

**CLINICAL, HEMATO-BIOCHEMICAL AND ULTRASONOGRAPHIC STUDY
IN ABOMASAL DISPLACEMENT IN COWS WITH TRIALS OF TREATMENT**

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ABSTRACT

This study was conducted on twelve Holstein dairy cows in a private farm at El-Khatatba, Minufya Governorate. Five cows were clinically healthy and considered as control. The other seven cows showed signs of abomasal displacement that involve anorexia, sudden drop of milk yield, scanty pasty faeces, colic and ketotic odour of the breath. By thorough clinical examination, four cows were affected with left displacement of abomasums (LDA) and three cows with right displacement of abomasum (RDA). The characteristic tinkling sounds were heard on simultaneous percussion and auscultation of the dorsal flank, especially on the cranial third of paralumbar fossa in LDA and between 9th and 12th ribs on the right dorso-lateral aspect of the body in RDA. All these signs were recorded 3-6 weeks after calving. Hematological examinations revealed a significant increase of PCV% and non-significant changes in RBCs, Hb concentration and WBCs. Serum biochemical analysis showed a significant decrease of serum sodium, potassium, chloride, calcium and glucose, while a significant increase in serum total proteins, ALT, AST, LDH, CPK, urea and creatinine was recorded. Ultrasonographic examination of abomasum showed a narrow echogenic line representing abomasal wall. Parts of abomasal folds were occasionally visible as elongated, echogenic and sickle-shaped structures. The dorsal abomasal cap was characterized by reverberation artifacts. The ingesta were visualized ventrally and appeared echogenic to hypoechogenic. In cows with LDA, the rumen was displaced by the abomasum more dorsally and the abomasum was visualized between the left abdominal wall and the rumen. While in cows with RDA, the liver was displaced medially from the right abdominal wall. Trials for medical treatment were successful in three cows only whereas, the other four cows were irresponsive to therapy and were salvaged. Therapeutic response was correlated to early diagnosis.

INTRODUCTION

Abomasum of adult dairy cow is a sac-like elongated organ lying on the lower right quadrant of the abdominal cavity extending from its abomasal attachment to the area of the eleventh or tenth rib where it is continued by the ascending duodenum (**Murray et al., 1991**). Abomasal displacement in cattle is found throughout the world. It is seen most commonly with increasing tendency in regions with intensive cattle breeding and husbandry (**Jubb et al., 1991, Pehrson and Shaver, 1992 and El-Gharieb et al., 1996**).

Abomasal diseases of dairy cattle are mostly associated with stress of situations, nutritional disorders and metabolic disturbances. These diseases include LDA, RDA, abomasal volvulus (AV), abomasal ulcer and impaction (**Schuch, 1993**). Abomasal displacement is one of the several conditions which causes post-calving digestive upset and may lead to confuse diagnosis of digestive and metabolic diseases in cattle (**Constable et al., 1992 b**).

A number of factors have been implicated in the etiology of abomasal displacement. The primary causative factor in abomasal displacement appears to be atony or hypotony of the abomasums (**Breukink and de Ruyter, 1976**). Dairy cattle with high milk production and being fed large quantities of grain, where exercise is limited may have abomasal atony. Other contributing factors decreased abomasal motility include metabolic disorders (hypocalcemia and ketosis), concurrent diseases as mastitis and metritis, changes of intra-abdominal organs especially in late pregnancy and genetic predisposition (**Robertson, 1968; Delgado-Lecaroz et al., 2000 and Radostits et al., 2000**). Sudden change in the feed before calving, in preparation for lactation or as a result of ownership promotes abomasal displacement and induces improper adjustment of flora and fauna (**Driskin et al., 1992**). **Geishauser (1995)** mentioned that there were loss of cholinergic excitatory and an increase of nitroergic inhibitory tone. He also suggested that this might cause decreased contractility of the antral muscles. **Sack (1968)** stated that a sudden onset of ruminal stasis, lack of appetite and fall in milk yield was also characteristic of acute intestinal obstruction, simple indigestion and abomasal displacement. **Constable et al. (1992 a)** mentioned that, the LDA was more frequent than

RDA in cows. They also added that ketosis was recorded in concurrent with LDA than RDA.

LDA is characterized by partial or complete displacement of the dilated abomasum between the rumen and left abdominal wall (**Dirksen, 1962**). Also, there was a high incidence in the period from 3-6 weeks after calving (**Constable et al., 1991**). In addition to this, there was a high incidence of LDA in winter due to higher frequency of calving and inactivity (**Martin, 1972**).

Clinical findings of abomasal displacement included inappetance, sometimes complete anorexia, marked drop of milk yield, varying degrees of ketosis and tinkling sounds on auscultation (**Radostits et al., 2000**). Abomasal displacement is characterized by dehydration and metabolic alkalosis due to continuous secretion of hydrochloric acid, sodium, potassium and chloride into the abomasum which becomes gradually distended and does not evacuate its contents into the duodenum (**Muyllé et al., 1990**).

Serum biochemical changes associated abomasal displacement included severe decrease in serum chloride, sodium, potassium, calcium and glucose. Also, there was significant increase in serum total proteins, urea, lactate dehydrogenase, aspartate aminotransferase and alanine aminotransferase (**Sevendesen, 1969; O'zkank and Poulsen, 1986 and El-Gharieb et al., 1996**).

By ultrasonography, the abomasum wall can be visualized as a narrow echogenic line. Parts of abomasal folds are occasionally visible as elongated echogenic sickle-shaped structures. The ingesta are visualized ventrally and appeared echogenic to hypoechogenic (**Braun et al., 1997a**). In cows with LDA, the rumen was displaced by the abomasum more dorsally and the abomasum is seen between the left abdominal wall and the rumen (**Braun et al., 1997b and Braun, 2003**). In RDA, the liver was displaced medially from the right abdominal wall (**Braun, 2003**).

The First step in the treatment of abomasal displacement depends on the correction of complication of ketosis and hypocalcaemia by intravenous administration of glucose 25% and calcium (**Hungerford, 1990**). Moreover, intravenous injection of isotonic saline (0.85%) and isotonic potassium chloride (1.1%) are indicated to overcome

metabolic alkalosis (**McGuirk and Butler, 1980**). Mechanical reposition of the displaced abomasum may also be successful (**Schuh, 1993**).

This work was aimed to study different methods diagnosis of abomasal displacement in cows including clinical, haemato-biochemical and ultrasonographic examinations. Also, there were trials of treatment.

MATERIALS AND METHODS

Animals

A total number of 12 Holstein dairy cows were used in the present study. The examined animals were allocated into 2 groups. Group I encompassed 5 clinically healthy cows and considered a control. Group II included 7 cows with clinical findings of abomasal displacement and subdivided into two sub-groups. Sub-group (A) involved 4 cows with LDA and sub-group (B) involved 3 cows with RDA.

Clinical Examination

Clinical examinations include inspection and recording of respiratory and pulse rates and body temperature. In addition, percussion and auscultation at the left or right rib cage were conducted (**Kelly, 1984**).

Collection of Abomasal Fluid for pH Determination

Paracentesis of the displaced abomasum as well as the normal abomasum was carried out using a 16-gauge needle to determine pH of the aspirated fluid (**Schuch, 1993**).

Urinalysis

Urine samples from all examined cows were collected for qualitative determination of pH, protein, glucose, ketone bodies, bilirubin in addition to blood and hemoglobin by comber test strips supplied by Boehringer Mannheim, Germany.

Hemato-biochemical Analysis

Two blood samples were collected from jugular vein of examined animals. The first sample was heparinized blood for determination of RBCs, Hb, PCV and WBCs

according to **Sastry (1985)**. The second sample was collected without anticoagulant, left to clot and non-hemolysed serum was obtained. Commercially available diagnostic kits were used for colorimetric determination of serum calcium (**Glinder and King, 1972**), sodium and potassium (**Henry et al., 1974**), chloride (**Feldkemp, 1979**), glucose (**Lott, 1975**), total proteins (**Doumas et al., 1981**), urea (**Faweet and Scott, 1960**), creatinine (**Young, 1990**), AST and ALT (**Reitman and Frankel, 1957**), LDH (**Tietz, 1986**) and CPK (**Rec, 1977**).

Ultrasonographic Examination of Abomasum

Sonographic examination was performed by the method described by **Braun et al. (1997a)**, using a 3.5 MHZ linear transducer and real-time scanner. Briefly, the examined region included the area at approximately 10 cm caudal to the xiphoid process. The cow was examined along the ventral midline and in the left and right paramedian regions. In animals where LDA is suspected, the transducer was placed on the dorsal region of the left 12th intercostal space. In animals where the RDA is suspected, the areas caudal to the last rib and the caudal two to three intercostal spaces on the right side were examined dorsoventrally with the transducer held parallel to the intercostal space (**Braun, 1997**).

Trials for Treatment

Treatment trials included intravenous administration of glucose 25% (El-Nasr Co., Cairo, Egypt) at a dose rate of 10 ml/kg B.Wt and calcium therapy (Cal-D-Mag (Pfizer) 1 liter / head (**Hungerford, 1990**). In addition, 2 liters of isotonic saline (0.85%), 1 liter of isotonic potassium chloride (1.1%) (McGuirk and Butler, 1980), and Neostigmine (1 mg/50kg) (Ameriya Pharmaceutical Industries, Cairo, Egypt) (**Radostits et al., 2000**) were intravenously injected.

On the other hand, there was a trial for reposition of displaced abomasum by casting the cow on the back followed by rolling to the right or left side according to the type of displacement and then stopping abruptly in the hope that abomasum will free itself (**Schuch, 1993**).

Statistical Analysis

All results were described as means \pm standard error (SE). All pairwise comparison of mean values of diseased cattle to those of control was analyzed using one-way analysis of variance (ANOVA) according to **Gelfert et al. (2006)**. Values were significantly different from control at $P < 0.05$ or $P < 0.01$.

RESULTS AND DISCUSSION

Abomasal displacement is among the most commonly encountered diseases in dairy veterinary practice (**Delgado-Lecaroz et al., 2000**). Cases with abomasal displacement were recorded within the period from 3-6 weeks after parturition, which was similar to that recorded by **Constable et al. (1991)**. Diseased cows were afebrile with normal pulse and respiratory rates. Ruminal sounds were reduced (Table 1). This result was agreeable to the result recorded by **El-Gharieb et al. (1996)**. Clinical examination of diseased cows indicated dullness, poor appetite, sudden drop of milk yield, ketotic breath, scanty pasty faeces, reduction of rumination and distention of the left or right side of the abdomen according the type of displacement. Also, high pitched resonant pings were audible on simultaneous percussion and auscultation of the dorsal flank, especially in the cranial third of the paralumbar fossa in cases with LDA. While in the right abomasal displacement, this diagnostic ping sound was heard on the right dorsolateral aspect of the body. On rectal palpation, the rumen was displaced medial than normal in left abomasal displacement, while in right displacement, the distended abomasum was palpated rectally. All these clinical findings were nearly similar to those recorded by **Buchanan et al. (1991)**, **Jubb et al. (1991)** and **Radostits et al. (2000)**.

Urine examination indicated ketonuria and aciduria (Table 2). This result was in accordance with the result of **El-Gharieb et al. (1996)**. Aciduria could be attributed to excretion of hydrogen ions and retention of sodium in the face of hypokalemia (**Anderson, 1980**). The ketonuria occurred in abomasal displacement could be primary and caused by the hypoglycemia that occurs peripartum and considered as a risk factor for abomasal displacement (**Rohrbach et al., 1999**) or may be secondary to abomasal displacement (**Itoh et al., 1998**).

Measuring abomasal fluid pH (Table 2) showed a significant increase in pH value in cases of LDA and RDA compared to control. The elevation of abomasal fluid pH could be attributed to that when fluid of rumen is transported to the abomasum and not mixed with acid of the abomasal contents due to decreased motility of the abomasum, the pH will rise. The increased pH of abomasum may enable the microbes in the fluid to produce methane and carbone dioxide, which are the main component of gas in a displaced abomasum (Svendsen, 1969). The rise in pH of abomasal contents can play an important role in the etiology of abomasal displacement in dairy cows due to continued fermentation with a poor motility of abomasum (Van Winden et al., 2002).

Blood picture in cows with abomasal displacement (Table 3) indicated a significant increase of PCV%, while non-significant changes of RBCs, Hb concentration and WBCs were recorded. This result coincided with those of El-Gharibe et al. (1996). The increase in PCV% is usually related to haemoconcentration and dehydration (Jubb et al., 1991 and Rohn et al., 2004).

Serum biochemical changes in cows with abomasal displacement (Tables 4 & 5) revealed significant decrease of serum sodium, potassium, chloride, calcium and glucose, while significant increase in serum total proteins, urea, creatinine, lactic dehydrogenase, creatine phosphokinase, alanine aminotransferase and aspartate aminotransferase (AST) was recorded. These results were agreeable with those of O'Zkank and Poulsen (1986), El-Gharieb et al. (1996) and Zadnik (2003). Metabolic alkalosis with hypochloremia and hypokalemia associated with abomasal displacement could be attributed to abomasal atony, continued secretion of hydrochloric acid into the abomasum and impairment of flow into the duodenum (Svendsen, 1969). The increased total serum proteins might be attributed to the hemoconcentration accompanied the displacement of abomasum. There was a significant increase of serum ALT and AST. This result could be attributed to disturbance in liver function (Voros and Karsai, 1987 and Lenz, 1993). Additionally, there was a significant increase in the serum enzymatic activity of lactate dehydrogenase (LDH) and creatinine phosphokinase (CPK) in LDA and RDA compared to control (Table 5). These results were in accordance to those obtained by El-Gharieb et al. (1996). The significant increases of the activities of these enzymes might be attributed to

affection of liver (LDH), heart and skeletal muscles (CPK) associated with the muscular exertion and excitement occurring during displacement. The significant increase in serum urea and creatinine usually regarded to dehydration and decreased renal blood flow (renal ischemia) (**Anderson, 1980**).

The ultrasonographic appearance of abomasum indicated that in control animals (Figure 1 and 2), the abomasal wall appeared as a narrow echogenic line following the abdominal wall and muscles. Parts of abomasal folds were visible as elongated, echogenic sickle-shaped structures. The dorsal abomasal cap was characterized by reverberation artifacts. The ingesta were visualized ventrally and appeared echogenic to hypoechogenic. This appearance was similar to that observed by **Braun et al. (1997a)**. In cows with LDA, the rumen was displaced by the abomasum more dorsally and the abomasum was seen between the left abdominal wall and the rumen (Figure 3). The abomasal gas cap is not visible because of the reverberation artifacts at the abomasal surface (Figure 4 and 5). This ultrasonographic appearance was similar to that detected by **Braun (1997 and 2003)**. These artifacts are caused by reflection of the ultrasound waves by abomasal gases and reverberation between the transducer and the abomasal surface (**Braun, 2003**). In the RDA, (Fig. 6) the liver that appears hypoechogenic was displaced medially from the right abdominal wall by the displaced abomasum with hypoechogenic ingesta. This observation coincided with that of **Braun (2003)**.

With regard to the treatment, three cows were responded to medical therapy of calcium, glucose, isotonic solutions and neostigmine and the manual reposition. The other four cows did not respond to medical therapy or manual correction and were salvaged, probable because reaching correct diagnosis in these four cows was delayed and therefore they were in advanced stage of the disease at the time of diagnosis. Consequently, this result imply that good prognosis of abomasal displacement is better attained with early diagnosis of affected cows (**El-Gharieb et al. 1996**).

In a conclusion, abomasal displacement was diagnosed in 7 dairy cows 3-6 weeks after parturition. It was associated with reduction in milk yield, anorexia, abdominal distention, scanty pasty faeces and ketotic breath. Affected cows had a higher PCV indicating haemoconcentration, while ketonuria and aciduria indicating ketosis.

Moreover, affected cows had hyponatremia, hypocalcemia, hypochloremia and hypoglycemia. Additionally, serum biochemical changes involved elevated AST, ALT LDH, CPK, urea and creatinine, suggesting liver or kidney dysfunction. The occurrence of displacement and its type was confirmed by ultrasonographic examination of displaced abomasum, suggesting that ultrasonography is a useful aid in diagnosis of abomasal displacement and determination of its type.

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Table (1): Clinical picture of apparently healthy cows and those with LDA and RDA.

Parameters \ Groups	Control	LDA	RDA
Body temperature °C	37.8 ± 0.7	36.8 ± 0.5	37.2 ± 0.4
Respiratory rate / min.	37.5 ± 0.6	39.7 ± 0.8	42.5 ± 0.7
Pulse rate / min.	72.6 ± 0.5	78.5 ± 0.7	81.0 ± 0.9
Ruminal movement / 2 min.	3 ± 0.00	2 ± 0.00	1 ± 0.00

Table (2): Urine analysis and abomasal fluid pH of apparently healthy cows and those with LDA and RDA.

Parameters \ Groups	Control	LDA	RDA
pH	8.3 ± 0.3	6.5 ± 0.2	6 ± 0.2
Protein	- ve	- ve	- ve
Glucose	- ve	- ve	- ve
Ketone bodies	- ve	+++ ve	+++ ve
Blood	- ve	- ve	- ve
Hemoglobin	- ve	- ve	- ve
Bilirubin	- ve	- ve	- ve
Abomasal fluid pH	2.2 ± 0.3	4.0 ± 0.2*	4.8 ± 0.2*

* Significant different from control at P < 0.5.

Table (3): Hematological parameters in control and those with abomasal displacement.

Parameters \ Groups	Control	LDA	RDA
Erythrocytic count (RBCs) $10^6/\text{Cumm}$	7.2 ± 0.5	7.5 ± 3.60	7.8 ± 3.41
Hemoglobin (gm/dl)	9.76 ± 0.51	10.4 ± 0.72	10.7 ± 0.92
Packed cell volume (PCV) %	34.6 ± 2.5	$44.2 \pm 3.0^*$	$46.5 \pm 3.5^*$
White blood cell count (WBCs) $10^3/\text{Cumm}$	7.8 ± 0.5	8.2 ± 0.42	8.4 ± 0.51

* Significant different from control at $P < 0.05$

Table (4): Levels of some serum parameters including sodium, potassium, chloride, calcium and total proteins in control and animals with abomasal displacement.

Parameters \ Groups	Control	LDA	RDA
Sodium (mEq/L)	142.5 ± 1.4	$132.0 \pm 1.75^*$	$131.3 \pm 1.91^*$
Potassium (mEq/L)	4.6 ± 0.21	$3.5 \pm 0.17^*$	$3.2 \pm 0.091^*$
Chloride (mEq/L)	99.2 ± 3.0	$78.0 \pm 1.1^*$	$75.0 \pm 1.5^*$
Calcium (mg/dl)	9.8 ± 0.2	$7.3 \pm 0.4^*$	$6.8 \pm 0.2^*$
Glucose (mg/dl)	55.0 ± 13.8	$40.2 \pm 15.5^*$	$38.2 \pm 12.7^*$
Total proteins (gm/dl)	7.4 ± 0.32	$8.1 \pm 0.41^*$	$8.4 \pm 0.53^*$

* Significant different from control at $P < 0.05$

**Significant different from control at P < 0.01

Table (5): Some liver and muscle enzymes and some kidney function parameters in control and those with abomasal displacement.

Parameters \ Groups	Control	LDA	RDA
Alanine aminotransferase (ALT) IU/L	20.1 ± 0.91	31.04 ± 0.62**	33.3 ± 0.83**
Aspartate aminotransferase (AST) IU/L	45.2 ± 0.64	73.02 ± 6.41**	78.00 ± 0.58**
Lactic dehydrogenase (LD) IU/L	440.3 ± 7.81	495.2 ± 8.21*	543.2 ± 9.52*
Creatine phosphokinase (CPK) IU/L	64.31 ± 0.81	95.20 ± 3.81**	88.5 ± 4.71**
Urea (mg/dl)	20.442 ± 2.1	52.0 ± 4.21**	55.3 ± 4.72**
Creatinine mg/dl	1.48 ± 0.031	2.05 ± 0.071*	2.1 ± 0.05*

* Significant different from control at P < 0.05

**Significant different from control at P < 0.01

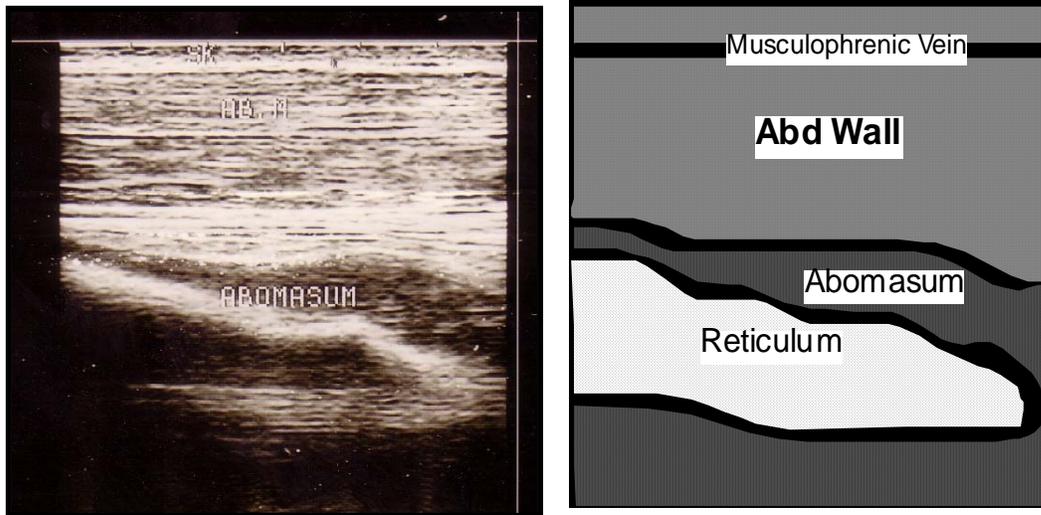


Figure 1: Ultrasonogram of normal abomasum viewed from the ventral midline caudal to the sternum. The abomasum is situated caudal to the reticulum. Notice the echogenic wall of the abomasum with hypoechoogenic ingesta.

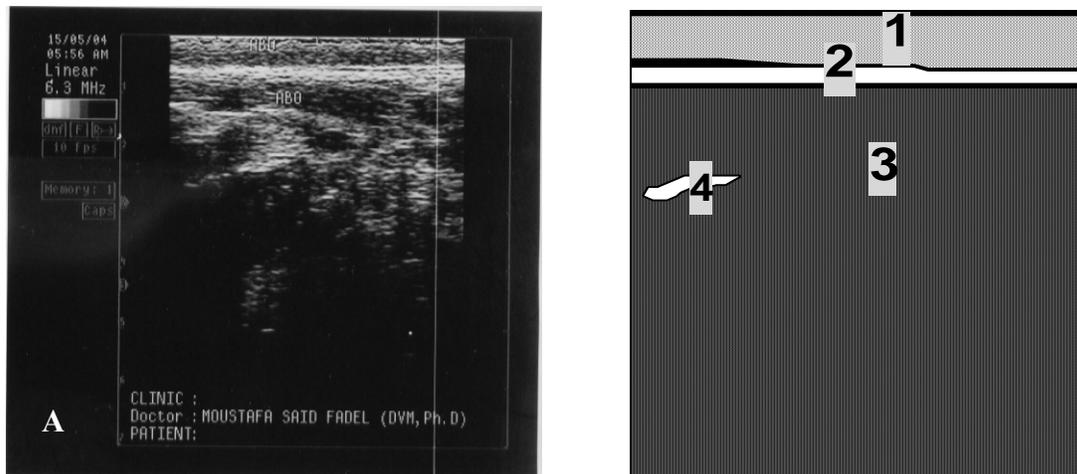


Figure 2: Ultrasonogram of normal abomasum imaged from the paramedian region showing (1) the abdominal wall, (2) the abomasal wall which appears as a narrow echogenic line (3) the abomasum with echogenic to hypoechoogenic ingesta and (4) the abomasal fold which appears as echogenic sickle-shape structure..

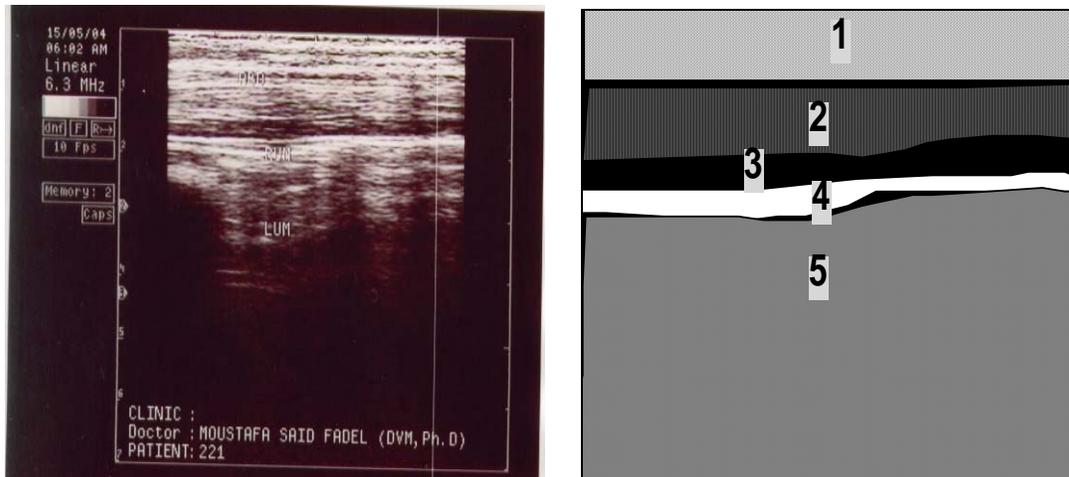


Figure 3. Ultrasonogram of left displacement of abomasum imaged from the dorsal region of the 12th intercostal space showing localization of the displaced abomasum between the abdominal wall and rumen. (1) Abdominal wall, (2) abomasum with hypoechogenic ingesta, (3) abomasal fluid, (4) rumen wall, (5) ruminal content.

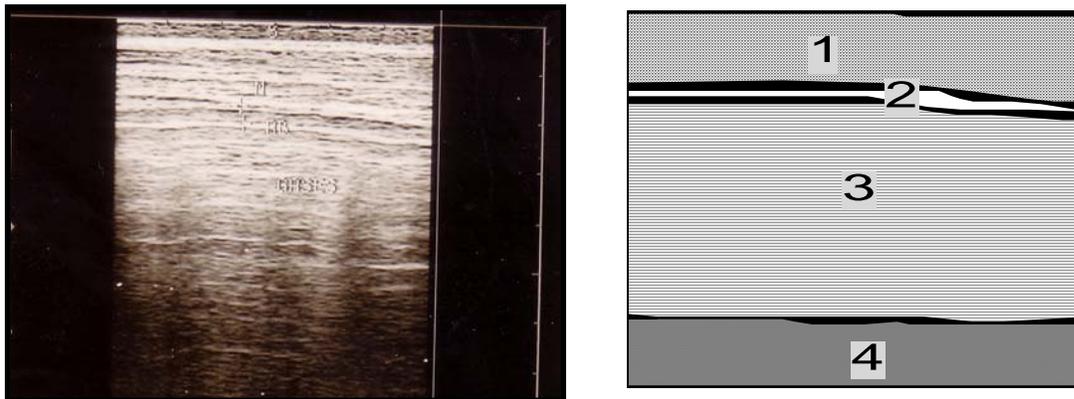


Figure 4: Ultrasonographic image of left displacement of abomasum viewed from the left dorsal region of the 12th intercostal space. The abomasal gas cap is not visible because of the reverberation artifacts at the abomasal surface. (1) Abdominal wall, (2) Abomasal wall, (3) Reverberation artifacts, (4) Abomasal fluid.

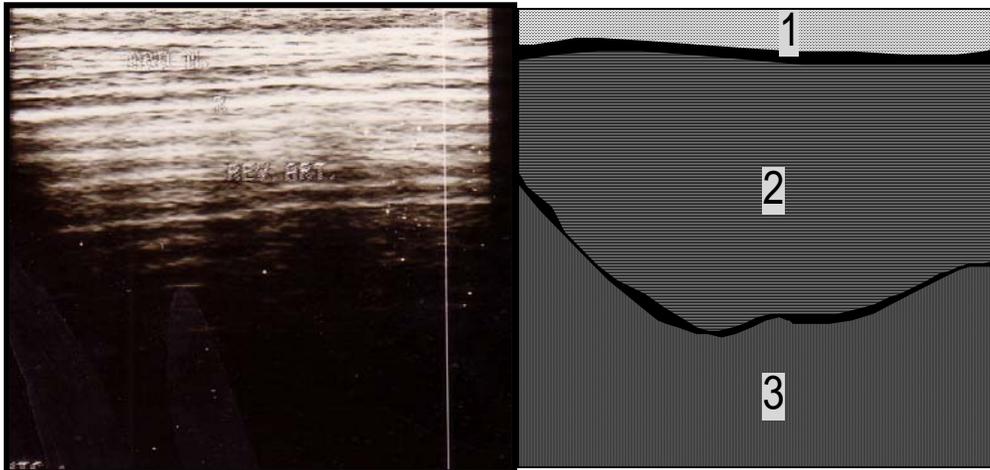


Figure 5: Ultrasonogram of left displacement of the abomasum imaged from the 12th intercostal space, showing the abomasal fluid /gas interface. Reverberation artifacts are visible instead of the abomasal gas cap.

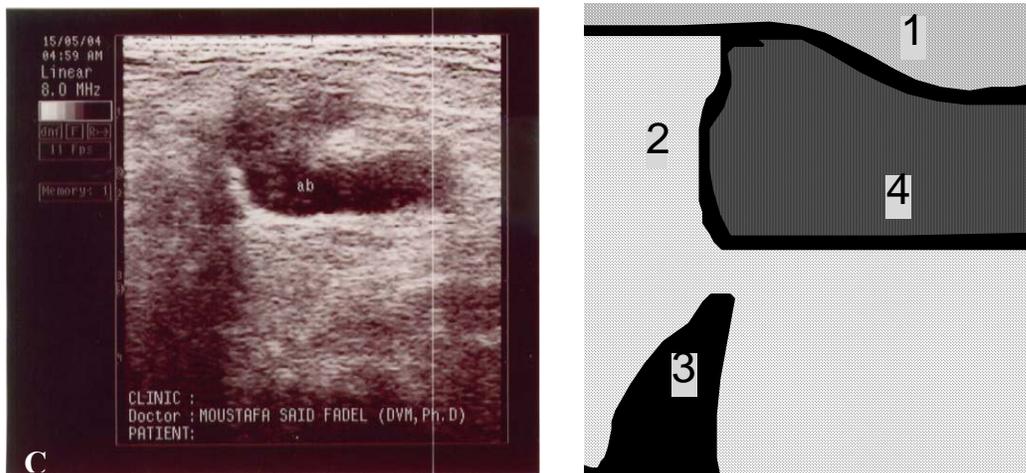


Figure 6: Ultrasonogram of right abomasal displacement imaged from the right side just behind the last rib showing the abomasum (4) between the right abdominal wall (1) and the liver (2) that appears as hypoechogenic and identified by the anechoic gall bladder (3).

دراسة إكلينيكية وفحص صورة الدم والتحليل البيوكيميائي و التشخيص بالموجات فوق الصوتية لإزاحة المعدة
الرابعة فى الأبقار مع محاولات للعلاج

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أجريت هذه الدراسة على عدد ١٢ بقرة من سلالة هولشتاين ، قسمت هذه الأبقار إلى مجموعتين ، إحتوت المجموعة الأولى على خمسة بقرات سليمة إكلينيكيًا واتخذت كمجموعة ضابطة .بينما إحتوت المجموعة الثانية على سبعة بقرات كانت تعاني من أعراض ناتجة عن حدوث إزاحة للمعدة الرابعة كان منهم اربعة بقرات حدث لهم إزاحة للمعدة الرابعة جهة اليسار بينما فى الثلاث بقرات الأخرى حدث إنحراف للمعدة الرابعة جهة اليمين .تم فحص هذه الحيوانات إكلينيكيًا وقد تبين ظهور الأعراض الآتية :خمول وتناقص فى الشهية وهبوط فى معدل إدرار اللبن وكان البراز غير متماسك وكميته صغيرة و كذلك تناقص فى عدد مرات الإجتار وسماع صوت رنين على الناحية اليسرى فى حالة إزاحة المعدة الرابعة للناحية اليسرى وكذلك سماع هذا الصوت على الناحية اليمنى فى حالة الإزاحة للناحية اليمنى .وقد أظهر تحليل البول ظهور أجسام كيتونية وزيادة فى الحامضية وقد بينت الفحوصات الدموية زيادة فى حجم الأجسام المضغوطة ونسبة الهيموجلوبين وعدم وجود أى تغير ملحوظ فى عدد خلايا الدم الحمراء والبيضاء كما أشارت التحاليل البيوكيميائية لمصل الدم إلى نقص معنوى ملحوظ فى مستوى الصوديوم والبوتاسيوم والكلوريد والكالسيوم والسكر .كما لوحظ أيضاً زيادة معنوية فى البروتين الكلى واليوريا ونشاط إنزيم اللاكتيك دى هيدروجينيز وكذلك إنزيمات وظائف الكبد مثل ناقلة الأمين الأسبرتية وناقلة الأمين الألانينية وقد أظهر التصوير بالموجات فوق الصوتية أن الجدار كان كثير التجانس وكذلك ظهرت أيضاً فى بعض الأحيان الحواجز الداخلية والتي ظهرت فى شكل المنجل وكانت كثيرة التجانس وقد ظهر المحتوى الغذائى فى الناحية الداخلية وقد اختلف فى التجانس وفى حالة الإزاحة اليسرى ظهرت المعدة الرابعة بين الجدار الخارجى الأيسر للبطن والكرش وفى الإزاحة اليمنى ظهرت المعدة الرابعة بين الجدار الايمن والكبد ، وبالنسبة للعلاج كان هناك بعض الاستجابة للعلاج بمحاليل الجلوكوز وكلوريد الصوديوم والبوتاسيوم المتماثل وكذلك الكال دى ماج مع محاولات للتعديل اليدوى لهذه الإزاحة وقد وجد ان الاستجابة للعلاج تكون افضل مع التشخيص المبكر.