

Glandular Gastritis in Commercial Finishing Pigs: An Abattoir-Based Study

Jaroslav Novotný¹, Zuzana Krepelková¹, Katarína Bárdová¹, Sandra Andrašková²,
Arpád Csörgő³

¹Clinic for Swine, University of Veterinary Medicine and Pharmacy in Košice, Komenského 73, 041
81 Košice, Slovakia

²Department of the Morphological Disciplines, University of Veterinary Medicine and Pharmacy,
Komenského 73, 04181 Košice, Slovakia

³ArVet Veterinary Clinic, Mlynársky rad 205/30, 930 05 Gabčíkovo, Slovakia

*For Correspondence

Assoc. prof. DVM. Jaroslav Novotný, PhD.
Clinic for Swine, University of Veterinary Medicine
and Pharmacy, Komenského 73, Košice, Slovakia

jaroslav.novotny@uvlf.sk

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Abstract: Gastritis of the glandular stomach is less extensively investigated in pigs than lesions of the pars oesophagea. This study aimed to evaluate the prevalence of glandular gastritis in commercial finishing pigs during abattoir monitoring in Slovakia and to characterize the distribution of macroscopic lesions of the pars oesophagea. A total of 873 stomachs from commercial finishing pigs originating from several farms were examined macroscopically. The glandular mucosa was evaluated for gastritis, while lesions of the pars oesophagea were classified into four severity grades. Selected stomachs were also examined histopathologically. Glandular gastritis was detected in 260 stomachs (29.8%). No macroscopic lesions of the pars oesophagea were observed in 502 stomachs (57.5%), whereas parakeratosis was recorded in 210 (24.1%), erosions in 141 (16.2%), and ulcerations in 20 (2.3%). Histopathological examination confirmed inflammatory and degenerative changes in the glandular mucosa. The findings indicate that glandular gastritis is relatively common in commercial finishing pigs. Systematic evaluation of both the glandular mucosa and pars oesophagea during abattoir examination may provide a more comprehensive assessment of gastric lesions in finishing pigs.



I. Introduction

Gastritis and gastric ulceration represent important multifactorial health problems in modern pig production. Under intensive production conditions, several factors may contribute to disruption of the physiological integrity of the gastric mucosa, including the physical structure of feed, feed particle size, and dietary fibre content [1–3]. While lesions of the non-glandular region of the stomach (*pars oesophagea*) have long been the subject of extensive research and routine veterinary diagnostics, considerably less attention has been paid to inflammatory and degenerative changes affecting the glandular region of the stomach (*pars glandularis*).

During inflammatory and ulcerative processes, the glandular gastric mucosa undergoes complex morphological changes. More severe lesions may involve damage or loss of the surface epithelium, disruption of gastric gland architecture, hyperaemia, granulation tissue formation, and neovascularization. The inflammatory process is characterized by infiltration of immune cells into the *lamina propria* mucosae and the spaces between the gastric glands [4].

The composition of the inflammatory infiltrate varies depending on the stage and duration of the pathological process. Acute inflammatory changes are predominantly associated with neutrophilic infiltration, whereas chronic gastritis is characterized mainly by lymphocytes, plasma cells, and other mononuclear cells. Chronic inflammation may also be accompanied by reparative changes, proliferation of granulation tissue, and epithelial hyperplasia surrounding the lesion [4].

Several factors may contribute to the development of gastritis in the glandular region of the stomach, including infection with *Helicobacter suis*. Colonization of the gastric mucosa by bacteria of the genus *Helicobacter* has been associated with inflammatory changes in the glandular mucosa and alterations in the gastric environment in pigs [5,6]. However, the aetiology of gastritis is multifactorial, and the relative importance of individual infectious and non-infectious factors may vary among farms.

Healing of glandular mucosal lesions involves restitution and proliferation of epithelial cells, re-epithelialization of the damaged surface, and activation of growth factors involved in restoration of the mucosal barrier and vascularization of the damaged tissue [7].

The aim of this study was to evaluate the prevalence of glandular gastritis in commercial finishing pigs during abattoir monitoring in Slovakia and to characterize the distribution of macroscopic lesions of the *pars oesophagea*.

II. Materials and methods

2.1. Abattoir monitoring and macroscopic examination

During 2025, a total of 873 stomachs from commercial finishing pigs originating from several farms in Slovakia were macroscopically examined as part of routine abattoir monitoring.

Following evisceration, the stomachs were opened along the greater curvature (*curvatura major*), emptied of gastric contents, gently rinsed with physiological saline, and subsequently examined macroscopically. The glandular region of the stomach and the non-glandular *pars oesophagea* were evaluated separately.

The glandular mucosa was assessed for the presence of macroscopic changes indicative of gastritis, particularly focal or diffuse hyperaemia, oedema, thickening of the gastric folds, and inflammatory exudate. The presence of erosions and ulcerations of the glandular mucosa was also recorded.

Lesions of the *pars oesophagea* were classified into four categories according to severity: score 0 – no macroscopic lesions; score 1 – parakeratosis; score 2 – erosions; and score 3 – ulcerations. The results of the macroscopic examination were recorded for each group of pigs included in the study.

2.2. Histopathological examination

For histopathological examination, representative samples of glandular mucosa showing macroscopic signs of gastritis, as well as samples without apparent macroscopic lesions, were collected from selected stomachs.



Tissue samples were fixed in 10% neutral buffered formalin, routinely processed, embedded in paraffin, and sectioned using a microtome. The sections were subsequently stained with haematoxylin and eosin (H&E). Histopathological examination included assessment of the integrity of the surface epithelium and gastric glands, the presence and characteristics of inflammatory cell infiltration, vascular changes, oedema, and the presence of necrotic changes.

2.3. Statistical analysis

The prevalence of gastritis in the glandular region of the stomach was expressed as the absolute number and percentage of affected stomachs among all examined stomachs. The distribution of the individual severity scores of *pars oesophagea* lesions was expressed in the same manner. The results were evaluated for the entire study population and descriptively compared among the individual sampling groups.

III. Results

3.1. Macroscopic findings

Gastritis of the glandular region was detected in 260 of the 873 examined stomachs (29.8%). Among the individual groups, the prevalence of gastritis ranged from 12.5% to 75.0% (Table 1).

No macroscopic lesions of the *pars oesophagea* were observed in 502 stomachs (57.5%). Parakeratosis (score 1) was recorded in 210 stomachs (24.1%), erosions (score 2) in 141 (16.2%), and ulcerations (score 3) in 20 (2.3%). Overall, lesions of the *pars oesophagea* (scores 1–3) were present in 371 stomachs (42.5%), while erosions and ulcerations (scores 2–3) were recorded in 161 stomachs (18.4%).

Table 1. Occurrence of glandular gastritis and *pars oesophagea* lesions in finishing pigs

Group	n	Gastritis n (%)	PE 0 n (%)	PE 1 n (%)	PE 2 n (%)	PE 3 n (%)
A	318	102 (32.1)	197 (62.0)	68 (21.4)	47 (14.8)	6 (1.9)
B	50	14 (28.0)	20 (40.0)	19 (38.0)	11 (22.0)	0 (0.0)
C	189	59 (31.2)	144 (76.2)	32 (16.9)	11 (5.8)	2 (1.1)
D	185	48 (25.9)	118 (63.8)	51 (27.6)	15 (8.1)	1 (0.5)
E	20	6 (30.0)	2 (10.0)	5 (25.0)	11 (55.0)	2 (10.0)
F	20	3 (15.0)	16 (80.0)	4 (20.0)	0	0
G	19	5 (26.3)	0	14 (73.7)	5 (26.3)	0
H	24	3 (12.5)	4 (16.7)	14 (58.3)	6 (25.0)	0
I	20	15 (75.0)	0	0	17 (85.0)	3 (15.0)
J	8	2 (25.0)	0	1 (12.5)	5 (62.5)	2 (25.0)
K	20	3 (15.0)	1 (5.0)	2 (10.0)	13 (65.0)	4 (20.0)
Total	873	260 (29.8)	502 (57.5)	210 (24.1)	141 (16.2)	20 (2.3)

PE 0 – no macroscopic lesions; PE 1 – parakeratosis; PE 2 – erosions; PE 3 – ulcerations; PE – *pars oesophagea*.

A representative macroscopic appearance of gastritis and erosive lesions in the glandular region of the stomach is shown in Fig. 1.





Fig. 1. Representative macroscopic lesions of the glandular region of the porcine stomach. (A) Marked hyperaemia of the glandular mucosa consistent with gastritis. (B) Erosive lesions and early ulcerative changes in the fundic region.

3.2 Histopathological findings

Histopathological examination of selected samples confirmed inflammatory and degenerative changes in the glandular mucosa. Damage and desquamation of the surface epithelium, disruption of the architecture of the fundic glands, and reduction or loss of gastric pits were observed. The inflammatory infiltrate consisted of polymorphonuclear leukocytes, lymphocytes, and plasma cells. Additional findings included hyperaemia, venous stasis, submucosal oedema, vascular proliferation, and focal necrotic changes. Representative histopathological changes in the fundic region are shown in Fig. 2.

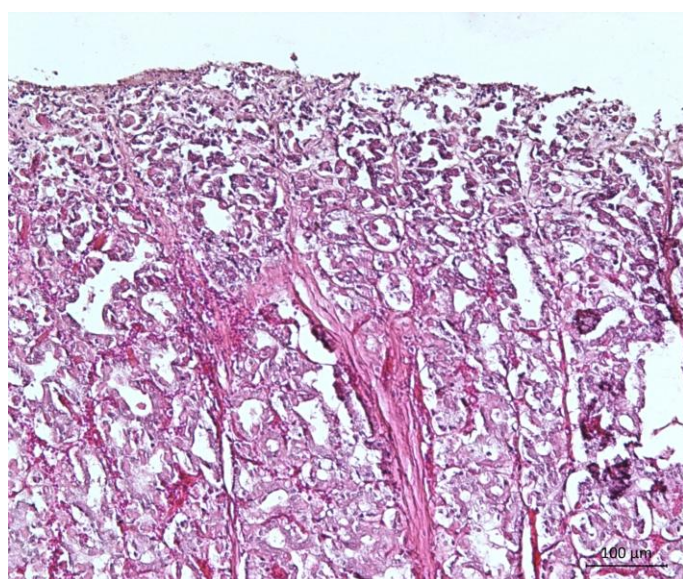


Fig. 2. Histopathological findings of gastritis in the fundic region of the porcine stomach. Denudation of the surface epithelium with exposure of the *lamina propria* and disruption of the normal architecture of the fundic glands (H&E, $\times 100$).



IV. Discussion

In the present study, gastritis of the glandular region of the stomach was detected in 29.8% of the examined commercial finishing pigs, indicating a relatively frequent occurrence of inflammatory changes in the glandular mucosa within the studied population. Although gastric lesions in pigs have been investigated for many years, research has focused predominantly on lesions of the *pars oesophagea*, whereas pathological changes of the glandular mucosa have received considerably less attention. Consequently, data on the occurrence of glandular gastritis in commercial pig populations remain relatively limited.

Macroscopic lesions of the *pars oesophagea* were detected in 42.5% of the examined stomachs, while erosions and ulcerations (scores 2–3) were present in 18.4%. The different characteristics of lesions affecting the glandular and non-glandular regions of the stomach are related to their distinct anatomical and physiological features. The *pars oesophagea* is lined by stratified squamous epithelium and lacks secretory glands, whereas the glandular mucosa possesses several protective mechanisms. The mucus-bicarbonate barrier, adequate mucosal blood flow, and the high regenerative capacity of the epithelium play important roles in maintaining mucosal integrity. Restitution and proliferation of epithelial cells, together with the activity of growth factors, contribute to rapid restoration of the damaged mucosal barrier [7].

Several nutritional and management-related factors may contribute to the development of gastric lesions in pigs. Physical characteristics of the feed, particle size, and dietary fibre content are considered particularly important. Mößeler et al. [1] demonstrated an association between feed processing and feeding method, characteristics of the gastric contents, and the development of gastric lesions. Similarly, Millet et al. [2] showed that feed particle size and crude fibre content influence gastric mucosal integrity in growing-finishing pigs. Finely ground feed may alter the consistency and stratification of gastric contents, thereby creating conditions that promote damage to the gastric mucosa. These factors may also be relevant when interpreting differences in the prevalence of gastritis among the groups examined in the present study, although the composition and physical characteristics of the diets were not systematically evaluated.

The prevalence of gastritis among the individual groups ranged from 12.5% to 75.0%. Such variability may reflect differences in housing conditions, feeding, animal health status, or environmental factors. In one of the examined groups, a high prevalence of gastritis was observed during a period of elevated environmental temperature. This observation is of interest in light of experimental evidence indicating adverse effects of heat stress on gastrointestinal barrier function in pigs [8]. However, given the observational nature of the present study and the absence of systematic monitoring of microclimatic conditions, no causal relationship between environmental temperature and the occurrence of gastritis can be established from these findings.

Histopathological examination of selected samples confirmed that macroscopic changes in the glandular mucosa were accompanied by inflammatory and degenerative alterations. Damage and desquamation of the surface epithelium, disruption of the architecture of the fundic glands, inflammatory cell infiltration, hyperaemia, venous stasis, oedema, and focal necrotic changes were observed. These findings are consistent with the general histopathological features of gastritis, in which neutrophils, lymphocytes, and plasma cells may be involved depending on the stage of the inflammatory process [4]. Histopathological examination therefore provides a useful complement to macroscopic assessment of the glandular mucosa and enables more precise characterization of the observed lesions.

The findings of this study highlight the importance of systematic evaluation of the entire stomach, rather than assessment of the *pars oesophagea* alone, during abattoir monitoring of pigs. Such an approach enables the detection of pathological changes affecting the glandular mucosa and provides a more comprehensive picture of the occurrence of gastric lesions in the examined populations. The observed variability in the prevalence of gastritis among groups also indicates the need for further research into the potential nutritional, environmental, management-related, and infectious factors involved in the development of glandular gastritis in pigs.



V. Conclusion

The present study demonstrates that glandular gastritis is a relatively common finding in commercial finishing pigs, affecting 29.8% of the examined stomachs. Histopathological examination of selected samples confirmed inflammatory and degenerative changes in the glandular mucosa. In addition, macroscopic lesions of the *pars oesophagea* were detected in 42.5% of the stomachs, with erosions and ulcerations accounting for 18.4% of the examined samples.

These findings support the systematic evaluation of both the glandular and non-glandular regions of the stomach during abattoir examination, as this approach may provide a more comprehensive assessment of gastric lesions in finishing pigs. A limitation of the present study is that potential nutritional, environmental, management-related, and infectious risk factors were not systematically investigated; therefore, the observed differences among groups cannot be interpreted as evidence of specific causal associations. Further studies combining abattoir monitoring with detailed assessment of these factors may help to identify conditions associated with the development of glandular gastritis and support the implementation of targeted preventive measures in commercial pig production.

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