Diagnosis and Management of Abomasal Disorders in Cattle

Thesis presented
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Submitted to Faculty of Veterinary Medicine
Mansoura University
For the Degree of Master of Vet. Med. Sciences (Veterinary Surgery)

(2007)
Acknowledgment

First of all I am greatly indebted in my work and success to our merciful "Allah" who gave me the ability to terminate this work.

Great appreciation profound gratitude and deepest thanks are offered to Prof. Dr. Adel E.I. Zaghloul, Prof. and head of Surgery Dept. Fac. Vet. Med., Mansoura University for his kind supervision, valuable advice, encouragement, keen suggestion during the course of this study and revision all the details that enabled me to finish this work.

Grateful thanks, deep-seated sincere appreciation are also extended to Dr. Gamal I. A. Karrouf, Associate. Prof. of surgery. Fac. Vet. Med., Mansoura University for his help, co-operation and supervision, continuous guidance, unlimited help, valuable advice, encouragement, keen suggestion during the course of this study and revision all the details that enabled me to finish this work.

Great appreciation profound gratitude and thanks offered to Prof. Dr. Taha A. Fouda, Prof. of internal medicine, Fac. Vet. Med., Mansoura University for his kind supervision, valuable advice, encouragement, keen suggestion during the course of this study.
I would like to express my grateful thanks, very great indebtedness to **Prof. Dr. Hussein M. EL-Maghraby**, Prof. of surgery and head of Surgery Dept. Fac. Vet. Med., Benha University for his help and co-operation in performance of the ultrasonographic part of this study.

I would like to express my grateful thanks, very great indebtedness, to **Prof. Dr. Ahmed F. El-Shaieb**, Prof. of pathology and head of Pathology Dept. Fac. Vet. Med., Mansoura University for his help and co-operation for preparation and reading of the histopathological specimens during the course of this study.

I wish also to extend my thanks to **Dr. Esam M. M. Mahmoud**, lecturer of surgery. Fac. Vet. Med., Mansoura University for his co-operation, valuable advice and encouragement.

Finally my thanks to every hand help me to complete this work and to all members of Surgery Dept. Fac. Vet. Med., Mansoura University for their help and encouragement.
Dedication

This work is dedicated to the world Prophet "Mohamed" (P.B.U.H.), to my parents, brothers, sisters and my lovely wife (El-Shiemaa).
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Introduction

Surgery of the bovine digestive tract has been the subject of renewed research interest as veterinarians continue to improve techniques to treat gastrointestinal problems that having great effect on milk production and body weight which necessitate culling the animal from the herd (Fox, 1980; Milian-Suazo et al., 1988; and Turner & Mcllwraith, 1989).

Abomasal disorders of dairy cattle are mostly predisposed by metabolic disturbance, lactation stress and nutritional inadequacies. They include left and right abomasal displacement (LDA&RDA), volvulus (AV), ulceration, impaction and abomasitis (Trent, 1990; Geishauser, 1995; and Simkins & Nagele, 1997).

Abomasal displacement is a multifactorial disease commonly occur in dairy cows aged from 3-10 years in the first 2-4 weeks postpartum and at other times in the lactation period (Coppock, 1974). It has been reported sporadically in beef breeds (Martin et al., 1978), bulls (Fennelly, 1984) and calves (Hawkins et al., 1986).

Abomasal volvulus usually associated with abomasal dilatation especially RDA. It commonly involves the omasum with occlusion of systemic circulation, necrosis, and luminal out flow obstruction which associate with a poor prognosis (Smith, 1978; Garry et al., 1988; and Trent, 1990).
Introduction

Abomasal impaction was frequently seen in dairy cattle, and also in calves, sheep, goats and buffaloes. It usually of non-specific cause and difficult to be established clinically; however, the ultimate diagnosis was made during the exploratory laparotomy (Blikslager et al., 1993; and Rebhun, 1995).

Abomasal ulcers are focal or multi-focal defects in the mucosa and muscularis of the abomasum of dairy cattle (Rebhun, 1982), calves (Wensing et al., 1986) and feedlot cattle (Jensen et al., 1992). The disease is associated with stress, concentrated rations and concurrent diseases (Rebhun, 1995).

Abomasitis is a sporadic disorder among adult cattle, calves, sheep and goat caused by many adverse effects of either dietary, environmental, infectious or parasitic stressors. It is characterized by diffuse, hemorrhagic to necrotizing inflammation of the abomasal mucosa (Dahlgren et al., 1984; and Roeder et al., 1988).

Diagnosis of the fore-mentioned disorders based on invasive and non-invasive techniques. The invasive ones are exploratory laparo-rumenotomy and necropsy findings after slaughtering or euthanasia (Mesaric et al., 1997), while non-invasive ones are case history, general appearance, simultaneous auscultation and percussion, ultrasonography, rectal palpation and laboratory examination (Braun et al., 1997; and Radostitis et al., 2000).

Medical and/or surgical treatment of the abomasal disorders depend on promotion of normal abomasal motility, correction of systemic electrolytes and
metabolic alteration, management of concurrent diseases and the liability of the disorders to be corrected surgically (Trent 1990; and Velden, 1991).

The aim of this study was to throw light on:

1. Ultrasonography of the normal abomasum in cattle.
2. Most common abomasal disorders occurring in cattle.
3. The role of clinical signs, physical examinations and exploratory laparorumenotomy in diagnosis of such disorders.
4. The role of laboratory investigation in making the diagnosis and predicting the prognosis of such cases.
5. The necropsy findings accompanying some of abomasal disorders.
6. The possibilities of medical and/or surgical treatment of such disorders.
Review of literatures

Anatomy of abomasum:

The ruminant stomach is composed of four structurally distinct parts. The first three parts (rumen, reticulum and omasum) are collectively called the forestomach which lined entirely by a nonglandular mucosa having a keratinized stratified squamous epithelium. The fourth part is the abomasum, lined by a glandular mucosa change abruptly from keratinized stratified squamous to simple columnar epithelium that similar to the simple stomach of other species (Habel, 1975; and Dellman & Eurell, 1998).

Abomasum is an elongated tubular sac located chiefly on the abdominal floor in the epigastric region, half way between the xiphoid process of the sternum and the umbilicus. It extend from the omaso-abomasal orifice through the fundus and the body to the pyloric antrum and pylorus (Habel, 1975; and Pavaux, 1983).

Dyce et al. (1996) found a difficulty to specify abomasal position and relations exactly. They are dependent on the fullness of the different parts of the stomach, intrinsic abomasal activity and contraction of the rumen and reticulum to which the abomasum is attached.
In healthy non-gravid cow, the fundus actually curves medially from the omasoabomasal orifice and rests on/or slightly to the left of midline under the ruminal recess. The body then crosses obliquely along the abdominal floor caudally and to the right to bend dorsocaudally to the omasum as the pyloric antrum (Church, 1979).

Pavaux (1983) and Dyce et al. (1996) added that, the pylorus lies at the ventral aspect of the 9th or 10th intercostal spaces and points dorsally or dorsocaudally to continue as the cranial duodenum. The pylorus of the cow has not a distinct sphincter but have a muscular projection into the lumen on the lesser curvature of the pylorus called the torus pyloricus.

The abomasum has lesser and greater curvatures which serve as attachment sites for the lesser and greater omentum. The parietal surface of the abomasum is not covered by omentum and lies between the lesser and greater omentum, while the visceral surface is covered by the deep wall of the greater omentum (Habel, 1975).

A number of neuro-humoral factors interact to regulate the volume and characters of the abomasal secretions, particularly of hydrochloric acid. The volume and the acidity of gastric secretions decreased when flow of ingesta into the abomasum is prevented, also when the abomasal and duodenal pH are reduced (Valminck et al., 1984).
Movement of ingesta through the abomasum was regulated by both local and systemic mechanisms to maintain a constant luminal pH of approximately 3.0. In addition to, abomasal activity is greatest at the pyloric antrum, which demonstrates strong peristaltic movement, whereas contraction of the body are variable and the fundus exhibits minimal activity (Church, 1979).

Abomasal disorders:

I-Left abomasal displacement:

The left displacement of the abomasum is a condition in which the abomasum was dilated with fluids, gases, or both and it was mechanically trapped in the left side of the abdominal cavity. Its displacement causes little to no outflow obstruction, decreased appetite and milk production (Coppock, 1974).

Abomasal displacement differs according to heritability, sex, age, season, husbandry, milk yield and concurrent diseases (Geishauser, 1995). In addition to feeding, mechanical influences, disturbances of the abomasal motor activity, collection of gases and dilatation of the abomasum are effective (Furll et al., 1999).

A number of etiological factors have been implicated for LDA. These may be grouped as factors that impair abomasal motility or inhanse gas production (Goff and Horst, 1997), factors that alter normal nutrient digestion
or utilization (Geishauser et al., 2000), and factors that result in anatomical arrangement of abdominal structures to enhance displacement (Radostitis et al., 2000).

LDA in cattle has found throughout the world with annual incidence ranged from 0.05-4.4 %. It was seen most commonly and with increasing tendency in regions with intensive cattle breeding and husbandry (Geishauser, 1995). However, the incidence of the disease in Egypt and probably in most other tropical countries is very low (5.7%) (Sigh et al., 1996; and Misk et al., 2003).

Abomasal displacement is a common disorder of Holstein-Friesian, Jersey and Guernsey cows (Jubb et al., 1991). Dairy cows were at higher risk of developing LDA than were beef cattle (Constable et al., 1992; and Roussel et al., 2000).

Misk et al. (2003) reported that, abomasal displacement in Egypt occurs most commonly in large sized, high producing dairy cows in the age group of 3-7 years. Displacement of the abomasum is very rare in buffaloes and native breed cattle and recorded in 5.7% of the studied cases.

Jubb et al. (1991) and Geishauser (1995) mentioned that, displacement of the abomasum can occur in cattle of all ages, the highest risk was found between the ages 4 and 7 years. Approximately 80% of the LDA cases were seen predominantly in the first month after calving.
Radostitis et al., (2000) and Vanwinden & Kuiper (2004) reported that, the occurrence of LDA in different seasons are not concise; in general most cases occur in winter. Besides season, there are housing system and quality, weather conditions (rainfall, low temperature and strong wind), altered exercise and feeding patterns caused by management practices may also contribute to development of LDA.

Displacement of the abomasum is mainly an illness of non pregnant cattle approximately 90% of all cases, and can occur during pregnancy but with low frequency (10%) in the 2nd and 3rd pregnancy (Godger and Ruppaner, 1984).

There is an association between the amount as well as the quality of the roughage feed and LDA. Roughage of poor quality and bad tastiness leads to a lowered feed intake with decreased LDA occurrence. However, total mixed ration, finely chopped base feed and pelletized or ground feed all promote displacement of the abomasum (Geishauser, 1995; and Shaver, 1997).

Massey et al. (1993) Geishauser et al. (2000) and Fouda etal. (2004) said that, Holstein cows that were hypocalcaemic (less than 4 mg/dl) at the time of parturition or 2weeks post partum had a relative risk of developing LDA 3.8 times than normocalcemic cows. Oral calcium administration with correction of an excess vitamin A and D after calving prevents both parturient paresis and DA.
Concurrent diseases are commonly related to LDA (54%) than RDA (30%). Diseases of the abomasal wall (mucosal erosions, ulcerations and wall adhesions) and liver are often commonly found (Trent, 1990). Also, diseases of reproductive organs, milk fever, diarrhea, aciduria, ketonuria, and other illness (impaction of the abomasum with sand, dilatation and displacement of the cecum, claw problems, foreign bodies and peritonitis) are common (Geishauser, 1995).

Geishauser et al., (1998) suggested that, endotoxins and mediators of inflammation could be a direct cause of LDA via motility disorders. In addition, DA was seen more often in herds with lameness problems.

Rohrbach et al. (1999) mentioned that, ketosis is a risk factor for LDA. It occurs as primary ketosis before the displacement and as secondary ketosis following the displacement of the abomasum. It was seen more often together with LDA than with RDA.

Disorders of abomasal motility and gas accumulation were widely accepted as being prerequisites of left abomasal displacement. It include; electrolytes disturbance, high concentration of volatile fatty acids (VFA) or increased amounts of histamine associated with tissue damage caused by concurrent diseases (Coppock et al., 1974). Also, a high osmotic pressure of the abomasal fluid, which leaded to distension of the abomasal wall (Breukink and Kuiper 1980).
Review of Literatures

Cows with LDA usually had a sudden onset of anorexia, decreased milk production, rise of temperature (39.5°C) and heart rate (100 beat/minute) accompanied by signs of moderate abdominal pain (Wallace, 1976). In addition to, an obvious bulge (slab-sided abdomen) developed in the anterior part of the upper left para lumber fossa and may extend up behind the costal arch (Dirksen, 1983). Simultaneous auscultation and percussion of the left abdomen revealed tympanic resonance (ping sound) over the last three ribs (10th to 13th) along or above a line drawn from the left tuber coxae to the elbow and from the elbow to the stifle (Radostitis et al., 2000).

Ultrasonography is a valuable technique for assessment of the size, dimensions, position, appearance and contents of the abomasum. It can be visualized approximately 10 cm caudal to the xiphoid process from the left and right Para median regions and from the ventral midline (Braun et al., 1997). It located immediately caudal to the reticulum between the craniodorsal blind sac of the rumen or the rumen and the ventral abdominal wall, while the bulk of the abomasum is situated to the right of the ventral midline, which are seen ultrasonographically as a moderate heterogeneous echogenic mass with echogenic stippling. Passive and slow movement of abomasal contents was frequently seen (Braun, 2003).

Ultrasonographic examinations of cows with LDA were performed from the 11th and 12th intercostal spaces on left side, whereas the rumen was displaced by the abomasum more dorsally. The ingesta that were visualized ventrally in
the abomasum appeared echogenic to hypoechogenic and in a few cases the abomasal folds were visible as elongated echogenic sickle shaped structure. Reverberation artifacts from the abomasal surface characterized the dorsal abomasal gas cap (El-Ghoul, 2001; and Braun, 2003).

Cows with LDA frequently show significant increase in total bilirubin, total proteins, urea, creatinine, alkaline phosphatase, glutamic pyrovate transaminase (GPT), glutamic oxaloacetic transaminase (GOT) activities, aspartate transaminase (AST), and packed cell volume (PCV) (El-Gharieb et al., 1996; and Rohn et al., 2004).

Ithon et al. (1998) and Fouda et al. (2004) mentioned that, the most prominent laboratory findings associated with LDA, include hypoglycemia, hypocalcaemia, hypokalemic metabolic alkalosis and elevated values of urea nitrogen and PCV.

The displaced abomasum at necropsy was trapped between the rumen and the left abdominal wall and contain variable amount of fluid and gas. In occasional cases, it is fixed in position by adhesions that usually arise from abomasal ulcers. Fatty liver is common in cows that died from LDA complications within a few days of parturition or following surgery (Radostitis et al., 2000).

LDA must be differentiated from those diseases causing anorexia, ketosis, reduced reticulorumenal motility and abnormal sounds on ausculto-percussion
of the left abdomen. They include; simple indigestion, ketosis, acute form of traumatic reticuloperitonitis, vagus indigestion, ruminal tympany, pneumoperitonium, fatty cow syndrome and posterior functional stenosis of the rumen (Baker, 1979; Rebhun, 1995; Misk et al., 2003; and Fouda et al., 2004).

The course of the disease in both LDA and RDA is highly variable and the animal may survive several weeks or even few months. Animal with RDA with AV appears more depressed and the signs are more pronounced in comparison to those observed with LDA. Also, animal having serum chloride levels equal to or below 79 µeq/L and heart rate equal to or greater than 100/minute have a poor prognosis (Singh et al., 1996; Radostitis et al., 2000; and Roussel et al., 2000)

Numerous medical (conservative) treatments have been used with variable success as calcium borogluconate 25%, neostigmine, saline and cathartics. Most of these drugs do not have direct effect on the abomasum but stimulate the gastrointestinal motility in general (Velden, 1991; and Rebhun, 1995).

Surgical Treatment:

Correction of LDA may be done non surgically by rolling or surgically by right flank omentopexy or pyloro-omentopexy, right paramedian abomasopexy, left flank omentopexy or abomasopexy and laparoscopy. All of these methods
involved decompression (deflation), replacement to normal position and fixation of the omentum or the abomasum to the body wall (Patty, 1981; Smith, 1981; Jean et al., 1987; and Singh et al., 1996).

The common surgical techniques for treating LDA were classified according to the restraint used. **Standing restraint including**: left paralumbar approach either abomasopexy or omentopexy (Utrecht method) (Smith, 1981), and right paralumbar approach including omentoabomasopexy or omentopexy (Dirksen method) (Dirksen, 1983). **Dorsal recumbency restraint**: via paramedian abomasopexy (Noordsy, 1989).

Left flank omentopexy is not appropriate for correction of RDA or AV, but when used in the presence of LDA, gas is decompressed and the abomasum is pushed ventrally to its normal position. Reported success rates for this technique ranged from 86-93 % (Steanhaut et al., 1974).

Left flank abomasopexy is indicated for the treatment of LDA. This technique offers direct fixation of abomasum to the ventral body wall and the surgery is preformed with the animal in the standing position. Also, adhesions or ulceration of the LDA can be visualized and dealt with, and an exploratory rumenotomy can be performed if indicated (Jean et al., 1987).

Intensive fluid therapy is usually necessary preoperatively and for several days postoperatively to correct dehydration and metabolic alkalosis and to restore normal abomasal motility. A mixture of sodium chloride 50-100 gm,
potassium chloride 50 gm and ammonium chloride 50-100 gm is given orally/daily along with the parenteral fluids necessary until the cow regains its normal appetite (Rehage et al., 1996; and Radostitis et al., 2000).

II-Abomasal volvulus:

Volvulus (torsion) of the abomasum can occur at different positions (at the omaso-abomasal opening or the reticulo-omasal opening), a long different axis (horizontual or vertical) and in different directions (left or right) resulting in ischemic necrosis, peritonitis, dehydration, vascular compromise and luminal out flow obstruction (Habel and Smith, 1981).

Smith (1984) and Baker (1986) mentioned that, abomasal dilatation is generally considered to be a potential cause of AV, where half of all RDA are seen with torsion although some cases of AV may occur with or without evidence of previous dilatation.

Cattle with AV had a visibly distended abdomen, depression and weakness, complete anorexia with progressive drop in milk production and dehydration (Habel and Smith, 1981). The heart rate and respiration are increased. The feces are usually scanty, soft and dark in color (Constable et al., 1991). Death usually occur in 48-96 hours from shock and dehydration or abomasal rupture (Radostitis et al., 2000).
Volvulus has a variable and less favourable prognosis. The prognostic values for non survival include; large quantity of fluid accumulation in the abomasum (> 30L) *(Smith, 1978)*, tachycardia (> 100/min) *(Smith, 1984)*, high anion gape (> 32.5) hypochloraemic, hypokalaemic metabolic alkalosis associated with hypocalcaemia *(Garry et al., 1988)*, and metabolic acidosis *(Trent, 1990)*.

*Trent (1990) and Constable et al. (1991)* reported that, cattle with omaso-abomasal volvulus are thought to have a lower survival rate than cattle with AV. Slaughter or euthanasia are options that should be considered in such cases with limited economic value or poor prognostic indicators.

The post-mortem findings in cattle with AV include; abomasal distension with brownish, sanguanous fluid, twisting of the abomasum with displacement of the omasum and reticulum *(Habel and Smith, 1981)*. Also, the abomasal wall is grossly hemorrhagic, edematous, necrotic and gangeranous indicated by its bluish or blackish coloration and may be ruptured *(Trent, 1990)*.
III- Abomasal impaction:

Abomasal impaction describes a set of condition in which abomasal contents are partially or completely prevented from exiting the abomasum while the abomasum retains a normal anatomical orientation. The outflow disturbance results in distention of the abomasum, alteration in digestion and in circulating electrolytes balance (Baker, 1979; and Blikslager et al., 1993).

No specific factor has been found to precede all cases of abomasal impaction in cattle. Primary abomasal impaction may be caused by extremely fibrous feed, lack of water, pica with subsequent heavy ingestion of sand or rocks or be idiopathic (Purohit et al., 1987). However, secondary abomasal impaction, which is more common include vascular or neurogenic damage secondary to AV, abdominal adhesion, lymphosarcoma, traumatic reticuloperitonitis and peritonitis associated with perforating abomasal ulcers (Rebhun, 1995).

Mitchell (1991) and Blikslager et al. (1993) mentioned that, the combination of low digestibility and excessive intake leads to excessive accumulation in the fore stomach and abomasum. Once impaction occurs, a state of subacute obstruction of the upper alimentary tract develops. Almost no ingesta or fluid move beyond the pylorus, dehydration, alkalosis, electrolytes imbalance and progressive starvation occur. The abomasal impaction is usually severe enough to cause permanent abomasal atony.
The clinical signs of abomasal impaction include complete anorexia, cessation of defecation and moderate to severe distension of the abdomen on the lower right and left quadrants with poor body condition. The abdomen appeared papple shaped (pear like on right side and apple like on left) when viewed from the rear. Rectal examination of adult dairy cattle usually finds enlargement of the rumen dorsal and ventral sacs. *(Baker, 1979; Cebra et al., 1996; and Misk et al., 2003).*

The course of the disease depends on the extent of the impaction and the severity of the acid-base and electrolytes imbalance. Severely affected cattle will die in 3-6 days after the onset of signs. Rupture of the abomasum has occurred in some cases and death from acute diffuse peritonitis and shock occurs within few hours *(Purohit et al., 1987; and Rebhun, 1995).*

*Smikins and Nagele (1997) and Misk et al. (2003)* found that, at necropsy of abomasal impaction, the abomasum was enlarged and dilated with thin wall and congested serosal membrane. The length reached up to 100 cm and the circumference was more than 80 cm. The contents appeared dry and molded inside the abomasum taking its shape but mostly homogenous in consistency. In several cases, the pylorus of the abomasum was found empty except of some abomasal fluids. Abomasal tears, ulcers and necrosis of the wall of the rumen, omasum or abomasum may occur.
Review of Literatures

The prognosis for abomasal impaction depends on the animal age, the original cause of impaction, the chronicity of impaction before treatment and the method of treatment. The prognosis is generally considered to be poor in adult cattle and in chronic cases (Baker, 1979; and Trent, 1990).

Rational treatment of abomasal impaction consist of correcting metabolic alkalosis, hypochloremia, hypokalemia and dehydration. Also, attempting to move the impacted material with lubricants and cathartics or surgically emptying the abomasum (Trent, 1990; and Simkins and Nagele, 1997).

Surgical correction of abomasal impaction consists of an abomasotomy through a right para median or a low right Para costal approach (Merritt and Boucher, 1967). Rumenotomy may be advised for emptying the rumen and infuse dioctyl sodium sulfosuccinate 25% (120-180ml/450kg B.w.) mixed with 10 L of warm water and 10 L of mineral oil directly into the abomasum through the reticulo-omasal orifice in an attempt to soften and promote the evacuation of the abomasal contents (Trent, 1990; Blikslager et al., 1993; and Rebhun, 1995).
IV-Abomasal ulcers:

Abomasal ulcers are focal or multi focal defects that penetrate the mucosa and muscularis of the abomasum. This distinguishes them from superficial erosions, which do not extend through the muscularis layer (Pearson et al., 1987; and Jensen et al., 1992).

Abomasal ulceration has been recognized in all groups of cattle particularly those under production or management stress (Rebhun, 1982; and Henninger, 1984), specifically veal calves, yearling feedlot cattle and high-production dairy cows (Smith et al., 1986; and Pearson et al., 1987).

Abomasal ulceration may occur secondary to other diseases such as lymphoma, left and right abomasal displacement, erosions of abomasal mucosa in viral diseases (Bovine viral diarrhea, Rinder pest, Bovine malignant catarrhal) and enteric form of enzootic bovine leucosis (Wass et al., 1981; and Murray et al., 1995).

Abomasal ulceration in suckling calves can be the cause of fatalities. Its main causes include, mechanical abrasions of the pyloric antrum, infection with bacteria such as Clostridium perfringes type A or unidentified fungi, abomasal localization of Trichostrongylus species together with Haemoncus species, trace mineral deficiency specially coper, and concurrent stress (Murray et al., 1995). The use of non steroidal anti-inflammatory agents can also lead to ulceration in
any age. In addition, the ingestion of heavy metals, dry weather, high stocking rates and application of fertilizers (Moriss et al., 2000).

Jensen et al. (1992) mentioned that, feeding of large levels of grains in feedlot cattle might be a risk factor associates with abomasal erosions. Ulcers developed during all seasons and all stages of fattening but more common during the first 45 days of winter initiating fattening than other times.

Fox (1980) Smith et al. (1983) and Rebhun (1995) described four types of abomasal ulceration which include: non perforating ulcer, bleeding ulcer, perforating ulcer with acute local peritonitis and perforating ulcer with diffuse peritonitis.

Whitlock (1980) and Rebhun (1995) stated that, abomasal ulcers with slight bleeding are the most difficult to diagnose because signs are not profound. A small quantity of tarry material (blood clots) mixed with normal faces were observed. This may occur only once or intermittently at variable intervals.

Most of the bleeding ulcers located in the greater curvature of the fundic area. They were uncommon in cows less than 2 years old and significantly more common in cows over 3 years old (Braun et al., 1991).

Perforating abomasal ulcer with localized peritonitis is an ulcer extends through the abomasal wall, including the serosal surface and the perforation is small (1-3mm), gastric contents bleed through it, which results in a localized
peritonitis and a syndrome similar to traumatic reticuloperitonitis. However, in case of diffuse peritonitis death occur within 24-48 hours after the onset of clinical signs (Johnson et al., 1981; Palmer and Whitlock, 1984; and Rebhun, 1995).

The clinical syndromes of abomasal ulceration varies depending on weather ulceration is complicated by hemorrhage or perforation. Abdominal pain, melena, pale mucous membranes, indigestion and dropped milk production are its common clinical findings in cattle. Some animals have abomasal ulceration at necropsy which was sub clinical (Wass et al., 1981; Braun et al., 1991; and Radostitis et al., 2000).

Murray et al. (1995) mentioned that, many cases of abomasal ulcers particularly in calves cause no apparent illness. Melena almost is a pathognomic sign of an acute bleeding ulcer of the abomasum. However, the presence of normal colored feces does not exclude the presence of chronic non-bleeding ulcers, which may be the cause of intractable indigestion.

Palmer and Whitlock (1984) and Rebhun (1995) reported that, the affected animal with perforating ulcer with localized peritonitis has fever of 39.4 - 40.6°C, rumen hypomotility or stasis, abdominal pain and an audible grunt with each expiration and the animal somewhat reluctant to move. They added that, the entire course of the disease may be per-acute with death within 6 hours or can be extended to 36-72 hours or longer if medical support is provided.
Abomasal ulcers in most cases are multiple and usually associated with acute abomasitis. They were linear, round, oval or irregular with sharp margins. The base of the ulcer showed brown discoloration and was sometimes hemorrhagic (Smith et al., 1986). The most frequent location of ulcers were the pylorus and were occasionally in the fundic region. Most ulcers are approximately circular, 2-4 cm in diameter or may be irregular up to 15 cm in size (Braun et al., 1991).

Most cases of perforating ulcers in cattle were walled of by omentum with the formation of a large cavity contains degenerated blood and necrotic debris. Adhesions may present between the ulcer and surrounding organs or abdominal wall (Palmer and Whitlock, 1984; and Cable et al., 1998).

Ibrahim (1991) found that, in a study of 650 cattle (420 fattening steers and 230 adults) and 530 buffalos (280 fattening steers and 250 adults) were examined in Cairo and Geiza abattoirs for presence of abnormalities and pathological changes in the omasum and abomasum. The abomasal abnormalities and pathological changes were abomasitis (2.6-12.9% in cattle and 1.2-5.7% in buffalos), ulcerations (0.9-4.8% in cattle and 0.4-1.8% in buffalos), impaction (0.0-0.5% in cattle and 0.0-0.4% in buffalos), displacement (0.0-0.4% in cattle and 0.0% in buffalos), worm infestation (28.0-72.0% in cattle and 19.0-48.0% in buffalos), and tumors only one case in buffalo.
Hosein et al. (1995) reported that, in a study of 83 emergency slaughtered and freshly dead calves aged from 1-5 months with histories of abdominal pain, some times diarrhea, tympany or sudden death. Necropsy findings revealed abomasitis, hyperemia, erosions, and ulcerations in the abomasal mucosa with greyish-brown to yellowish fluid in 18% of examined calves. Bacteriological examination of abomasal contents and tissue specimens from ulcerated mucosa yielded Clostridium perfringes type A (9.6%), Campylobacter jejuni (3.6%) and E-coli (1.2%).

Diagnosis of small abomasal ulcers was difficult unless the lesion has caused hemorrhage or peritonitis. Definitive diagnosis was generally based on observation during surgery or postmortem examination (Tullners and Hamilton, 1980). The clinical findings of chronic ulceration can resemble many other diseases of the alimentary tract (lymphosarcoma, volvulus, ileus, tumors and intestinal ulcers) but the presence of occult blood in the faeces and hematological evidence of hemorrhagic anemia were suggestive (Braun et al., 1991).

Rebhun (1982) and Rebhun (1995) treated bleeding abomasal ulcers in cows by blood transfusion (4-10 liters), fluid therapy (5-20 liters of sodium chloride and glucose solution), and 3-4 liters of sodium chloride solution containing gelatin as a plasma volume expander. The cows also received 2 gm vitamin C, 2 gm vitamin K, 500 ml of a solution containing 35 gm calcium gluconate and 60 gm magnesium hypophosphite intravenously, magnesium
oxide 1 gm/kg body weight orally and metoclopramide 30 gm 7-9 times at 8 hours intervals.

Recent studies on the treatment of abomasal ulcers describe the effectiveness of type 2 anti-histaminic blockers and anti-acids administered orally. The recommended dosages include cimetidine (50-100 mg/ kg per os /8 hours), or ranitidine (10-50 mg /kg per os / 8 hours). The use of aluminum hydroxide and magnesium hydroxide compounds (50 ml containing 5.0 gm of aluminum hydroxide and 4.5 gm of magnesium hydroxide per os/ 8 hours) also, increase gastric pH. However, doses may have adverse effects such as metabolic alkalosis, diarrhea, and hypomagnesaemia (Simon et al., 1985; Braun et al., 1991; and Ahmed et al., 2002).

Surgical correction of abomasal ulcers has been attempted with some limited success. The presence of multiple ulcers may require the radical excision of a large portion of the abomasal mucosa. The surgical approach requires a laparotomy and exploratory abomasotony to determine the presence and location of the ulcer (Tulleners and Hamilton, 1980).
V-Abomasitis:

Abomasitis is a sporadic disorder among cattle, calves, sheep and goats. It is characterized by diffuse, hemorrhagic to necrotizing inflammation of the abomasal mucosa, frequently involving the deeper layers of the abomasal wall in severe or chronic cases. Emphysema and edema of the abomasal wall may be present also (Dahlgren et al., 1984; Mills et al., 1994; and Metre & Callan, 2006).

Many causes of abomasitis have been postulated: dietary coarse roughage feeds, environmental or physical stress, infections with various microorganisms either bacteria such as C.perfringens, Salmonella and Pseudomonas, mycotic as Mucor species, viral like Bovine viral diarrhea and Rinder pest or parasitic infestation by Ostertagia or Trichostrongylus species (Lilley et al., 1985; Mills et al., 1990; and Mendez et al., 1995).

Jensen et al., (1976) mentioned that, abomasal ulcers, erosions and abomasitis are frequently observed at necropsy or at inspection of slaughtered animals, while their diagnosis on clinical basis is still obscure and seldom detected. However, their general clinical signs include: abdominal distension, abdominal pain, diarrhea, and death (Roeder et al., 1988).

The postmortem findings of abomasitis were acute hemorrhagic, necrotizing inflammation of the mucosa with emphysema of the abomasal wall
which was grossly thickened with varying degrees of edema (Roeder et al., 1988; and Mills et al., 1994). In addition to multiple whitish tiny nodules with parasitic infestation (Gregory et al., 1985; Mendez et al., 1995; and Songer et al., 2005).

Abomasitis can be treated with I/V fluid therapy, parenteral antibiotic with the antitoxin for C.perfringens (Rebhun, 1995). In addition to decompression of the abomasum via percutaneous ventral abomasocentesis or abomasotomy (Metre and Callon, 2006).
Materials and Methods

The present study was conducted on a total number of 2180 cattle, from those 173 cattle (56 Holstein, 59 mixed breed and 58 native cattle) aged from 6 months to 10 years and of both sexes, and suffering from one or more abomasal disorders (LDA, AV, impaction, ulcers, abomasitis and worm infestation). The affected animals were belonged to a private animal farm stations at Dakahlia Province (34 cattle), animals that admitted to the Mansoura Veterinary Teaching Hospital (11 cattle) and those surveyed at Mansoura abattoir (128 cattle). In addition to ten clinically healthy dairy cows were selected and served as control groups for laboratory examination and five ones were subjected to ultrasonographic examination. The later was performed at surgery dept. Fac. Vet. Med. Benha University.

Diagnosis and categorization of the affected animals were based on the well-informed case history, presenting clinical findings, simultaneous auscultation and percussion of the left abdominal wall in an area marked by a line drawn from the tuber coxae to the point of elbow and from the elbow toward the stifle (Fig.1). Exploratory laparo-rumenotomy and laboratory investigations were performed. In addition to, histopathological examinations were performed in some disorders.
Materials and Methods

Ultrasonographic examination of the normal abomasum:

Ultrasonographic examination of the abomasum was performed in a standing five cows by applying a 8 MHz linear transducer to the ventral aspect of the abdomen 10 cm caudal to the xiphoid process in the transverse and sagittal planes from the left and right paramedian regions and from the ventral midline according to Braun et al. (1997).

Samples and sampling protocol

Two types of blood samples were obtained from each animal by jugular venipuncture. The first blood samples (whole blood) were used for determination of PCV. The second blood samples were used for separation and collection of serum for biochemical analysis of the selected parameters (Coles, 1986).

The biochemical analysis for the selected parameters were measured spectrophotometrically using the commercially available kits, specifically chloride, sodium, potassium, and urea nitrogen according to Fawcett (1960). Phosphorus, calcium, magnesium and total protein according to Henry (1964). Aspartate transaminase (AST) and glucose according to Triender (1969). Alkaline phosphatase and creatinine according to Feldkamp (1974). Total bilirubin and serum albumen according to Varely et al. (1980) and packed cell volume was determined according to Coles (1986).
Materials and Methods

Medical treatment of LDA

The recommended treatment regimen for diseased cows with LDA were intravenous injection of Ringer’s solution & Dextrose 5%* at the rate of 10 ml/kg body weight, 20% Calcium solution at the rate of 2 L/head/daily and Paraffin oil 5 liters/head orally.

Surgical treatment of LDA

Surgical correction of LDA was performed through standing left flank approach; either by left flank omentopexy (17 cows) and left flank abomasopexy (15 cows) according to Smith (1981).

Preoperative preparation and analgesia

Feed were withheld for 24 hours prior to surgery. The left para-lumbar fossa is prepared for aseptic surgery. Pre-operative antibiotic include intramuscular injection of Gentamycin sulphate** 3-4mg/Kg.B.W. and Benzyl pencillin*** 20,000-30,000 i.u./kg.b.wt. Sedation was conducted by I.M. injection of Xylazine Hcl 2%**** 0.05mg/Kg.B.W. Local analgesia in standing position was achieved by using inverted L-shape infiltration of 2% Lidocaine Hcl*****.

** Gentamycin sulphate (Garavet): Memphis Co. for pharm. Cairo, A.R.E.
*** Benzyl penicillin: Nile Co. for Pharm. Cairo, A.R.E.
**** Xylazine Hcl (Xylaject): The Egyptian Co. for Chemical Pharmaceutical (ADWIA) A.R.E.
***** Lidocaine Hcl (Debocaine): El-Nasr Pharm. Chemical Co. for Al-Debeiky Pharma-A.R.E.
Materials and Methods

Surgical techniques

Left flank omentopexy (Utrecht method):

Left flank omentopexy was performed in 17 cows with LDA in which the abdomen was entered through 15-20 cm long vertical incision in the left para-lumbar fossa 8-10 cm distal to the transverse process of the lumbar vertebrae and 2 cm caudal and parallel to the last rib. The abomasum usually lies under the incision (Fig.2). The attachment of the greater omentum along the abomasum was located (Fig.3). The abomasum was decompressed using a 13 to 14 gauge needle attached to infusion set to allow the accumulated gas to be defelated. After complete abomasal deflation the needle and tube were carefully removed. A needle threaded with about 2 meters of hernial tape was passed in and out of the greater omentum adjacent to the pylorus or greater curvature in the form of cross mattress sutures over a length of about 7cm. About a meter of the hernial tape should extend from each end of the suture line (Fig.4). The abomasum was then carefully pushed to its normal position. The cranial end of the suture was attached to a large straight sharp needle which was carried in guarded position under the hand protection along the internal body wall and forced through the ventral midline, 10-15cm caudal to the xiphoid. An assistant grasps the needle with the tape. A second needle was passed by the same manner 10cm caudal to the cranial suture. The two suture ends were pulled by an assistant (Fig.5). While the surgeon palpates to insure that no structures are trapped between the abomasum sutures and body wall. The two suture free ends


Materials and Methods

were tied in place firmly. The abdomen was lavaged with 2 liters of warm physiological saline solution prior to routine closure of the celiotomy incision.

Left flank abomasopexy:

Left flank abomasopexy was performed in 15 cows with LDA as the technique was similar to the left flank omentopexy except that the used suture materials was polygalactin 910 * or silk.No.3 ** placed in a simple continuous seromuscular manner or in cross fashion in the greater curvature of the abomasum (Fig.6&7). The suture material ends were then brought through the ventral abdominal wall as omentopexy (Fig.8).

Post-operative care:

The pre-operative antibiotic as well as fluid therapy (10ml/Kg.B.Wt.) were continued for five successive days with roughages supplementation. Such animals were examined 3 times after surgery (3, 14 and 60 days) to check the rumen motility, presence or absence of ping sound, milk production and feed consumption. The areas of the needle penetration were examined for signs of pain and degrees of tissues reactions. The suture ends at the ventral body wall were cutted at 10-14 days as close as possible to the skin allowing the sutures to recede into the skin and abomasum. Also the laparotomy skin sutures were removed.

* Coated Vicryl: Ethicon L.T.D./U.K.
Materials and Methods

Abomasal impaction treatment

Cows with abomasal impaction were subjected to the traditional methods for treatment of indigestion and all failed to relieve the conditions. Consequently all cases have been subjected to the surgical intervention by laparo-rumenotomy for assessment of the gross lesions and provide a method for infusion of medication directly into abomasum. Rumenotomy was performed by Weingerth’s technique in standing position according to Singh et al. (1996) (Fig.9&10). Liquid paraffin (3-5 L) was pumped through a tube placed in the reticulo-omasal orifice into the omasum and abomasum to soften the contents (Fig.11).

Necropsy study of abomasal disorders

This part of the study was conducted on 128 animals that introduced to the abattoir. In addition to the animals that emergency slaughtered in the farms after failure of their treatment. These animals were examined for any abomasal lesions after slaughtering. Out of the total surveyed animals at abattoir. The affected animals were found to be involved either with abomasal erosions and ulceration, abomasitis, parasitic infestation, AV and impaction. The post-mortem examination was confined to intact abomasum which had been severed from the rest of the gastrointestinal tract immediately after slaughter. The mucosal surface of the abomasum was inspected after a longitudinal incision had been made along the greater curvature. The incised abomasum was placed in a water bath to wash away feed particles. The mucosa was examined, and the
Materials and Methods

type, number, and location of lesions were recorded.

**Histopathological examination:**

Several sections (1x1x1cm) from the affected abomasum were collected in 10% neutral buffered formalin, cleared in xylene, embedded in paraffin, cut at 5µm, and stained with Haematoxylin and Eosin (H&E) and examined microscopically according to Bancroft et al. (1996).

**Statistical analysis**

The obtained data were statistically analyzed. The mean values and standard error (SE) were calculated and the significance was determined according to Snedecor and Cochran (1982).
Fig (1): An area of tympanitic resonance (ping sound) marked by a line drawn from the tuber coxae to the point of elbow and from the elbow toward the stifle heard over the left side of the abdominal wall of adult cattle.
**Fig (2):** A distended abomasum lies immediately under the left laparotomy incision(a) and upward the rumen(b) in a Holstein cow.

**Fig.(3):** The same case in Fig.(2) showing the omental attachment(a) to the distended abomasum(b).
**Materials and Methods**

**Fig.(4):** The same case in Fig.(2) showing abomasal deflation by using a 13-14 gauge needle (white arrow) attached to infusion set, and the cross mattress suture by using hernial tape (black arrow) in the omental attachment to the abomasum for left flank omentopexy.

**Fig (5):** The same case in Fig.(2) showing the ends of the hernial tape (arrow) pulled outside the ventral abdominal wall, 10 cm cranial to the umbilicus.
Materials and Methods

**Fig (6):** Two cross mattress sutures passed in a seromuscular fashion in the greater curvature of the abomasum for left flank abomasopexy by using No.3 polygalactin 910(Vicryl)- Ethicon L.T.D./ U.K.

**Fig (7):** Left flank abomasopexy by using silk(No.2) inserted seromuscularly in a simple continuous manner through the greater curvature of the abomasum.
**Materials and Methods**

**Fig (8):** The two free ends of polygalactin 910 pulled outside the ventral abdominal wall with interval 10cm in between for complete the left flank abomasopexy.

**Fig (9):** Pre-operative preparation of the left flank for laparorumenotomy.
Fig (10): A left flank laparorumenotomy by Weingerth's technique.

Fig (11): Ruminal incision after evacuation of the ruminal contents and pumping of liquid paraffin (3-5L) through a tube placed in the reticulo-omasal orifice into the omasum and abomasum for treatment of abomasal impaction.
Results

In the present study a total number of 173 diseased cattle were categorized as (34 cattle with LDA, 1 cattle with AV, 4 cattle with abomasal impaction, 35 cattle with abomasal erosions and ulcerations, 40 cattle with abomasitis and 59 cattle with abomasal worm infestation) as shown in (Table.1) and (Fig.12).

Table (1): Categorization of the examined animals according to their disease condition:

<table>
<thead>
<tr>
<th></th>
<th>The examined cattle</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Holstein</td>
<td>Mixed</td>
</tr>
<tr>
<td>Left abomasal displacement</td>
<td>34</td>
<td>-</td>
</tr>
<tr>
<td>Abomasal volvulus</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td><strong>Abomasal impaction</strong></td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Abomasal erosions and ulcerations</td>
<td>3</td>
<td>19</td>
</tr>
<tr>
<td>Abomasitis</td>
<td>7</td>
<td>21</td>
</tr>
<tr>
<td>Abomasal parasitism</td>
<td>9</td>
<td>17</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>56</td>
<td>59</td>
</tr>
</tbody>
</table>
Results

Fig (12): The different abomasal disorders in different breeds.
**Results**

I- Normal abomasal sonogram

In the five examined animals, the bulk of the abomasum is situated to the right ventral midline at the ventral abdominal wall. The different layers of the ventral abdominal wall were appeared as a narrow bands of varying echogenisity. The wall of the abomasum appears at the most as a thin echogenic line. However, the abomasum is easily differentiated from neighboring organs by the ultrasonographic appearance of its contents, which are seen as a heterogenous moderately echogenic mass with echogenic stippling (Fig.13). Parts of the abomasal folds can occasionally be seen as a sickle-shaped echogenic structures within the content of the abomasum. Passive and slow movement of the abomasal contents is frequently seen (Fig.14).
**Fig. (13):** The different layers of the ventral abdominal wall appeared as a narrow bands of varying echogenility (A) and the wall of the abomasum appeared at the most as a thin echogenic line (B).

**Fig. (14):** The abomasal wall appeared as a thin echogenic line (A) and parts of the abomasal folds can occasionally be seen as a sickle-shaped echogenic structures within the content of the abomasum (arrow).
II- Left displacement of the abomasum:

LDA were recorded in 34 Holstein cows. The age of the affected cows were ranged from 2–7 years (Fig.15), 27 cases had calved recently (2-4 weeks), six cases were presented after 2–7 months post-partum and one case was present 2 months pre-partum, with a history of reduced milk yield, varying degrees of reduced appetite and scanty feces. Simultaneous auscultation and percussion, of the middle to upper half of the left abdomen under the last five intercostal spaces (9th - 13th) revealed a specific left tympanic resonance (ping) sound for LDA (Fig.16). A varies degrees of dehydration and rumen stasis were also evident. Rectal examination revealed medial displacement of the rumen and left displacement of the abomasum, which appeared as a soft fluctuant or emphysematous mass in the left abdominal wall.

The peak of disease occurrence was recorded in the late autumn (11 cases) and winter (15 cases) as apposed to spring (5 cases) and summer (3 cases) months (Fig.17). The predominant concurrent diseases were ketosis (5 cases), hypocalcaemia (7 cases), metritis (3 cases), traumatic reticulitis (1 cases), retained placenta (2 cases), abomasal ulcer (2 cases), mastitis (2 cases), abortion (2 cases), and foot root (3 cases) (Fig.18). In addition to seven cases were diagnosed without apparent concurrent diseases (Table 2). Twins was present only in one cow.
**Results**

Table (2): The clinical data in 34 affected cattle with LDA:

<table>
<thead>
<tr>
<th>Date Cases</th>
<th>Age (years)</th>
<th>Breed</th>
<th>Season</th>
<th>Days from parturition to diagnosis (unless otherwise noted)</th>
<th>Concurrent diseases</th>
<th>Twins</th>
</tr>
</thead>
<tbody>
<tr>
<td>Case 1</td>
<td>2</td>
<td>Holstein</td>
<td>Autumn</td>
<td>12 days P.P</td>
<td>Retained Placenta</td>
<td>-</td>
</tr>
<tr>
<td>Case 2</td>
<td>4</td>
<td>Holstein</td>
<td>Winter</td>
<td>30 days P.P</td>
<td>Metritis</td>
<td>+</td>
</tr>
<tr>
<td>Case 3</td>
<td>2</td>
<td>Holstein</td>
<td>Winter</td>
<td>7 days P.P</td>
<td>Hypocalcaemia</td>
<td>-</td>
</tr>
<tr>
<td>Case 4</td>
<td>3</td>
<td>Holstein</td>
<td>Winter</td>
<td>23 days P.P</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Case 5</td>
<td>5</td>
<td>Holstein</td>
<td>Summer</td>
<td>210 days P.P</td>
<td>Ketosis</td>
<td>-</td>
</tr>
<tr>
<td>Case 6</td>
<td>3</td>
<td>Holstein</td>
<td>Autumn</td>
<td>15 days P.P</td>
<td>Abomasal ulcer</td>
<td>-</td>
</tr>
<tr>
<td>Case 7</td>
<td>2</td>
<td>Holstein</td>
<td>Winter</td>
<td>10 days P.P</td>
<td>Mastitis</td>
<td>-</td>
</tr>
<tr>
<td>Case 8</td>
<td>3</td>
<td>Holstein</td>
<td>Winter</td>
<td>53 days P.P</td>
<td>Ketosis</td>
<td>-</td>
</tr>
<tr>
<td>Case 9</td>
<td>3</td>
<td>Holstein</td>
<td>Spring</td>
<td>100 days P.P</td>
<td>Hypocalcaemia</td>
<td>-</td>
</tr>
<tr>
<td>Case 10</td>
<td>2</td>
<td>Holstein</td>
<td>Autumn</td>
<td>15 days P.P</td>
<td>Metritis</td>
<td>-</td>
</tr>
<tr>
<td>Case 11</td>
<td>6</td>
<td>Holstein</td>
<td>Winter</td>
<td>10 days P.P</td>
<td>Abortion</td>
<td>-</td>
</tr>
<tr>
<td>Case 12</td>
<td>4</td>
<td>Holstein</td>
<td>Winter</td>
<td>37 days P.P</td>
<td>Ketosis</td>
<td>-</td>
</tr>
<tr>
<td>Case 13</td>
<td>7</td>
<td>Holstein</td>
<td>Summer</td>
<td>150 days P.P</td>
<td>Foot rot</td>
<td>-</td>
</tr>
<tr>
<td>Case 14</td>
<td>3</td>
<td>Holstein</td>
<td>Autumn</td>
<td>13 days P.P</td>
<td>Ketosis</td>
<td>-</td>
</tr>
<tr>
<td>Case 15</td>
<td>2</td>
<td>Holstein</td>
<td>Winter</td>
<td>16 days P.P</td>
<td>Hypocalcaemia</td>
<td>-</td>
</tr>
<tr>
<td>Case 16</td>
<td>5</td>
<td>Holstein</td>
<td>Autumn</td>
<td>18 days P.P</td>
<td>Abortion</td>
<td>-</td>
</tr>
<tr>
<td>Case 17</td>
<td>7</td>
<td>Holstein</td>
<td>Winter</td>
<td>15 days P.P</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Date Cases</td>
<td>Age (years)</td>
<td>Breed</td>
<td>Season</td>
<td>Days from parturition to diagnosis (unless otherwise noted)</td>
<td>Concurrent diseases</td>
<td>Twins</td>
</tr>
<tr>
<td>------------</td>
<td>------------</td>
<td>-------</td>
<td>--------</td>
<td>-------------------------------------------------------------</td>
<td>---------------------</td>
<td>-------</td>
</tr>
<tr>
<td>Case 18</td>
<td>6</td>
<td>Holstein</td>
<td>Spring</td>
<td>10 days P.P</td>
<td>Hypocalcaemia</td>
<td>-</td>
</tr>
<tr>
<td>Case 19</td>
<td>6</td>
<td>Holstein</td>
<td>Autumn</td>
<td>100 days P.P</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Case 20</td>
<td>4</td>
<td>Holstein</td>
<td>Autumn</td>
<td>19 days P.P</td>
<td>Foot rot</td>
<td>-</td>
</tr>
<tr>
<td>Case 21</td>
<td>4</td>
<td>Holstein</td>
<td>Spring</td>
<td>90 days P.P</td>
<td>Traumatic reticulitis</td>
<td>-</td>
</tr>
<tr>
<td>Case 22</td>
<td>3</td>
<td>Holstein</td>
<td>Autumn</td>
<td>60 days Pre. P</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Case 23</td>
<td>4</td>
<td>Holstein</td>
<td>Winter</td>
<td>16 days P.P</td>
<td>Retained Placenta</td>
<td>-</td>
</tr>
<tr>
<td>Case 24</td>
<td>3</td>
<td>Holstein</td>
<td>Autumn</td>
<td>70 days P.P</td>
<td>Abomasal ulcer</td>
<td>-</td>
</tr>
<tr>
<td>Case 25</td>
<td>3</td>
<td>Holstein</td>
<td>Winter</td>
<td>18 days P.P</td>
<td>Mastitis</td>
<td>-</td>
</tr>
<tr>
<td>Case 26</td>
<td>3</td>
<td>Holstein</td>
<td>Winter</td>
<td>7 days P.P</td>
<td>Ketosis</td>
<td>-</td>
</tr>
<tr>
<td>Case 27</td>
<td>4</td>
<td>Holstein</td>
<td>Spring</td>
<td>20 days P.P</td>
<td>Hypocalcaemia</td>
<td>-</td>
</tr>
<tr>
<td>Case 28</td>
<td>4</td>
<td>Holstein</td>
<td>Spring</td>
<td>10 days P.P</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Case 29</td>
<td>3</td>
<td>Holstein</td>
<td>Summer</td>
<td>21 days P.P</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Case 30</td>
<td>5</td>
<td>Holstein</td>
<td>Autumn</td>
<td>11 days P.P</td>
<td>Hypocalcaemia</td>
<td>-</td>
</tr>
<tr>
<td>Case 31</td>
<td>2</td>
<td>Holstein</td>
<td>Winter</td>
<td>15 days P.P</td>
<td>Metritis</td>
<td>-</td>
</tr>
<tr>
<td>Case 32</td>
<td>2</td>
<td>Holstein</td>
<td>Winter</td>
<td>30 days p.p</td>
<td>Hypocalcaemia</td>
<td>-</td>
</tr>
<tr>
<td>Case 33</td>
<td>5</td>
<td>Holstein</td>
<td>Autumn</td>
<td>43 days p.p</td>
<td>Foot root</td>
<td>-</td>
</tr>
<tr>
<td>Case 34</td>
<td>4</td>
<td>Holstein</td>
<td>Winter</td>
<td>17 days p.p</td>
<td>-</td>
<td>-</td>
</tr>
</tbody>
</table>

P.P = Post partum

Pre.P = Pre partum
Pre-operative laboratory findings:

The affected cows with LDA showed decrease in serum chlorides, sodium, potassium and calcium, and in blood glucose in comparison to control cows. Two cows showed a significant hypochloremia (78mEq/l) and hypocalcaemia (4.8mg/dl) which could not be with-stand the risk of surgical interference and were sold for slaughtering. The preoperative mean of PCV exceeded the normal range with higher blood urea, serum creatinine, AST, serum albumen, serum alkaline phosphatase, serum total protein and total serum bilirubin in comparison to control cows as shown in table (3&4).
Results

Table (3): Mean values±SE of serum electrolytes in control cows and in affected cows with LDA before surgical interference:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Controlled cows</th>
<th>Affected Cows</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorides mEq/l</td>
<td>92.87 ± 2.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>84.24 ± 1.84&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Sodium mEq/l</td>
<td>147.63 ± 7.1&lt;sup&gt;a&lt;/sup&gt;</td>
<td>130.69 ± 3.21&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Potassium mEq/l</td>
<td>4.20 ± 0.37&lt;sup&gt;a&lt;/sup&gt;</td>
<td>2.71 ± 0.11&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Calcium mg/dl</td>
<td>12.02 ± 1.01&lt;sup&gt;a&lt;/sup&gt;</td>
<td>6.18 ± 0.23&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Mean with the same superscripts are not significantly different at (P< 0.05).

Table (4): Mean values±SE of blood biochemical and hematological parameters in control cows and in affected cows with LDA before surgical interference:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Controlled cows</th>
<th>Affected Cows</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total protein gm/dl</td>
<td>7.3±0.42&lt;sup&gt;a&lt;/sup&gt;</td>
<td>9.9±0.39&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Urea mg/dl</td>
<td>25.13±1.6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>59.41 ± 7.3&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Glucose mg/dl</td>
<td>67.69 ± 18.33&lt;sup&gt;a&lt;/sup&gt;</td>
<td>49.72 ± 3.92&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>A.S.T.u/l</td>
<td>78.72 ± 5.02&lt;sup&gt;a&lt;/sup&gt;</td>
<td>103.84 ± 8.81&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Alkalnie phosphatase u/l</td>
<td>58.91 ± 5.12&lt;sup&gt;a&lt;/sup&gt;</td>
<td>88.97 ± 3.88&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Albumine mg/dl</td>
<td>4.93 ± 0.2&lt;sup&gt;a&lt;/sup&gt;</td>
<td>4.47 ± 0.27&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Total bilirubine mg/dl</td>
<td>0.8 ±0.04&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.23 ± 0.06&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Creatinine mg/dl</td>
<td>0.95 ± 0.03&lt;sup&gt;a&lt;/sup&gt;</td>
<td>1.94 ± 0.1&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>P.C.V %</td>
<td>34.6± 1.4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>41.67 ± 1.56&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

Mean with the same superscripts are not significantly different at (P< 0.05).
The affected animals showed no great response to medical therapy and were subjected for surgical intervention through left flank omentopexy (17 cows) and left flank abomasopexy (15 cows).

Out of 32 operated cows, 27 cows were recovered without complications and still in their reproductive herd producing nearly a normal quantity of milk as judged by owners/manager (Table 5) and (Fig.19). During laparotomy 2 cows showed severe adhesion between the greater curvature of the abomasum and the peritoneum secondary to a perforating abomasal ulcer and were sold for slaughter. Also, 3 cows were culled because of these cows did not reach the normal level of productivity.
**Fig. (15):** The relation between age per year and LDA occurrence in affected cows.
**Fig. (16):** The typical area of the ping characteristic to LDA located in an area between 9\textsuperscript{th}-13\textsuperscript{th} intercostal spaces in the middle to upper third of the abdomen of adult cattle on the drown region.
Fig. (17): The relation between seasonal variation within the year and LDA occurrence.
Results

Fig. (18): The most common concurrent diseases associated with LDA in affected cows.
**Results**

**Table (5):** Illustrate the mean values of milk production (kg/day) in affected cows with LDA pre-and post-operatively:

<table>
<thead>
<tr>
<th>Case</th>
<th>Mean lactation display (kg / day)</th>
<th>Case</th>
<th>Mean lactation display (kg / day)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post (20-60day)</td>
<td>Pre</td>
</tr>
<tr>
<td>Case 1</td>
<td>11</td>
<td>28</td>
<td>Case 18</td>
</tr>
<tr>
<td>Case 2</td>
<td>7</td>
<td>7(culled)</td>
<td>Case 19</td>
</tr>
<tr>
<td>Case 3</td>
<td>9</td>
<td>31</td>
<td>Case 20</td>
</tr>
<tr>
<td>Case 4</td>
<td>6</td>
<td>27</td>
<td>Case 21</td>
</tr>
<tr>
<td>Case 5</td>
<td>8</td>
<td>30</td>
<td>Case 22</td>
</tr>
<tr>
<td>Case 6</td>
<td>4</td>
<td>Slaughtered*</td>
<td>Case 23</td>
</tr>
<tr>
<td>Case 7</td>
<td>4</td>
<td>22</td>
<td>Case 24</td>
</tr>
<tr>
<td>Case 8</td>
<td>6</td>
<td>13(culled before operation)</td>
<td>Case 25</td>
</tr>
<tr>
<td>Case 9</td>
<td>5</td>
<td>26</td>
<td>Case 26</td>
</tr>
<tr>
<td>Case 10</td>
<td>9</td>
<td>31</td>
<td>Case 27</td>
</tr>
<tr>
<td>Case 11</td>
<td>4</td>
<td>27</td>
<td>Case 28</td>
</tr>
<tr>
<td>Case 12</td>
<td>10</td>
<td>35</td>
<td>Case 29</td>
</tr>
<tr>
<td>Case 13</td>
<td>7</td>
<td>6(culled)</td>
<td>Case 30</td>
</tr>
<tr>
<td>Case 14</td>
<td>11</td>
<td>29</td>
<td>Case 31</td>
</tr>
<tr>
<td>Case 15</td>
<td>9</td>
<td>30</td>
<td>Case 32</td>
</tr>
<tr>
<td>Case 16</td>
<td>3</td>
<td>22</td>
<td>Case 33</td>
</tr>
<tr>
<td>Case 17</td>
<td>12</td>
<td>31</td>
<td>Case 34</td>
</tr>
</tbody>
</table>

* Showing abomasal ulcerations with severe adhesion between the abomasum and the peritoneum.
Fig. (19): The amount of milk production in diseased cows with LDA pre-and post-operatively.
Results

Post-operative laboratory findings:

The mean values of blood and serum biochemical and hematological parameters for the operated cows were nearly similar to that of control cows as shown in table (6&7) and illustrated by (Fig.20&21).

**Table (6):** Mean values±SE of serum electrolytes in control cows and in affected cows with LDA after surgical interference:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Controlled cows</th>
<th>Affected Cows</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorides mEq/l</td>
<td>92.87 ± 2.04</td>
<td>99.12 ± 1.9</td>
</tr>
<tr>
<td>Sodium mEq/l</td>
<td>147.63 ± 7.1</td>
<td>145.24 ± 6.1</td>
</tr>
<tr>
<td>Potassium mEq/l</td>
<td>4.20 ± 0.37</td>
<td>4.60 ± 0.51</td>
</tr>
<tr>
<td>Calcium mg/dl</td>
<td>12.02 ± 1.01</td>
<td>9.49 ± 0.84</td>
</tr>
</tbody>
</table>

Mean with the same superscripts are not significantly different at (P< 0.05).

**Table (7):** Mean values±SE of blood biochemical and hematological parameters in control cows and in affected cows with LDA after surgical interference:

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Controlled cows</th>
<th>Affected Cows</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total protein gm/dl</td>
<td>7.3±0.42</td>
<td>7.25±0.1</td>
</tr>
<tr>
<td>Urea mg/dl</td>
<td>25.13±1.6</td>
<td>35.58±1.52</td>
</tr>
<tr>
<td>Glucose mg/dl</td>
<td>67.69±18.33</td>
<td>98.12±21.75</td>
</tr>
<tr>
<td>A.S.T.u/l</td>
<td>78.72±5.02</td>
<td>68.32±3.4</td>
</tr>
<tr>
<td>Alkaline phosphatase u/l</td>
<td>58.91±5.12</td>
<td>56.81±4.33</td>
</tr>
<tr>
<td>Albumine mg/dl</td>
<td>4.93±0.2</td>
<td>4.48±0.12</td>
</tr>
<tr>
<td>Total bilirubine mg/dl</td>
<td>0.8±0.04</td>
<td>0.78±0.03</td>
</tr>
<tr>
<td>Creatinine mg/dl</td>
<td>0.95±0.03</td>
<td>0.9±0.04</td>
</tr>
<tr>
<td>P.C.V %</td>
<td>34.6±1.4</td>
<td>32.53±1.24</td>
</tr>
</tbody>
</table>

Mean with the same superscripts are not significantly different at (P< 0.05).
**Fig. (20):** The mean values of some serum electrolytes in healthy (control) cows and in diseased cows with LDA pre and post operatively.
**Results**

*Fig. (21)*: The mean values of some blood biochemical and hematological parameters in healthy (control) cows and in diseased cows with LDA pre and post operatively.
Results

III- Abomasal volvulus:

AV was diagnosed in a Holstein cow with a history of complete anorexia, sharp drop in milk production and tachycardia (120/min). The feces were scanty and dark in color. A marked depression, weakness and dehydration were obvious with animal recumbency within 24-36 hours from the disease occurrence. The blood and serum biochemical analysis for this cow revealed a significant hypochloremic (76mEq/l), hypokalemic (1.9mEq/l) metabolic alkalosis associated with hypocalcaemia (4.3mg/dl) which indicate a poor prognosis. This cow not respond to the traditional medical treatment and could not be with-stand the risk of surgical interference (exploratory laparotomy), so sold for slaughtering.

After slaughtering the necropsy findings revealed a distended abomasum with brownish, sanguineous fluid and twisted with omasal involvement. The abomasal and omasal mucosa was grossly hemorrhagic, edematous and gangrenous indicated by bluish or blackish coloration with multiple ulceration. The histopathological examinations showed necrosis with mucinous degeneration and focal replacement of the abomasal mucosa with numerous leukocytic infiltration (fig.22).
Fig. (22): The necropsy findings of abomasal volvulus with omasal involvement represented by hemorrhagic, edematous and gangrenous mucosa with multiple ulceration (a) and characterized histopathologically by coagulative necrosis of the mucosa, numerous mucinous degeneration and focal replacement of the tunica mucosa with numerous leukocytic infiltration (b) H&E x130.
Results

IV-Abomasal impaction:

Abomasal impaction was diagnosed in 2 Holstein and 2 mixed breed cows. The affected animals had a history of complete loss of appetite, deteriorated body conditions which might be masked by abdominal over distension which appeared papple-shaped (pear like on right side and apple like on the left one) when viewed from the rear and failure of the animals to defecate (Fig.23). Physical examinations revealed fluid-filled rumen with gurgling sound on auscultation of the left side of the abdomen which extended to the left flank. Moderate to severe degrees of dehydration had been observed and manifested by sunken eyes, folding of skin and loss of its elasticity.

Laparo-rumenotomy revealed that the abomasum was impacted and dilated occupying most of the ventral abdominal cavity displacing the rumen dorsally with omasal impaction and dilatation. The consistency of the abomasal contents were hard and difficult to pit under pressure. The contents of the rumen were frothy and large amount of aqueous, bad smell ruminal fluid gush out from the dilated rumen. Medical treatment of the impacted abomasum by direct administration of liquid paraffin inside the omasum through a tube placed in the reticulo-omasal orifice gave a good result in one case out of the four cases. The other three cows which not respond to treatment were sold for slaughtering. Necropsy findings showed that abomasum as a large bag with increased dimensions (90cm in length and 70cm in circumference). On cut section it was over packed by ingesta taking its shape which varies from hard to fragmented material and yellowish to brownish in colour. The normal soured odour of the abomasum was not detected (Fig.24).
Fig. (23): A Holstein cow showing abdominal distension (papple-shaped abdomen) in case of abomasal impaction (rear view).
Results

Fig. (24): Impacted abomasum appeared as a large bag with increased dimensions (a). On cut section, it was over packed by hard ingesta taking its shape (b).
Results

V - Abomasal erosions and ulcerations

Abomasal ulcerations were diagnosed in 35 cattle (10 cases (7 calves and 3 dairy cows) were clinically diagnosed and 25 cases (1 calf, 22 feedlot steer and 2 dairy cattle) were diagnosed by necropsy examinations). The clinically affected animals showed abdominal pain manifested by arched back and girding on teeth with foamy salivation, sudden onset of anorexia, excessive water intake, decreased milk production, melina (scanty black and tarry feced), weakness and anaemia manifested by pale mucous membranes and dehydration (Fig.25).

Abomasal ulcers were seen at necropsy in the fundic and pyloric regions or both of the abomasum as a focal areas of superficial erosions to deep ulcers. They appeared as a whitish, grayish or reddish area of different shapes, varies in sizes from 0.25cm-11cm and about 1-20 ulcers/abomasum in different locations of the abomasum with shallow to deep base according to the degree of ulceration. According to their shape, 3 types of ulcers were differentiated (Fig.26, 27, 28&29) and (Table 8, 9&10):

1-Linear ulcers:

Linear ulcers were mostly seen at the margin of prominent abomasal folds of the fundus or deep between them and some times at the body of the abomasum aligned longitudinally as linear areas measuring from 0.1-0.3 cm in diameter and 5-11cm in length. They were dark red in color. It characterized histoathologically by acute to subacute necrosis of the superficial epithelium which was swollen and had structural loss of the superficial cell layer without including the glandular zone in lamina propria. Also subjacent capillaries were
dilated, thick walled, and thrombosed. Mucosal defect were minimal in this type. Also, lines of intense hyperemia surrounded areas of erosions and ulceration were present on the edges of the abomasal folds or the pyloric region (Fig.30).

2-Circular (rounded) ulcers:

Circular ulcers were seen mostly at the pyloric region and rarely at the fundic region. They noticed as sharply defined dark reddish or yellowish spots with depressed centers and varied diameter from 0.25cm - 1.5cm. Minimal inflammatory reaction or swelling was present in the mucosa surrounding the lesion. It represented histopathologically by tissue damage deep and extensive at lesions centers as sometimes the lesions visible from the serosal side. Changes consisted of necrosis of the mucosal and submucosal epithelium often accompanied by hemorrhage (Fig.31).

3-Irregular Ulcers:

Irregular ulcers were scattered as irregular lines or radiation over the mucosa of both fundic and pyloric regions with more occurrences in fundic region (Fig.29a) Histopathologically there were local destruction of the mucosa including the sub mucosa. The mucosa was replaced by granulation tissue. A severely reduced number of tubular glands were present. There was a marked increase in connective tissue (Fig.32).
**Results**

**Table (8):** Percentage of the 3 types of abomasal ulcers in slaughtered cattle:

<table>
<thead>
<tr>
<th>Ulcer types</th>
<th>% of altered abomasa (n = 25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Linear (n = 9 )</td>
<td>36%</td>
</tr>
<tr>
<td>Circular (n = 11)</td>
<td>44%</td>
</tr>
<tr>
<td>Irregular (n = 5)</td>
<td>20%</td>
</tr>
<tr>
<td>Total altered (n = 25)</td>
<td>100%</td>
</tr>
</tbody>
</table>

**Table (9):** Topographical distribution of the various types of abomasal ulcers in slaughtered cattle:

<table>
<thead>
<tr>
<th>Ulcer types</th>
<th>Fundus</th>
<th>Pylorus</th>
<th>Fundus and pylorus</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Linear (n=9)</td>
<td>6 (66.7%)</td>
<td>1 (11.1%)</td>
<td>2 (22.2 %)</td>
<td>9 (36 %)</td>
</tr>
<tr>
<td>Circular (n=11)</td>
<td>1 (9.1%)</td>
<td>7 (63.6%)</td>
<td>3 (27.3 %)</td>
<td>11 (44 %)</td>
</tr>
<tr>
<td>Irregular (n=5)</td>
<td>3 (60 %)</td>
<td>1 (20%)</td>
<td>1 (20 %)</td>
<td>5 (20)</td>
</tr>
<tr>
<td>Total</td>
<td>10 (40 %)</td>
<td>9 (36 %)</td>
<td>6 (24 %)</td>
<td>25 (100 %)</td>
</tr>
</tbody>
</table>
**Table (10):** The age, breed, and sex distribution of various types of abomasal ulcers in slaughtered cattle:

<table>
<thead>
<tr>
<th>Ulcer's types</th>
<th>Data (n)</th>
<th>Age (Year)</th>
<th>Breed</th>
<th>Sex</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>0-1</td>
<td>1-2</td>
<td>&gt; 2 – 6</td>
</tr>
<tr>
<td>Linear (n = 9)</td>
<td>-</td>
<td>7</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Circular (n=11)</td>
<td>1</td>
<td>8</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Irregular (n=5)</td>
<td>-</td>
<td>4</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
<td>19</td>
<td>5</td>
<td>2</td>
</tr>
</tbody>
</table>

- Linear (n = 9): 1 (4%) 76% 20% 8% 60% 32% 88% 12%
- Circular (n=11): 1 (9%) 19% 3% 12% 9% 18% 22% 14%
- Irregular (n=5): 1 (5%) 11% 3% 6% 24% 10% 20% 8%
Fig. (25): Melina in case of abomasal ulceration in mixed breed calf laying on the ground.
Results

Fig. (26): The topographical distribution of the different types of abomasal ulcers in the examined animals.

Regions of ulcers in abomasum

Fig. (26): The topographical distribution of the different types of abomasal ulcers in the examined animals.
Fig. (27): The relation between abomasal ulcers and the age per year of examined animals.
Fig. (28): The relation between abomasal ulcers and the breed of examined animals.
**Fig. (29):** The relation between abomasal ulcers and the sex of examined animals.
Fig. (30): Abomasum showing a long linear ulcer(11cm) at a fundic fold(a) represented microscopically by periglandular leukocytic infiltration with complete loss of mucosal epithelium H&E x130(b).
**Fig.(31):** Abomasum showing two large rounded ulcers at the fundic region (a) characterized microscopically by focal replacement of the tunica mucosa with numerous leukocytic infiltration and fibrous connective tissue (F.C.T.) proliferation (b) H&E x130.
Fig. (32): Abomasum showing multiple irregular ulcerations at the pyloric part of the abomasum (a) characterized microscopically by coagulative necrosis of the mucosal epithelium beside focal replacement of the mucosal glands by numerous leukocytic infilteration (b) H&E x520.
Results

VI- Abomasitis

Abomasitis was detected in 40 slaughtered cattle. Macroscopically the serosal surface showed mild degree of congestion. On cut section its wall was occasionally edematous, or contain few amount of ingesta which appeared slimy in nature with disappearance of the normal soured abomasal odour. Its mucosal surface appeared hyperemic in focal or diffuse manner. Microscopically, focal areas of desquamated epithelium with severe leukocytic infiltration, mainly macrophages were detected. In some other cases focal areas of coagulative necrosis in the glandular zone were seen and adjacent to them a zone of inflammatory cells were extending to include the muscularis mucosa. Mild to marked multifactorial edema of the sub mucosa with a distinct and prominent inflammatory components observed. Also, small indistinct strips of catarrhal exudates consisted of small areas of detached cells were seen on the abomasal surface (Fig.33).

VII- Abomasal parasitism

In 59 infested examined abomasum, there were multiple small yellowish - red bite marks (parasitic nodules) varies between 15-17 nodules widely distributed all over the abomasum especially on the body. These nodules were presented as focal minute raised areas (pin point size) with depressed centers which giving the abomasum surface a granular appearance. Histopathologically the abomasal parasitic nodules were composed mainly of focal superficial eroded folds infiltrated with and surrounded by leukocytic infiltrations. Also in other cases fibrous connective tissues proliferation in the interstitial tissue with edema of the submucosal were detected (Fig.34).
Fig.(33): Abomasum showing catarrhal abomasitis and erosions of the pyloric region represented by mucosal desquamation with loss of normal mucosal rugae and local lines of hyperemia, congestion and hemorrhage (a) which characterized histopathologically by edematous and necrotic mucosa infiltrated with mononuclear cells of mainly macrophages and widely separate the mucosal glands(b) H&E x520.
**Fig. (34):** Abomasum showing multiple pin point size parasitic nodules in the fundic region of the abomasum giving its surface a granular appearance (a) which characterized histopathologically by leukocytic infiltration with F.C.T. proliferation in the interstitial tissue with edema in the submucosa (b) H&E x130.
Discussion

Disturbances of the forestomach can result from a variety of disease conditions. The types of disturbances range from simple cases of indigestion to vagus nerve lesions and abomasal disorders. Abomasal diseases of dairy cattle are mostly associated with metabolic disorders, lactational stress, and nutritional disturbances. It is recognized more frequently now than ever before. These diseases include left and right abomasal displacement, volvulus, ulcers and impaction (Geishaufer, 1995; and Radostits et al., 2000).

Ultrasonographic evaluation of the abomasums is a valuable supplementary technique appears to provide a practical, rapid, noninvasive and accurate method for determination of abomasal position, appearance and contents. Any change in these normal parameters indicates that there is any abomasal disorder. The results of ultrasonography that conducted on clinically healthy cows indicate that the abomasum is easily accessible by ultrasonography because it is situated immediately adjacent to the ventral abdominal wall. This is in agreement with Braun et al. (1997) El-Ghoul (2001) Braun (2003) and Wittek et al. (2005).

The abomasal wall in clinically healthy cows could be partially recognized as a distinct echogenic line. It could be attributed to the heterogeneous abomasal contents with echogenic stippling which consisted of
fluid, partially digested food, gas bubbles, small stones and sand, and its thin sub mucosa as mentioned by Braun et al. (1997).

The abomasal folds could be imaged ultrasonographically as a sickle-shaped echogenic structures but they could only be followed for a short distance. This may be because the abomasal folds are undulating and thus not always visible on a two dimensional image and the ultrasound waves were absorbed by the heterogeneous contents of the abomasum before they reached the folds. Similar observation was noticed by Braun et al., (1997) and El-Ghoul (2001).

The abomasum in LDA, partially or completely slides under the rumen and dorsally along the left body wall leading to pulling of the abomasum and cranial duodenum. It results in a partial impairment of abomasal flow leading to abomasal gas accumulation, electrolytes pooling with subsequent systemic alterations and depressed gastrointestinal motility and appetite (Trent, 1990; and Geishauser et al., 1998).

Abomasal displacement cause economic losses due to treatment costs, decreased milk yield, extended interval form calving to conception, loss of body weight, culling and death (Martin et al., 1978; Milian-Suazo et al., 1988; and Grohn et al., 1998)
Discussion

It was postulated by many authors a number of etiologic factors have been implicated for LDA. Such factors that alter the abomasal motility or enhance gas production include dietary modifications with increased concentrates and reduced crude fibers in the immediate pre-or postpartum period. Genetic and mechanical effects may also have a role in the development of the condition (Grymer 1980; Nocek et al., 1983; and Markusfeld, 1986).

Twins may be associated with abomasal displacement. It was recorded in the present study in a case of LDA. This could be attributed as mentioned by Markusfeld (1987) and Constable et al. (1992) to the reduced rumen fill by enlarged uterus and escape of rumen fluid to the abomasum which enhance gas production and allow abomasal displacement..

The pregnancy was a predisposing factor for LDA because of the enlarged uterus was pushing the rumen to the front and the abomasum to the left, at the same time lifting the rumen slightly dorsally and thus enabling the abomasum to insert itself under the rumen. After parturition the pressure reduces and the abomasum especially if it is atonics or distended with gas or feed as the cow feed heavily on grain, become displaced between the rumen and left abdominal wall. (Wallace, 1974; and Constable et al., 1992).

In the present study 80% to 90% of LDA were recorded within one month post-partum (transition period). Similar observation was recorded by
Daniel (1983) and Martens (2000) who mentioned that this period from 2 weeks pre-partum to 2 - 4 weeks post-partum is the major risk period in the etiology of LDA as it is characterized by pre-partum intake depression, followed by increase in postpartum intake, positional changes of the abdominal viscera during late pregnancy or ruminal atony.

LDA occur more commonly in countries where feed consists of high grains and concentrates than roughages (Constable et al., 1992; and Shaver, 1997). Contradictory the incidence of this disease in Egypt and probably most other tropical countries is very low due to higher proportion of roughages than grains in the feed (Singh et al., 1996; and Misk et al., 2003). The present study show a high number of LDA which have diagnosed in private farms supplying high grain feeds than roughages. This result is in agreement with Fouda et al. (2004).

Reduced feed intake associated with the variety of concurrent diseases was noticed prior to LDA in this study. This could be attributed to decreasing rumen fill and reducing the rumen effectiveness as a major barrier against displacement, this enables the abomasums to shift to the left and finally dislocates clinically (Grymer, 1980; and Geishauser, 1995).

The time around calving and early lactation seems to be a period in which cows are highly susceptible to metabolic and reproductive disorders. In the
Discussion

present study about 79.4% of cows diagnosed with LDA have at least one concurrent disease such as milk fever, ketosis, metritis and abomasal ulcers. This was agreed with the result of Klerx and Smolders (1997) Heuer et al. (1999) and Kim and Suh (2003) who attribute this condition to the high energy demand for nutrients, and decreased activity of the immune system in this period.

Ketosis which diagnosed prior to the occurrence of LDA has been implicated as a risk factor for LDA in our study. This could be attributed to low dry matter intake in ketotic cows, which would reduce rumen fill and volume, reducing fore-stomach motility and potentially abomasal motility which offers low resistance to DA (Erb and Grohn, 1988; and Constable et al., 1992).

The other concurrent diseases associated with LDA were metritis, retained placenta, mastitis, abortion, and foot rot. This could be attributed as mentioned by Correa et al. (1993) and Geishauser (1995) to endotoxemia which reduce abomasal emptying and predisposing it to displacement.

The breed predisposition for abomasal displacement in dairy breed cattle has a significant effect. Phenotype may be also have an important role in development of LDA, because large abdominal size has been associated with increased risk of LDA by providing the abomasum more room to displace (Erb and Martin, 1978; and Constable et al., 1992).
Discussion

The risk of developing LDA increased with age up to 7 years with the greatest risk at 2.5 to 5.5 years of age. This result agreed with that recorded by Markusfeild (1986) Constable et al. (1992) and Zadink et al. (2001) who added that the youngest cows had a higher risk of developing LDA, which showed the longest period between the first recognizable signs to the confirmed diagnosis. Also greater exposure to factors that induce abomasal atony.

In the present study the highest number of LDA was recorded in the late autumn and winter seasons. It may be related to factors that induce abomasal atony, seasonal changes in rumen volume in corresponding to seasonal variation in roughage, higher frequency of calving or relative inactivity and low environmental temperatures in winter increase the energy requirement of cows. This attribution was reported by Cameron et al. (1998) Zadink et al. (2001) and Silva et al. (2004).

Diagnosis of LDA was based on simultaneous auscultation and percussion of tympanic area in the left abdominal wall centered on the 9th -13th rib (Smith et al., 1982). Also, the presence of partial anorexia, decreased fecal volume, decreased milk production and the presence of characteristic serum electrolytes alteration as hypochloraemic metabolic alkalosis with variable degrees of hypokalaemia and hyponatremia as reported by Trent (1990) and Delgado-Lecaroz et al. (2000).
The ping sound could be heard by simultaneous auscultation and percussion at the left abdominal wall in case of LDA, may be resulted from gas accumulation in the displaced abomasum, as described by Smith et al. (1982) and Radostits et al. (2000). However, it may heard in many other disease conditions as traumatic reticulitis, hydro-allantois, frothy tympany and diaphragmatic hernia, so exploratory laparorumenotomy makes it possible to diagnose LDA accurately in doubtful cases which has been recommended as a last diagnostic resort (Trent, 1990; Singh et al., 1996; and Misk et al., 2003).

Our results revealed a hypochloremia accompanied the cases suffered from LDA. This could be attributed to continuous secretion of chloride ions into the abomasum which was refluxed through omasal canal due to pyloric stenosis or obstruction and consequently returned to rumen (Geishauser and Sech, 1996; and Rohn et al., 2004).

Hypokalaemia in our study was explained by anorexia associated with the displacement or due to potassium shift into the cell during alkalosis to permit hydrogen exchange and its continued loss by the kidney, or may explained by shift in potassium ion from the extracellular to the intracellular fluid spaces or to the intestinal or bone system, as a result of starvation or impaired renal function as mentioned by Hafez and Mottelib (1994) Delgado- Lecaroz et al. (2000) and Fouda et al. (2004).
In this study the decreased sodium ions in the blood serum of diseased cows with LDA may be due to its reduced absorption from the gastrointestinal tract with continuous secretion of isotonic fluid into the abomasum or due to the renal losses as reported by Svendsen (1969) Whitlock (1976) and Dirksen (1983).

Dehydration in affected cows with LDA in comparison to control group was a fact which reflected in an elevated total protein, packed cell volume and blood urea. This observation could be explained by withdrawal of circulating fluid from the vascular system into the high osmotic pressure static gastrointestinal tract. These results were in parallel with those reported by Dirksen (1983) and Rehun (1995).

Increased values of blood urea nitrogen were observed in diseased cows with LDA. It was usually a result of dehydration and reduced renal tissue perfusion with blood because of haemocentration with consequent reduced glomerular filtration and eventual accumulation of by-products normally excreted through the kidneys (El-Gharieb et al., 1996; and Geishauser et al., 2000).

Hypoglycemia was evident in diseased cases with LDA in this study. It was often related to the severity of ketosis and moderate starvation with consequent reduction of absorption from the gastrointestinal tract because of
Discussion

reduced motility resulted from secondary hypocalcaemia in these conditions as mentioned by Massey et al. (1993) and Rohn et al. (2004). However, these results were disagreed with the findings of Meirhaeghe et al. (1988) who mentioned that, high basal blood glucose level was associated with abomasal displacement.

Hypocalcaemia was observed in LDA cases in the present study. These results were in concern with that obtained by Daniel (1983) Massey et al. (1993) Shaver (1997) Rohrbach et al. (1999) and Delgado-Lecaroz et al. (2000) who mentioned that the inappetence developed with abomasal displacement and reduced calcium absorption from the gut causing abomasal atony.

Our study revealed that the activity of alkaline phosphatase and AST were greatly increased with higher values of bilirubin and albumen in diseased cows with LDA, especially in the first two weeks postpartum. This could be attributed to disturbance in liver function or a result from protein mobilization from muscles in order to deliver glycogenic amino acids as glucose precursors (El-Gharieb et al., 1996; Itohn et al., 1998; Herdt, 2000; and Rohn et al., 2004).

In the present study 2 cows diagnosed with LDA, had a significant hypochloremia and hypocalcaemia which could not with stand the risk of surgical interference for LDA correction. This result was in concern with Massey et al. (1993) Singh et al. (1996) Delgado-Lecaroz et al. (2000) and
Discussion

Zadnik et al. (2001) who reported that animals having serum chloride level equal to or below 79µEq/L, hypocalcaemia below 4mg/dl and heart rate equal to or greater than 100/min have a poor prognosis.

The choice of either non absorbable or absorbable suture material was probably had no great importance as long as the material retains tension for long enough sufficient for adhesions to be formed. This is in agreement with Turner and Mellwraith (1989) who mentioned that the use of both non absorbable suture materials (silk and hernial tape) and absorbable suture materials (polygalactin 910 No.3) were related to the type of pexy. So the hernial tape was used in omentoexy to avoid the tearing while the silk or vicryl were used for abomasopexy.

Left flank omentopexy was used for treatment of LDA with reported success rate 94.1%. The abomasum was anchored to the ventral body wall by fixation of the greater omentum resulting in formation of adhesions between the omentum and peritoneum. No signs of fistulation or suture sinuses were noticed. Nearly similar observation was reported by Steanhaut et al. (1974) Smith (1981) and Trent (1990).

The results of the present study indicated that left flank abomasopexy was successfully used for correction of LDA cases with reported success rate 86.6%. It allow good exposure by palpation of the LDA with variable amount of greater omentum visible to the incision, minimal stress on the animal, minimal restraint
requirement and more familiar orientation of the viscera during exploration and offers direct fixation of abomasum to the ventral body wall. This was in agreement with Baker (1973) Turner and Mellwraith (1989) and Singn et al. (1996). While 2 cows with LDA have concurrent perforating abomasal ulceration with severe adhesion of the greater curvature with the surrounding tissues, have a poor chance for survival and were sold for slaughtering. This findings were compatible with that reported by Cable et al. (1998).

The success rate after LDA surgery in our study were 84.4% of the operated animals which were remained productive. Two cows showed abomasal adhesion with peritoneum due to perforating abomasal ulcerations. Also, 3 cows were culled from the herd as they not return to their normal productivity. This is in agreement with Steanhaut et al. (1974) Trent (1990) and Hirvonen and Pyorala (1998).

Reduction of the incidence of LDA in a dairy herd can be achieved by optimal nutrition and management during the dry period. Avoid a negative energy balance prepartum by avoiding over conditioning, and by providing optimal feed to cows in late gestation. Ensuring adequate intake of a high fiber diet to dairy cows during late pregnancy will help to reduce the incidence of LDA. (Constable et al., 1992; El-Gharieb et al., 1996; and Cameron et al., 1998).

In this study AV with omasal involvement was diagnosed in a Holstein cow with a history of anorexia, sharp drop in milk production, dehydration, tachycardia
and a significant hypochloraemic, hypocalcemic metabolic alkalosis associated with hypocalcaemia. The affected cow could not withstand the risk of surgical interference. Slaughtering was the option in such case due to poor prognostic indicators. This was in agreement with that recorded by Garry et al. (1988) Constable et al. (1991) and Delgado-Lecaroz et al. (2000).

Necropsy findings of AV were diagnostic in which the abomasum was distended with a large volume of brownish, sanguineous fluid. Its wall was grossly hemorrhagic, edematous, necrotic and gangrenous with congested omasal mucosa. Nearly similar observations were reported by Habel and Smith (1981) Hoffsis and McGurik (1986) and Trent (1990).

The onset of abomasal impaction in cattle was gradual. The body condition was poor, anorexia was present and the rumen motility and quantity of faces were decreased. Abomasal contents were generally firm and the rumen usually distended with fluid contents. Similar findings were reported by Baker (1979) Breukink and Kuiper (1980) Cebra et al. (1996) and Misk et al. (2003).

The presence of chopped roughage, finely crushed grains and sands in combination with excessive intake and low digestability leads to excessive accumulation in the forestomach and abomasum which enhance abomasal atony and primary impaction. Secondary abomasal impaction usually associated with traumatic reticuloperitonitis or diaphragmatic hernia (Mitchell, 1991; Ibrahim, 1991; and Misk et al., 2003).
Abomasal impaction is usually difficult to be established clinically in large sized cattle, but a history of gradual abdominal distension, loss of appetite, reduced fecal output, failure of traditional treatment and postmortem findings were diagnostic. However, the ultimate diagnosis for abomasal impaction was made via the exploratory laparo-rumenotomy. Similar remarks were mentioned by Baker (1979) Trent (1990) and Misk et al. (2003).

Treatment of abomasal impaction depends on the severity and chronicity of the disease and value of the animal. It includes; slaughter, medical therapy, rumenotomy and abomasotomy. Medical trials for treatment of abomasal impaction are usually unsatisfactory, but correction of electrolytes and acid base imbalance is so necessary and will be helpful. Also, supportive therapy before surgery such as parenteral calcium solution and electrolytes are indicates in this condition (Rebhun et al., 1988; Radostitis et al., 2000). In this study abomasal impaction was surgically corrected only in one case through emptying the abomasum by laparo-rumenotomy with direct administration of liquid paraffin into the omasum as described by Blikslager et al. (1993) Simkins and Nagele (1997) and Fouda et al. (2004).

Necropsy findings of abomasal impaction were diagnostic in which the impacted abomasum was enlarged and dilated with thin wall. The length reached up to 90 cm and the circumference was more than 70 cm. The contents appeared dry and homogenous in consistency taking the abomasal shape. Similar
Abomasal ulceration occurs commonly in cattle with particular concern in animals under intensive production or management stress especially veal calves, yearling feedlot steer and high producing dairy cows. It can represent an added therapeutic challenge when present with other surgically manageable conditions. Similar remarks were reported by Pearson et al. (1987) Rebhun (1995) and Ahmed (2001).

The pathogenesis of abomasal ulceration is a combination of several factors: decreased mucosal protection mechanisms, increased peptic secretions, altered gastrointestinal tract motility, nutritional deficiencies, gastritis and infections (Pearson et al., 1987). Lesion formation is expanded by corrosive factors such as acid, pepsin, bile salts and change in mucosal blood flow caused by stress (Livingston et al., 1991). Two possible mechanisms occur at the onset of ulcer formation, focal ischemia with infection or surface cell necrosis induced by acid when mucosal blood flow is reduced (Rebhun, 1995).

Diagnosis of abomasal ulceration was difficult clinically even with presence of some clinical signs as they were variable according to the type, site, number, extend or the cause of the ulcers. Definitive diagnosis was generally based on observation during surgery, necropsy or at inspection of slaughtered cattle. This is in agreement with Smith et al. (1983) who mentioned that, the
prevalence of abomasal ulcers in clinical cases was 2.17%, however, at abattoir the prevalence may reach 6%.

In the present study there was increase in incidence of abomasal erosions and ulcerations in male (88%) than female (12%) cattle. This high incidence may be attributed to the great number of male at risk of heavy grain feeding for fattening. This was agreed with Pearson et al. (1987) and Ibrahim (1991).

The gross and histopathological appearance of different forms, types, and numbers of ulcers seen in different areas of the abomasal mucosa were similar to that reported by Smith et al. (1986) Braun et al. (1991) Ibrahim (1991) and Misk et al. (2003).

Linear ulcers were mostly seen in the fundus specially on fold edges. This concise with the result of Jensen et al. (1992) and Misk et al. (2003) who mentioned that possible mechanical abrasions occur because of folds edges project into the lumen where digesta flow rate is high and contact with moving particles of feed and geoparticles is possible. However, round ulcers were the most common type seen in the pyloric part of the abomasum. This result is in agreement with Braun et al. (1991) who said that partial blockage of the pyloric exit delaying abomasal emptying and rendering the mucosa susceptible to ulceration.
Discussion

Irregular ulcers were seen in both fundic and pyloric mucosa with high occurrence in fundic one. Most ulcers were seen as focal bleeding ulcers which cause chronic melina, however, many ulcers were seen as non bleeding or healed and were difficult to be determined at necropsy. Similar findings were reported by Braun et al. (1991) and Misk et al. (2003).

The postmortem examinations and histopathological alterations of abomasitis were nearly similar to that reported by Roeder et al. (1988) Ibrahim (1991) Hosein et al. (1995) and Songer et al. (2005) who said that abomasitis is not concerned with certain age in cattle and usually co-existed with many diseases and stress conditions. It frequently observed at necropsy or at inspection of slaughtered cattle, while its diagnosis on clinical basis is still obscure.
Summary and Conclusion

The present study was conducted on a total number of 2180 cattle, from those 173 cattle (56 Holstein, 59 mixed breed and 58 native cattle) aged from 6 months to 10 years and of both sexes, and suffering from one or more abomasal disorders (LDA, AV, impaction, ulcer, abomasitis and worm infestation). The affected animals were belonged to a private animal farm station at Dakahlia Province (34 cattle), and cases that admitted to the Mansoura Veterinary Teaching Hospital (11 cattle). In addition to the cases surveyed at Mansoura abattoir (128 cattle). These animals were subjected to well informed case history, clinical examinations as well as histopathological examinations for some cases of abomasal disorders. Also, ten clinically normal dairy cows were selected and served as control groups for laboratory examination and five ones were subjected to ultrasonographic examination. The later was performed at Surgery Dept. Fac. Vet. Med. Benha University.

Ultrasonographic examinations revealed that the bulk of the abomasum is situated to the right ventral midline at the ventral abdominal wall. The different layers of the ventral abdominal wall were appeared as narrow bands of varying echogenisity. The wall of the abomasum appears as a thin echogenic line. However, the abomasum contents were seen as a heterogenous moderately echogenic mass with echogenic stippling. Parts of the abomasal folds can
occasionally be seen as a sickle-shaped echogenic structures within the content of the abomasum. Passive and slow movement of the abomasal contents is frequently seen.

LDA were diagnosed clinically and confirmed by laboratory tests in 34 Holstein cows with a history of reduced milk yield, varying degree of reduced appetite, scanty feces with evidence of tympanitic resonance on left side of the abdomen. The peak of disease occurrence was seen in the late autumn and winter. From these animals 2 cows had a significant hypochloremia and hypocalcaemia which can not with stand the risk of surgical interference and sold for slaughtering, and the other 32 cows were subjected to left flank omentopexy and left flank abomasopexy. Satisfactory results with complete recovery were obtained in 27 cows with success rate 84.4% after the third follow up (60 days). While 2 cows during laparotomy showed severe adhesions between the abomasum and peritoneum secondary to a perforated abomasal ulceration and sold for slaughtering. Also 3 cows were culled because of these cows not reach the normal level of productivity.

AV was diagnosed in a Holstein cow with a history of complete anorexia, sharp drop in milk production, tachycardia, marked depression and dehydration, and were sold for slaughtering. The necropsy findings showed a distended abomasum with large volume of fluid and hemorrhagic, edematous, necrotic and gangrenous abomasal wall with omasal involvement.
Summary and conclusion

Abomasal impaction was diagnosed in 2 Holstein cows and 2 mixed breed cows with a history of anorexia, rumen stasis, varied degrees of dehydration with abdominal over distention. These animals were showed no response to the traditional medical treatment, and consequently subjected to laparo-rumenotomy in combination with medical treatment by direct administration of liquid paraffin inside the omasum through a tube placed into the reticulo-omasal orifice. Good result was obtained only in a cow while the other 3 cases which not respond to the treatment were sold for slaughtering. After slaughtering, the impacted abomasum appeared as a large bag with increased dimensions over packed by hard ingesta taking its shape.

Abomasal ulcerations were diagnosed in 35 cattle {10 cases (7 calves and 3 dairy cows) were clinically diagnosed and 25 cases (1 calf, 22 feedlot steer and 2 dairy cattle) were diagnosed by necropsy examinations}. The clinically affected animals showed abdominal pain manifested by arched back and girding on teeth with foamy salivation, sudden onset of anorexia, excessive water intake, decreased milk production, melina (scanty black and tarry feced), weakness and anaemia manifested by pale mucous membranes and dehydration. Different types, numbers, locations and sizes of abomasal erosions and ulcers were differentiated during the abattoir necropsy examinations. According to their shape, three types of ulcer were differentiated: Linear ulcers (36%) were mostly seen in the fundic folds, circular ulcers (44%) were seen mostly at the pyloric region and irregular ulcers (20%) were seen in both fundic and pyloric regions.
Summary and conclusion

The frequency of abomasal ulcers per examined abomasa were ranged from 1-20 lesions.

Different forms of abomasitis were recorded in 40 cattle appeared either hemorrhagic, necrotizing or edematous. In addition to 59 animals with parasitic abomasitis in which the abomasum showed multiple small yellowish-red bite marks (parasitic nodules) distributed all over the abomasum especially on the body give its surface a granular appearance.

From proceeding results it could be concluded that:

Ultrasonographic appearance of the normal abomasum provides a practical, rapid, non invasive and accurate basis for the prediction of the abomasal position and its differentiation from the neighbouring organs.

LDA was commonly recorded in Holstein cows aged from 2.5-5.5 years and subjected to the stress of high intense production. Also, feeding and management practices that prevent other post partum disorders reduce the risk of LDA and so reduce its economic losses from milk, culling or death of the affected animals.

The establishment of the invasive and noninvasive methods in combination with laboratory investigatin provides good diagnostic data for surgical interference of LDA and its differentiation from other digestive disorders of the same medical history.
Summary and conclusion

Tachycardia, marked depression and significant hypochloremic, hypokalemic metabolic alkalosis associated with hypocalcaemia were bad prognostic indicators for surgical interference.

Left flank omentopexy and left flank abomasopexy could be used with high success rate for the treatment of LDA, with special preference for left flank omentopexy.

Surgical intervention coupled with medical treatment of early cases of abomasal impaction seemed to be helpful in prognosis and recovery of these cases.

The necropsy and histopathological examinations are essential diagnostic tools in the recognition of different types of abomasal ulcers and abomasitis, and in confirmation the diagnosis of AV and abomasal impaction. Abomasal ulcers and impaction mainly related to the feed steer animals, thus it is mainly a feeding problem, so correction of ration should be considered for avoidance of this problem in these animal groups.

The economic effect of abomasal disorders is an important factor in cattle milk and meat manufacture, as they resulting in physical discomfort, reduce feed consumption, weight gain and milk production.
References


Reference


Reference


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Nocek, J.E.; English, J.E; and Braund, D.G. (1983): Effects of various forage feeding programs during dry period on body condition and subsequent lactation,


Reference


المختص العربي

59 وال - مِنْ ذِكْرِ (حَسَنَةٍ) ۚ ذُکْرُ اَنْفَسِي ۖ وَلَا تَكُونِي ضَحَياً
ۚ بِذَٰلِكَ نُفِرْنَآ ۖ فَأَنتُمْ لَا تَكُونِ عَلَيْنَا حَارِيًّا
ۚ إِنَّا نَجْعَلُكُمْ مَرْجَعًا فِي الْأُمُورِ ۖ وَلَا تَكُونُوا
ۚ فِي هَٰذِهِ قَرْنَآءٍ يَوْمًا وَرَبَّيْنَ ۖ وَلَا يَكُونُ
ۚ وَكَيْفَ مَعْلَمُ الْقُرْآنِ ۖ وَلَا يَكُونُ تِلْكَ مَا ذَهَبَذُ
ۚ إِنَّا نَجْعَلُكُمْ مَرْجَعًا فِي الْأُمُورِ ۖ وَلَا تَكُونُوا
ۚ فِي هَٰذِهِ قَرْنَآءٍ يَوْمًا وَرَبَّيْنَ ۖ وَلَا يَكُونُ
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المدخل العربي

- يَكَّ رَيْيَتَ لِلرَّجُلِينَ فَلَيْنَكَ إِنْ تُرَيْتَ بَيْنَهُمْ سَيَّ وَاللَّهُ رَبِّيَّاهُمْ. يَلَوَّنْ ذَلِكَ لِلرَّجُلِينَ ذَلِكَ حَيَّ وَاللَّهُ رَبِّيَّاهُمْ.

- يَلَوَّنْ ذَلِكَ لِلرَّجُلِينَ ذَلِكَ حَيَّ وَاللَّهُ رَبِّيَّاهُمْ.
الملخص العربي

> يُعتقد أن تُوجِّه المُؤسسات للمُستخدمين لاستبدال المنتجات القديمة بحلول 2024. يُشيرٌ التقرير إلى أن نسبة المستخدمين الذين يفضلون استخدام المنتجات الجديدة تؤخذ في الاعتبار. مُستهلكون يواجهون تحديات في الوصول إلى المنتجات الجديدة. ومع ذلك، فإن العديد من المستهلكين يختارون استخدام المنتجات القديمة بسبب التكلفة أو الافتراضات الخاصة بهم.

: المصدرين

: المُؤسسات

: المستهلكون

: تحديات

: التكلفة
الملخص العربي

"الجيم" و"الفا" أَوَّلَتْ لَهَا نُفَاعًا، إِنَّهَا أَقْبَالُ لِلنَّاسِ، وَتَأْتِي بِنَفْعٍ مَّتَاعًا.

"اليوم" تَأْتِي بِنَفْعٍ مَّتَاعًا وَأَقْبَالُ لِلنَّاسِ. وَتَأْتِي بِنَفْعٍ مَّتَاعًا وَأَقْبَالُ لِلنَّاسِ.
السادة المشرفين و مساعديهم

عنوان الرسالة: التشخيص والتعامل لاضطرابات المعي الرابع في الماشية
اسم الباحث: طبر السيد احمد عوض الشافعي

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<td>طه عبد المنعم فودة</td>
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وكيل الكلية لشؤون الدراسات العليا والبحث

عميد الكلية

السيد الشريف السعيد

1. عادل التابعي زغلول
2. محمد محمد فودة
السادة أعضاء الحكم والمناقشة

عنوان الرسالة: التشخيص والتعامل لاضطرابات المعي الرابع في الماشية

اسم الباحث: طرابي أحمد عوض الشافعي

الإشراف:

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لجنة الحكم والمناقشة:

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على أن يكون الثالث و الرابع معا بصوت واحد.

رئيس القسم

وكيل الكلية لشئون الدراسات العليا والبحوث

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أ.د. عادل التابع إبراهيم زغلول

أ.د. السيد الشرياني السعيد
التشخيص والتعامل لاضطرابات المعي الرابع في الماشية

 رسالة مقدمة من

 ط. ب. السيد أحمد عوض الشافعي

 تحت إشراف

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 أستاذ مساعد الجراحة والتخدير والأشعة
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 أستاذ الأمراض الباطنة
 كلية الطب البيطري جامعة المنصورة

 2007

 في العلوم البيطرية
 (الجراحة البيطري)

 (2007)