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Concurrent Infections of Porcine Circovirus 2 and Swinepox Virus in Crossbred Piglets

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ABSTRACT

The study was conducted during September and October, 2024 at a local farm in Bareilly, Uttar Pradesh, India to investigate the etiologies and pathology of concurrent viral infections in piglets. Post mortem examinations of three crossbred piglet carcasses (38-day-old male, 42-day-old female and 50-day-old female) were conducted having history of reduced feed intake, severe dullness, progressive lethargy, diarrhoea and multifocal cutaneous circumscribed scabs followed by death. Necropsy revealed pericarditis with serous fluid accumulation, pulmonary consolidation with edema, hepatic congestion and necrosis, intestinal lymph nodes congestion, renal tubular degeneration and characteristic cutaneous pock lesions with umbilicated crusts. Histopathology showed interstitial pneumonia, hepatic sinusoidal congestion, sloughing of intestinal villus epithelium, lymphoid depletion in lymph nodes and characteristic swine pox lesions like vacuolar degeneration of keratinocytes in the epidermis with eosinophilic intracytoplasmic inclusion bodies. All tissue samples from the three affected piglets were found to be positive for porcine circovirus 2 (PCV2) and swinepox virus (SWPV) by PCR amplification of *ORF2 and P42* genes, respectively. Organ-wise viral distribution analysis revealed PCV2 predominantly in lymphoid tissues, heart and lungs, while SWPV was predominantly detected in skin and lymphoid tissues. Based on pathomorphological and molecular investigations, the cause of death of the piglets was diagnosed as concurrent infections of PCV2 and SWPV in piglets maintained under poor biosecurity conditions. PCV2-induced immunosuppression likely exacerbated swinepox severity through enhanced viral replication and systemic dissemination.

KEYWORDS: Concurrent infections, pathology, piglet, porcine circovirus 2, swinepox virus

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1. INTRODUCTION

Porcine circovirus type 2 (PCV2) is a non-enveloped, single-stranded circular DNA virus belonging to the family *Circoviridae* and genus *Circovirus* (Afolabi et al., 2017; Rosario et al., 2017). PCV2 causes porcine circovirus-associated diseases (PCVAD) with different clinical manifestations including post-weaning multisystemic wasting syndrome (PMWS), porcine dermatitis and nephropathy syndrome (PDNS) and respiratory disease complex (Liu et al., 2020; Kim et al., 2024) leading to significant economic losses to swine producers worldwide including India (Borah et al., 2024). The most significant clinical form of PCV2 is systemic disease (PCV2-SD) which is characterized by wasting, ill-thrift and occasionally respiratory or digestive signs (Baro et al., 2015; Wilfred et al., 2018). PCV2 replicates preferentially in immune cells, particularly lymphocytes and macrophages, resulting in lymphoid depletion through apoptosis-mediated cell death and functional impairment of dendritic cells (Zhai et al., 2019; Zhang et al., 2020). Pathologically, there is lymphoid depletion with histiocytic and granulomatous inflammation, particularly in lymph nodes and other lymphoid organs (Du et al., 2019; Shi et al., 2021). The severity of these lesions reflects the viral burden, with higher PCV2 loads observed in clinically diseased pigs (Patterson et al., 2015). In addition, PCV2 is implicated in reproductive failure characterized by fetal myocarditis and has been linked to porcine dermatitis and nephropathy syndrome through immune-mediated vascular injury (Palinski et al., 2017; Segales et al., 2022; Vidyarani et al., 2024).

The molecular pathogenesis of PCV2-induced immunosuppression involves multiple mechanisms where the virus directly disrupts cellular processes and antagonizes host antiviral responses (Feher et al., 2023). The viral capsid protein independently induces apoptotic responses through PERK-mediated pathways, resulting in marked depletion of lymphocytes and macrophages in lymphoid tissues (Zhang et al., 2019). This mechanism explains the profound immunosuppression characteristic of PCV2-SD and the marked increased susceptibility of infected animals to concurrent and secondary infections (Pan et al., 2018). Swinepox virus (SWPV) is an enveloped double stranded DNA virus belonging to the family *Poxviridae*, genus Suipoxvirus and causes cutaneous disease primarily in piglets up to four months of age (Afonso et al., 2002). Swinepox is characterized by a progressive cutaneous eruption beginning with petechiae that evolve through stages of papules, vesicles, pustules and eventually crusts with characteristic crateriform appearance (Kaiser et al., 2021). Swine pox primarily affects the skin but may cause mild lesions in superficial lymph nodes and the disease is typically self-limiting in juvenile and adult pigs (Kaiser et al., 2021; Opriessnig et al., 2025). The main skin lesions are hydropic degeneration and eosinophilic intracytoplasmic inclusion bodies (Pereira et al., 2020) of epidermal keratinocytes. These inclusion bodies are pathognomonic for poxviral infections and represents viral replication sites. Concurrent viral infections have been well-established as critical determinants in the severity and progression of PCVAD (Opriessnig et al., 2012; Ouyang et al., 2019). However, infections of PCV2 with other viruses like PPV, PRRSV, ASFV and CSFV and secondary bacterial infections were well documented worldwide (Chen et al., 2019; Dundon et al., 2022; Kim et al., 2022; Chrun et al., 2023). The synergistic effects of concurrent infections are primarily mediated through PCV2-induced immunosuppression, which impairs both innate and adaptive immune responses, thereby creating a permissive microenvironment for enhanced replication and tissue invasion by secondary pathogens (Feher et al., 2023). However, limited information exists regarding concurrent PCV2 and SWPV infections and their specific pathogenic interactions in naturally infected pigs. Furthermore, recent global surveillance studies reveal ongoing evolution and genotypic diversity of PCV2, with PCV2d now predominating worldwide and emerging as a concerning variant with potential implications for vaccine efficacy (Nascimento et al., 2025; Franzo et al., 2025). The present study was conducted to identify the etiologies and to understand the pathology of concurrent infections in naturally infected piglets maintained under field conditions.

2. MATERIALS AND METHODS

2.1. Outbreaks and sampling

The outbreak investigation was carried out at a local farm in Bareilly, Uttar Pradesh, India in the month of September and October, 2024 and further investigations were performed at the Division of Pathology, ICAR-IVRI, Bareilly. The affected herd comprised 8 adult pigs (2 male and 6 female) and 24 grower pigs (11 male and 13 female) maintained under a backyard production system. Adult pigs were vaccinated annually only against classical swine fever. Animals were managed under semi-scavenging conditions, with adult pigs allowed to graze outside the premises and weaning was not routinely practiced. Overall farm hygiene and biosecurity measures were poor. Approximately one year before the current episode, a respiratory outbreak affected pigs of all age groups during the monsoon season, with 100% morbidity. Clinical signs included nasal discharge and dyspnoea, which persisted for about 2-3 weeks; however, no mortality was recorded at that time. In the current episode, similar respiratory signs were observed predominantly in piglets, but with increased severity, characterized by marked dyspnoea and respiratory distress, resulting death of 3 piglets. The three piglet carcasses (38-day old crossbred male, 42-day old crossbred female and 50-day old crossbred female) were submitted to the post mortem facility at Division of Pathology, ICAR-IVRI, Bareilly, Uttar Pradesh with the reduced feed intake, marked dullness, progressive lethargy and diarrhoea, leading to death. The piglets had cutaneous multifocal crusty necrotic, dark brown, elevated circumscribed scabs over the snout, abdomen and limbs. A detailed necropsy was carried out and gross pathological findings of various vital organs

were recorded. Samples were collected from vital organs for further studies.

2.2. Pathology

Collected tissue samples were fixed in 10% neutral buffered formalin, routinely processed through graded alcohols for dehydration, cleared in xylene, and embedded in paraffin wax. Sections of approximately 4-5 µm thickness were cut using a microtome and stained with haematoxylin and eosin (H&E) for microscopic examination (Luna, 1968). Special histochemical stains like Masson's trichrome for the evaluation of fibrosis and Shorr's stain for the demonstration of inclusion bodies were performed.

2.3. Molecular detection

Genomic DNA was extracted from representative tissue samples collected in ice using commercially available DNeasy Blood and Tissue Mini Kit (Qiagen, USA). The extracted DNA was quantified and used as a template for PCR amplification.Molecular confirmation of PCV2 and SWPV was accomplished by PCR targeting the *ORF2* and *P42* genes, respectively. Sequences of oligonucleotide primers used in the study are presented in Table 1 and the respective thermal cycling conditions for the used primers are presented in Table 2. The tissue samples were additionally screened for other pathogens including porcine circovirus 3, porcine parvovirus 1, African swine fever virus (Vidyarani et al., 2025) and *Streptococcus* spp. using published primers.

3. RESULTS AND DISCUSSION

External necropsy examination of piglets revealed fair carcass condition, presence of rigor mortis in the hindlimb, moderately congested conjunctival mucous membrane, congested snout and cyanotic discolouration of the body extremities. Multiple coalescing crusty, dark brown, elevated scabs of 0.5-1 cm diameter on the skin of ear pinnae, periorbital and snout region (Figure 1). Similar lesions were observed on the flank region and limbs (Figure 2). The lesions were characterized by umbilicated centres with rough, dark brown to black crusts. These lesions are characteristic for swinepox virus infection and represent progressive stages of cutaneous

Table 1: Sequences of oligonucleotide primers used in the study

Sl. No.	Primer	Sequence 5'-3'	Targetregion	Expected productsize	Reference
1.	PCV2-F	CGGATATTGTAGTCCTGGTCG	*ORF2*	481bp	Ellis et al. (1999)
	PCV2-R	ACTGTCAAGGCTACCACAGTCA			
2.	SP-F	ATAGCATCGTTTTGTTGTAATCTT	*P42*	880bp	Mech et al. (2018)
	SP-R	CGTATTAAAACTAACGAACGCATG			

Table 2: PCR Thermal cycling conditions for the primers used in the study

Sl. No.	Primer	Initial Denaturation	Denaturation	Annealing	Extension	Final Extension
1.	PCV2 (*ORF2*)	94°C, 2min	94°C, 1min	56°C, 45sec	72°C, 1min	72°C, 7min
2.	SPV (*P 42*)	95°C, 5min	95°C, 50sec	58°C, 50sec	72°C, 1min	72°C, 15min

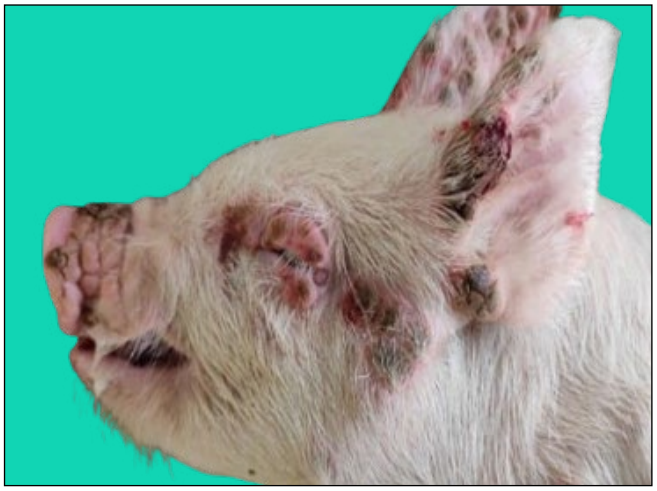

Figure 1: Photograph showing multiple coalescing crusty, dark brown, elevated scabs on the ear pinnae, snout and periorbital region of the affected piglet

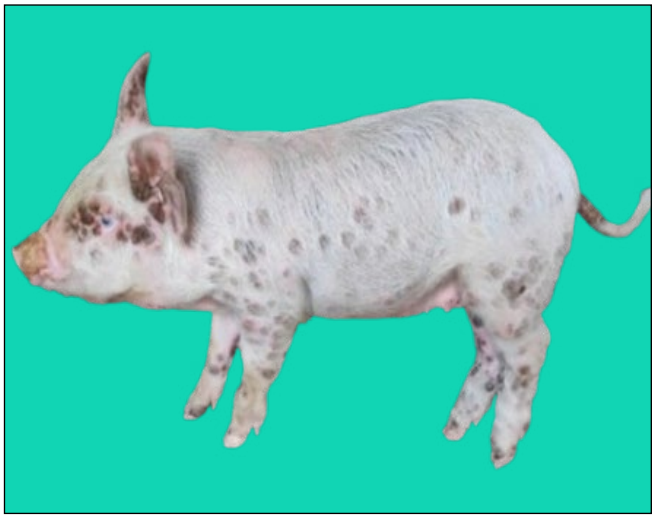

Figure 2: Multifocal dried, dark brown, elevated scabs on the flank region and limbs

eruptions (Kaiser et al., 2021). The age of piglets (38–50 days) represents peak susceptibility, as declining maternal antibodies and developing adaptive immunity render these animals unable to effectively control either pathogen (Shen et al., 2012).

Gross examination of internal organs revealed the presence of serous fluid (around 15–20 ml) in the pericardial cavity with slightly thickened and congested pericardium (Figure 3). The diaphragmatic lobes of the lung were congested with diffusive consolidation of left lung lobes (Figure 4) consistent with severe pneumonia. Similar pulmonary findings have been documented in PCV2-infected piglets, where interstitial pneumonia and pulmonary consolidation are characteristic lesions (Opriessnig et al., 2024). Liver showed multiple diffusive ecchymotic haemorrhages and focal necrotic patches on the parietal surface of all the liver lobes, suggesting direct viral hepatic injury and secondary inflammatory damage. Hepatic hemorrhages in PCV2 infection correlate directly with viral burden and immunosuppression severity (Zheng et al., 2025). Intestine showed congested mesenteric blood vessels with enlarged mesenteric lymph nodes (Resendes and Segales, 2015) and yellow faecal impacted small intestine indicating gastrointestinal inflammation and altered intestinal motility (Deng et al., 2023). Confirmation of PCV2 and SWPV was achieved by PCR amplification of the *ORF2* gene, yielding a 481-bp amplicon (Figure 5), and the *P42* gene, yielding an 880-bp amplicon (Figure 6), respectively. The collected tissue samples from the three affected cases were further analyzed for organ-wise viral detection using PCR. Organ-wise viral distribution analysis revealed that PCV2 was predominantly detected in lymphoid tissues (lymph nodes and spleen: +++), heart (+++), and lungs (+++), consistent with the lymphotropic nature of PCV2 and its documented ability to establish systemic infection (Opriessnig et al., 2010). SWPV was predominantly distributed in skin tissues (+++) and lymphoid tissues (+++), confirming its primary dermatotropism while demonstrating systemic dissemination in concurrent infections (Opriessnig et al., 2025). The tissue samples were additionally screened for other pathogens including porcine circovirus 3, porcine parvovirus 1, African swine fever virus and *Streptococcus* spp., and all samples tested negative.

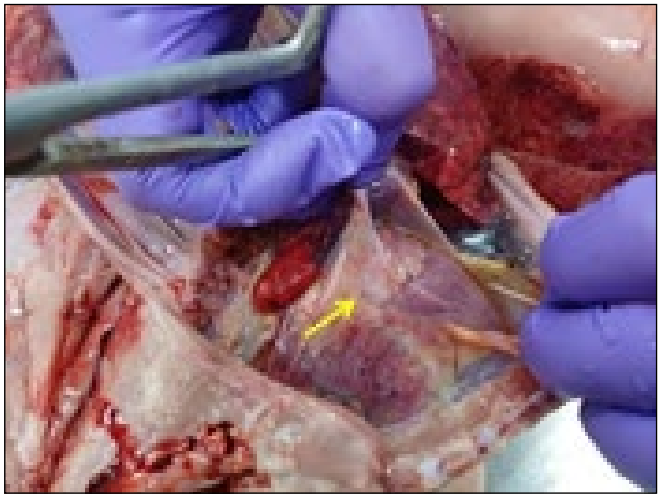

Figure 3: Presence of serous fluid in the pericardial cavity (arrow) with slightly thickened and congested pericardium in a piglet

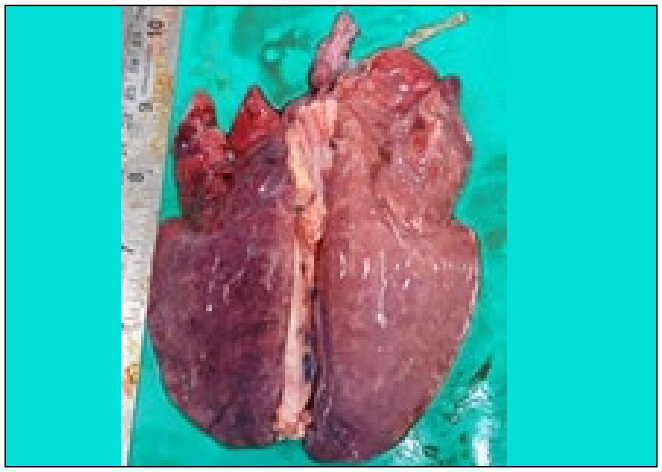

Figure 4: Congestion of diaphragmatic lobes of the lung with diffusive consolidation of left lung lobes in a piglet

Histopathological examination of lung showed thickened interstitium, congested pulmonary vessels (Figure 7) with inflammatory cell infiltration, moderate pulmonary edema (Figure 8) and lumen of bronchi containing eosinophilic debris. Masson's trichrome staining of lung tissue demonstrated

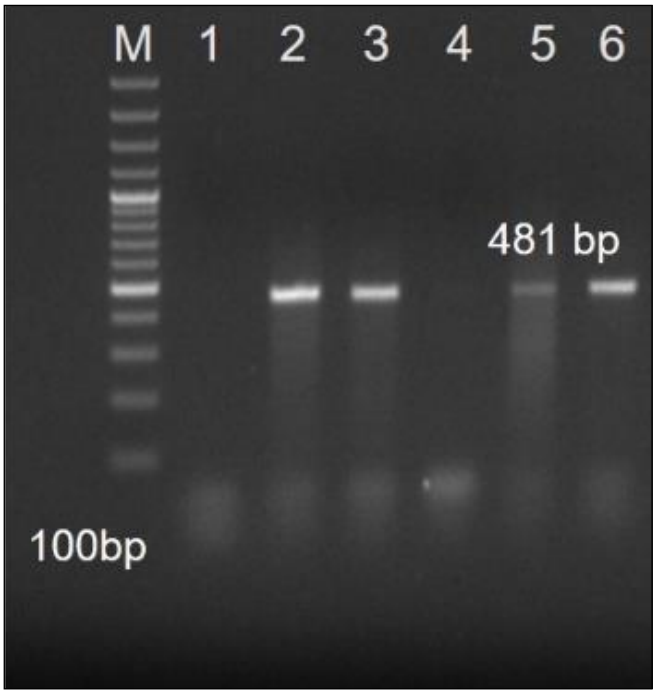

Figure 5: Agarose gel showing amplification of 481 bp amplicons of PCV2 (ORF2 gene) (Lane M, 2, 3, 4, 5 and 6: Ladder 100 bp, 38-day old piglet pooled sample, 42-day old piglet pooled sample, negative control, 50-day old piglet pooled sample and positive control, respectively)

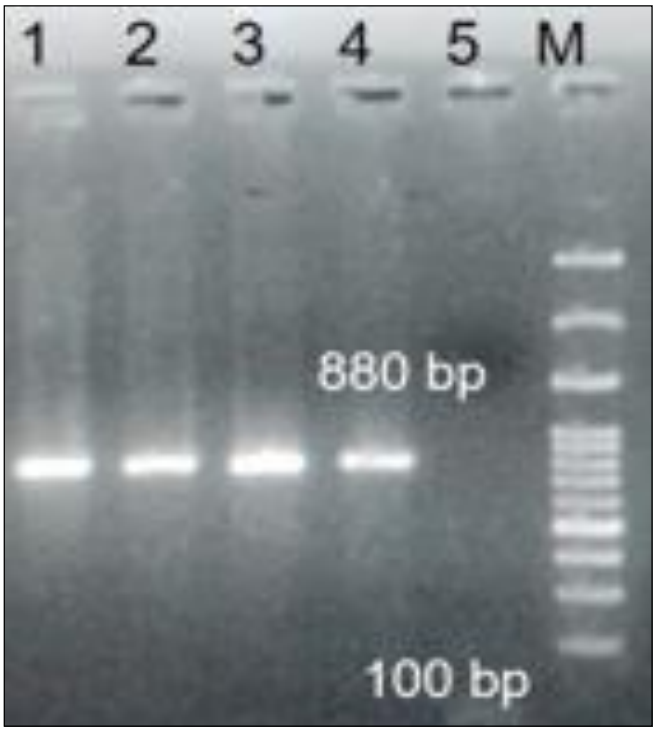

Figure 6: Agarose gel showing amplification of 880 bp amplicons of SWPV (P42 gene) (Lane M, 1, 2, 3, 4 and 5:Ladder 100 bp, 38-day old piglet pooled sample, 42-day old piglet pooled sample, 50-day old piglet pooled sample, positive control and negative control, respectively)

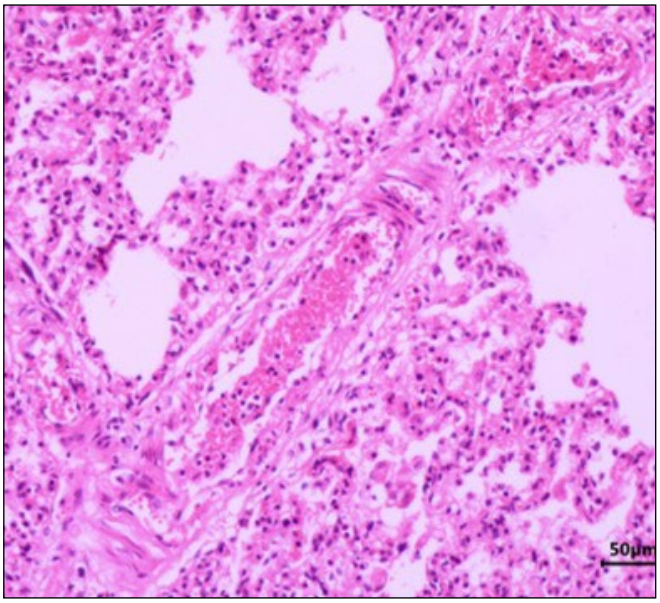

Figure 7: Photomicrograph of lung section showing congested pulmonary vessels (H&E X100)

moderate fibrotic changes shown by blue collagen deposition in the interstitium. These findings are consistent with PCV2-associated interstitial pneumonia, which represents one of the major manifestations of porcine respiratory disease complex

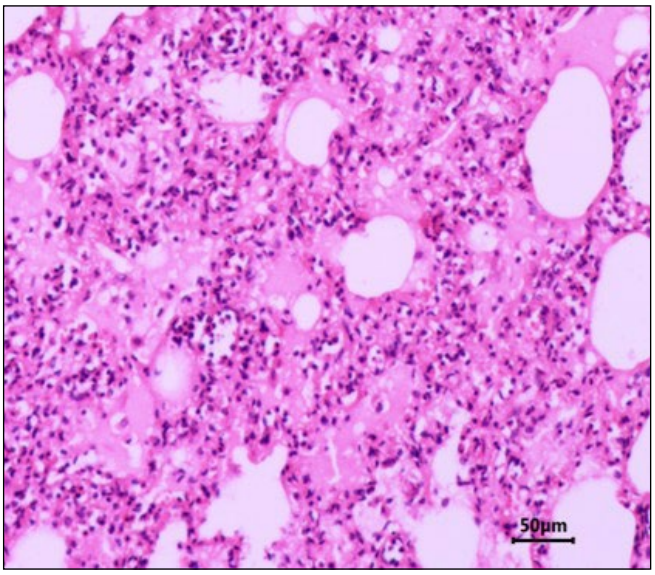

Figure 8: Lung section showing moderate pulmonary edema (H&E X100)

(PRDC) in PCV2-infected animals (Opriessnig et al., 2024). Liver showed congested portal vessels with distorted sinusoidal space containing erythrocytes and inflammatory cells. PCV2-induced hepatic lesions including sinusoidal congestion, periportal inflammatory infiltration and hepatocellular necrosis have been well-documented as characteristic features of systemic PCV2 disease (Chianini et al., 2003; Segales et al., 2022). Intestine showed submucosal congestion and sloughed off villus epithelium (Figure 9). These intestinal changes are consistent with viral enteritis and the extensive epithelial destruction might be the cause of diarrhoea observed in these piglets (Opriessnig et al., 2024).

Kidney showed tubular degeneration and necrosis with

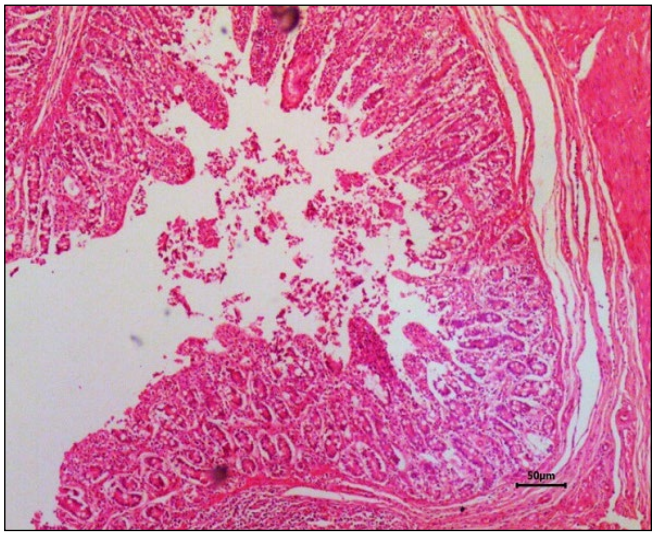

Figure 9: Intestinal section showing sloughed off villus epithelium (H&E X100)

inflammatory cell infiltration in interstitium. PCV2-associated renal pathology manifesting as tubular necrosis and interstitial nephritis has been documented in naturally infected piglets (Opriessnig et al., 2010). Lymph node showed mild lymphoid depletion (Figure 10). Quantitative multiplex immunohistochemistry studies have demonstrated that PCV2 infection causes significant reduction in both CD20+ B lymphocytes and CD3+ T lymphocytes within lymphoid

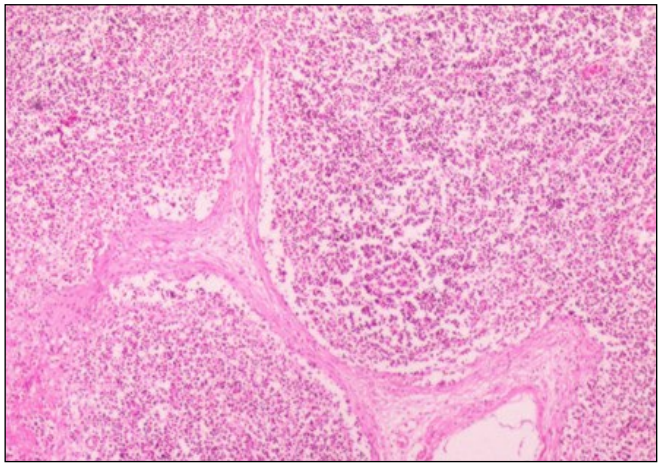

Figure 10: Section of lymph node showing mild lymphoid depletion (H&E X100)

follicles, with particularly marked depletion of CD4+ T helper cells (D'Annunzio et al., 2025). The degree of lymphoid depletion observed directly correlates with PCV2 antigen levels and viral burden within tissues (Chianini et al., 2003).

Skin showed concentric lamellar hyperkeratinization of the epidermis (Figure 11), hydropic degeneration of epidermal keratinocytes with eosinophilic cytoplasmic inclusions (Figure 12). Shorr's staining revealed distinct magenta-colored intracytoplasmic inclusion bodies within epidermal keratinocytes. Ballooning degeneration of keratinocytes is a hallmark cytopathic effect of poxviral infection resulting from viral-induced perturbation of cellular homeostasis and protein synthesis (Parekh et al., 2023). The dermis showed moderate mononuclear cell infiltration with lymphocytes and macrophages representing the secondary inflammatory response to viral infection (Kaiser et al., 2021).

The herd was not vaccinated against porcine circovirus and was maintained under poor hygienic condition. This likely predisposed the animals to disease occurrence. A previous history of a severe respiratory disease outbreak in the herd suggests the presence of underlying health and biosecurity challenges. The age of the piglets (38, 42 and 50 days) is significant, as young weaned pigs are particularly susceptible to PCV2-associated disease due to declining maternal antibodies and an immune system still in development (Shen et al., 2012). This developmental stage also corresponds to peak susceptibility to swinepox in the 4-month-or-younger

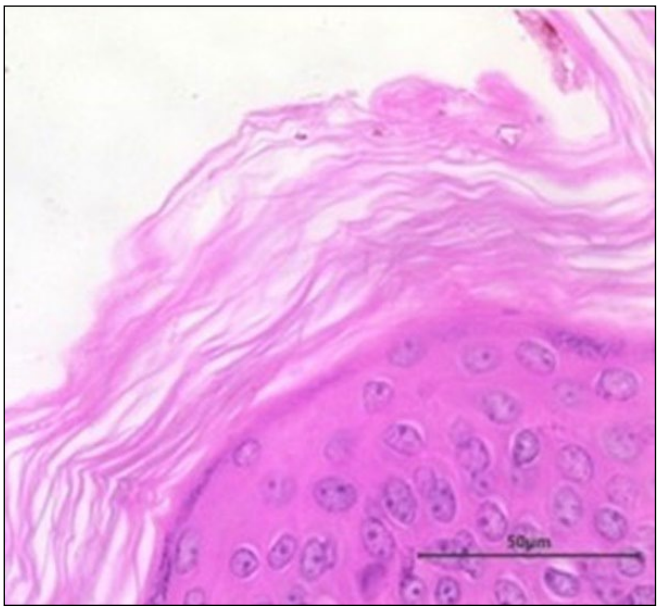

Figure 11: Skin section showing concentric lamellar hyperkeratinization of epidermis (H&E X400)

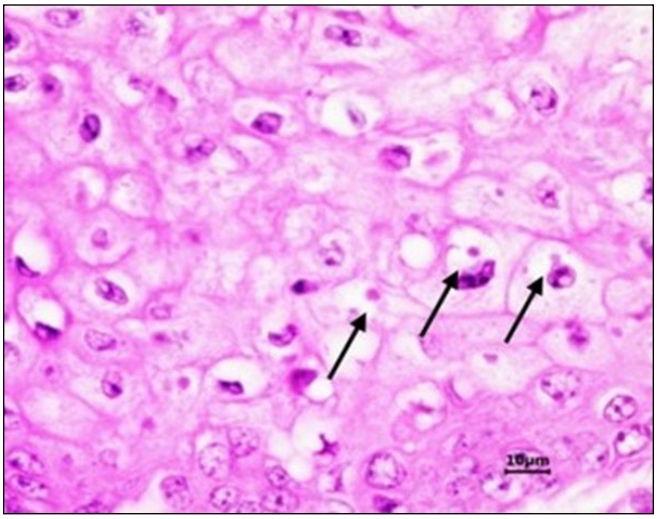

Figure 12: Skin section showing hydropic degeneration of keratinocytes of epidermis with eosinophilic cytoplasmic inclusions-arrows (H&E X400)

age group. The presence of cutaneous crusty lesions over the snout and abdomen is characteristic of swinepox virus infection (Kaiser et al., 2021). The adult pigs were allowed to graze outside the farm premises and might have acted as potential sources for the introduction of infectious agents into the herd, while mortality remained confined to piglets. The absence of ectoparasites on clinical examination indicates that swinepox virus transmission through lice was unlikely, indicating probable introduction through direct contact.

4. CONCLUSION

The present study documented a rare case of concurrent infections of PCV2 and swinepox virus in crossbred

piglets. PCR-based organ-wise viral distribution highlighted the lymphotropic nature of PCV2 and the dermatotropic predominance of SWPV, with evidence of systemic dissemination.

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