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Research article

PATHOMORPHOLOGICAL CHARACTERISTICS OF RESPIRATORY INFECTIONS IN PIGS FROM DIFFERENT PRODUCTION CATEGORIES AND AT THE SLAUGHTER LINE

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In the complex conditions of intensive pig breeding, respiratory diseases remain a significant health and economic challenge, despite substantial progress in diagnostics and control measures. This study analyzes the pathoanatomical and histopathological changes in the respiratory organs of dead pigs from different production categories, as well as in clinically healthy fattening pigs inspected on the slaughter line. The research was conducted on a farrow-to-finish pig farm in southern Serbia without immunoprophylaxis against respiratory infections. A total of 182 animals were examined: 50 suckling piglets, 50 weaned pigs, 30 pre-fattening pigs, and 52 fattening pigs.

Macroscopic and microscopic analysis of lungs, trachea, tonsils, and tracheobronchial lymph nodes revealed interstitial pneumonia predominated in young pigs categories (suckling and weaned), while older pigs (pre-fattening) showed purulent, fibrinous, and mixed broncho-interstitial pneumonia, often with pleural adhesions. Among fattening pigs, 82.7% showed no macroscopic lung changes. In cases of interstitial pneumonia, histology revealed type II pneumocyte hyperplasia, fibrosis, and inflammatory cell accumulation in the interstitium. Purulent bronchopneumonia exhibited neutrophilic granulocytes and epithelial desquamation, while fibrinous bronchopneumonia showed extensive fibrin deposits.

Catarrhal inflammation of the trachea was most common in weaned pigs (38.0%), while tonsillitis occurred most frequently in suckling piglets (24.0%) and weaned pigs (28.0%). Reactive lymphadenitis in the tracheobronchial lymph nodes was highest in pre-fattening pigs (63.3%), suckling piglets (44.0%) and weaned pigs (40.0%).

Although pathomorphological findings alone cannot confirm an etiological diagnosis, they guide further diagnostic investigations and emphasize the need for improved control, prevention, and diagnostic programs for respiratory infections in pigs.

Keywords: gross changes, histopathological changes, pigs, respiratory infections

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INTRODUCTION

In the complex conditions of intensive pig farming, despite significant advancements in diagnostics and control, respiratory diseases remain the main health and economic challenge [1]. Porcine Respiratory Disease Complex (PRDC) is a multifactorial disease caused by numerous viral and bacterial pathogens, as well as unfavorable environmental factors. Morbidity ranges from 10% to 40%, and mortality from 2% to 20% [2,3]. The pathogenesis of PRDC involves numerous etiological factors, with infectious agents playing a leading role [4-7]. These agents can be divided into four groups: viral respiratory pathogens (porcine reproductive and respiratory syndrome virus (PRRSV), porcine circovirus type 2 (PCV2), porcine cytomegalovirus (PCMV), and porcine respiratory coronavirus (PRCV)); primary bacterial respiratory pathogens (*Mycoplasma hyopneumoniae*, *Actinobacillus pleuropneumoniae*, and *Bordetella bronchiseptica*); opportunistic bacterial respiratory pathogens (*Pasteurella multocida*, *Streptococcus suis*, and *Haemophilus parasuis*); and septicemic causes of pneumonia (*Salmonella enterica*, *Actinobacillus suis*, and *Arcanobacterium pyogenes*) [8,9].

In addition to infectious agents, predisposing factors also play a significant role. Technological systems that do not consistently apply the all-in/all-out principle and involve animals from different locations, as well as young breeding animals introduced without an internal diagnostic protocol, enable the continuous circulation of infections within the herd. Moreover, farms often fail to implement time when the pigstyes are vacant within the prescribed timeframes, which hinders the harmonization of the immune status of all animals in the group.

Bronchopneumonia, or exudative pneumonia, is the most common form of lung inflammation in pigs and is typically accompanied by cranioventral lung consolidation. It is most often caused by bacteria, mycoplasmas, or food aspiration. Interstitial pneumonia, on the other hand, is of viral etiology and usually arises from aerogenic damage to the alveolar epithelium, hematogenous damage to pulmonary capillaries, or the local release of toxic metabolites within the lungs. Based on morphological characteristics, the findings differ significantly between acute and chronic forms of interstitial pneumonia [10].

One of the main pathogens associated with the porcine respiratory disease complex and the most significant pathogen in modern intensive pig farming in Europe is *Mycoplasma hyopneumoniae*, the causative agent of enzootic pneumonia in pigs [5]. It primarily affects pigs during the grower and finisher phases, but in herds lacking immunity, pigs of all age categories can become infected [11]. Clinically, it is characterized by a dry, non-productive cough and causes cranioventral lung consolidation [12].

Microscopically, pneumonia caused by *Mycoplasma hyopneumoniae* is characterized by an alveolar exudate rich in neutrophilic granulocytes. As the disease progresses, peribronchial and perivascular accumulations of lymphocytes and monocytes are observed, which, in severe cases, can lead to airway narrowing [13]. Additionally,

there is an increase in goblet cell numbers and hyperplasia of bronchial glands [14]. *Mycoplasma hyopneumoniae* facilitates the development of secondary infections and further damages the respiratory system [15].

In PRRS virus infection, the lungs of pigs exhibit mild to moderate multifocal interstitial pneumonia characterized by thickened interalveolar septa, septal infiltration with mononuclear cells, and type II pneumocyte hyperplasia. In cases of secondary bacterial infection, alveolar exudate composed of macrophages and necrotic debris is present, occasionally accompanied by polymorphonuclear cells [16].

In swine influenza, macroscopic changes are typical of viral broncho-interstitial pneumonia. The lungs are red and consolidated, with pronounced interstitial edema, while the bronchial and mediastinal lymph nodes may be enlarged. The most prominent microscopic changes include necrosis and desquamation of bronchial and bronchiolar epithelium [17,18]. The PCV-2 virus is associated with multisystemic wasting in piglets, often accompanied by co-infections with PRRS virus, influenza virus, and *Mycoplasma hyopneumoniae*, which contribute to more severe forms of the disease [19]. According to Savić et al. [20], the PCV-2b strain is dominant in the pig population in Serbia.

In a study conducted between 2011 to 2012 on ten pig farms located in the northern and central regions of Serbia, 235 lung tissue samples from naturally infected pigs were examined. The samples were analyzed using PCR to detect the presence of PCV2, PRRS, SIV, *Mycoplasma hyopneumoniae*, *Pasteurella multocida*, *Actinobacillus pleuropneumoniae*, *Haemophilus parasuis*, *Streptococcus suis* and *Arcanobacterium pyogenes*. A total of 49 different combinations of viral and bacterial pathogens were identified in the analyzed samples. Monobacterial infections were detected in 150 samples, while polybacterial infections were found in 85 examined samples. The most frequently detected virus was PCV2, whereas *Pasteurella multocida* was identified as the most significant secondary bacterial respiratory pathogen in the tested samples [21].

The porcine respiratory disease complex is common problem on pig farms in Serbia [21,22], however, no comprehensive study has been conducted on the morphological characteristics of respiratory diseases in different production categories in southern Serbia. The objectives of this study were to investigate the presence, characteristics and frequency of gross and histopathological changes in the respiratory organs of dead pigs across and within different production categories, as well as to analyze morphological changes in clinically healthy pigs from the finishing category at the slaughter line, originating from the same farm as the dead animals.

MATERIAL AND METHODS

The study was conducted in the period from October 2020 to June 2021 on a farrow-to-finish pig farm production system in southern Serbia, with a complete production system from farrowing to finishing, capacity of 1500 breeding sows. The farm did not implement immunoprophylaxis against respiratory diseases. Farm biosecurity measures

were focused on external biosecurity ensuring strict entry control of employees, visitors, and transport vehicles. The farm was fully fenced, with a designated and controlled entry point for both people and vehicles. When introducing new breeding animals, internal quarantine measures were implemented. The internal biosecurity protocols on the farm included separate housing based on production categories. Furthermore, internal protocols for cleaning, washing, and disinfection were applied in accordance with the farm's production process technology, The all-in/ all-out system, including an empty period for the designated stable, was implemented at all production stages.

Postmortem examinations and sampling of organs and tissues from piglets in the suckling (50 animals), weaned (50 animals), and grower (30 animals) production phases were performed at the necropsy hall of the Veterinary Specialist Institute Niš. Organ and tissue examinations and sampling from finishers (52 animals) originating from the same farm as the dead piglets were carried out at the slaughter line.

Macroscopic examination of the sampled organs from dead piglets and finishers at the slaughter line involved assessing and classifying visible morphological changes described in the literature [10]. This formed the basis for preliminary macroscopic diagnoses, categorized as follows: no changes (BO), purulent bronchopneumonia (PBP), fibrinous bronchopneumonia (FBP), interstitial pneumonia (IP), pleuropneumonia (PP), pulmonary hyperemia and edema (PHE), pulmonary hyperemia (PH), tracheal hyperemia (TH), catarrhal tracheitis (CT), tonsillar hyperemia (TONH), tonsillitis (TON), hemorrhagic tonsillitis (HTON), lymphadenitis (LDTB), and hemorrhagic lymphadenitis (HLDTB).

Following the sampling of target organs (lungs, trachea, tonsils, and tracheobronchial lymph nodes), tissues were fixed in neutral buffered formalin, processed, and embedded in paraffin blocks. Tissue sections, 4 – 5 μm thick, were cut using a microtome and subsequently stained using the standard hematoxylin and eosin (HE) staining method. Tonsil sampling was not performed on finishing pigs at the slaughter line due to the specific procedures involved in carcass processing.

Histological evaluation of the tissue sections was conducted using an Olympus BX51 microscope, with an Olympus Color View III camera for creating photodocumentation. Microscopic changes were described and systematically categorized to establish a database of pathohistological diagnoses based on the literature [10]: no changes (BO), fibrinous bronchopneumonia (FBP), purulent bronchopneumonia (PBP), interstitial pneumonia (IP), broncho-interstitial pneumonia (BIP), pulmonary hyperemia and edema (PHE), hemorrhages (HEM), pronounced pulmonary interstitium/ bronchiolitis/peribronchiolitis (PPI/BL/PBL), catarrhal tracheitis (CT), catarrhal desquamative tracheitis (CDT), catarrhal mucinous tracheitis (CMT), hyperemia of tonsils/lymph nodes (HIPT/LN), tonsillitis (TON), erosive tonsillitis (ETON), lymphoid hyperplasia of tonsils/lymph nodes (LHT/LN), depletion of lymphoid follicles in tonsils/lymph nodes (DLFT/LN), reactive lymphadenitis (RLDTB), and hemorrhagic lymphadenitis (HLDTB).

Statistical data processing was performed in the statistical package SAS 9.3 (SAS Institute Inc., 2002-2010). The choice of evaluation and testing methods depended on the structure and characteristics of the data. The research results are presented tabularly, using photos and microphotos. Testing of the distribution of frequencies by categories and changes (pathoanatomical and pathohistological) was performed using the non-parametric χ^2 test, at three levels of significance ($p<0.05$; $p<0.01$; $p<0.001$). The original p value from the output is displayed to four decimal places.

RESULTS

Statistically significant differences in the prevalence of various pathological changes within and between the examined production categories were identified in the lungs ($p<0.0001$) and tracheobronchial lymph nodes ($p<0.001$) (Table 1).

Table 1. Frequency of pathological changes within and between the examined production categories

Organ	Pathological change	Production category				Total	p value
		suckling piglets n=50	weaned piglets n=50	growing piglets n=30	finishing pigs n=52		
Lungs	BO	12	5	0	43	60	<0.0001
	FBP	4	12	1	0	17	
	PBP	5	11	6	0	22	
	IP	11	9	12	2	34	
	PP	0	2	3	0	5	
	PHE	7	9	4	4	24	
	PH	11	2	4	3	20	
Trachea	BO	47	41	29	51	168	0.0224
	TH	3	5	1	1	10	
	CT	0	4	0	0	4	
Tonsils	BO	38	32	24	/	94	0.4929
	TONH	4	3	1	/	8	
	TON	7	12	5	/	24	
	HTON	1	3	0	/	4	
Tracheobronchial lymph nodes	BO	37	30	16	49	132	0.0003
	LDTB	13	16	12	3	44	
	HLDTB	0	4	2	0	6	

Legend: **BO** – no changes; **FBP** – fibrinous bronchopneumonia; **PBP** – purulent bronchopneumonia; **IP** – interstitial pneumonia; **PP** – pleuropneumonia; **PHE** – pulmonary hyperemia and edema; **PH** – pulmonary hyperemia; **TH** – tracheal hyperemia; **CT** – catarrhal tracheitis; **TONH** – tonsillar hyperemia; **TON** – tonsillitis; **HTON** – hemorrhagic tonsillitis; **LDTB** – lymphadenitis; **HLDTB** – hemorrhagic lymphadenitis.

The most prevalent pathological changes observed in the lungs of suckling piglets were interstitial pneumonia (IP, 22.0%), hyperemia (HP, 22.0%), and pulmonary edema (PHE, 14.0%). In weaned piglets, fibrinous bronchopneumonia (FBP, 24.0%), purulent bronchopneumonia (PBP, 22.0%) and interstitial pneumonia (IP, 18.0%) were almost equally represented, while interstitial pneumonia was the dominant finding in growing piglets (IP, 40.0%). Among finishing pigs examined at the slaughter line, the majority of lungs showed no visible gross changes (BO, 82.7%). Tracheal changes, such as tracheal hyperemia (TH) and catarrhal tracheitis (CT), were approximately equally represented in weaned piglets. Macroscopically evident tonsillitis was most prevalent in weaned piglets (TON, 24.0%), whereas lymphadenitis (LDTB) was observed in a higher percentage of dead animals (26–40%) across the first three production categories.

Hyperemia and pulmonary edema were most frequently observed in carcasses of weaned piglets. Macroscopically, such lungs appeared shiny, with rounded edges, and ranged in color from gray-red to red-blue (Figure 1). The bronchi and trachea typically contained a large amount of pale pink frothy fluid.

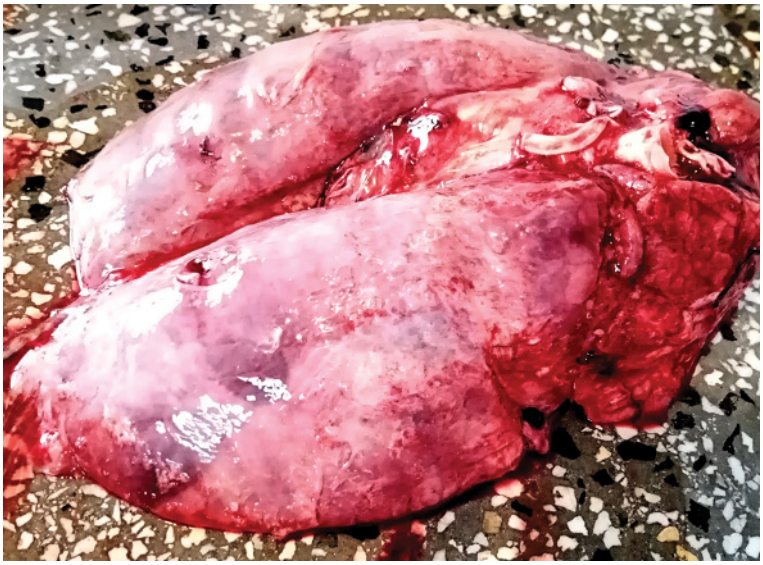

Figure 1. Pig, lung. Gross changes in the lungs: Pulmonary hyperemia and edema.

In animals with interstitial pneumonia, all lung lobes were diffusely affected, with the lungs exhibiting a light red color and a rubbery consistency (Figure 2A). In advanced cases of purulent bronchopneumonia, the lungs, due to the consolidation of individual lobules, resembled a checkerboard pattern in appearance (Figure 2B). Fibrinous bronchopneumonia was often accompanied by yellow fibrin deposits on the pleura (Figure 2C) and thoracic wall (Figure 2D), as well as the formation of pleural adhesions.

Figure 2. Pig, lung. Gross changes in the lungs with pneumonia. **A)** Interstitial pneumonia; **B)** Bronchopneumonia purulenta; **C)** Fibrinous pleuropneumonia; **D)** Fibrinous deposits on the pleura.

In dead piglets, histopathological examination of the lungs affected by interstitial pneumonia revealed hyperplasia of type II pneumocytes, reduced alveolar lumen, and thickened alveolar walls (Figure 3A). In prolonged cases, there was an accumulation of mononuclear cells in the lung interstitium, presence of hyperplastic type II pneumocytes within the alveoli, hyperplasia of peribronchial lymphatic tissue, and fibrosis. In the lungs affected by purulent bronchopneumonia, microscopic examination showed numerous neutrophilic granulocytes, macrophages, and desquamated cells within the alveoli (Figure 3B). In extreme cases, these cells completely obliterated the lumina of bronchi, bronchioles, and alveoli, accompanied by hyperplasia of local lymphatic tissue. Microscopic analysis of lung tissue sections with fibrinous bronchopneumonia revealed fibrin accumulation in the bronchoalveolar spaces (Figure 3C). In some cases, morphological features of both bronchopneumonia and interstitial pneumonia were observed simultaneously, classified as broncho-interstitial pneumonia (Figure 3D).

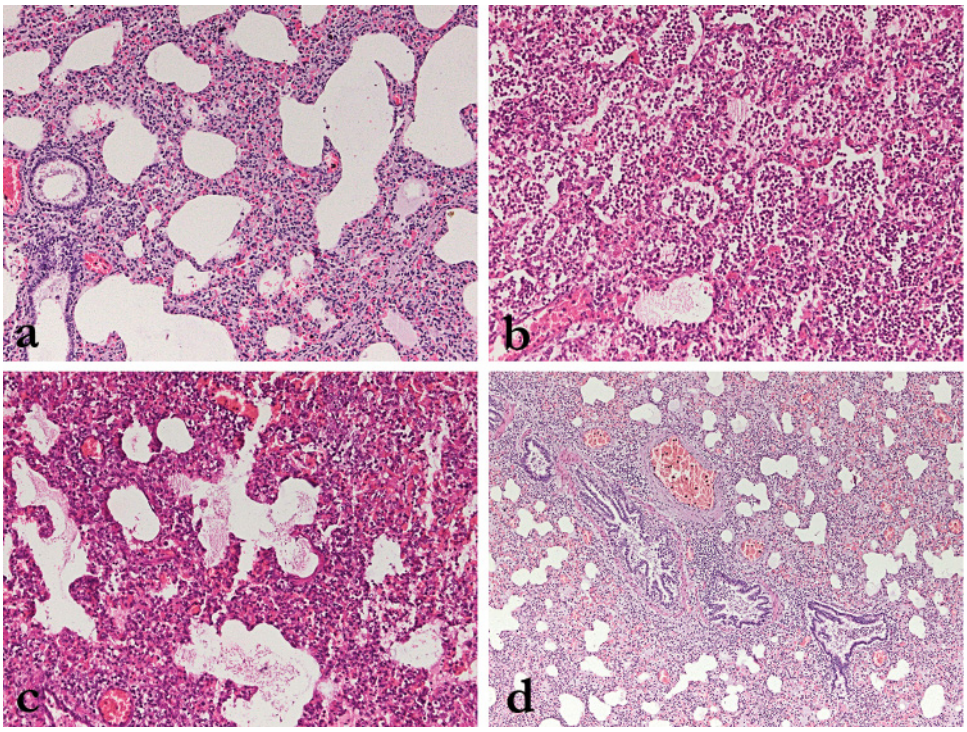

Figure 3. Pig, lung. Microscopic changes in the lungs with pneumonia. **A)** Interstitial pneumonia – hyperplasia of type II pneumocytes and narrowing of the alveolar lumen, HE, x20; **B)** Purulent bronchopneumonia – neutrophilic granulocytes, macrophages, and desquamated cells in the alveoli, HE, x20; **C)** Fibrinous bronchopneumonia – fibrin in the bronchoalveolar spaces, HE, x20; **D)** Interstitial pneumonia with purulent components – hyperplasia of type II pneumocytes, narrowing of the alveolar lumen, and neutrophilic granulocytes in the lumen, HE, x10.

Inflammation of the tracheal mucosa was observed as mild catarrhal tracheitis, characterized by moderate hyperplasia of the surface epithelium and the presence of occasional neutrophilic granulocytes and lymphocytes in the lamina propria (Figure 4A). In desquamative tracheitis, erosion of the surface epithelium was observed, along with numerous desquamated epithelial cells and an increased presence of inflammatory cells in the subepithelial layer of the mucosa (Figure 4B). Microscopic findings in the cases of tonsillitis were characterized by varying degrees of lymphoid hyperplasia, inflammatory cell infiltration, and cellular debris within the surface epithelium, subepithelial zone, and tonsillar crypts (Figure 4C). Fibrosis was also noted in prolonged chronic processes. Reactive lymphadenitis was characterized by increased vascularization and hypercellularity without clear organization into lymphoid follicles (Figure 4D).

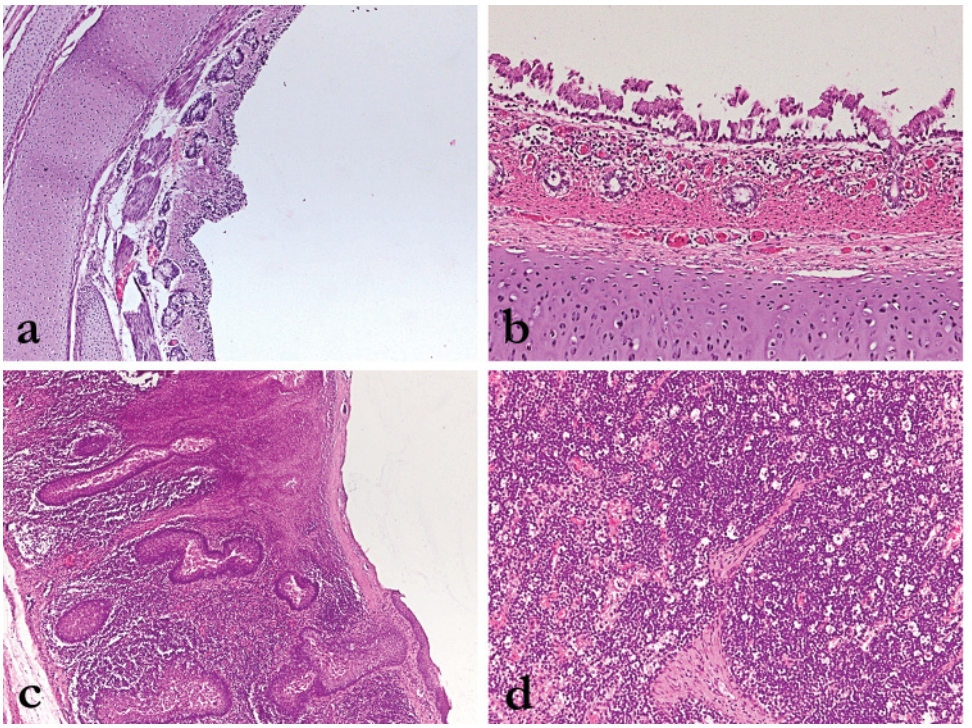

Figure 4. Pig, trachea, tonsils and tracheobronchial lymph nodes. Microscopic morphological changes in trachea, tonsils and tracheobronchial lymph nodes. **A)** Catarrhal tracheitis – epithelial hyperplasia with neutrophilic granulocytes and lymphocytes in the lamina propria, HE, x20; **B)** Desquamative catarrhal tracheitis – desquamated epithelial and inflammatory cells in the subepithelial mucosal layer, HE, x20; **C)** Tonsillitis – inflammatory cell infiltration in the surface epithelium, subepithelial zone, and tonsillar crypts, HE, x10; **D)** Reactive lymphadenitis – disrupted lymph node architecture and hypercellularity, HE, x20.

Statistically significant differences were also determined in the prevalence of various histopathological changes within and between examined production categories in the lungs ($p<0.0001$), trachea ($p<0.0001$), tonsils ($p<0.05$) and tracheobronchial lymph nodes ($p<0.001$) (Table 2).

In dead piglets from different production phases (farrowing, weaning, and pre-fattening), interstitial pneumonia (IP) was the predominant finding (20.0%, 22.0%, and 26.7%, respectively). Additionally, a significant proportion of histopathological changes were characterized by prominent pulmonary interstitium, bronchiolitis, and peribronchiolar cellular infiltration (PPI/BL/PBL: 24.0%, 12.0%, and 26.7%). Purulent bronchopneumonia (PBP) and the mixed form of broncho-interstitial pneumonia (BIP) were nearly equally represented across all phases (8.0%, 12.0%, and 10.0%). In the fattening category, 9.6% of the examined tissue samples showed prominent pulmonary interstitium with bronchiolitis and peribronchiolar cellular infiltration (PPI/BL/PBL), while interstitial pneumonia was observed in 5.8% of the samples.

Table 2. Frequency of histopathological changes within and between the examined production categories

Organ	Histopathological change	Production category				Total	p value
		suckling piglets n=50	weaned piglets n=50	growing piglets n=30	finishing pigs n=52		
Lungs	BO	16	5	4	43	68	<0.0001
	FBP	1	5	3	0	9	
	PBP	4	6	3	0	13	
	IP	10	11	8	3	32	
	BIP	4	6	3	0	13	
	PHE	0	9	1	1	11	
	HEM	3	2	0	0	5	
	PPI/BL/PBL	12	6	8	5	31	
Trachea	BO	35	17	16	51	119	<0.0001
	CT	12	19	11	1	43	
	CDT	3	7	2	0	12	
	CMT	0	7	1	0	8	
Tonsils	BO	30	18	15	/	63	0.0206
	HIPT	5	9	4	/	18	
	TON	12	14	3	/	29	
	ETON	2	4	4	/	10	
	LHT	1	0	3	/	4	
	DLFT	0	5	1	/	6	
Tracheobronchial lymph nodes	BO	13	8	2	45	68	<0.0001
	RLDTB	22	20	19	1	62	
	HLDTB	0	2	0	0	2	
	HIPLN/HEM	9	13	6	0	28	
	LHLN	3	1	2	5	11	
	DLFLN	3	6	1	1	11	

Legend: **BO** – no changes; **FBP** – fibrinous bronchopneumonia; **PBP** – purulent bronchopneumonia; **IP** – interstitial pneumonia; **BIP** – broncho-interstitial pneumonia; **PHE** – pulmonary hyperemia and edema; **HEM** – hemorrhages; **PPI/BL/PBL** – pronounced pulmonary interstitium/bronchiolitis/peribronchiolitis; **CT** – catarrhal tracheitis; **CDT** – catarrhal desquamative tracheitis; **CMT** – catarrhal mucinous tracheitis; **HIPT** – hyperemia of tonsils; **TON** – tonsillitis; **ETON** – erosive tonsillitis; **LHT** – lymphoid hyperplasia of tonsils; **DLFT** – depletion of lymphoid follicles in tonsils; **RLDTB** – reactive lymphadenitis; **HLDTB** – hemorrhagic lymphadenitis; **HIPLN** – hyperemia of lymph nodes; **LHLN** – lymphoid hyperplasia of lymph nodes; **DLFLN** – depletion of lymphoid follicles in lymph nodes.

Excluding the fattening category, the predominant histopathological finding in the trachea was catarrhal tracheitis, most prevalent in the weaning category (CT, 38.0%). Tonsillitis was most commonly observed in suckling piglets (TON, 24.0%) and during the weaning phase (TON, 28.0%). Histological changes in the tracheobronchial lymph nodes were most frequently manifested as reactive lymphadenitis (RLDTB), predominantly in the suckling piglets (44.0%), weaning (40.0%), and pre-fattening categories (63.3%).

DISCUSSION

The pathomorphological changes identified in this study across different pig production categories are consistent with the findings of previous research [3,22,23]. Considering the fact that the etiology of swine pneumonia is often multifactorial, the morphological manifestations of pneumonia are frequently non-uniform [19]. The nature of these lesions corresponds to the etiological agent predominantly present at the time of necropsy. In general, viral pneumonias are initially characterized by acute interstitial pneumonia, during which pneumotropic viruses cause degeneration and desquamation of type I pneumocytes, followed by their replacement with type II pneumocytes, leading to pulmonary fetalization. Secondary bacterial infections gradually alter the morphological characteristics of lung lesions towards purulent bronchopneumonia, which, in some cases, progresses to apostematous or fibrinous bronchopneumonia, often associated with pleuropneumonia [24].

In our study, interstitial pneumonia predominated in the suckling piglet category, which may indicate the dominant role of viral infections at this age. In older production stages, such as weaning and grower-finisher phases, there was a significantly higher incidence of purulent, fibrinous, and mixed broncho-interstitial pneumonias. This suggests that viral infections in younger age categories create favorable conditions for secondary bacterial infections, which gradually alter the morphological characteristics of lung lesions over time [25]. It should be noted that no immunoprophylaxis was implemented on the studied farm against any of the most commonly described causative agents of respiratory diseases in intensive swine production. Therefore, bacterial infections such as *Mycoplasma hyopneumoniae* may play a significant role in all age categories, including suckling piglets and breeding animals [11]. A study conducted on Macedonian commercial pig farms, which combined serological analysis and lung lesion assessment, confirmed a high percentage of EP-like lesions in finishing pigs. The high *Mycoplasma hyopneumoniae* seroprevalence observed was most likely a result of vaccination. These findings further indicate that farms with higher *Mycoplasma hyopneumoniae* seroprevalence tend to have lower lung lesions score (LLS), highlighting the importance of a multifactorial approach in evaluating *Mycoplasma hyopneumoniae* infections on farms [26].

Previous studies [2,21,27] indicate that interstitial pneumonia of varying severity can, in most cases, be associated with PCV-2 or the combined effects of PCV-2 and

Mycoplasma hyopneumoniae. These findings suggest that within the porcine respiratory disease complex (PRDC), PCV-2 contributes to creating conditions favorable for the entry of other respiratory pathogens and may also act as a secondary causative agent. Furthermore, the PRRS virus, as the sole pathogen, can also be linked to the development of interstitial pneumonia [16]. In this study, histopathological examination of the lungs of experimentally infected pigs revealed mild to moderate multifocal interstitial pneumonia, characterized by thickening of interalveolar septa, septal infiltration with mononuclear cells, hyperplasia and hypertrophy of type II pneumocytes, and the presence of alveolar exudate composed of macrophages and necrotic debris, occasionally accompanied by polymorphonuclear cells [16]. Unlike European studies, research conducted on swine populations in America [4] identified the PRRS virus and swine influenza virus as the predominant pathogens in PRDC.

In this study, complicated purulent bronchopneumonias could be associated with the combined effects of PCV-2, *Mycoplasma hyopneumoniae,* and PRRS virus [19].

It is believed that most pigs raised under intensive farm conditions are infected with respiratory pathogens at some point in their lives, with lesions in the respiratory system being observed in a significant number of animals at the slaughter line [22,28]. The presence of lung lesions in pigs has been associated with reduced carcass value and poorer pork quality [3,28]. Morphological changes in the lungs were detected in 17.3% of clinically healthy fatteners, a figure comparable to the findings of Christensen and Enoe (1999), who reported lung lesions in 25% of fatteners [29]. Other studies [30,31] indicate a similar prevalence of lesions observed at the slaughter line, whereas earlier research reported significantly higher prevalence rates, such as 37% [32] and 45% [33]. Microscopic changes predominantly included varying degrees of interstitial prominence, focal to multifocal thickening of alveolar septa, and mild to moderate bronchiolar and peribronchiolar cellular infiltration, consistent with the findings of Hansen et al. (2010). Such thickening of alveolar septa, accompanied by bronchiolitis and peribronchiolitis, is not always associated with the presence of respiratory pathogens, suggesting the possibility that these changes represent a defensive response of the lungs to environmental factors characteristic of intensive pig farming (e.g., dust particles, ammonia) [3].

CONCLUSION

This study identified varying degrees of prevalence and severity of morphological changes in the respiratory organs of pigs across different production categories. Interstitial pneumonia was predominant in younger age groups, while purulent, fibrinous, and mixed broncho-interstitial pneumonias were significantly more prevalent in older production categories.

Pathomorphological changes alone are insufficient for establishing an etiological diagnosis but provide valuable direction for further diagnostic investigations, as well as directing further control measures in pig farm production.

Understanding the key pathogens involved in the porcine respiratory disease complex, as well as the consequences and effects of their interactions on the progression, morphological expression, and outcomes of the disease, is essential for implementing effective control and prophylactic strategies in intensive pig farming. To this end, further comprehensive research in this area is certainly necessary to be continued.

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Authors' contributions

IJ participated in the study design, sample collection, and conducted pathoanatomical and histopathological examinations as well as statistical analysis. MP contributed to the study design, coordinated sample collection and examinations, performed the final revisions, and helped draft the manuscript. JPR was involved in the final revisions and helped draft the manuscript. MS contributed to data analysis and manuscript revision. VM contributed to the study design and data interpretation. SAK participated in the study design, performed histopathological examinations, and analyzed data. IV conceived the study, participated in its design, conducted histopathological examinations, analyzed data, and performed the final revisions.

Declaration of conflicting interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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PATOMORFOLOŠKE KARAKTERISTIKE RESPIRATORNIH INFEKCIJA KOD SVINJA IZ RAZLIČITIH PROIZVODNIH KATEGORIJA I NA LINIJI KLANJA

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U složenim uslovima intenzivnog uzgoja svinja, uprkos velikom napretku koji je postignut u oblasti dijagnostike i kontrole, respiratorne bolesti predstavljaju značajan zdravstveni i ekonomski problem. Ovo istraživanje se fokusira na analizi patoanatomskih i patohistoloških promena na respiratornim organima uginulih svinja u različitim proizvodnim kategorijama, kao i kod klinički zdravih tovljenika na liniji klanja. Istraživanje je sprovedeno na farmi svinja od prašenja do tova na jugu Srbije, koja nije primenjivala mere imunoprofilakse protiv uzročnika respiratornih infekcija. Ukupno su ispitane 182 jedinke: 50 iz kategorije prasadi na sisi, 50 iz odgoja, 30 iz predtova i 52 tovljenika.

Makroskopskim i mikroskopskim pregledom pluća, dušnika, tonzila i traheobronhalnih limfnih čvorova ustanovljeno je da je kod mladih kategorija svinja (prasad na sisi i prasad u odgoju) dominirao nalaz intersticijalne pneumonije, dok su kod starijih jedinki (predtov) češće bile prisutne gnojne, fibrinozne i mešovite bronhointersticijalne pneumonije, često praćene pleuralnim priraslicama. Kod tovljenika je kod 82,7% pregledanih jedinki plućno tkivo bilo bez makroskopskih promena. Histološkom analizom je ustanovljena hiperplazija pneumocita tipa II, fibroza i nakupljanje inflamatornih ćelija u intersticijumu pluća kod intersticijalne pneumonije, kao i prisustvo neutrofilnih granulocita i deskvamisanih epitelnih ćelija kod gnojnih bronhopneumonija, odnosno fibrina kod fibrinoznih bronhopneumonija.

Kataralno zapaljenje sluznice dušnika je bilo najzastupljenije u kategoriji odgoja (38.0%), dok je tonzilitis najčešće zabeležen kod prasadi na sisi (24,0%) i u fazi odgoja (28,0%). Reaktivni limfadenitis na traheobronhalnim limfnim čvorovima je bio najzastupljeniji u kategoriji predtova (63,3%), kod prasadi na sisi (44,0%) i u odgoju (40,0%).

Iako patomorfološke promene same po sebi nisu dovoljne za postavljanje etiološke dijagnoze, one usmeravaju dodatna dijagnostička ispitivanja i naglašavaju potrebu za unapređenjem programa kontrole, profilakse i dijagnostike respiratornih infekcija svinja.