



Histopathological lesions in gastric and duodenum organs in dogs (*Canis familiaris*) suspected of *Escherichia coli* infection

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Abstract

It is widely known that *Escherichia coli* (*E. coli*) can infect the gastrointestinal tract of various animals and damage some tissue organs. Through histopathological examination of the gastric and duodenal organs can provide valuable information relating to how the *E. coli* infection impacts the animal. A male dog age $\pm$ 1.5 months, body weight 0.6 kg diagnosed with colibacillosis was euthanized. The dog has never been vaccinated and has not been dewormed. Examination of pathology is carried out macroscopically and microscopically. The standard of microbiological examination for *E. coli* isolation was conducted. Then, histopathological observation of gastric and duodenum organs was performed. The results showed that there were histopathological lesions in gastric and duodenum organs in dogs (*canis familiaris*) suspected of *E. coli* infection.

Keywords: *Escherichia coli*, colibacillosis, dog, duodenum, gastric

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Background

Dogs are animals that live side by side with humans. Animal health needs to be considered because some diseases that infect dogs and cats are zoonotic diseases that can infect humans. Diseases in dogs can be caused by viruses, bacteria, fungi, and parasites. Good maintenance and care will improve dog health. Pet health can be maintained by providing balanced nutritional intake, vaccination, good environmental sanitation, and periodic health checks to the veterinarian (Natasya et al., 2021).

According to studies, dog gastrointestinal infections may be caused by certain *Escherichia coli* strains, such as enterotoxigenic strains of *E. coli*. Due to the toxins that *E. coli* creates, the intestinal lining cannot function normally, which causes an increase in fluid secretion and subsequent diarrhea (Buetin, 1999). Dogs' susceptibility to *E. coli* infections can also be affected by other factors such as

stress, nutritional changes, and weakened immune systems (Mondo et al., 2019).

Case Report

A male dog age $\pm$ 1.5 months, body weight 0.6 kg diagnosed with colibacillosis was euthanized. A dog was suspected with *E. coli* infection. The dog has never been vaccinated and has not been dewormed. Histopathological examination of gastric and duodenum organs was carried out macroscopically and microscopically. *Escherichia coli* isolation was conducted based on the standard of microbiological examination.

The purpose of this examination is to diagnose the dog diseases by carrying out anatomical pathology and microbiology examinations. The results of this examination are expected to be used to determine dog disease and can be used as basic information for disease management and prevention as well as treatment for dogs

with the same disease symptoms for the next maintenance period.

Discussion

Several anatomical changes were observed based on the examination of gastric and duodenum. The gastric was found with diffused hemorrhage and the duodenum had ecchymosis (Figure 1). This finding may direct the diagnoses to gastroenteritis condition. Gastroenteritis is an inflammation of the mucous membrane of the gastric and small intestine characterised by vomiting and diarrhoea which results in fluid electrolyte loss leading to dehydration and electrolyte balance symptoms. Enteritis is a medical condition characterised by inflammation of the intestinal mucosa leading to impaired intestinal function where intestinal peristalsis and secretion are increased, but intestinal function and absorption are reduced resulting in clinical symptoms of diarrhoea. Enteritis can usually occur in conjunction with gastritis and is therefore referred to as gastroenteritis (Schaer, 2008).

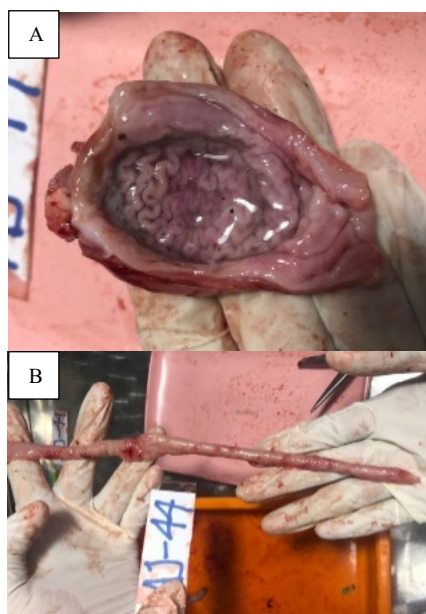


Figure 1. (A) Gastric has diffuse haemorrhage; (B) Duodenum has ecchymosis.

Based on microscopic examination, rupture of the gastric pit epithelium, haemorrhage and infiltration of inflammatory cells in the tunica sub mucosa were found (Figure 2). Haemorrhage can be

recognised by the presence of blood spots with small spots or large spots. According to Smith and Jones (1961), haemorrhage consists of two types, namely small haemorrhage and large haemorrhage. Small haemorrhages can be characterised by bleeding in the form of blood spots and are no larger than the tip of a pin called petechiae, while haemorrhages with rather large spots on the surface of the body or tissue are called ecchymosis (Simarmata et al., 2020).

Further examination continued by observing lesions in the duodenal organs, macroscopic changes in the dog's duodenum were found ecchymosis (Figure 3). Then, it was also observed the presence of *E. coli* infection, followed by the microscopic observation that in the duodenal organ there were ruptures in the cylindrical layer epithelium, rupture villi, hyperemia, haemorrhage and desquamation in the tunica submucosa.

Microscopic observations showed inflammatory cell infiltration and epithelial erosion can occur due to inflammatory processes caused by organism activity in accordance with the dog's clinical symptoms of diarrhoea and lethargy due to lack of nutritional intake. Diarrhoea is caused by organisms invading the intestinal villi so that there will be a vascular response as well as a cellular response to the infectious agent resulting in an inflammatory process. Haemorrhagic conditions can be caused by damage to the vascular endothelium due to the activity of the worms so that blood extravasates into the interstitial villi (Haryo et al., 2021).

Histopathological results showed that there were differences in the severity of infiltration of inflammatory cells. The dominant inflammatory cells in the intestinal tissue are neutrophils. The degree of severity of infiltration of inflammatory cells is also influenced by the length of time inflammation occurs, in addition to the severity of infiltration of inflammatory cells is also influenced by the number of foreign agents, such as bacteria that infect a tissue in an individual. The more foreign agents that enter the body, the more inflammatory cell responses will be seen in the inflammatory

process. *Escherichia coli* that has villi will attach to the small intestine, then will release enterotoxins which cause a decrease in sodium absorption and stretch the intestinal lumen and increase intestinal

peristalsis which causes diarrhoea. These disturbances can cause damage to the small intestine (Rahmawandani, 2014).

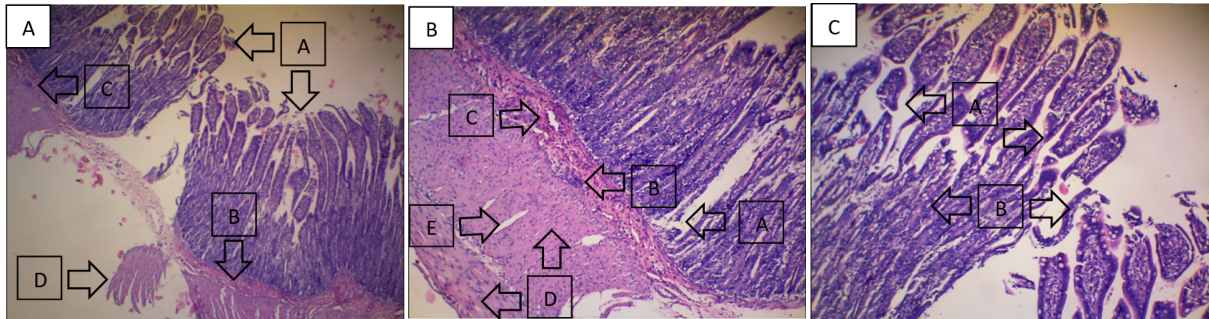


Figure 2. Histological lesions in gastric; Description: I. (A) Rupture of mucosa layer, (B) Inflammatory cell infiltration in submucosa, (C) Edema in muscularis, (D) Rupture of serosa layer (HE, 40x); II. (A) Rupture and inflammatory cell infiltration in the epithelium of the mucosal layer, (B) Inflammatory cell infiltration, (C) Capillary dilation in the mucosal muscularis, (D) Inflammatory cell infiltration of the sub mucosal layer (arrow up) and external muscularis (arrow left), (E) Dilatation of the submucosa (HE, 100x); III. (A) Mucosal layer rupture resulting in *irregular faveola gastrica*, (B) Necrotised cylindrical layer epithelium, (C) Inflammatory cell infiltration, (D) Necrotised zymogenic and prenatal cells, (HE, 400x).

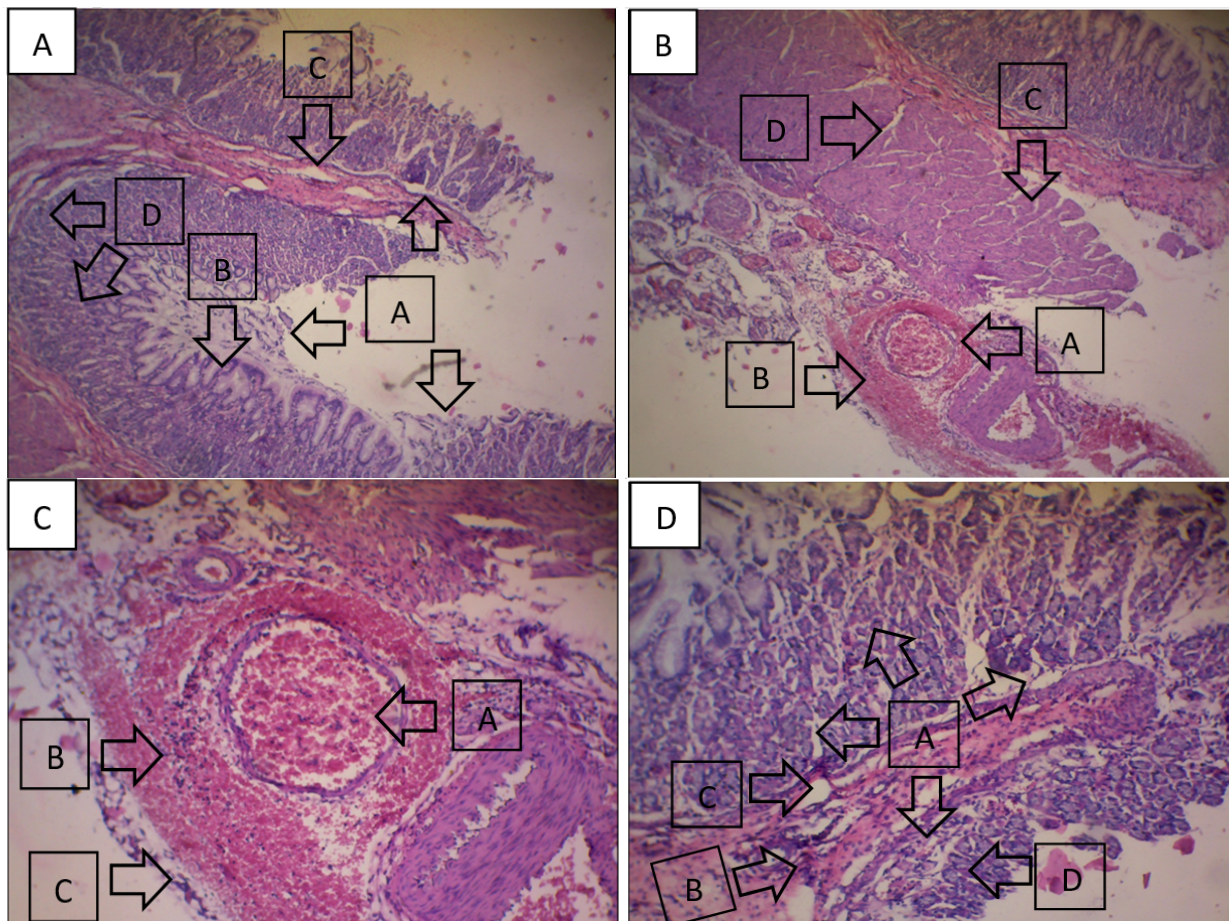


Figure 3. Histopathological lesions of duodenum; Description I. (A) Rupture of villi, (B) Inflammatory cell infiltration, (C) Dilatation of submucosal tunica, (D) Rupture of Brunner and intestinal glands (HE, 40x); II. (A) Hyperemia, (B) Haemorrhage, (C) Inflammatory cell infiltration, (D) Dilatation of sub-mucosa; III. (A) Hyperemia, (B) Haemorrhage, (C) Inflammatory cell infiltration, (D) Dilatation of sub-mucosa.

Inflammatory cell infiltration (HE, 400x); D. (A) Rupture of villi, (B) Inflammatory cell infiltration, (C) Dilatation of sub-mucosal tunica, (D) Rupture of Brunner's gland and intestine (HE, 400x).

The *E. coli* was isolated from the duodenum. According to Kandi (2015), *E. coli* is Gram-negative, naturally occurring microorganisms that have metallic green colonies and size of bacterial colony 2-3 mm. These microorganisms are found in the digestive tracts of both humans and animals.

Figure 4 shows the results of *E. coli* isolation, Gram staining bacteria, and identification using biochemical test.

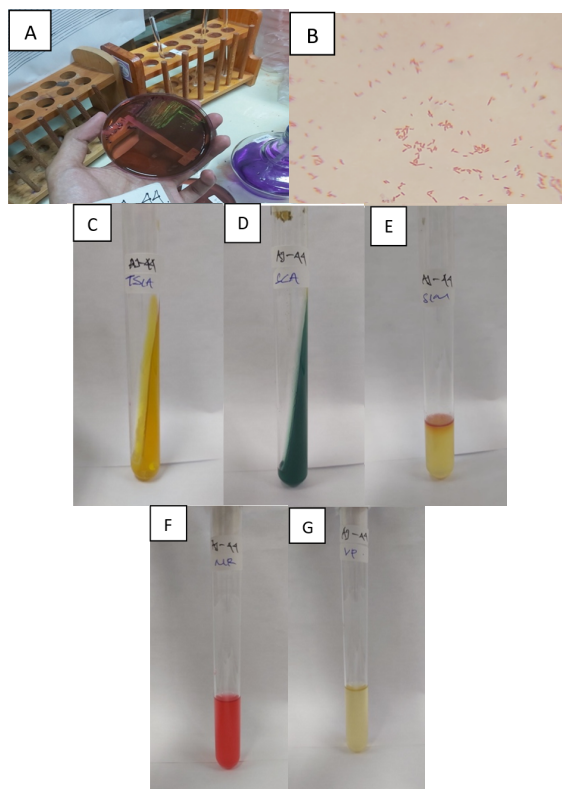


Figure 4. (A) Bacterial culture on metallic green EMBA media; (B) Bacterial staining identified *E. coli*; Biochemical tests include: (C) TSIA (+); (D) SCA (-); (E) SIM (+); (F) MR (+); (G) VP (-).

Bacteria suspected to be *E. coli* on Eosin-methylene blue (EMBA) media were then continued with biochemical tests, namely Triple sugar iron agar (TSIA) (+), Citrate (-), Motility (+), Methyl Red (MR) (+), and Voges Proskauer (VP) (-) tests. TSIA test results: Slant: (+); Butt: (+); H₂S: (+) which means, the slant part shows a yellow colour which means it ferments sucrose and lactose (acid). In the deep butt section, it shows a yellow colour which means fermenting

glucose (acid) and there is a cavity containing gas (H₂S) (Sari et al., 2019). The Simmons citrate agar (SCA) test results are negative, there is no colour change from green to blue, if the microbes use citrate, the acid will be removed from the medium, causing an increase in pH and changing the colour of the medium from green to blue (Mahon et al., 2015). Sulfide indol motility (SIM) test results are positive, there is a red ring on the surface of the media, indole test with peptone medium rich in amino acid tryptophan inoculated and allowed to grow for 24 hours. Therefore, *E. coli* will make the enzyme tryptophanase which will form indole (Sari & Apridamayanti, 2014). Methyl red test results are positive. There is a colour change to red after adding methyl red. This indicates the presence of mixed acid fermentation (methylene glycon) (Radji, 2012). Voges Proskauer test results are negative. After incubating for 24 hours and tested with Alpanaphthol and 10% KOH there is no colour change to pink or red, indicating that *E. coli* is unable to convert glucose into acetyl methyl carbinol (Suardana et al. 2014).

The pathogenicity of *E. coli* begins with the bacteria entering the gastrointestinal tract and attaching to the intestinal epithelium. Bacteria then settle in the intestine which can stimulate the production of toxins or endotoxins in the digestive tract and can lead to inflammation of the intestine resulting in decreased carbohydrate absorption resulting in hypoglycemia. The result of intestinal inflammation can lead to an increase in gastric acid, causing symptoms of nausea, vomiting, resulting in a lack of fluid volume and a high risk of nutrient deficiency, resulting in hypoglycemia and protein energy malnutrition. There is an increase in intestinal mortality resulting in increased fluid and electrolyte secretion which can lead to fluid and electrolyte disturbances such as potassium and sodium resulting in hypokalemia which causes spasms and abdominal cramps resulting in pain. In addition to increased intestinal permeability,

intestinal inflammation can also cause fluid-like faces, excessive frequency of feces, and liquid consistency, as well as acidic excretion, all of which can disrupt the integrity of the epidermis. The intestine requires the ability to absorb these substances, which results in the release of fluid-like faces and excessive frequency of feces. Dehydration and hypovolemic shock can develop from higher levels of fluid and electrolytes because of an increase in intraluminal pressure (Try, 2011). Predisposing factors also determine the pathogenicity of *E. coli*, including animal age, immune status, feed and environment, and intensity of infection with pathogenic strains (Quinn et al., 2016).

Conclusion

Based on the results it was concluded that there were histopathological lesions in gastric and duodenum organs in dogs (*canis familiaris*) suspected of *E. coli* infection. Further research is needed to observe influence of helminth infestation.

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