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RESEARCH ARTICLE

Study on Abomasal Ulcer in Sheep in Iraq

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Abstract

Background: Abomasal ulcers are an erosion of the lining of the abomasum that can result in a variety of clinical signs. The exact underlying cause is still unknown, but abomasal ulceration has been associated with stress, proliferation of bacteria within the gut, abrasions of the abomasum due to roughage, and trichobezoars. This study was conducted at Al-Diwaniyah Abattoir, Al-Diwaniyah city, south of Iraq, with the objectives of to assess the presence of abomasal ulcer in sheep, identifying types of abomasal ulcer.

Material and methods: The investigation was carried out in the abattoir and examination of 56 abomasum of sheep to detect the ulcers. Abomasal fluid pH was measured immediately post cut of abomasal And study the gross lesions with histopathological changes

Results: From total of 56 (46 male and 10 female) sheep were examined, 17.85% were found abomasal ulcer at slaughter. There was no significant difference ($P \geq 0.05$) between sex of sheep in the prevalence of the abomasal ulcers. No-significant differences ($p \geq 0.05$) in the pH values were observed between ulcerated and non-ulcerated abomasal. The abomasal ulcers were showed as a single 7 (70%) and multiple 3(30%) ulcer were found in abomasal. Gross observations of ulcer was differed between the ulcer and other. Histological study of ulcer revealed deposition of granulation tissue with extended through of mucosa, metaplasia of gastric mucosa and atrophy of gastric glands, with infiltration of inflammatory cells.

Conclusion: It is concluded that the detection of this level of prevalence of abomasal ulcer in sheep is the most not only because of its mortality and morbidity but also it contributes a lot for reduced production, productivity and complications associated with it.

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Introduction

Abomasum is the main stomach of ruminants in lower right quadrant of the abdomen that will be affected primarily or secondarily by infections, parasites and foreign bodies somehow prohibit proper nutrition and prehension of essential substances for metabolism, inducing to anorexia, weakness, emaciation and death [1].

Marshall [2]; Tharwat and Ahmed[3] defined abomasal ulcers that a local process of auto digestion of the mucosa. It develops under the influence of acid peptic activity, or it is the result of pathphysiological conditions under which it cannot oppose the destructive action of the gastric juice, as the balance between protective and destructive processes is altered.

Whitlock[4] and Braun *et al.*[5] mentioned that the etiology of abomasal ulcers is still unclear and they an important cause of indigestion in dairy cattle and its occur in several forms and produce different clinical signs.

Abomasal ulcers were divided into four types, depending on the severity of the lesions. Nonperforating abomasal ulcers either or not with severe blood loss are classified as type 1 and 2 ulcers, respectively, while perforating ulcers with local and diffuse peritonitis are classified as type 3 and 4 ulcers[3].

The clinical signs associated with abomasal ulcers depend on the severity of the ulceration. If the ulcers are not very deep and have not eroded into the blood vessels, the animal might show no clinical signs or perhaps only mild signs of discomfort. If the ulceration penetrates the blood vessels in the deeper layers of the abomasum (the submucosa) the animal will bleed into the abomasum. This can cause anemia and thus pale mucous membranes along with digested blood in the feces. With perforating ulcers, all layers of the abomasum are affected and the contents of the abomasum leak into the abdominal cavity. This is very irritating and causes peritonitis and significant pain [4].

The etiology of perforating abomasal ulcers in sheep has not been determined. However, various theories have been proposed. These theories have suggested that main categories includes nutritional deficiencies, especially copper, microbiological agents, stress and abrasive agents, such as, roughages, geosediments, and trichobezoars[6]. It was also hypothesized that high luminal VFA concentrations should cause an overload of cellular pH and volume regulative systems. This might cause cellular swelling and cellular or epithelial damage, which may alter the mucosal barrier so that protection against back diffusion of protons is compromised. The investigators also postulated that high carbohydrate or high concentrate diets may lead to high VFA concentrations in rumen fluid since damage (ulceration) of the abomasal epithelium is found often in animals such as sheep fed concentrate-rich diets.[7].

This study aimed to investigate and presence of abomasal ulcer and study some aspects related to obtaining this effect. Due to lack of researches concerning with causes, macroscopic and histological changes associated with it.

In Iraq, and due to lack of researches concerning whether there causes or macroscopic and histological changes associated with it, so study aimed to investigate and presence of abomasal ulcer and study some aspects related to obtaining this effect.

Materials and methods

1-Sample collection

Fifty six abomasal samples were collected from newly slaughtered sheep in slaughterhouse of Al-Diwaniyah city, in south of Iraq. Sheep were of both sexes with different ages. They included 46 males and 10 females with age range between 3 months and 2.5 years. Immediately after slaughter, after take out the contents of the stomach and intestines of the carcass, by a clean knife, abomasum separated from its anterior part associated with omasum and its posterior part with small intestine, and which linked in both ends, and each sample is placed in nylon bags and transported directly into the laboratory of clinical pathology at the College of Veterinary Medicine, University of Qadisiyah.

2- Measurement of pH:

Abomasal fluid pH was measured immediately post cut of abomasal by used a digital pH meter apparatus, the pH meter was calibrated with buffer of pH 4, 7 and 9 before each sampling period, the probe was cleaned with distilled water between samples, the probe was inserted into the abomasal fluid directed and results were recorded .

3- Examination of samples:

In laboratory, the abomasal sample was put in clean container and examined to show any change in outer surface. By clean scissor the abomasal of the animals was opened along the greater curvature and the ingesta emptied in clean flask, and abomasums washed in water to free ingesta.

4- Physical examination included:

a- Abomasal exam:- Examination of the abomasum before opening to determine which a impaction, dilatation and emptying defect.

b-examination of ingesta:- Color and consistency of ingesta was recorded. The abomasal mucosa was examined, and the type, number, and location of ulcers were recorded [5].

5- Histopathology

Tissue samples were collected from the ulcers for histological examination. The samples were fixed in 10% neutral formalin and sent to the pathology laboratory.

Results and Discussion:

All animals were examined in pre-slaughter were clinically healthy and except two animals (lamb suffered from acute enteritis and ewe suffer from lack of appetite).

Results of physical examination:

The results of physical examination of abomasums were showed impaction in nine samples, and three abomasums were dilated and only four cases were emptied.

The color of ingesta showed yellowish-green, milky, olive and brown color, and consistency is fluidity, semi fluid and dry.

The abomasal impaction, dilation and emptying defect in sheep was recorded by many researchers and the results of current study agreement with [8] whose reported that Suffolk sheep primarily affected with Abomasal emptying defect this syndrome is characterized by distension and impaction of the abomasum. Also [9] were reported some physiological aspects of abomasal contents which included grayish-brown to yellow fluid and which containing sand, dirt and hairballs.

Results of pH:

The results of abomasal pH recorded in animals and were within the normal range, and the mean of all sample is 3.61 while the mean of pH in ulcerated abomasum and non-ulcerated was 3.48 ± 0.245 and 3.639 ± 0.127 respectively. No-significant differences ($p \geq 0.05$) in the pH values were observed between ulcerated and non-ulcerated abomasal (table, 1).

No study confirms the effect of ulcer on the values of gastric pH except [10] pointed out that 5% of the horses with moderate to severe ulceration had a significantly lower pH than horses with mild or no ulceration This agreed with the results of the current study.

Table (1): pH value in ulcerated and non-ulcerated animals

Sheep state	pH value \pm SE
Sheep without abomasal ulcer	3.639 ± 0.127 a
Sheep with abomasal ulcer	3.480 ± 0.245 a

Results of ulcer:

Fifty six sheep (46 male and 10 female) were sampled. All sampled sheep were clinically healthy in preslaughter examination. The results of the prevalence of the abomasal ulcer in both sexes and in different age groups. All ulcer of abomasal were classified as type1, ten(17.85%) sheep had abomasal ulcer which is eight(17.39%) male and two (20%) female. There was no significant difference ($P \geq 0.05$) between sex of sheep in the prevalence of the abomasal ulcers. The abomasal ulcer was less reported in 1-2 years old of sheep comparison in young and old animals, which are 3(10.71%), 5(22.72%) and 2(33.33%) respectively. The significant difference ($p \leq 0.05$) was reported between group-3 of animals at age more than 2 years with group-2 of animals at age between 1-2 years. The abomasal ulcers were showed as a single or multiple ulcer in same sample, which 7 (70%) as single and 3(30%) was multiple. Female animal showed affect only with singles ulcer 2(100%) are shown in Table(2).

Unclear clinical signs in animals that suffer from abomasal ulcer was due to type of ulcer, our results agreed with who mentioned [11] and [12] that type 1 of abomasal ulcer although presence of ulcer and erosion without perforating mucosa the clinical signs are often absent or nonspecific.

Tajik *et al* [13] found that there is no effect of sex and age of the animal on the occurrence of abomasal ulcer in their study in prevalence of abomasal ulcers in water buffalo, and our study differed only effect of age where we found that age a significant effect on the occurrence of abomasal ulcer.

Table(2): show prevalence of abomasal ulcer in both sexes, different ages, percentage infected of animals.

		Male	female	age			
				*G1 < 1 year	*G2 >1year- <2years	*G3 >2 years	
Number of Sheep		56	46	10	22	28	6
Number of infected Sheep (100%)		10(17.85%)	8 (17.39%) A	2(20%) a	5(22.72) ab	3(10.71%) a	2(33.33%) b
Type of ulcer	I	10(100%)a	8(100%)A	2(100%)a	5(100%)a	3(100%)a	2(100%)a
	II		-(0%)B	-(0%)b	-(0%)b	-(0%)b	-(0%)b
	III		-(0%)B	-(0%)b	-(0%)b	-(0%)b	-(0%)b
	IV	-(0%)b	-(0%)B	-(0%)b	-(0%)b	-(0%)b	-(0%)b
Number of ulcer in same sample	Single	7(70%)a	5(62.5%)a	2(100%) a	2(40%)a	3(100%)a	2(100%) a
	Multiple	3(30%)b	3(37.5%)b	-(0%)b	3(60%)b	-(0%)b	-(0%)b

*G1 : Group 1, G2: Group2,G3:Group3

Similar letters refers to the non-significant differences between groups while different letters refers to significant differences at ($p < 0.05$)

Gross examination of the structure of the ulcerated abomasum showed that The abomasal ulcers were single and multiple and which localized in the corpus and pylorus, and mainly round, oval or irregular in shape. The margins were mainly smooth, and an abomasal hemorrhagic ulcer was seen in the abomasal body. The mucosal surface of the abomasum is pale, but has irregularly roughened appearance indicating a case of hyperplasia (Figure, 1 and 2). Same results of gross lesion of abomasal ulcer were showed by [14].

It can be supposed that the transport of heifers was the cause of multiple ulcerations, as it is a fact that long transport can be considered as a stress factor. Besides, heifers were diagnosed with bronchopneumonia, which could have in turn, contributed to the development of ulcers as hypoxia decreases the resistance of the gastric mucosa and hypercapnia increases the secretion of hydrochloric acid [15].

Histological examination showed that abomasal ulceration was characterized by focal to multifocal, sharply demarcated areas of coagulation necrosis with deposition of granulation tissue that extending through the mucosa, tortuous gastric glands with metaplasia of gastric mucosa into intestinal shape were also recognized in many cases, (figure:1). Figure (2,3): also showed deposition of granulation tissue tortuous and atrophy of gastric glands with infiltration of inflammatory cells. Edges of ulcer are obliterated by deposition of collagen tissue with atrophy of gastric glands and infiltration of inflammatory cells. In the other figure, showed abomasal ulcer characterized by thickening in the edges, infiltration of inflammatory cells and complete atrophy of gastric glands (figure, 4 and 5).

The result of current study for histopathological examination were compatible with mentioned [13] and [14] that area of ulceration is revealed a moderate inflammatory infiltrate in the lamina propria with coagulate necrosis and showed infiltration of inflammatory cells in the mucosa, both acute and chronic inflammatory reactions, a reduced number of tubular glands.

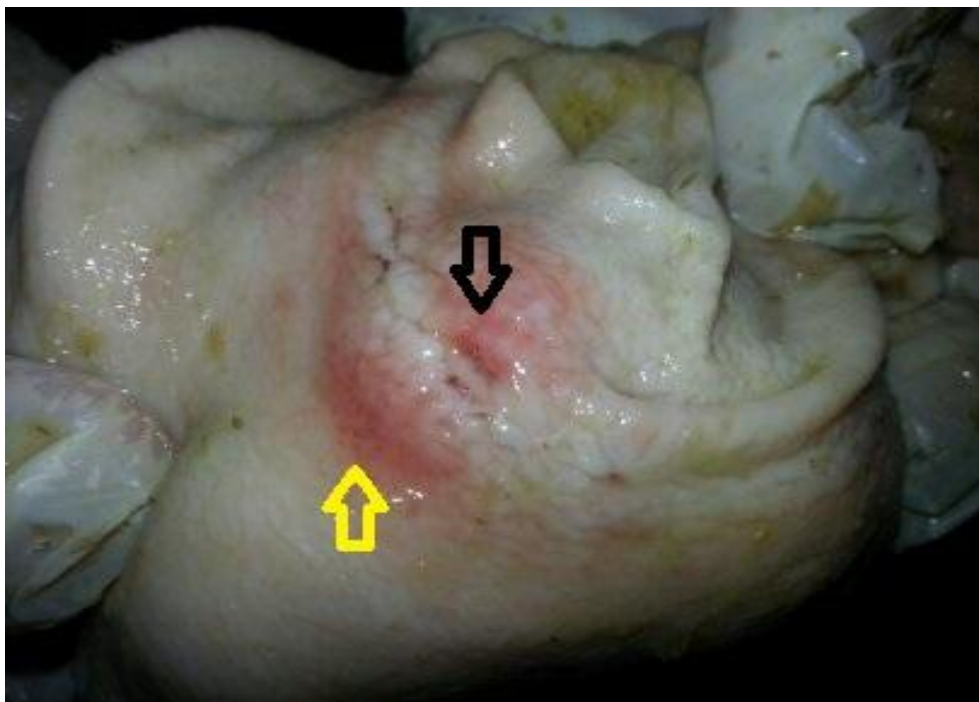


Figure (1): Description of the lesions: an abomasal ulcer was seen in the greater curvature of the abomasum, in the pyloric region near the pyloric orifice (black arrow) surrounding by an area of petechial hemorrhage (yellow arrow). The ulcer was shallow and characterized by elongated hemorrhagic center.

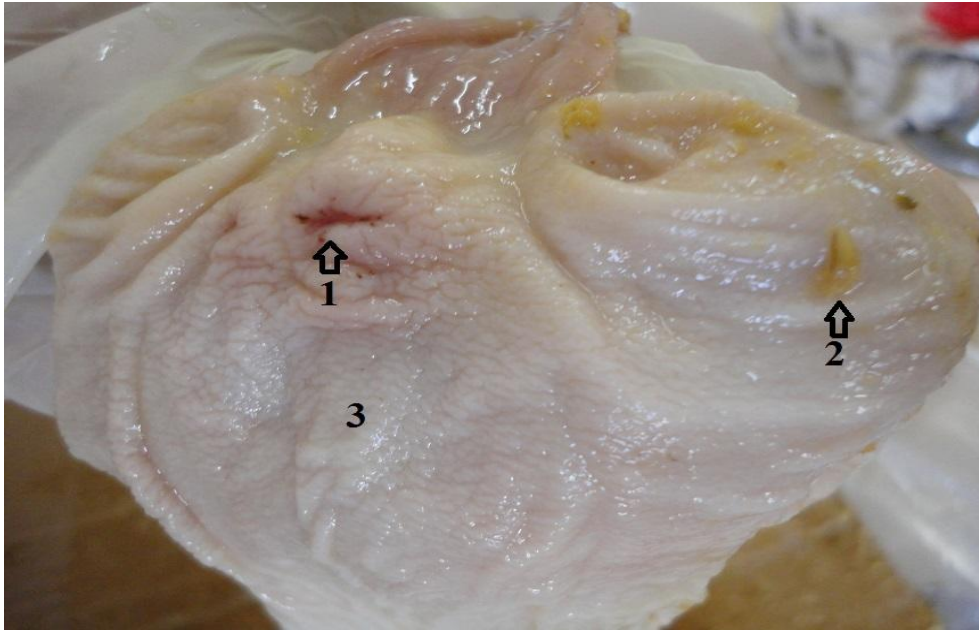


Figure (2): Description of the lesions: an abomasal hemorrhagic ulcer was seen in the abomasal body (1), a healed ulcer also occur surrounding by yellow color and its orifice is shallow or obliterated (2). The mucosal surface of the abomasum is pale, but has irregularly roughened appearance indicating a case of hyperplasia (3).



Figure (1): section of abomasum showed gastric ulcer (1), with deposition of granulation tissue (2), tortuous gastric glands (3) with metaplasia of gastric mucosa into intestinal shape (4). H&E, 100X.

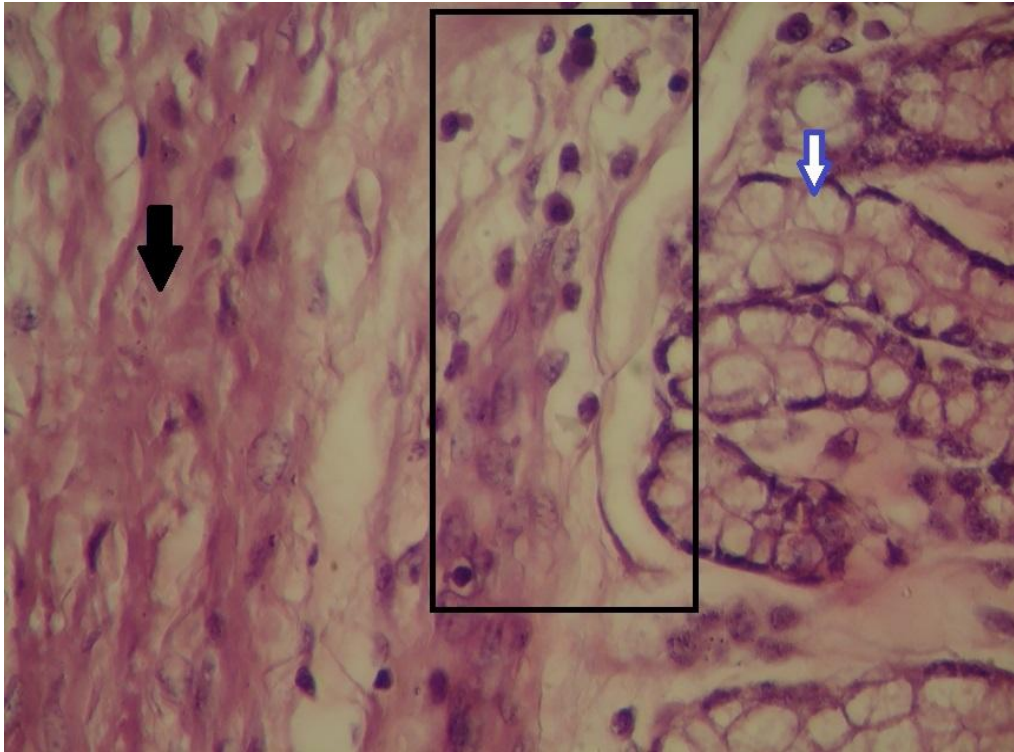


Figure (2): section of abomasum showed deposition of granulation tissue (black arrow), tortuous and atrophy of gastric glands (Blue arrow) with infiltration of inflammatory cells (Box). H&E, 100X.

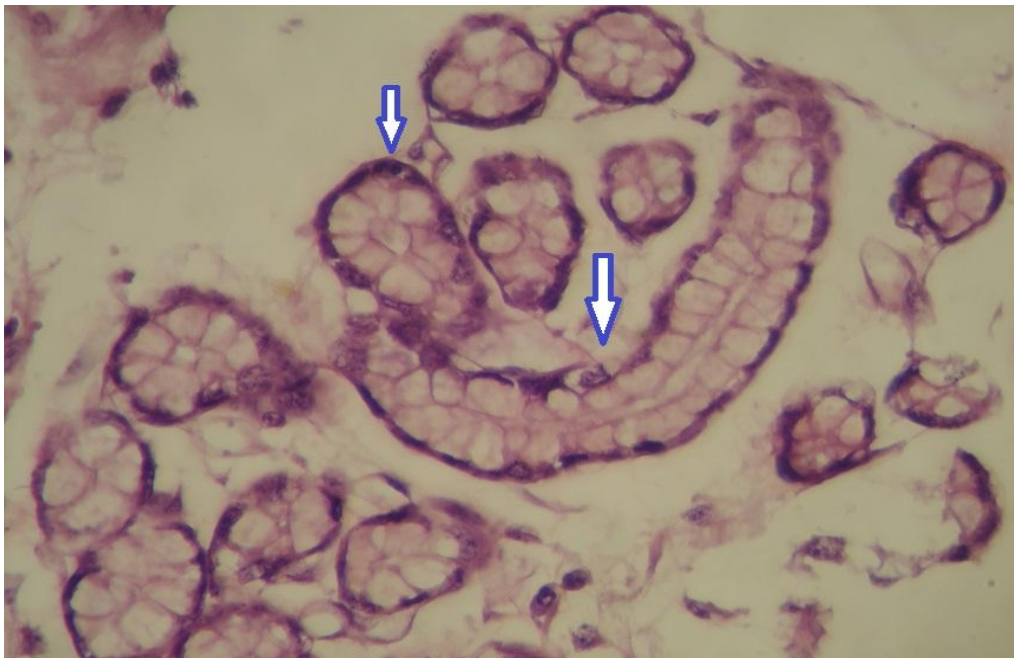


Figure (3): section of abomasum showed tortuous and atrophy of gastric glands (Blue arrow). H&E, 400X.

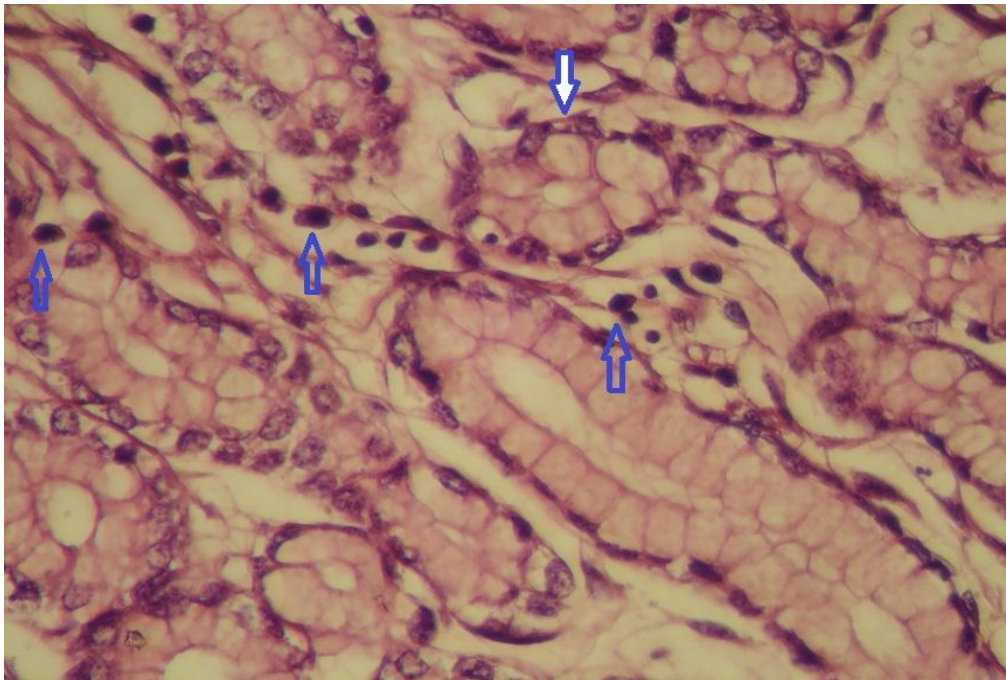


Figure (4): section of abomasum showed infiltration of inflammatory cells between abomasal glands (Blue arrow). H&E, 100X

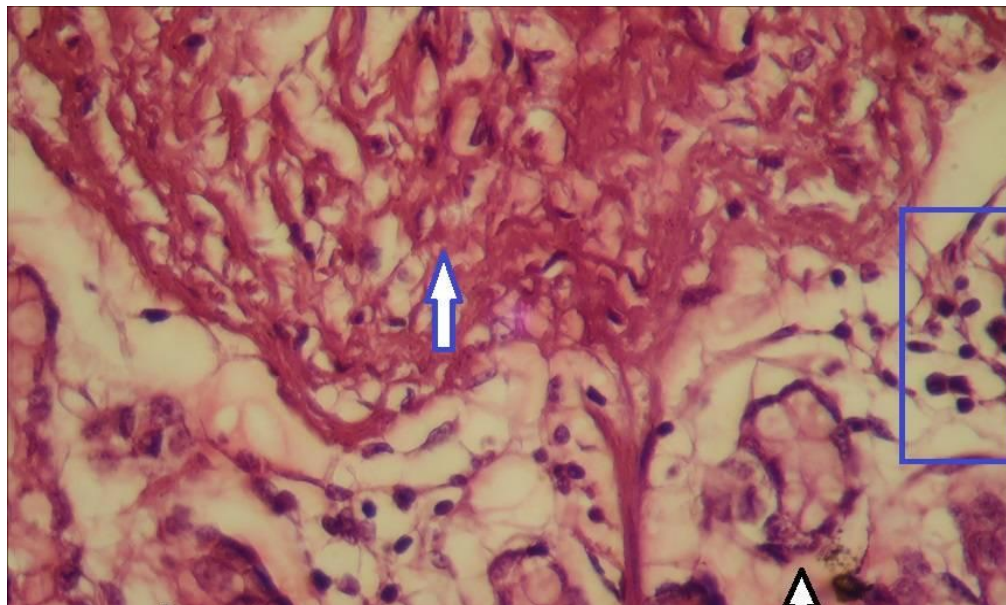


Figure (5): section of abomasal mucosa showed obliteration edges of gastric ulcer by deposition of collagen tissue (Blue arrow), atrophic gastric glands (black arrow) and infiltration of inflammatory cells (Box). H&E, 100X.

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