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# HISTOLOGICAL CHANGES IN STOMACHS OF APPARENTLY HEALTHY DOGS INFECTED WITH *HELICOBACTER*

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*The aim of our study was to determine the prevalence and the intensity of infection with Helicobacter sp. in a group of 23 apparently healthy dogs in order to describe the morphological features of Helicobacters, to detect histopathological changes in the gastric mucosa and to evaluate if there is a correlation between infection and eventual alterations in the stomach.*

*Helicobacters were detected in the stomach of all dogs: they were spirally shaped with five to twelve turns. According to the difference in width and length between the turns we formed a group resembling H. salomonis and H. heilmannii and a group resembling H. felis and H. bizzozeronii. The width of the narrower spiral bacteria, resembling H. felis and H. bizzozeronii, varied from 0.29 to 0.5 µm and the length between the turns was 0.4 µm. The wider, "screw-like" bacteria, which resembled H. salomonis and Helicobacter heilmannii had numerous turns and measured from 0.55 to 0.72 µm, whereas the length between turns varied from 0.55 to 0.95 µm.*

*Helicobacters were present in all examined parts of the stomach, i.e. in the fundus, corpus and antrum at equal density. They colonised the gastric mucus, gastric pits and gastric glands. A mild and a moderate infection in 13% of the dogs and a severe infection in 74% of the dogs was determined. Only a mild chronic gastritis was diagnosed in the gastric fundus in 16 dogs, of gastric corpus in 18 dogs and in the antral region in 18 dogs. We concluded that gastritis is not directly linked to the helicobacter infection in our dogs.*

*Key words: dog, gastritis, helicobacteriosis, histopathology*

## INTRODUCTION

*Helicobacters* are gram-negative, curved to spiral, motile, non-sporulating bacteria, which can be found in the gastrointestinal and hepatobiliar system of various domestic and wild animal species and humans (Neiger and Simpson, 2000). They are highly prevalent in the stomach in healthy dogs and in dogs with gastrointestinal disorders (Peyrol *et al*., 1998; Yamasaki *et al*., 1998; Vajner *et al*.,

2000) colonising gastric mucus, gastric glands, gastric pits and parietal cells in all parts of the stomach (Neiger and Simpson, 2000).

It is known that helicobacter infection is in correlation with chronic gastritis, ulcerations and gastric adenocarcinoma in humans (Simpson and Burrows, 1997; Švec *et al*., 2000), gastritis in cats (Hermanns *et al*., 1995) and ulcers in pigs (Queiroz *et al*., 1996; Szeredi *et al*., 2005; Appino *et al*., 2006), but it is still under debate, if *Helicobacters* cause any alteration in gastric mucosa of dogs.

In our study we necropsied 23 dogs from a big branch, which showed no gastrointestinal disorders. We determined the prevalence of infection, characterised the intensity of infection, established morphologic features of *Helicobacters*, detected histopathological changes in gastric mucosa and tried to find out if there is any correlation between the infection and the severity of infection, and changes in the stomach.

## MATERIAL AND METHODS

*Animals*

Twenty-three mixed breed dogs – 10 males and 13 females, at the age from 4 months to 12 years (mean 4 years), which lived freely in a group in a small village, were euthanized. All dogs were fed home prepared food. Dogs were apparently healthy, without any signs of gastrointestinal disorders.

*Necropsy and histologic examination*

All dogs were necropsied immediately after euthanasia. Stomachs were removed, opened along the smaller curvature and rinsed under tap water to remove food particles. Gastric mucosa was examined macroscopically for the presence of alterations. From each dog samples from three gastric parts, i.e. fundus, corpus and antrum were taken for histopathologic examination and put in 10 % buffered formalin for 24 hours. Formalin fixed samples were routinely embedded in paraffin and cut in 6 $\mu$m sections. Sections, stained with touluidin blue, were examined with a light microscope at high magnification (1000 x) for the presence of *Helicobacters* and infection intensity. With touluidin blue *Helicobacters* stain intensive blue. The sections, stained with hematoxylin and eosin (HE), were examined with light microscope at magnification of 200 x and 400 x for the evaluation of histological changes in all layers of the stomach.

The morphology of *Helicobacters* was visualised at high magnification (1000 x). Using the program "Lucija G" (Optoteam, Austria) the length and the width of 100 *Helicobacters* were measured, as well as the distance between the turns and the number of turns.

The immunohistochemical method was performed on paraffin tissue sections of 10 stomachs. Paraffin sections were treated with microwaves for 10 minutes and after cooling, endogenous peroxidase activity was quenched in 3% hydrogen peroxide in phosphate buffered saline (PBS). For immunolabelling we used commercial polyclonal antibodies against *H. pylori* (DAKO, Denmark), diluted 1:10 and 5% bovine serum albumine in PBS. Tissue sections were incubated in primary antibodies for 60 minutes at room temperature. LSAB®2

System HRP kit, DAKO (DAKO, Denmark) was applied as a visualisation system and 3.3-diaminobenzidine tetrahydrochloride (DAB) was used as a substrate. Sections were counterstained with haematoxylin, dehydrated and mounted with resin: *Helicobacters* stained golden-brown.

The intensity of the infection was defined by the bacteria count at high magnification (1000 x): mild – less than 5 *Helicobacters* per section; moderate – 5 to 20 *Helicobacters* per section; severe – more than 20 *Helicobacters* per section.

For histological diagnosis we used the same criteria as Happonen *et al*. (1998) proposed to use for the evaluation of histological changes. The criteria for histological diagnosis are presented in Table 1.

Table 1. Criteria for histological diagnoses of gastric samples (Happonen *et al*., 1998). Number of each cell type is the mean of three fields, viewed at magnification, x 400 and the number of lymphocyte aggregates is the number in a sample at magnification x 200

| Histological Diagnosis | Criteria |
|---|---|
| Normal gastric mucosa | 0 neutrophils, 0-10 lymphocytes and plasma cells, no aggregates, normal epithelium |
| Mild acute gastritis | 1-10 neutrophils |
| Moderate acute gastritis | 10-50 neutrophils |
| Severe acute gastritis | more than 50 neutrophils |
| Mild chronic gastritis | 10-50 lymphocytes and plasma cells, ≤2 aggregates, normal epithelium |
| Moderate chronic gastritis | 10-50 or more lymphocytes and plasma cells, ≥3 aggregates, normal epithelium |
| Severe chronic gastritis | 10-50 or more lymphocytes and plasma cells, ≥3 aggregates, epithelial changes |

## RESULTS

There were no macroscopically visible changes in the gastric mucosa in any of the examined dogs.

*Helicobacters* were detected in stomachs of all dogs by histological examination, using the toluidine blue staining method. A mild and a moderate infection was determined in 13% of dogs (n=3), and a severe infection in 74% of dogs (n=17).

The immunohistochemical method gave us a strong positive reaction in all ten stomachs. *Helicobacters* were clearly visible, stained golden-brown (Fig. 1). *Helicobacters* were spirally shaped with five to twelve turns, 3.24 – 8.61 $\mu$m long

(5.6 $\mu$m in average) and they differed in width and length between the turns. The width of the narrower spiral bacteria varied from 0.29 to 0.5 $\mu$m and the length between the turns was 0.4 $\mu$m. The wider, "screw-like" bacteria, with numerous turns, had from 0.55 to 0.72 $\mu$m in wideness and the length between turns varied from 0.55 to 0.95 $\mu$m. On the basis of morphological properties we formed two groups of helicobacters: a group of the wider "screw-like" bacteria with numerous turns, which resembled *H. salomonis* and *Helicobacter heilmannii*, and a group of the narrower spiral bacteria with high density turns resembling to *H. felis* and *H. bizzozeronii*.

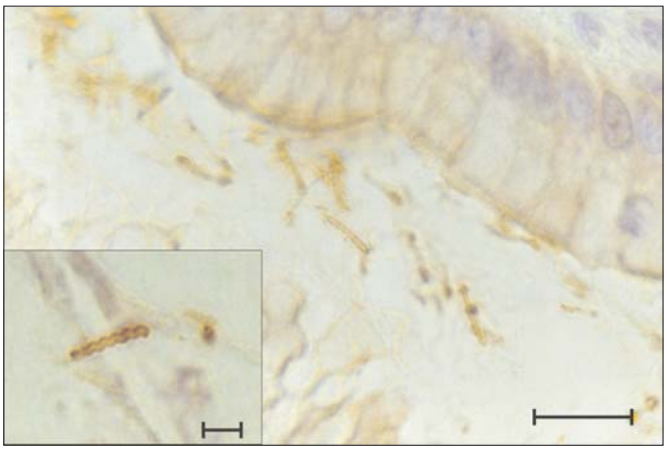

Figure 1. *Helicobacters* in the gastric mucus of a dog's stomach. Immunohistochemistry, anti-*H. pylori* antibody, LSAB, DAB substrate, counterstained with haematoxylin, 1000 x

*Helicobacters* were located in all the examined parts of the stomach, i.e. in the fundus, corpus and antrum. The density of bacterial population was the same in all parts of the stomach, in each of examined dogs. *Helicobacters* colonised gastric mucus, gastric pits and gastric glands lumen, but they were absent from the connective tissue of the lamina propria, submucosa, tunica muscularis mucosae and tunica serosa. The highest number of bacteria was found in the gastric mucus and in gastric pits. Gastric glands lumens were less severely infected.

The gastric mucus covered intact epithelium in the stomach of 22 dogs, only in the fundus of one dog there was no gastric mucus and the epithelium was depleted. The gastric pits were wider in the fundus of 17 dogs, in the corpus of 16 dogs and in the antrum of 11 dogs. In the fundus of one of the dogs they were completely missing. We found compressed and partly degenerated gastric glands in the fundus of 14 dogs, in corpus of four dogs and in antrum of 20 dogs. Gastric glands were missing in the fundus of one of the dogs. In all stomachs we noticed only very small foci of fibroblasts in propria, but in the fundus of one of the dogs there was also a severe segmental fibrosis with strong lymphocytic and

plasmacellular infiltration. In the the fundus of four dogs, corpus of three dogs and antrum of one of the dogs there were less then 10 lymphocytes and plasma cells per field; in all other dogs the number of them varied from 10 to 50 per field. In the corpus of one of the dogs we also found numerous disseminated eosinophilis just under the epithelium and in the antrum of one a diffuse neutrophil infiltration. Single lymphocyte aggregates were detected in the fundus of six, in the corpus of two and in the antrum of four dogs. In the fundus of one of the dogs and in the corpus of two there were also severe multifocal haemorrhages. We found hypertophy of the muscular part of the mucosa in the fundus and corpus of five dogs and in the antrum of 11 dogs. There were no changes in the submucosa of 22 dogs, but in the antrum of one of the dogs the mucosa was severely infiltrated with lymphocytes. The tunica muscularis and lamina serosa were intact in all dogs.

On the basis of histopathological changes in gastric mucosa we diagnosed a mild chronic gastritis of the gastric fundus in 16 dogs, of gastric corpus in 18 dogs and in the antral region in 18 dogs (Fig. 2). A moderate or even a strong chronic gastritis were not diagnosed. We found erosive gastritis of the gastric fundus and antrum in one of the dogs and a focal eosinophilic gastritis in the corpus of one. The gastric mucosa was normal in the fundus of five dogs and in the corpus and antrum of four dogs. Types of diagnosed gastritis and the intensity of infection are presented in Table 2.

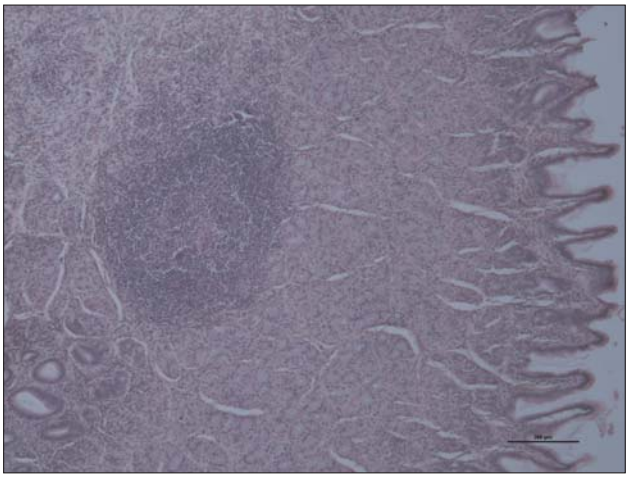

Figure 2. Fundus of a dog. A lymphocyte aggregate with a germinative centre in the gastric mucosa – a mild chronic gastritis. HE, 40 x

We haven't found any correlation between the infection and the intensity of the infection and the histopathological changes in the gastric mucosa.

Table 2. Types of diagnosed gastritis in all examined parts of the stomach and intensity of infection

| Dogs number | Fundus | Corpus | Antrum | Intensity of infection |
|---|---|---|---|---|
| 1 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 2 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 3 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 4 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 5 | Acut erosive gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 6 | Normal gastric mucosa | Normal gastric mucosa | Mild chronic gastritis | Severe |
| 7 | Mild chronic gastritis | Eosinophil gastritis | Mild chronic gastritis | Severe |
| 8 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 9 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 10 | Mild chronic gastritis | Mild chronic gastritis | Normal gastric mucosa | Severe |
| 11 | Normal gastric mucosa | Normal gastric mucosa | Acute erosive gastritis | Moderate |
| 12 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 13 | Normal gastric mucosa | Mild chronic gastritis | Mild chronic gastritis | Mild |
| 14 | Mild chronic gastritis | Mild chronic gastritis | Normal gastric mucosa | Mild |
| 15 | Normal gastric mucosa | Normal gastric mucosa | Normal gastric mucosa | Mild |
| 16 | Normal gastric mucosa | Mild chronic gastritis | Normal gastric mucosa | Severe |
| 17 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 18 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 19 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Moderate |
| 20 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 21 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Severe |
| 22 | Mild chronic gastritis | Mild chronic gastritis | Mild chronic gastritis | Moderate |
| 23 | Normal gastric mucosa | Normal gastric mucosa | Mild chronic gastritis | Severe |

## DISCUSSION

Spiral bacteria were found in stomachs of all of the examined dogs. Performing immunohistochemistry in ten dogs it was confirmed that they belong to the genus *Helicobacter*. Pirarat *et al*. (2003) and Prachasilpchai *et al*. (2007) also showed that commercial polyclonal antibodies against *H. pylori* are very sensitive for the detection of *Helicobacters* in paraffin embedded samples from dogs' stomachs. Differences in morphology indicate that our dogs had been infected with two or more different species of *Helicobacters* – the fact was confirmed by Van den Bulk *et al*. (2005), who have successfully isolated and determined *Helicobacter felis*, *H. bizzozeronii*, and *H. salomonis* from stomachs of our dogs. Mixed infection with two or even more species of *Helicobacters* are often reported by several authors (Happonen *et al*., 1998; Jalava *et al*., 1998; Peyrol *et al*., 1998; Cattoli *et al*., 1999). A mild and a moderate infection were determined in 13 % and a severe infection in 74% of dogs in our study. Peyrol *et al*. (1998) described a mild to moderate infection in 42% and a severe infection in 58% of asymptomatic laboratory beagles.

The density of *Helicobacters* was the same in the fundus, corpus and antrum in each of our dogs. Cattolie's *et al*. (1999) and Happonen's *et al*. (1996, 1998) research gave the same results. Scanziani *et al*. (1997) found *Helicobacters* more often in the fundus and antrum. Japanese scientists (Yamasaki *et al*., 1998) on the other hand discovered that the fundus is more heavily colonised by *Helicobacters* than pylorus. After experimental infection of dogs with *Helicobacter*, Simpson *et al*. (1999) found the largest number of bacteria in the fundus, corpus and cardia. On the basis of our results and the results of other scientists, we can conclude that the fundus is the optimal part of a dog's stomach for the detection of *Helicobacters*.

*Helicobacters* colonised gastric mucus, gastric pits and gastric glands lumina: the highest number was found in the gastric mucus and in gastric pits, but they were not found in the connective tissue of the lamina propria, submucosa, tunica muscularis mucosae and tunica serosa. Our findings are similar to those described in literature, although some authors found *Helicobacters* also in parietal cells (Peyrol *et al*., 1998; Simpson and Burrows, 1999; Vajner *et al*., 2000). With the light microscope, used in our study, it was not possible to establish, if *Helicobacters* were also within the parietal cells.

We found an inflammatory cells infiltration and an increased number of lymph follicles in the lamina propria, altered width and depth of gastric pits, some compressed and degenerated gastric glands, focal fibrosis of propria and hypertrophy of the muscular layer of the mucosa in many dogs. Submucosa, tunica muscularis and tunica serosa were completely normal. Similar changes were also described by other authors (Peyrol *et al*., 1998; Simpson *et al*., 1999; Vajner *et al*., 2000). Some scientists also found atrophy of gastric glands (Scanziani *et al*., 1997; Peyrol *et al*., 1998; Vajner *et al*., 2000). Peyrol *et al*. (1998) also described necrosis of epithelial cells and lymphocytes. After experimental infection of beagles with *H. pylori* Rossi *et al*. (1999) even found erosions in the antrum.

On the basis of Happonen's *et al*. classification (1996) of chronic gastritis, made accordantly to the Updated Sydney System from 1996, we diagnosed only a mild superficial chronic gastritis of gastric fundus in 16 dogs, of gastric corpus in 18 dogs and in the antral region of 18 dogs. Fourteen parts of the stomach were without microscopic changes. Taking in consideration that all 23 dogs from our study were infected (17 of them heavily), we can conclude that chronic gastritis in the studied dogs is not directly linked to helicobacter infection. Happonen *et al*. (1996), who found only a mild chronic gastritis in infected dogs without gastrointestinal disorders and Eaton *et al*. (1996), who diagnosed a mild or a moderate gastritis in many infected dogs, think that even a very strong infection doesn't have any influence on healthy dogs. Scanziani *et al.* (1997) and Yamasaki *et al.* (1998) on the other hand concluded that changes (i.e. infiltration with lymphocytes, erosions and ulcers), found in the stomach of infected dogs could be caused by *Helicobacters*. Peyrol *et al.* (1998) found a mild or a moderate gastritis when the stomach was colonized with *H. bizzozeronii*, necrosis of lamina epithelialis in dogs which harbored *H. felis*, and necrosis of epithelial cells and glands' atrophy when the stomach was infected with both species, i. e. *H. bizzozeronii* and *H. felis*. They concluded that the species of *Helicobacter*, colonizing the stomach, is of key importance for the development of changes in the gastric mucosa (Peyrol *et al*., 1998).

In spite of a 100% helicobacter infection and colonization of all parts of the stomach in all dogs only a mild chronic gastritis was found in some parts of stomachs, therefore we concluded that *Helicobacters*, found in stomachs of our dogs, didn't cause gastritis. Maybe the criteria for the evaluation of histological changes in the gastric mucosa of dogs should be re-evaluated and 10-50 lymphocytes and plasma cells and ≤2 aggregates per field in the lamina propria and an epithelium without alteration are normal findings in a gastric mucosa of dogs. Perhaps the alterations, described in the literature, are caused only by some of *Helicobacter* species and/or their interaction, or they may be only the consequence of individual host response to infection.

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**HISTOLOŠKE PROMENE U ŽELUCU NA IZGLED ZDRAVIH PASA SA *HELICOBACTER* INFEKCIJOM**

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SADRŽAJ

Ciljevi naših istraživanja su bili da odredimo zastupljenost i intenzitet infekcije bakterijama roda *Helicobacter* kod 23 naizgled zdrava psa, da opišemo morfološke karakteristike ovih bakterija, patohistološke promene na sluzokoži želuca i da utvrdimo eventualnu korelaciju između infekcije i oštećenja ovog organa. *Helicobacter* je dokazan u želucima svih ispitivanih pasa i bio je spiralnog oblika sa pet do dvanaest zavoja. Imajući u vidu razlike u širini i dužini zavoja bilo je moguće podeliti bakterije u dve grupe. U prvoj su bili *H. salomonis* i *H. heilmannii* sa brojnim širim zavojima nalik zavrtnju širine 0,55 do 0,72 μm i razmakom između njih od 0,55 do 0,95 μm. U drugoj grupi su bili *H. felis* i *H. bizzozeronii* čiji su zavoji znatno uži, širine 0,29 do 0,50 μm i sa razmakom od 0,40 μm. Bakterije su nađene u svim delovima želuca: fundusu, telu i antrumu i bile su zatupljene u istoj meri. One su naseljavale sluzokožu, želudačne nabore i gastrične žlezde. Slaba do umerena infekcija je dokazana kod 13% pasa a izražena kod 74%. Hronična infekcija slabog intenziteta je dokazana u fundusu kod 16 pasa, a u telu (corpus) i antralnom delu kod 18 pasa. Na osnovu ovih nalaza mogli smo da zaključimo da kod ovih pasa gastritis nije bio povezan sa infekcijom bakterijama roda *Helicobacter*.