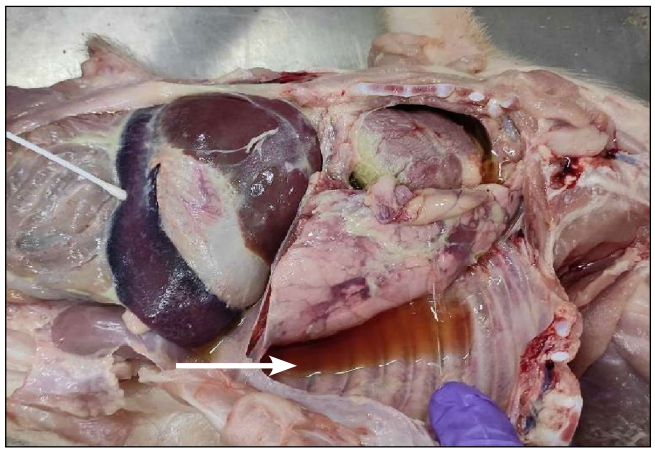

Figure 1: Hydrothorax with presence of straw coloured fluid in thoracic cavity and yellowish white fibrinous deposition over the right lateral surface of lungs

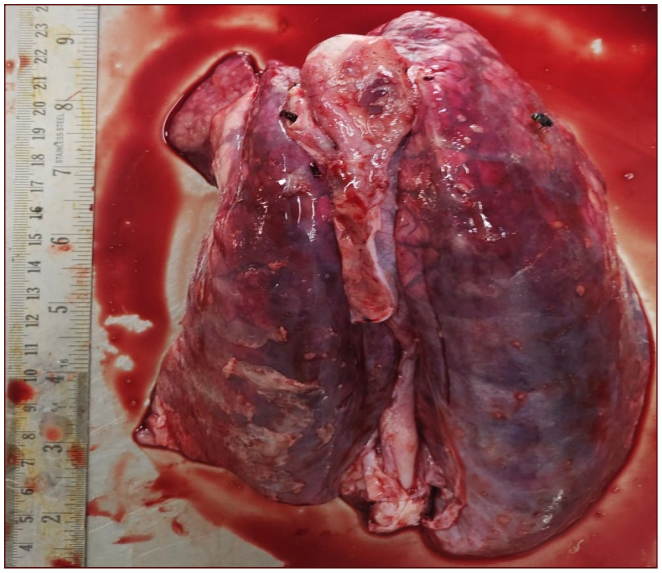

Figure 2: Enlarged, oedematous, severely haemorrhagic lung with fibrinous adhesion of left lobes to the pericardium as well as fibrinous thickening present over the posterior surface of left diaphragmatic lobe

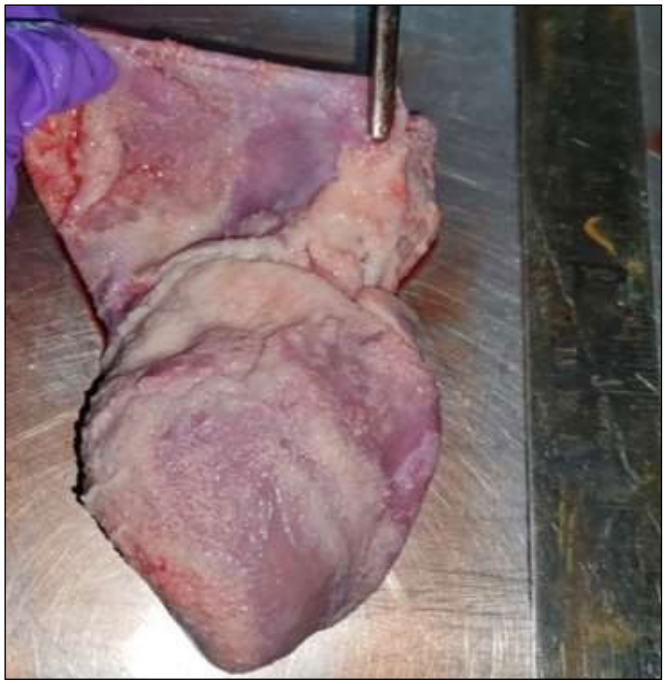

Figure 3: Heart showing inner pericardial and epicardial surfaces with covered with yellowish fibrinous material deposition

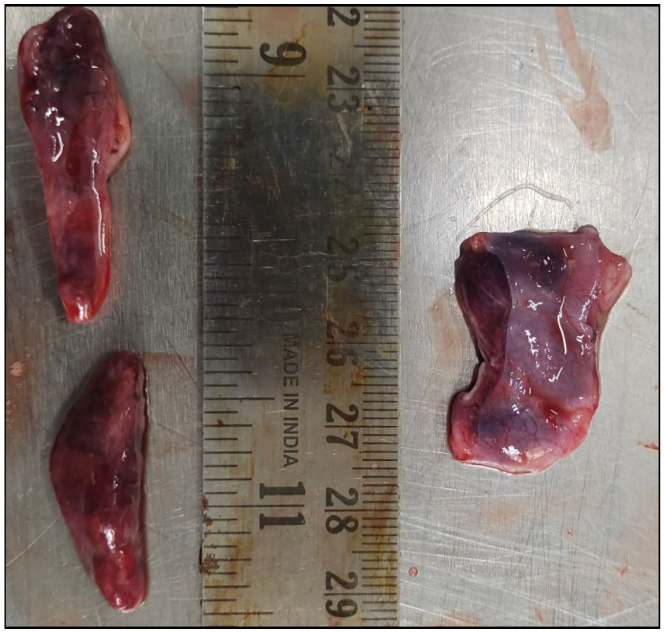

Figure 4: Mediastinal lymph node enlarged with oedema and haemorrhages

showed mild enlargement. Intestine showed thickened mucous membrane with enlarged mesenteric lymph node and mesenteric congestion. Kidney showed focal areas of petechial haemorrhages over the capsule. The brain of all animals showed severe leptomeningeal congestion on the ventral aspect.

Molecular analysis confirmed the presence of PCV2 in all the samples (n=10) by amplification of a 481 bp product targeting ORF2 (Figure 5). *Streptococcus suis* was detected in six piglets (n=6) by amplification of 688 bp product targeting the *gdh* gene (Figure 6). PPV1 was also detected in all the samples (n=10) by amplification of a 265 bp product targeting the NS1 gene (Figure 7). All samples were found to be negative for PCV3.

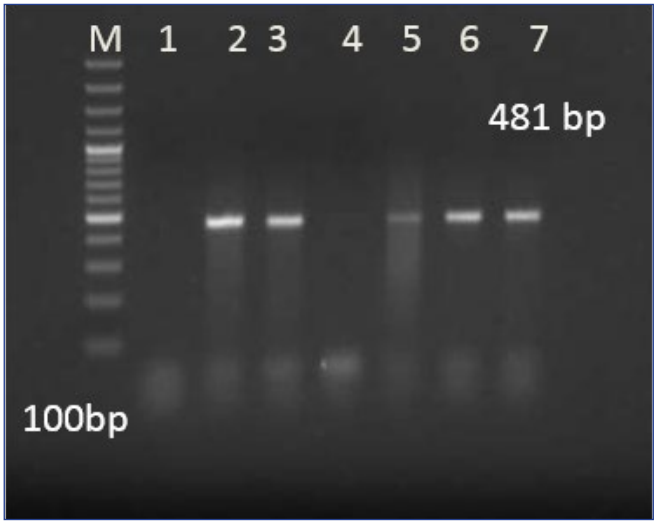

Figure 5: 1.5% agarose gel showing amplification of 481 bp amplicons of PCV2 (ORF-2). Lane M: Ladder (100 bp), Lane 1: NTC, Lane 2: Positive control, Lane 3: Sample ID: 274 A, Lane 4: Negative sample, Lane 5: Sample ID: 99A, Lane 6: Sample ID: 91 A. Lane 7: Sample ID: 92 A

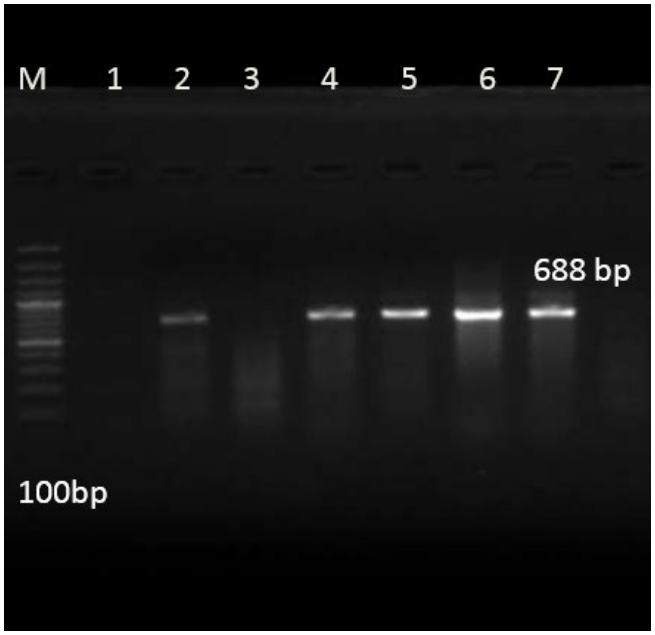

Figure 6: 1.5% agarose gel showing amplification of 688 bp amplicons of S. suis (gdh), Lane M: Ladder (100 bp), Lane 1: NTC, Lane 2: Positive control, Lane 3: Negative sample, Lane 4: Sample ID: 274; A, Lane 5: Sample ID: 99 A, Lane 6: Sample ID: 90 A, Lane 7: Sample ID: 91 A

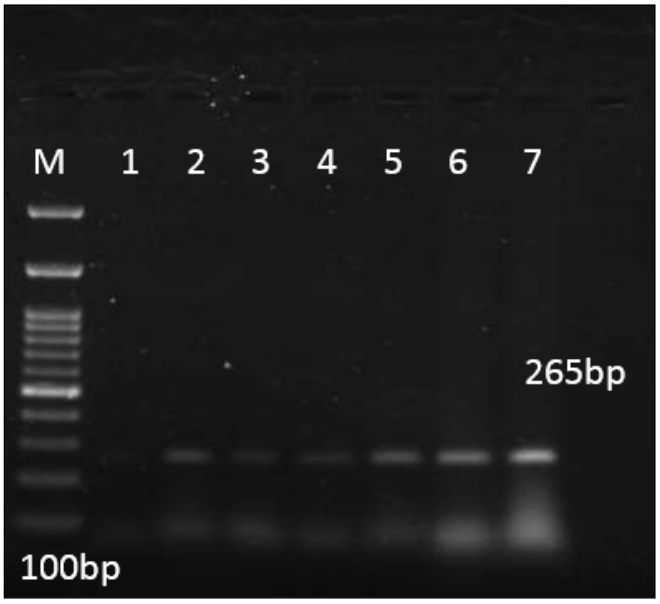

Figure 7: 1.5% agarose gel showing amplification of 265 bp amplicons of PPV1(NS1). Lane M: Ladder (100 bp), Lane 1: NTC, Lane 2: Positive control, Lane 3: Sample ID: 74 A, Lane 4: Negative sample, Lane 5: Sample ID: 90A, Lane 6: Sample ID: 91 A, Lane 7: Sample ID: 92 A

Microscopically lungs exhibited two forms of pneumonia: interstitial and broncho-interstitial pneumonia. Broncho-interstitial pneumonia was noticed in 6 piglets while interstitial pneumonia in 4 piglets. Broncho-interstitial pneumonia was observed in association with PCV2 and PPV1 coinfections with *Streptococcus suis* while coinfection of PPV1 and PCV2 led to interstitial pneumonia. In cases of interstitial pneumonia, lungs showed severe inflammatory cell infiltration within the alveolar spaces (Figure 8), widened alveolar septa due to macrophage activation, and congested alveolar capillaries. In bronchointerstitial pneumonia, there was bronchial epithelial denudation, mononuclear cells infiltration and neutrophils within the bronchial lumen, and mononuclear cell and neutrophil infiltration in the alveolar septa. Brown and Brenn staining of lung showed multiple discrete patches of blue-stained bacterial colonies predominantly located in the peribronchial and interstitial regions in six samples (Figure 9). The lymph nodes showed marked lymphoid depletion, necrotic masses, and giant cell formation within the lymphoid follicles (Figure 10). Histopathologically heart revealed myocardial fibres degeneration with mononuclear cells infiltration and thickening of the epicardium (Figure 11). Masson's trichrome staining in heart revealed predominantly blue stained collagenous fibrosis with minimal red fibrinous deposition over epicardium layer consistent with chronic organized pericarditis (Figure 12). The palatine tonsils exhibited necrosis and depletion of lymphoid cells. Spleen

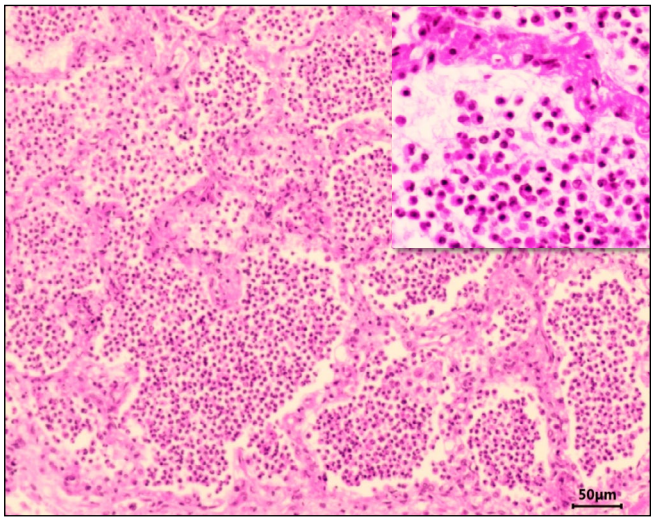

Figure 8: Alveolar spaces showing severe inflammatory cell infiltration. H&E, 4X. Inset, H&E, 20X

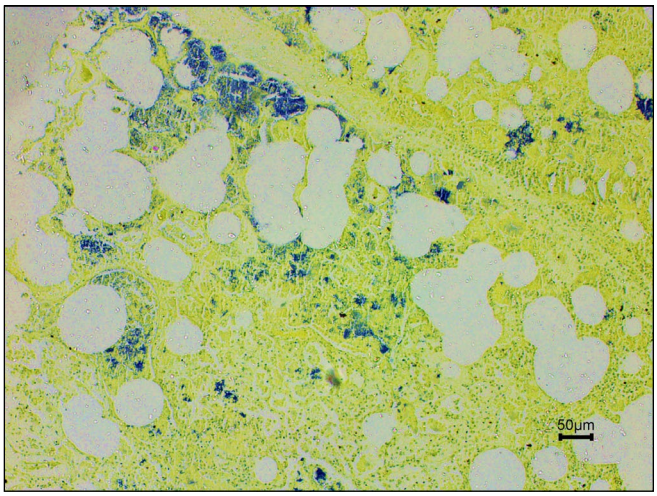

Figure 9: Lung showing multiple discrete patches of blue-stained bacterial colonies predominantly located in the peribronchial and interstitial regions. Brown and brenn 10X

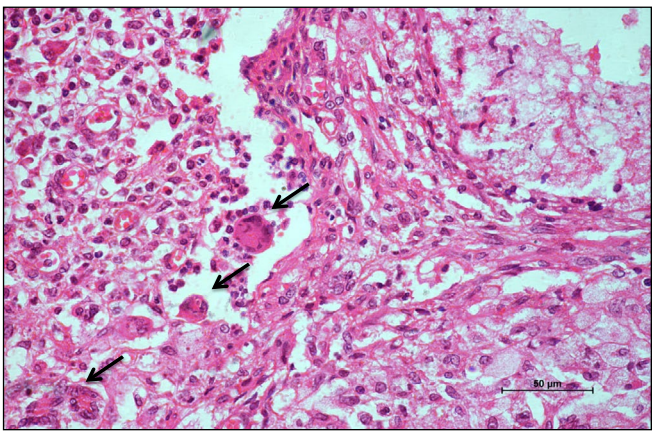

Figure 10: Lymph node showing lymphoid depletion, necrotic mass and giant cells formation in lymphoid follicles H&E 40X

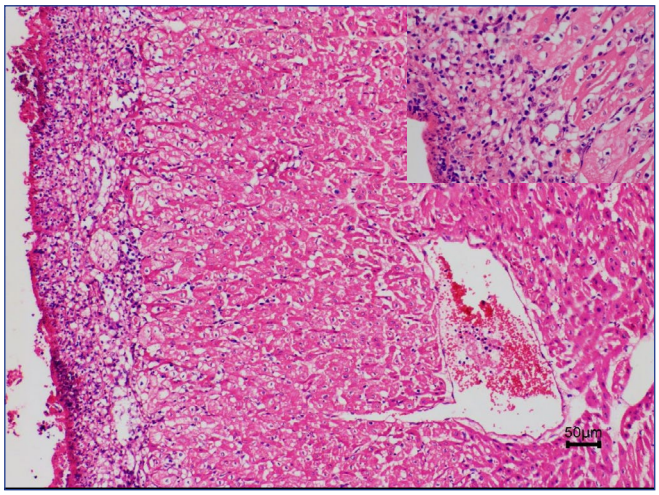

Figure 11: Heart showing thickened epicardium with marked mononuclear cells infiltration and degeneration within myocardial fibres. H&E,10X

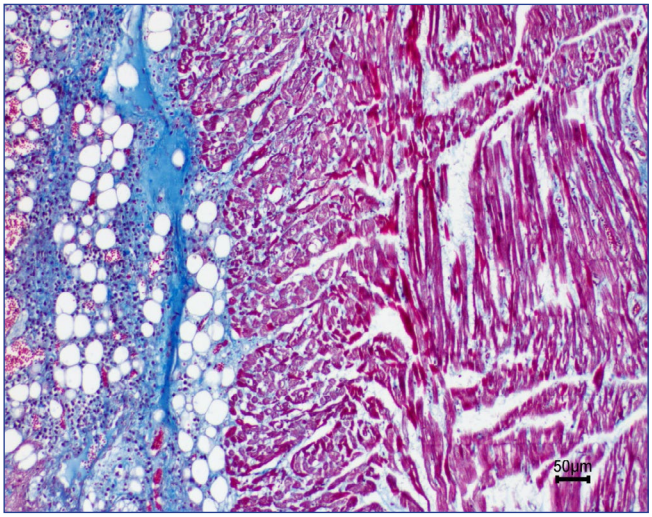

Figure 12: Heart showing blue stained showed predominantly blue collagenous fibrosis with minimal red fibrinous material. MST, 40X

showed lymphoid depletion in the PALS area with congested blood vessels. The liver demonstrated distortion of hepatic cords, severe dilation of sinusoids, and infiltration of mononuclear cells. Kidney section showed glomerular atrophy and mild mononuclear cells infiltration in the interstitium. Intestine revealed lymphoid depletion in the GALT area with severe mononuclear cells infiltration within the lamina propria of colon and jejunum and giant cells formation within the lymphoid tissue. Brain sections exhibited congestion in the cerebral blood vessels. Immunohistochemical examination demonstrated positive labeling for PCV2 antigen in bronchial epithelium (Figure 13A), while the corresponding control sections showed no immunostaining (Figure 14A and B). PPV1 antigen was demonstrated in mononuclear cells within the lamina

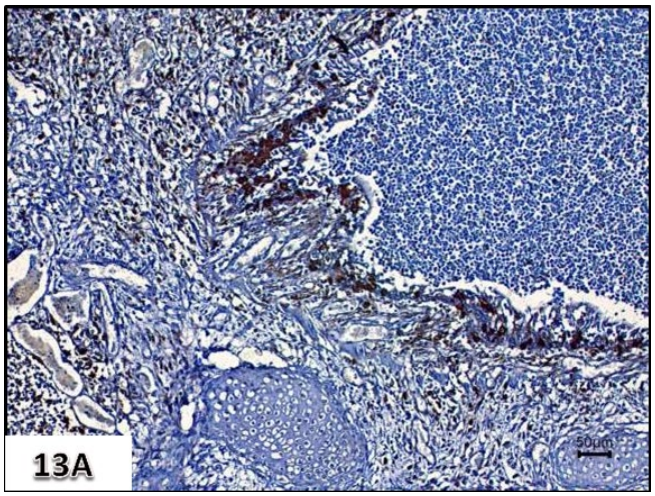

Figure 13 A: Demonstration of PCV2 antigen in bronchial epithelium. IP-DAB-MH10X

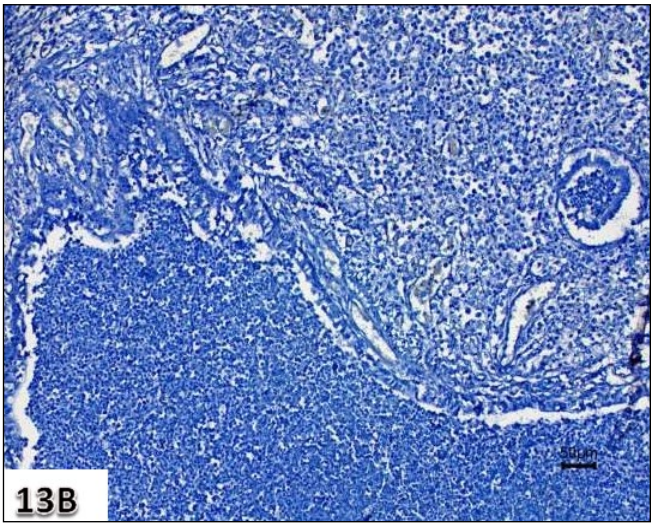

Figure 13 B: Control without primary antibody, IP-DAB-MH10X

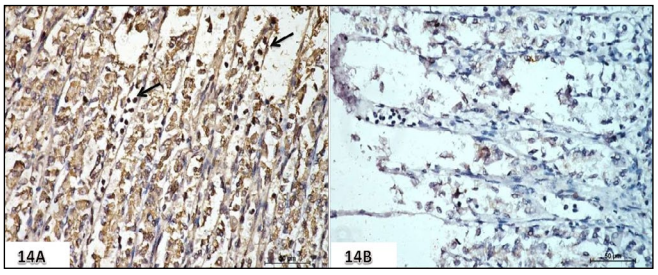

Figure 14: A: Positive immunolabeling of PPV1 antigen in mononuclear cells within lamina propria of intestine IP-DAB-MH 40X; B: Control showing no immunostaining for PPV1 antigen. IP-DAB-MH 10X

propria of the intestine (Figure 15A) and in myocardium cells (Figure 16A), whereas the negative controls remained unstained (Figure 14B, Figure 15B).

Both gross and histopathological examination confirmed

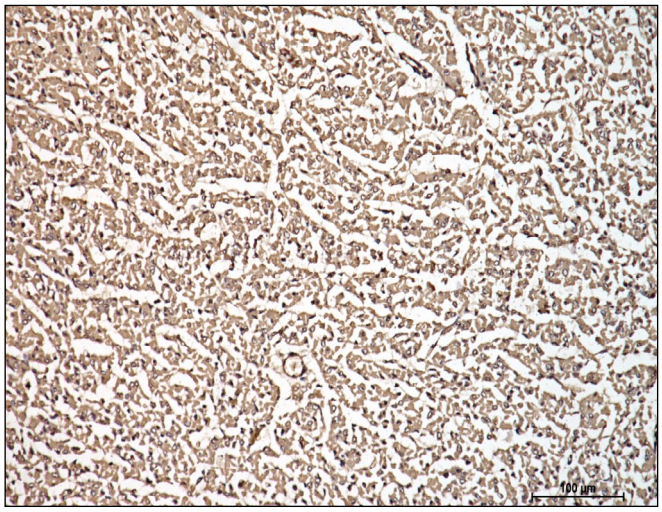

Figure 15A: Positive immunolabeling of PPV1 antigen within myocardium cells. IP-DAB-MH 20X

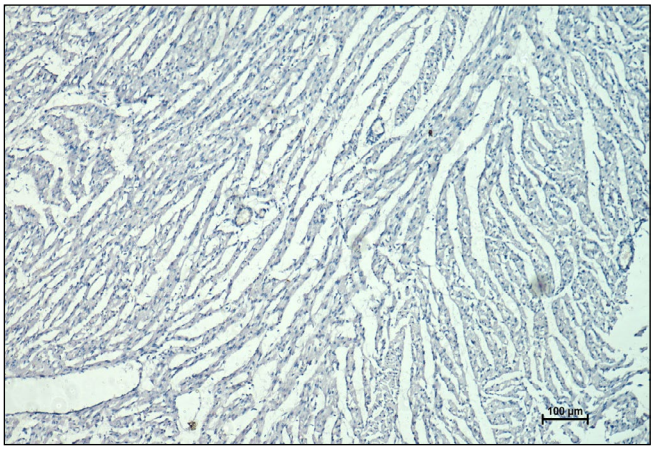

Figure 15B: Control showing no immunostaining for PPV1 antigen. IP-DAB-MH 10X

multisystemic nature of PMWS, with severe interstitial to broncho-interstitial pneumonia, hepatic necrosis, interstitial nephritis, granulomatous enteritis and cerebral congestion, consistent with previous reports of systemic involvement due to multiple pathogens (Segales et al., 2005; Sairam et al., 2023). This suggested that concurrent infection in PMWS might intensify lesion severity and broaden the range of organs tropism (Pallares et al., 2002). Severe wasting and multisystemic involvements were already seen due to PPV1 and PCV2 coinfections in the previous experimental studies (Allan et al., 1999; Sairam et al., 2023).

The cardiac lesions, including fibrinous pericarditis and mononuclear cell infiltration in the myocardium, were also compatible with earlier findings in piglets infected with PCV2 and PPV 1, where myocardial lesions and hydrothorax were reported more prominently in coinfected animals (Pallares et al., 2002). Chronic fibrinous pericarditis as indicated by blue stained fibrous tissue in masson's trichrome stain and other myocardial affections were mainly seen in cases with coinfection of *Streptococcus suis* which was a consistent finding with the previous studies involving bacterial pathogen mainly *Haemophilus parasuis* and *Mycoplasma hyorhinisin* in such cases (Nedbalcova et al., 2006; Ustulin et al., 2021). *Streptococcus suis* infection in piglet mainly involved in cases of endocarditis and polyserositis **(**Tram et al., 2021) but cases of pericarditis have also been seen (Yang et al., 2026).

The respiratory lesions like fibrinous pleuritis, pulmonary edema, hemorrhage, and inflammatory cell infiltration in the alveolar spaces, were in agreement with previous reports that PCV2 infection commonly involves the lungs and contributes to respiratory compromise (Rosell et al., 2000). Interstitial pneumonia and severe mononuclear cells infiltration with pulmonary oedema, consolidation and thickened alveolar septa have been well documented PCVAD lesions in previous reports **(**Rosell et al., 2000; Opriessnig and Langohr, 2013). Severe lymphoid depletion, giant cell formation, and necrotic changes in lymphoid tissues were classical lesions of PCV2 infection and have been widely described in PMWS/PCVAD-affected pigs (Allan and Ellis, 2000; Opriessnig and Langohr, 2013).

In the present cases, the presence of these lesions together with antigen detection in bronchial epithelium further supports the role of PCV2 in producing tissue damage and systemic disease. The detection of PPV1 antigen in the intestine and myocardium, along with PCR confirmation, indicated active involvement of porcine parvovirus in the disease process (Sairam et al., 2023). Earlier work has shown that PPV1 was often clinically mild when present alone, but coinfection with PCV2 could result in more severe pathological changes and worse clinical outcome (Pallares et al., 2002; Segales et al., 2005, Seeliger et al., 2007). Similarly, published studies have reported that PCV2 and PPV1 coinfection may aggravate fetal and piglet lesions, particularly in lymphoid and cardiac tissues. It was been seen that PPV1 favours the replication of PCV2 in the lymphoid system of an animals (Allan et al., 1999; Miłek et al., 2020), thereby enhancing its pathogenicity and contributing more on lymphoid depletion and development of secondary bacterial infection. Piglets born to dams naturally infected with PCV2, possessing maternal antibodies, could still become infected and develop viremia, underscoring incomplete protective efficacy of antibody. Comparable patterns were observed with porcine parvovirus, where maternal antibodies afford only partial shielding, insufficient to fully prevent offspring infection (Dias et al., 2012).

The detection of *Streptococcus suis* alongside PCV2 and PPV1 was also significant, because coinfection with bacterial pathogens has been shown to worsen disease severity in piglets (Opriessnig and Langohr, 2013; Das et al., 2025;

Thomas et al., 2026). *S. suis* involvement in the herd is of great concern to the piglet population as well as its potential risk to human population causing meningitis (Tram et al., 2021). The distribution of antigen in affected tissues further strengthens the association between pathogen presence and the observed histopathological damage. Moreover, vaccination against PCV2 and PPV1 was the most effective measure in breeding stock to reduce viral load. This extended the importance to strengthen the farm management with proper biosecurity measures.

4. CONCLUSION

The multifactorial pathogenic association of PCV2, PPV1 and *Streptococcus suis* in most of pig showing PMWS. The coinfections induced multisystemic wasting disease with severe and diverse pathological lesions in piglets in farm settings.

5. ACKNOWLEDGEMENT

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