

The prevalence of subclinical gastroduodenal ulceration in Dachshunds with intervertebral disc prolapse

S M Dowdle^{a*}, K E Joubert^a, N E Lambrechts^a, R G Lobetti^{a†} and A D Pardini^b

ABSTRACT

Endoscopy was used to determine the prevalence of subclinical gastroduodenal ulceration in 30 Dachshunds undergoing decompressive surgery for acute intervertebral disc prolapse. The endoscopy was performed on the day of admission and on the 3rd or 4th day after surgery. Three regions of the stomach (cardia, corpus and pylorus) and the proximal duodenum were visually inspected and biopsy samples were taken for histopathology. The combination of visual and microscopic changes were then used to determine the prevalence of subclinical gastroduodenal ulceration in this population. An overall prevalence of 76 % was calculated from these findings. Ulcerogenic medication administered prior to admission did not appear to influence the prevalence. This result identifies a need for veterinarians to be aware of this potentially severe complication and warrants the use of prophylactic anti-ulcer medication in spinal surgery patients.

Key words: canine, dog, endoscopy gastric ulcer, intervertebral disc disease, spinal cord.

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INTRODUCTION

Intervertebral disc prolapse (IVDP) is the most common neurological condition seen in the dog, particularly in the chondrodystrophic breeds^{3,11,13,14,16,21,25,27}. In human medicine, the correlation between spinal cord injury and gastroduodenal ulceration (GDU), as well as the tendency to misdiagnose the latter, has been recognised for many years^{15,23}. Gastric erosions are defined as superficial mucosal defects that do not penetrate the lamina muscularis mucosae, while gastric ulcers penetrate this layer and may even perforate it. If mucosal damage exceeds the reparative process, then erosions can progress to ulcers⁹. Less severe lesions that occur in the early development of an ulcer manifest as inflammation of the mucosa, causing hyperaemia and submucosal haemorrhages.

Gastroduodenal ulceration is reported to frequently accompany IVDP in the

dog^{4,7,15,19,25,26}. Until recently, however, the actual prevalence and severity of this complication had not been determined²⁰. A 2 % mortality rate has been surmised in cases of IVDP associated with gastrointestinal complications¹⁹.

Most authors concur that the aetiology of GDU, in general, is multifactorial. It is usually secondary to systemic disease, trauma, stress, hypovolaemia or administration of ulcerogenic drugs^{5,19,20,25,28}. In IVDP cases, it is postulated that autonomic dysfunction caused by spinal cord compression leads to hypersecretion of gastric acid and pepsin, with resultant GDU^{5,15}.

Ulcerogenic drugs such as corticosteroids and non-steroidal anti-inflammatory drugs (NSAID) are commonly used in patients with spinal injury and have been shown to be beneficial in reducing local oedema, providing analgesia and limiting the 'autoinflammatory' cycle that may lead to the progression of ascending haemorrhagic necrosis of the spinal cord²⁷. However, corticosteroid therapy predisposes the patient to GDU and perforation by inhibiting the action of prostaglandins, which are important in maintaining gastroduodenal mucosal integrity by enhancing secretions of bicarbonate and mucus^{2,5,6,17,20,24}. Similarly, NSAIDs increase the likelihood of gastroduodenal

haemorrhage and ulceration by affecting prostaglandin-mediated defence mechanisms of the bowel wall^{2,5,17,18,24–26,28}.

In the past, an accurate antemortem diagnosis of gastrointestinal complications was made only in 1 of every 4 cases presented^{6,25}. Clinical signs such as melaena, intermittent vomiting, abdominal pain and decreased haematocrit, were used as indicators of the presence of gastric ulceration and haemorrhage^{17,25,26,28}. The rapid onset of gastrointestinal complications, together with the fact that very few clinical signs may be present, means that this condition is often recognised too late for appropriate treatment protocols to be implemented effectively¹⁷. For this reason, a prophylactic approach to gastroduodenal complications in high-risk patients, such as those presenting with spinal cord injury, has been recommended^{5,25}.

Endoscopic examination provides a safe, non-invasive and sensitive technique for the early diagnosis of gastric mucosal lesions (submucosal haemorrhage and hyperaemia) as well as obvious ulcerations^{8,10,12}. In addition, it facilitates visualisation of the colour and integrity of multiple sites of the gastric mucosa, without the risk of dehiscence or peritonitis associated with surgical exploration⁶. It has also been shown to be more accurate for detecting subclinical GDU than diagnostic imaging modalities like ultrasonography or contrast gastrography¹².

The purpose of this study was to determine the prevalence of subclinical GDU in patients with IVDP, before and after undergoing decompressive spinal surgery, in order to formulate a risk profile for these animals.

MATERIALS AND METHODS

The Animal Use and Care Committee of the Faculty of Veterinary Science (OVAH), University of Pretoria, approved the research reported in this project.

Thirty Dachshunds admitted to the Onderstepoort Veterinary Academic Hospital with clinical signs suggestive of acute thoracolumbar or lumbar IVDP, were assigned to this study after myelographic confirmation. This sample size was chosen using statistical analysis

^aDepartment of Companion Animal Clinical Studies, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

^bEquine Research Centre, Faculty of Veterinary Science, University of Pretoria, Private Bag X04, Onderstepoort, 0110 South Africa.

*Author for correspondence. Present address: Ridgeway Veterinary Hospital, PO Box 1311, Randpark Ridge, 2156 South Africa. E-mail: sdowdle@mweb.co.za

†Present address: Bryanston Veterinary Hospital, PO Box 67092, Bryanston, 2021 South Africa.

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(SPSS, SPSS Inc., Chicago) based on a sample population of 3000 Dachshunds with a 90 % confidence interval and an estimated expected prevalence of 30 % and a 10 % accuracy range.

Dogs of any age, sex or weight were accepted into the trial. A full clinical and drug history was obtained. The duration of clinical signs before admission, treatments administered prior to admission and any previous history of IVDP were recorded. Clinical examination and serum chemistry (urea, creatinine, alkaline phosphatase, alkaline transaminase, total serum protein, albumin and globulin levels) were performed to rule out concurrent diseases that may have predisposed the dogs to GDU. A full neurological examination, including assessment of ambulation, flexor and extensor reflexes, deep pain sensation, tail response to vocal stimulation and proprioception, was performed to assess the severity and location of the spinal cord injury.

Following premedication with diazepam (0.2 mg/kg) (Valium, Roche Pharmaceutical, Isando) and morphine (0.4 mg/kg) (Morphine Sulphate, Micro HealthCare, Bethlehem), anaesthesia was induced with propofol (6 mg/kg) (Diprivan, Astra-Zeneca, Sandton). Intravenous Ringer's lactate (Sabax, Adcock Ingram, Johannesburg) was administered throughout the procedures to maintain blood pressure. Anaesthesia was maintained with halothane (Fluothane, Astra-Zeneca, Sandton) delivered in oxygen through an out-of-circuit, precision vaporiser and a non-rebreathing anaesthetic circuit. The fresh gas flow was maintained at twice the minute volume. Heart rate, respiratory rate and pulse oximetry (Satellite plus, Datex, Helsinki) were monitored throughout the procedure. A lumbar myelogram was performed within the 1st 6 hours of admission to the hospital, in order to confirm and locate the compressive spinal cord lesion¹⁴.

Gastroduodenoscopy was performed immediately after the myelogram, but prior to surgery. A 1-m-long, flexible endoscope (Olympus GIF XQ200) was used with a 2.8 mm biopsy channel and a mobile tip. In each case, the cardia, corpus, pylorus and proximal duodenum were evaluated, using the following criteria: colour and appearance of mucosa, presence and distribution of submucosal haemorrhages (erosions) and/or obvious ulceration. Three to 4 random, pinch biopsies were taken through the biopsy channel from each of these anatomical locations and placed in 10 % buffered formalin. Samples were dehydrated, embedded in paraffin, sectioned and

stained with haemotoxylin and eosin (H&E) for light microscopy.

The following histopathological changes were sought: infiltration of neutrophils and macrophages (indicating inflammation), disruption of the mucosal lining and haemorrhage into the submucosa (indicating erosions) and obvious ulcerations (lesions disrupting the mucosa and penetrating the muscularis layer). Patients with evidence of erosions and/or obvious ulceration were then designated as having (positive) evidence of ulceration, while those without were considered negative. Results from gastroduodenoscopy and histopathology were combined to determine an overall prevalence of GDU for this population. All gastroduodenoscopy procedures were videotaped and photographs taken of areas of interest.

Following gastroduodenoscopy, corticosteroids (methylprednisolone (30 mg/kg), Solucortef, Upjohn Pharmacia, Isando) and antibiotics (amoxicillin (20 mg/kg), Amocillin, Caps Pharmaceuticals, Isando) were administered intravenously. Decompression of the spinal cord was then performed following the technique described by Lubbe¹⁶. Post-operative amoxicillin (20 mg/kg) and morphine (0.2 mg/kg) were administered at 4 and 12 hours post-surgery.

All patients remained hospitalised until the follow-up gastroduodenoscopy. This was repeated on the 3rd or 4th day after surgery, following the induction of anaesthesia with propofol. The same 4 areas of interest mentioned above were examined and biopsied.

Data were divided into 2 groups, positive or negative, based on the above criteria. Student's *t*-tests and the Mann-Whitney Rank Sum tests were used to determine differences between the 2 groups. These differences included severity of spinal cord injury, duration of injury prior to admission, treatment with ulcerogenic drugs, length of general anaesthesia, age, weight and sex. For correlation between variables and the prevalence of gastric ulceration, Fisher's

exact test was used. McNemar's test was used for non-parametric data. *P* was set at ≤ 0.05 . The data were statistically analysed using Sigma Stat ver. 4 (Jandel Scientific, Milwaukee).

RESULTS

No dogs were excluded from this study, but 1 dog did not receive a post-operative endoscopic examination as it was an undue anaesthetic risk. Thirteen animals were male (3 neutered) and 17 female (12 neutered). Median age and weight were 5.6 ± 1.9 years and 6.8 ± 1.7 kg, respectively. Six dogs had suffered a previous incident of IVDP. Twenty-one dogs had received treatment prior to referral with corticosteroid and/or a NSAID (Table 1). The median duration of clinical signs prior to admission was 11.4 ± 18.9 hours. No dogs showed clinical signs of GDU at the time of admission. The median duration of anaesthesia from induction for the myelogram until the completion of surgery was 160.8 ± 31.2 minutes, while surgery lasted 78.5 ± 20.5 minutes.

Neurological deficits based on criteria recommended by Lubbe¹⁶ were present in 21 dogs. The other 9 dogs presented with thoracolumbar or lumbar spinal pain only. Four of the animals were considered to have severe neurological deficits, that is, limited or no deep pain sensation was present in either pelvic limb. A summary of the anatomical location of IVDP lesions is given in Table 2, and is in agreement with locations indicated in previous studies¹⁶. The results of the gastroduodenoscopy and histopathology examinations are presented in Table 3.

Endoscopic lesions identified ranged from mucosal hyperaemia (Figs 1, 4) to submucosal haemorrhage (Figs 1, 2) and obvious ulceration (Fig. 3). The incidence of these lesions is presented in Table 4.

No correlation was found between the prevalence of GDU and administration of ulcerogenic drugs prior to the admission. Similarly, no correlation was found between GDU and the severity of neurological signs, the duration of signs prior to

Table 1: Potentially ulcerogenic drugs administered to patients prior to admission.

| Drug or drug combination | Number of patients (<i>n</i> = 18) |
|---------------------------------|-------------------------------------|
| Prednisolone | 6 |
| Dexamethazone | 3 |
| Phenylbutazone/prednisolone | 2 |
| Flunixin meglumine | 1 |
| Flunixin meglumine/prednisolone | 1 |
| Phenylbutazone | 1 |
| Ketoprofen | 1 |
| Meloxicam | 1 |
| Methylprednisolone | 1 |
| Prednisolone/dexamethazone | 1 |

Table 2: Distribution of intervertebral disc prolapse lesions.

| Disc space | Number (n = 30) | Prevalence (%) |
|------------|-----------------|----------------|
| T11-T12 | 2 | 6.67 |
| T12-T 13 | 8 | 26.67 |
| T13-L1 | 10 | 33.33 |
| L1-L2 | 5 | 16.67 |
| L2-L3 | 2 | 6.67 |
| L3-L4 | 2 | 6.67 |
| L4-L5 | 0 | 0 |
| L5-L6 | 1 | 3.33 |

Table 3: Number and percentage of patients with evidence of gastroduodenal ulceration at various anatomical sites in the gastrointestinal tract as assessed by either gastroduodenoscopy or histopathology. The overall incidence was calculated from pre- and post-surgical results when a positive result was found with either one of the methods used to assess gastroduodenal ulceration.

| Sampling stage | Cardium | | Corpus | | Pylorus | | Duodenum | |
|--|---------|------|--------|------|---------|------|----------|------|
| | (n) | (%) | (n) | (%) | (n) | (%) | (n) | (%) |
| Pre-surgery (n = 30) | | | | | | | | |
| Gastroduodenoscopy | 6 | 20 | 5 | 16.7 | 15 | 50 | 1 | 3.3 |
| Histopathology | 7 | 23.3 | 7 | 23.3 | 15 | 50 | 6 | 20.0 |
| Post-surgery (n = 29) | | | | | | | | |
| Gastroduodenoscopy | 4 | 13.8 | 5 | 17.3 | 14 | 48.2 | 1 | 3.3 |
| Histopathology | 7 | 24.1 | 10 | 34.5 | 12 | 41.4 | 8 | 27.6 |
| Overall incidence of ulceration | | | | | | | | |
| Pre-surgery | 11 | 36.7 | 10 | 33.3 | 21 | 70 | 6 | 20.0 |
| Post-surgery | 8 | 27.6 | 12 | 41.4 | 17 | 58.6 | 8 | 27.6 |

admission, the length of the procedure (general anaesthetic or surgery), age, weight or sex. The overall prevalence of GDU-positive dogs was 76 %.

DISCUSSION

In 1975, Hoerlein¹¹ reported various non-neurological complications following decompressive spinal cord surgery in dogs, especially the occurrence of gastric, duodenal and colonic ulceration. Toombs^{25,26} reported the presence of colonic perforation following neurosurgical procedures with concurrent corticosteroid treatment. They concluded that antemortal diagnosis in these cases was difficult and that a prophylactic approach to these complications was warranted. Moore¹⁹ showed in a retrospective study, based on clinical signs, that the prevalence of gastrointestinal complications (pancreatitis, gastrointestinal haemorrhage, ulceration, and perforation) was as high as 15 % in dogs with IVDP, with a mortality rate of approximately 2 %.

In the present study, the overall prevalence of subclinical GDU was 76 % based on either endoscopic or histopathological evidence of ulceration at any 1 of the 4 above-mentioned regions in the gastrointestinal tract. Histopathology identified mucosal changes and ulcerations that were not evident during gastroduodenoscopy, thus increasing the sensitivity of detecting mucosal changes.

The highest overall prevalence of ulceration occurred in the pyloric region of the stomach, both before and after surgery. Histopathology of the proximal duodenum revealed that, in this region, endoscopic visualisation was much less sensitive for detecting mucosal pathology than histopathology. The apparent lower prevalence of GDU in the post-surgery groups was not statistically significant. Cases in which ulcers were visualised, but histopathology was negative, could be explained by the fact that biopsy samples were taken randomly from the 4 identified regions, thus visualised mucosal changes were not necessarily biopsied.

In a similar study, Neiger *et al.*²⁰ used

endoscopic examination to determine the incidence of GDU in dogs with acute intervertebral disc disease and concurrent corticosteroid use. All dogs in their study received dexamethasone (2 mg/kg IV) on day 1 of admission followed by prednisolone (1 mg/kg PO) daily for the duration of hospitalization. Histopathology was not performed, and their results indicated an overall prevalence of 76 % GDU-positive dogs. There was no significant decrease seen in the incidence of GDU with concurrent anti-ulcer treatment. They concluded either that anti-ulcer medication was instituted too late to prevent mucosal lesions from forming or that the drugs and dosages used were not

Table 4: Incidence of endoscopic lesions identified in both pre- and post-surgery procedures. This table provides the number and percentage of patients with specific endoscopic findings related to gastroduodenal ulceration.

| Parameter | Number | Percentage |
|------------------------------|--------|------------|
| Pre-surgery (n = 30) | | |
| Obvious ulceration | 1 | 3.3 |
| Submucosal haemorrhage | 12 | 40.0 |
| Hyperaemia | 5 | 16.7 |
| Normal | 12 | 40.0 |
| Total | 30 | 100 |
| Post-surgery (n = 29) | | |
| Obvious ulceration | 3 | 10.3 |
| Submucosal haemorrhage | 9 | 31.0 |
| Hyperaemia | 5 | 17.2 |
| Normal | 12 | 41.5 |
| Total | 29 | 100 |

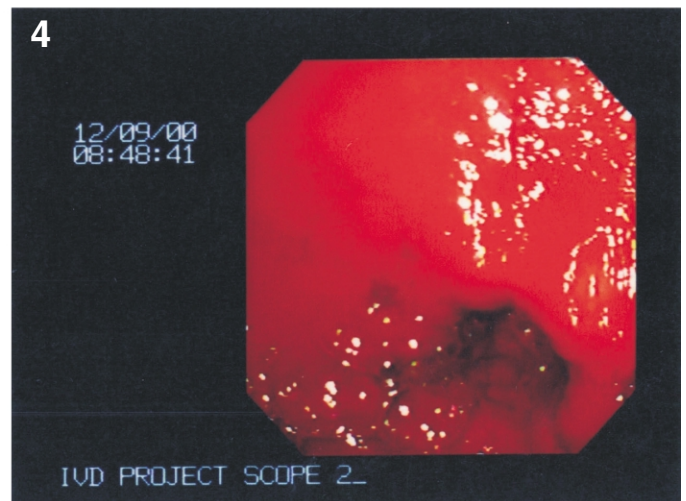
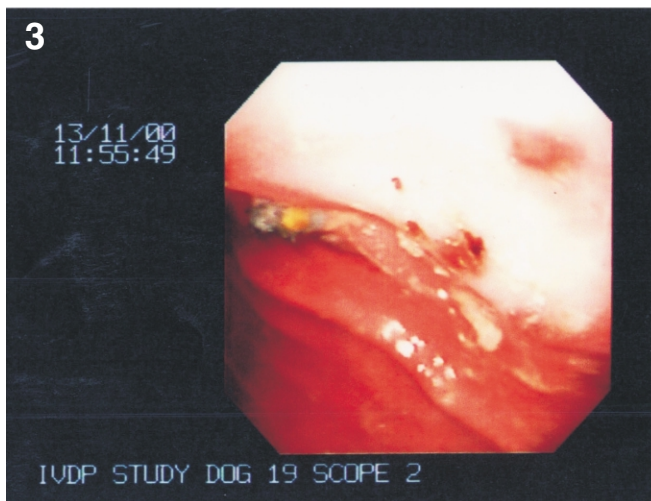
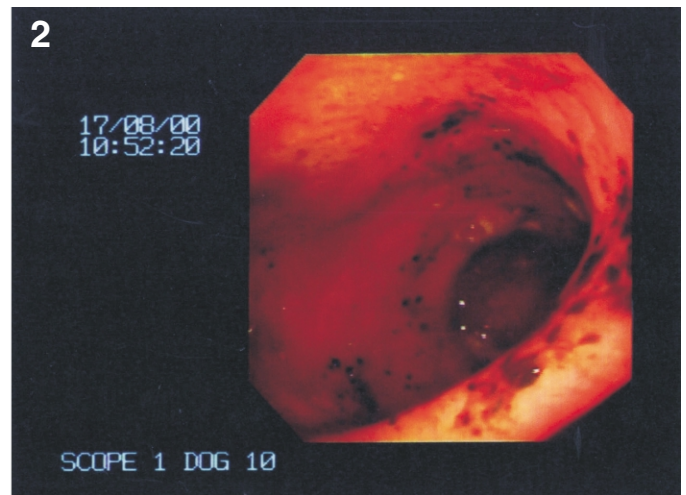
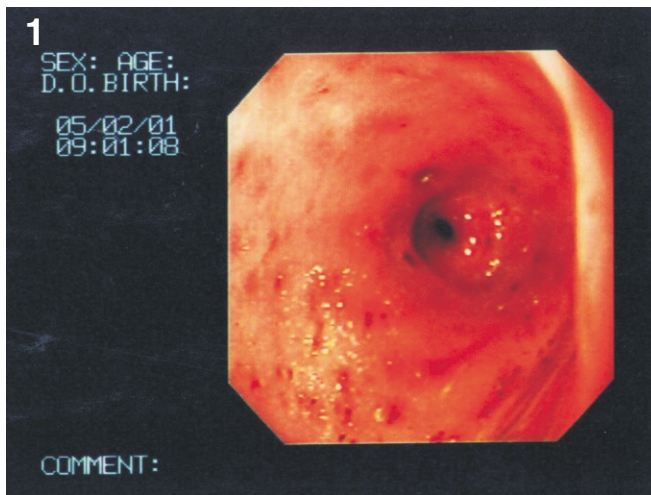


Fig. 1–4: 1: Endoscopic view of the corpus of the stomach showing multifocal, mucosal hyperaemia, with more severe, submucosal haemorrhages occurring in the pylorus; 2: severe, diffuse, submucosal haemorrhages in both the corpus and pyloric regions of the stomach of a dog, as seen using endoscopy on the day of admission; 3: endoscopic view of the minor incisure of the corpus of the stomach showing obvious, gastric ulceration, with complete penetration of the mucosal layer; 4: severe, diffuse, mucosal hyperaemia noted on endoscopy of the pyloric region, on the third day after surgery.

effective in acute intervertebral disc disease cases.

The prevalence of GDU in the present study was similar to that described by Neiger²⁰, in spite of our use of histopathology. This either suggests that endoscopic visualisation alone is a sufficiently sensitive diagnostic modality, or that the incidence in Neiger's study would have been higher had histopathological examinations been performed.

No correlation was found, in this study, between the use of ulcerogenic drugs and the prevalence of ulceration, or between severity and duration of spinal cord compression with GDU. This may be an indication of insufficient sample size. More importantly, it indicates that ulcerogenic drugs probably play a lesser part in the pathophysiology of GDU in dogs with IVDP, and autonomic dysfunction caused by compression of the spinal cord could play a more dominant role than has previously been reported.

The prevalence of gastroduodenal haemorrhage and ulceration in dogs with neurological disease that have not received any form of ulcerogenic drug treatment has not yet been reported²². In this study, 11 dogs had not received any form of ulcerogenic drug prior to admission. Of these, 6 presented with submucosal haemorrhage and/or obvious ulceration. Of the 19 dogs that received ulcerogenic drugs, 13 showed visual signs of submucosal haemorrhages and/or obvious ulceration. This supports the view that GDU in IVDP cases is not primarily related to ulcerogenic medication.

Future research should be directed at strategies to reduce the incidence of gastric ulcers in dogs with IVDP. This may involve judicious use of ulcerogenic drugs in the pre-surgical period and earlier surgical intervention. The early use of prophylactic anti-ulcer medication is recommended in all patients presenting with IVDP.

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