

Prevalence of gastro-oesophageal ulcers in grower-finisher pigs in the Northern Province of South Africa

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ABSTRACT

Ulceration of the gastric *pars oesophagea* is a common problem in intensive pig production that is often detected at slaughter. A survey was carried out at the Pietersburg abattoir in the Northern Province during a 6-month period. In total, 4320 pig stomachs were examined. Gastro-oesophageal ulcers were observed in 5.1 % of the stomachs, gastric erosion in 15.2 %, and hyperkeratosis in 18.9 %. Time of slaughter was found to affect the prevalence of gastric lesions in the pig.

Key words: abattoir, erosion, gastric ulcers, pigs, South Africa.

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INTRODUCTION

Gastric ulceration in grower-finisher pigs, especially of the *pars oesophagea*, is observed worldwide in all areas where intensive pig production occurs. Haemorrhage from severe erosive lesions usually results in sporadic deaths, and losses in the stock population may be up to 1 % or higher. Previous abattoir studies have revealed that erosion, the precursor to ulcers in the *pars oesophagea*, may be present in 32–65 %¹ of slaughtered pigs.

The objectives of this survey were to establish the prevalence of gastric ulcers in abattoir pigs in the Northern Province, to assess the severity of the lesions in the *pars oesophagea* and to compare these with those previously reported.

MATERIALS AND METHODS

Pietersburg abattoir, where an average of 250 pigs are slaughtered per working day, was used for the survey. Random visits were made to the abattoir between July 1996 and February 1997. The abattoir receives pigs from intensive farms around the central region of the province. The pigs for slaughter had a live weight of 60–150 kg. Usually the pigs were

slaughtered within 2 h of arrival at the abattoir and stomachs were examined within 20 min of slaughter. Stomachs were recovered from carcasses on the slaughter line and cut open with a pair of scissors from the cardia to the pyloric sphincter along the lesser curvature. They were drained of their contents and the internal surfaces washed with running water. The *pars oesophagea* was then examined for gross pathological changes to the epithelium. Lesions were classified as either normal, hyperkeratotic, erosive or ulcerative as previously described¹¹. Briefly, the classification is as follows:

Normal: smooth, white glistening surface with no thickening.

Hyperkeratotic: light to dark yellow discoloration and keratinisation of the epithelial layer with thickening of the epithelium (*pars oesophagea*), and extreme surface-folding with formation of rugae.

Erosion: filament-like erosions with haemorrhage along the entire length of affected areas.

Ulceration: complete epithelial loss with exposure of the underlying muscular layer.

During each visit to the abattoir, at least 20 tissue samples were collected from the affected areas of the stomach, fixed in 10 % formalin and processed for histopathological examination. Those that could not be examined at Louis Trichardt Veterinary Laboratory were submitted to the Onderspoort Veterinary Institute, Pretoria.

The following time intervals were recorded:

1. Time between loading on farm and slaughter.
2. Time between last meal and slaughter.
3. Time between slaughter and *pars oesophagea* examination.

RESULTS

A total of 4320 pig stomachs was examined. The prevalence of gastro-oesophageal ulceration was 5.1 %, hyperkeratosis 18.9 % and erosion 15.20 % (Table 1). The time between loading the animals and arrival at the abattoir ranged from 20 min to 2 h and this was also approximately the time between the last meal and slaughter. In some cases animals were kept overnight before slaughter owing to technical or labour issues. The time between slaughter and stomach examinations was 15–20 min. More lesions were observed in stomachs of pigs kept overnight.

In the group with gastric ulcers, the gross lesions varied from acute with blood clots, to subacute to chronic gastric ulceration with possible perforation. Incidental observations were the presence of trichobezoar and plastic materials in the stomachs.

Histopathological examination revealed multifocal fibrino-purulent epithelial necrosis with haemorrhage. Other histopathological findings include coagulative necrosis, thrombi in blood vessels, leucocytic infiltration that varied from fairly heavy to light with neutrophils and macrophages as the main cell types. The necrosis was generally confined to the superficial layer of the *pars oesophagea*.

DISCUSSION

At 5.1 % the prevalence of gastric ulcers in pigs in the Northern Province is low compared with figures reported from other countries. For example, a figure of 19.6 % was reported in the USA², 18.3 % in Australia⁷, while 17.7 % was reported in Slovenia¹⁰ and 11 % in Canada⁴. However, lower figures of 2.5 % and 2.3 % were reported in England^{8,9} and 2.8 % in

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Table 1: Prevalence of lesions encountered in the *pars oesophagea* of pigs slaughtered in the Northern Province, South Africa ($n = 4320$).

	Number	Percentage
Normal	2627	60.8
Hyperkeratotic	816	18.9
Erosions present	657	15.2
Gastro-oesophageal ulcers present	220	5.1

Zimbabwe⁵. The low rate of gastric ulceration in pigs in the Northern Province may be a reflection of the high management standard on the farms that supply the abattoir and the correct feed particle size offered to the pigs.

Most of the active ulceration observed in this study was superficial with no involvement of the lamina propria. The lesions were fairly mild, as would be expected in pigs that survive to slaughter age. It is difficult to estimate the age of the lesions; a few showed evidence of healing by fibrosis and could therefore be considered chronic.

The aetiological agents for gastric ulceration in pigs are varied, including genetic predisposition, high levels of unsaturated fatty acids in the feed and finely-ground feed¹², management systems such as mixing and regrouping pigs of different ages, abrupt changes of feed both in quantity and quality or excessive medication. Other important nutritional factors are high levels of copper in feed, high levels of nitrofurans, feed with mould and mycotoxins or diets with low levels of protein, sulphur amino acids, phosphorus, zinc, selenium and vitamins⁶.

Ulcers can also develop as a result of infections with organisms such as *Helicobacter pylori* or secondary infections with *Escherichia coli*, *Streptococcus suis*, *Klebsiella pneumoniae*, *Lactobacillus* spp., moulds and yeasts¹⁰. Ulcers may also be one of the symptoms of the following infections: African swine fever, foot-and-mouth

disease, hog cholera, chronic erysipelas, rotavirus, clostridial infections, *Campylobacter* spp., *Ascaris suum*, *Hyostrongylus rubidus* and *Candida albicans*, and may occur in pigs with liver diseases⁶.

Pigs held overnight had a higher prevalence of gastric lesions than those slaughtered within 2 h of arrival at the abattoir. It has been suggested that gastric ulceration lesions can change and become more extensive between the time that the pigs leave the farm and when they are slaughtered⁴. We tend to support this as more gross lesions were observed in the *pars oesophagea* of pigs slaughtered more than 6 h after their last meal. The economic significance of this condition has also been assessed in study in which 2 herds with a high prevalence of severe erosive lesions gained 50–70 g/pig/day less than pigs with no lesions³. It is our recommendation that in view of this apparently little known and significant decrease in growth rate due to erosion, with the added possibility of mortality, attempts should be made to identify the aetiological agent for gastric ulceration in pigs on affected farms. Suggestions to ameliorate the problem and reduce production losses include feeding coarse feed and control of medication.

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REFERENCES

1. Ball R O, Friendship R M, Bubenik G A, Ayles H L 1996 Effect of dietary melatonin supplementation on gastric ulcers in swine. *Proceedings of the 14th International Pig Veterinary Society Congress, Bologna, Italy*, 7–10 July 1996: 694
2. Curtin T M, Goetsch G D, Hollandbeck R 1963 Clinical and pathologic characterization of esophagogastric ulcers in swine. *Journal of the American Veterinary Medical Association* 143: 854–860
3. Elbers A R W, Hessing M J C, Tielen M J M, Vos J H 1995 Growth and oesophagogastric lesions in finishing pigs offered pelleted feed *ad libitum*. *Veterinary Record* 136: 588–590
4. Friendship R M, Ball R O, Ayles H L 1996. The relationship between growth performance and gastric ulcer severity. *Proceedings of the 14th International Pig Veterinary Society Congress, Bologna, Italy*, 7–10 July 1996: 695
5. Makinde M O, Obwolo M J 1990 Abattoir survey of gastric ulcer in pigs in Zimbabwe. *Zimbabwe Veterinary Journal* 21: 116–123
6. Palomo A, Delalleau J, Pujads P, Carmona L, Sanchez M, Garcia A, Bravo J 1996 Study about etiological factors in gastroesophageic ulcer complex in growing-finishing pigs. *Proceedings of the 14th International Pig Veterinary Society Congress, Bologna, Italy*, 7–10 July 1996: 712
7. Penny R H C, Edwards M J, Mulley R 1972 Gastric ulcer in the pig – a New South Wales abattoir survey of the incidence of lesions of the *pars oesophagea*. *British Veterinary Journal* 128: 43–49
8. Penny R H C, Hill F W G. 1973 Abattoir observations of ulceration of the stomach (*pars oesophagea*) of the pig. *Veterinary Annual* 1973: 55–60
9. Reed J H, Kidder D E 1970 Post mortem survey of the relationships between the bile staining and ulceration of the oesophageal portion of pig's stomach. *Research in Veterinary Science* 11: 438–440
10. Senk L 1986 Incidence, morphology, morphogenesis and complications of gastric ulcer in post mortem examined pigs in Slovenia during 1963–1983. *Proceedings of the 9th International Pig Veterinary Society Congress, Barcelona, Spain*, 15–18 July 1986: 378
11. Straw B, Henry S, Melssen J, Doster A, Moxley R, Rogers D, Webb D, Hogg A 1992 Prevalence of lesions in the *pars oesophagea* of normal and sick pigs. *Proceedings of the 12th International Pig Veterinary Society Congress, The Hague, The Netherlands*, 17–20 August 1992: 386
12. Taylor D J, 1983 *Pig diseases* (3rd edn). Burlington Press. Cambridge