INVESTIGATIONS ON ETIO-PATHOLOGY OF VOMITING IN DOGS

By

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THESIS

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DECLARATION

I hereby declare that the thesis entitled "**INVESTIGATIONS ON ETIO-PATHOLOGY OF VOMITING IN DOGS**" is a bonafide record of research work done by me during the course of research and that the thesis has not previously formed the basis for the award to me of any degree, diploma, associateship, fellowship or other similar title, of any other University or Society.

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CERTIFICATE

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Dedicated
To
My Parents and Sister

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Introduction

1. INTRODUCTION

Vomiting in dogs is a common clinical complaint presented to the small animal clinician. It is a major diagnostic challenge because of the wide variety of gastrointestinal and metabolic disorders that caused this symptom. Although vomiting does not always indicate a serious disorder, it is frequently a sign of many life-threatening diseases. Many clinicians have a preconceived impression that the reported vomiting is a result of a "gastric disorder". As all animals with gastric diseases do not vomit, a detailed history and thorough physical examination is essential to decide a diagnostic and therapeutic plan.

Many a times there may be a need for diagnostic imaging techniques to make or confirm a diagnosis. For a clinician in India many of the diagnostic tools such, as endoscopy is not available. Further, these techniques are invasive and have additional risk of anaesthetic procedures. Non-invasive and economical diagnostic tools such as ultrasonography and radiography are now within the reach of many clinicians in India.

Radiography provides excellent anatomic information but is limited to the evaluation of some tissues or organs like liver.

Ultrasonography is frequently performed in addition to radiography, but may be performed independent of the radiography. Ultrasound is superior to radiography in some circumstances but has limitations in other areas (Burk and Ackerman, 1996). Ultrasound provides information about size, shape and location of structure and details of architecture of the structure being studied. Ultrasonic scanning is safe, fast, repeatable and does not require tranquillisation or special preparation of the patient. It can be effectively used to evaluate a parenchymal organ. Earlier because of the presence of gas in gastrointestinal tract little attention was given to its sonographic evaluation. In the last decade there was a tremendous growth in the knowledge of gastrointestinal ultrasonography (Penninck *et al.*, 1989 and 1990, Penninck and Tidwell, 1997 and Lamb and Grievson, 1999) paving way for use of ultrasonic scanning effectively in gastrointestinal disorders.

While a vomiting animal is a diagnostic challenge for the clinician due to wide variety of etiologies that can cause vomiting, it is a therapeutic challenge too. Profuse and protracted vomiting can lead to dehydration, loss of electrolytes and acid-base derangements. While specific therapy is required to correct the underlying etiology, therapeutic measures are required to alleviate

the inconvenience of vomiting and metabolic derangements resulting out of this condition.

Considering these views this study "Investigations on etio-pathology of vomiting in dogs" was taken up to:

1. Evaluate ultrasonography and radiography as diagnostic tools in vomiting dogs.
2. Assess hydration status, and serum biochemical changes in vomiting dogs.
3. Correlate clinico-pathologic changes with ultrasonography and radiography.

Review of Literature

2. REVIEW OF LITERATURE

2.1 Definition

Vomiting is a reflex act co-ordinated through complex neural integration that results in the evacuation of gastric contents through the mouth (Twedt, 1983 and Strombeck and Guileford, 1991).

Vomiting referred to forceful ejection of gastric and occasionally proximal small intestinal contents through the mouth. This occured during sustained contraction of the abdominal muscles during which the cardia of the stomach is elevated and opened and the stomach is contracted. The vomiting act involved three stages: nausea, retching and vomiting (Tams, 1995).

Washabau and Elie (1995) described vomiting as a centrally mediated neurological reflex that required co-ordinated actions of the gastrointestinal, musculoskeletal and nervous systems.

Vomiting is a complex reflex act that is initiated by stimulation of the conceptualized vomiting or emetic centre located

in medulla oblongata of the brain. The driving force behind vomiting is contraction of the abdominal muscles and diaphragm causing intra-thoracic pressure changes from negative during retching to positive during vomiting (Twedt, 2000).

2.2 Etiology

2.2.1 Vomiting in general

Murdoch (1991) cited that many healthy dogs would vomit once weekly. Vomiting frequency with complete high obstruction was hourly and that with incomplete low intestinal obstruction was once every few days. It was also stated that with most gastric lesions, vomiting occurs shortly after eating. Higher the lesion in the alimentary tract shorter was the interval between eating and vomiting. At the other extreme it was described that vomiting in low intestinal obstruction and those associated with metabolic changes or systemic illness, eating–vomiting interval as variable.

First clinical sign of a chronic, previously asymptomatic disorder was the acute onset of vomiting and inflammatory bowel disease was an example for such a condition (Tams, 1992).

Acute vomiting included cases that had clinical signs persisted for one to seven days. Acute self-limiting vomiting was often characterised by a history of infrequent vomiting of food, mucous, bile or foreign material and were caused by dietary indiscretion or exposure to chemicals or plants. Chronic vomiting was often more than five to seven days or might occur periodically and were those associated with weight loss, inappetence and lethargy and these required serious evaluations (Leib, 1997a).

Johnson *et al.* (2000) observed that vomiting was a common clinical sign associated with many gastrointestinal or non gastrointestinal disorders of dogs and might be caused by benign self-limiting disorders such as acute gastritis or serious life threatening disorders such as acute pancreatitis, intestinal obstruction or acute renal or hepatic failure. Chronic or persistent vomiting was always an indication for further work up.

2.2.2 Vomiting due to gastrointestinal disorders

Murray *et al.* (1972) and Penninck and Tidwell, (1997) reported that major clinical feature with gastric ulceration were vomiting, hematemesis, melena, weight loss and anemia.

Chronic gastritis had vomiting that was infrequent and might or might not be associated with eating (Wingfield and Twedt, 1986) while acute gastritis had transient onset of acute vomiting exacerbated by eating and drinking (Strombeck and Guileford, 1991).

Fonda *et al.* (1989) observed consistent signs in gastric carcinoma as intermittent and unrestrained vomiting or hematemesis and was not related to feeding.

Jergens *et al.* (1992) observed diarrhea, vomiting, melena, abdominal pain and weight loss in idiopathic inflammatory bowel disease associated with gastroduodenal ulceration-erosion in dogs and cats.

Chronic intermittent vomiting according to Tams (1992) was the common presenting complaint in veterinary medicine with no specific time relation to eating and variable appetite. Depending on the cause (chronic gastritis, irritable bowel syndrome, and gastric motility disorders) might have diarrhoea, lethargy, inappetence, and salivation. He stated that 30 per cent of patients with colitis were presented with vomiting.

The predominant clinical sign in dogs and cats with colitis associated with inflammatory bowel disease was diarrhoea. Weight loss and vomiting occurred less frequently and might be related to concurrent involvement of stomach and/or small intestine and not to colonic inflammation. (Leib and Matz, 1995)

Willard, (1995) stated that motility deficits may occur in some animals which resulted in retention and vomiting of gastric contents.

2.2.3 Vomiting due to gastrointestinal tract obstructions

2.3.1.1 Pyloric stenosis

Pyloric stenosis and pylorospasm were important and common causes of persistent vomiting in young dogs. Vomiting might be seen at varying intervalsafter feeding and vomitus had food from the last meal. Animals, which vomited only froth or gastric secretion, were unlikely to have pyloric stenosis (Pearson, 1970).

Pearson, (1979) opined that predominant clinical sign in pyloric stenosis in the dog was vomiting with variable frequency (several times each day to once or twice a week) and in minority cases can be projectile in nature.

Happe *et al.* (1981) Clark (1985), Sikes *et al.* (1986), Bellenger *et al.* (1990) and Polese *et al.* (1991) observed vomiting, weight loss, diarrhoea, poor coat, poor appetite, polydipsia and depression as the common clinical signs of chronic hypertrophic pyloric gastropathy in dogs.

2.3.1.2 Intussusception

Wilson and Burt (1974) opined that vomiting in cases of intussusception may be due to frank obstruction of the bowel or due to reflex vagal stimulation from the viscera involved.

Weaver (1977) reported that the commonest triad of presenting signs in intestinal intussusception as vomiting, diarrhoea and a palpable abdominal mass.

Levitt and Bauer (1992) observed that the presenting signs in dogs and cats with intussusception were varied and non specific and may be seen in a number of acute abdominal conditions and they included vomiting, bloody mucoid diarrhoea and a palpable abdominal mass.

Evans *et al.* (1994) and Habeeb (2000) recorded intussusceptions concurrently in dogs having linear foreign bodies.

Lamb and Mantis (1998) quoted that clinical signs in dogs with intestinal intussusceptions as a result of partial intestinal obstruction, bacterial overgrowth, ischemia or infar ction of the intussusceptum and localized peritonitis.

2.3.1.3 Foreign bodies

Many gastric foreign bodies were asymptomatic or caused only occasional vomiting and their presence were often revealed during radiographic examination of other structures (Gibbs and Pearson, 1973).

Felts *et al.* (1984) observed in cats that as duration of obstruction increased, frequency of vomiting decreased.

Evans *et al.* (1994) noted that linear foreign bodies (LFBs) were most commonly lodged at the pylorus in dogs.

Jayaprakash *et al.* (1999) reported a case of linear foreign body (cotton thread) in a dog presented with the history of vomiting and hematochezia.

Jose (2001) observed that the predominant clinical sign in upper alimentary tract obstruction as vomiting with frequency of four times a day and vomitus frothy or white in colour. In lower alimentary tract obstruction it was reported that constipation was predominant with less frequent vomiting (three times or less a day) and vomitus frothy or yellow in colour with partially digested food.

2.2.4 Vomiting due to drugs

Gastric ulceration might be a primary lesion resulting from chronic administration of steroid or non-steroidal anti-inflammatory drugs (Moore, 1992).

Gastric and intestinal erosions and potentially serious ulceration might develop with the use of non-steroidal anti-inflammatory drugs. Nephrotoxicity might also occur resulting in acute renal failure (Tams, 1992).

Perforating gastric ulcers were associated with prolonged Ibuprofen therapy. Mild superficial gastric mucosal erosion and hemorrhagic lesions were more frequent than gastric ulcers shortly after administration of aspirin. Acute acetaminophen toxicosis produced hepatic necrosis and signs of depression, vomiting and abdominal pain could be attributed to hepatic failure (Villar *et al.*, 1998).

2.2.5 Vomiting due to hepatobiliary disorders

Mullowney and Tennant (1982) observed signs of depression, dehydration, pyrexia, icterus, vomiting and diarrhoea in a case of choledocholithiasis.

Ascites was the most common clinical sign reported by Sevelius (1995) in chronic hepatitis and cirrhosis followed by decreased appetite, lethargy, vomiting and diarrhoea.

Leib (1997b) reported that chronic gastritis or gastric or duodenal ulcer disease may be present in patients with liver disease and vomiting was a common sign in acute or chronic liver disease. It was also reported that abnormal metabolism of gastrointestinal peptides or stimulation of the chemoreceptor

trigger zone by circulating toxins associated with hepatic encephalopathy may be involved in the pathophysiology.

2.2.6 Vomiting due to renal disorders

Osborne *et al.* (1972) opined that vomiting is a common clinical sign in uremia due to stimulation of vomiting centre and it may be also due to ulcerative gastritis and hemorrhagic ulcerative gastritis.

Increased serum calcium prevailing in hypercalcemic nephropathy depressed the excitability of nervous tissue and the contraction of muscles. The loss of tonus of gastrointestinal tract as a result caused gastric atony with anorexia, vomiting and constipation (Osborne and Stevens, 1977).

Mikiciuk *et al.* (1989) stated that chronic partial urinary tract obstruction, chronic leptospirosis, canine adenovirus-1 infection, pyelonephritis and glomerulonephritis produce chronic tubulointerstitial disease.

Renal failure with uremia could produce gastric mural and mucosal mineralization, ulceration and vomiting (Moore, 1992).

Schulman and Krawiec (2000) stated that uremic gastroenteritis could be manifested as vomiting, diarrhoea or anorexia. Vomiting resulted due to prevailing hypokalemia and acidosis in renal failure or uremic toxemia triggering chemoreceptor trigger zone. It was also stated that hypergastrinemia results in renal failure and initiated a vicious cycle to produce hydrochloric acid and this state could produce pyloric spincter incompetency, biliary reflex and delayed gastric emptying.

Prolonged duration of decreased appetite, vomiting, diarrhoea, weight loss, or a combination of these states, were more consistent with the diagnosis of chronic renal failure (Vaden, 2000).

2.2.7 Vomiting due to miscellaneous disorders

Hardy and Osborne (1977) reported that pyometra occur in middle aged or older bitches with signs of depression, polydipsia/polyuria, purulent or sanguineous vaginal discharge, vomiting and diarrhoea.

Moore (1992) quoted that vomiting formed a common presenting sign of animals with hypoadrenocorticism and dogs with

primary neurologic disease such as cerebellar infarcts or vestibular disease.

Vomiting from systemic or metabolic causes might be acute or chronic. In these cases there was generally no direct correlation with eating and the content of vomitus was unpredictable (Tams, 1992).

Hess *et al.* (1998) attributed vomiting in acute pancreatitis to secondary gastrointestinal inflammation and local pancreatic inflammation.

2.3 Physical examination

Wilson and Burt (1974) and Weaver (1977) reported that in intestinal intussusception, abdominal palpation revealed an elongated mass and if missed were because of their craniodorsal location. It was also reported that pain on palpation was minimal.

Evans, *et al.* (1994) reported that in dogs with gastrointestinal linear foreign bodies physical examination revealed dehydration (53 per cent), pain on palpation (47 per cent) and a palpable abdominal mass (38 per cent).

In a dog with jejunal intussusception and acute renal failure, Kantrowitz *et al.* (1988) observed marked dehydration, depression and dementia, oral ulcers, and uremic breath odour. On abdominal palpation, bilaterally enlarged smooth kidneys and a painful tubular mass in the left cranioventral abdomen was also detected.

2.4 Hydration status

Vomiting caused fluid depletion and the dehydration increased with each vomiting due to the inability to take sufficient oral maintenance fluids (Twedt, 1983).

Bellenger *et al.* (1990) observed dehydration in dogs with vomiting due to chronic hypertrophic pyloric gastropathy and it was evidenced by clinical signs, packed cell volume (PCV), total plasma protein and/or blood urea concentration.

Evans *et al.* (1994) reported that physical examination revealed dehydration in cases with gastrointestinal linear foreign bodies.

Greco (1998) listed physical parameters that reflected hydration status as skin turgor, moistness of mucous membranes, heart rate, pulse character, capillary refill time, and jugular vein

distension. The laboratory tests that helped in determining the extent of dehydration were PCV, urine specific gravity and total plasma protein concentration. The clinical signs cited as the cause for rapid dehydration were vomiting, severe small bowel diarrhoea rapid and voluminous blood loss, polyuria, third space fluid shifts and insensible loss as a result of excessive panting or fever.

2.5 Ultrasonography

2.5.1 Ultrasonography of gastrointestinal system

Penninck *et al.* (1989) described the normal sonographic appearance of stomach, small bowel and colon in normal healthy dogs. They also stated that ultrasonographic scanning of abdomen offers information not available by other modalities such as radiographic contrasts studies or endoscopy. It was suggested that pathological thickening should be suspected when the stomach wall measures more than 6-7 mm and when the bowel wall measures more than 5mm.

Penninck *et al.* (1990) observed sonographically localized thickening of gastric wall (15 mm) in local severe gastritis; localized and circumferential thickening of pylorus in hypertrophic pyloric stenosis; generalized ileus, thickened

duodenum and fluid accumulation in parvovirus enteritis and homogenous to inhomogenous mass in gastrointestinal neoplasias.

Nyland and Mattoon (1995) cited that gastrointestinal inflammation was characterized by extensive and symmetric wall thickening with retained wall layer identification, whereas gastrointestinal neoplasia was characterized by localized, asymmetric wall thickening with disrupted wall layer identification. Exceptions to these rules cited were generalized, symmetric thickening noted with intestinal lymphosarcoma and disrupted wall layer identification with severe necrotic inflammation.

Penninck and Tidwell (1997) described ultrasonographic features of gastric ulcer as a wall defect or "crater" in the centre of the thickened gastric wall.

Poor intestinal wall layer definition, focal thickening and large mesenteric lymph nodes with hypoechoic changes were observed by Baez *et al.* (1999) in inflammatory bowel disease of cats.

Lamb and Grievson (1999) reported that ultrasonographic features in dogs with gastric lymphoma as sessile masses that involved all layers of the gastric wall with ulceration and lymphadenopathy and those with gastric leiomyoma or leiomyosarcoma as focal mass with involvement of the gastric antrum and thickening of the muscular layer of the gastric wall.

2.5.2 Ultrasonography of gastrointestinal tract obstruction

Fleischer, *et al.* (1980) cited a second ultrasonographic pattern in intussusception as a dense echogenic center surrounded by a single hypoechoic ring in humans. It was also stated that this pattern was less specific and can also be observed in neoplasia and infiltrative bowel disease.

Penninck *et al.* (1990) stated that the sonographic appearance of gastric foreign objects such as ball might be variable depending on the physical properties (cohesion, elasticity, air content). It was also stated that the curvy linear shadowing was highly suggesting of a ball, although it may also represent a large amount of gas and/or ingesta in the stomach and administration of water should help to confirm the diagnosis of gastric foreign object.

Pennick *et al.* (1990), Lamb and Mantis, (1998) and Prathaban *et al.* (2001) reported multiple concentric hyperechoic and hypoechoic rings as the ultrasonographic feature of intestinal intussusception in dogs.

Tidwell and Penninck (1992) opined that acoustic pattern arising from each foreign body varied depending on its physical properties and interaction with ultrasound beam.

Ultrasonographically chronic hypertrophic pyloric gastropathy in dogs was diagnosed by Biller *et al.* (1994) by visualizing an evenly thick hypoechoic layer surrounding the pyloric lumen.

Watson, (1997) reported that ultrasonography of a large craniodorsal abdominal mass that showed laminated appearance in some views typical of an intussusception in a case of gastroduodenal intussusception.

2.5.3 Ultrasonography of hepatobiliary system

Cartee (1981) reported ultrasonographic detection of small cirrhotic liver surrounded by ascitic fluid and opined that the ascitic fluid helped in better visualization of liver.

Nyland and Park (1983) stated that ultrasonography might be used to evaluate non-invasively a wide variety of dieases affecting the canine liver. Hepatic mass lesions, parenchymal pathology, gall bladder and biliary disease and vascular abnormalities may be detected and characterised by ultrasonography.

Feeney *et al.* (1984) described ultrasonographic appearance of hepatic tumors which ranged from focal, large mixed hyperechoic or hypoechoic masses. It was also stated that ultrasonography can be used to characterize the mass architecture, confirm the organ of origin and extent of involvement of the organ and to identify any metastases.

Voros *et al.* (1991) reported multifocal alterations in hepatic neoplasia, hepatic cirrhosis, generalized mycosis and unifocal lesions in hemangiosarcoma and nodular hyperplasia. Diffuse ultrasonographic alterations such as hyperechoic liver of normal or enlarged size in lymphosarcoma and hepatic lipidosis; hyperechoic 'bright' but small liver in atrophic cirrhosis; hypoechoic to normal intensity liver of normal size in liver dystrophy and hepatic venous distension. It was suggested that clinicolaboratory and ultrasonographic finding when combined provided correct diagnosis.

Diez-Bru (1994) stated diffuse hyperechogenicity as the ultrasonographic feature in hepatic fatty infiltration or cirrhosis and with cirrhosis, the parenchyma is often heterogeneous and the contour nodular. Diffuse hypoechogenicity was described as a feature in hepatic venous congestion, acute hepatitis or lymphosarcoma and lymphosarcoma may also appear as multiple hypoechoic foci.

Diez-Bru *et al.* (1997) opined that distension of gall bladder is not but dilation of cystic duct together with distension of the gall bladder as the sign of biliary obstruction.

Nyland and Mattoon (1995) cited that normal liver is equal or slightly more echogenic than the cortex of the right kidney at the same scanning depth and instrument gain settings and spleen had greater echo intensity than the liver.

Farrar *et al.* (1996) observed hypoechoic, heteroechoic or hyperechoic, masses in cases of dogs with hepatic abscesses.

Normal liver had sharp borders and uniformly homogenous parenchyma with speckled echos. Normal gall bladder was an anechoic structure to the right of midline (Bhadwal *et al.*, 1999).

2.5.4 Ultrasonography of the kidneys

Konde *et al.* (1986) opined that ultrasonography appeared to be more sensitive than radiography in differentiating the internal characteristics of renal lesions.

Walter *et al.* (1987) reported that ultrasonographic patterns were most specific for focal or multifocal or diffuse neoplasia. It was also reported that ultrasonography was least specific for diffuse parenchymal disease without architectural disruption such as glomerulo or interstitial nephritis, renal tubular necrosis, and nephrocalcinosis.

Increase in renal cortical echogenicity can be observed in some acute renal diseases and in chronic renal diseases due to fibrosis and scarring of tissues. This finding in chronic renal disease often is associated with reduced renal size and irregular margins (Kantrowitz *et al.*, 1988).

Lamb (1990) cited conditions that caused increased renal cortical echogenicity as nephritis, acute tubular necrosis, nephrocalcinosis, hypercalcemic nephropathy and end stage kidney disease. Decreased cortical echogenicity was attributed to lymphosarcoma. Focal hyperechoic kidney lesions listed include

primary or secondary neoplasia and cysts containing blood or other proteinacious material, while focal hypoechoic kidney lesions included renal cysts and abscess.

Ultrasonography in dogs suffering form chronic renal failure revealed small kidneys, loss of architectural details and hyperechoic periphery with anechoic core (Bhadwal and Mirakhur, 2000).

Byun and Kim (1998) reported high echointensity of the renal parenchyma and emergence of halo in the corticomedullary junction associated with ethylene glycol toxicosis in dogs.

2.5.5 Ultrasonography of other organ systems

Diez-Bru *et al.* (1997) observed ultrasonographically, mass in pancreatic region associated with pancreatic carcinoma causing extra hepatic biliary obstruction.

Hypoechoic pancreas and hyperechoic peripancreatic mesentery in addition to enlarged irregular pancreas, peritoneal effusion and evidence of duodenitis and extra hepatic biliary

obstruction were suggestive of acute pancreatitis (Hess *et al.*, 1998)

Vijayakumar *et al.* (1998) reported that ultrasonographic finding of anechoic fluid filled uterine horns with echogenic debris dorsal and cranial to urinary bladder as suggestive of pyometra.

Hoerauf and Reusch (1999) observed ultrasonographically measurable reduction in size of the adrenal glands in dogs associated with hypoadrenocorticism.

2.6 Radiography

2.6.1 Radiography of gastrointestinal system

2.6.1.1 Survey radiography

In cases of pyloric stenosis, the plain radiographs showed dilated stomach often containing fluid and food remnants and more significantly a layer of mineral debris that accumulated as a result of delayed emptying (Pearson, 1970, Gibbs and Pearson, 1973 and Pearson, 1979).

Kantrowitz and Biller (1992) opined that pyloric out flow obstruction may not result in any radiographic abnormalities.

It was also stated that fluid filled dilatation of the stomach is sometimes seen with chronic outflow abnormalities.

2.6.1.2 Contrast radiography

Gastric emptying time implies the time taken for stomach to begin to empty – not to empty completely. The normal stomach begins to empty within 30 minutes and if there is delay for an hour or more there is probably some degree of pyloric stenosis or pylorospasm (Pearson, 1970).

Pearson (1979) described that "beak, string and tit" signs as the radiographic pattern of pyloric occlusion.

In dogs, the stomach and duodenum, generally are filled with contrast medium in the radiographs taken immediately after administration. The proximal one-third of the small intestine is filled within 30 minutes and entire small intestine at 3 hours. The stomach is empty and cecum filled at six hours. At 24 hours only colon and cecum contained contrast medium (Kleine, 1985b).

Fonda *et al.* (1989) reported radiographic details of gastric cancer as thickening or roughening of gastric wall, loss of

normal rugal fold pattern, pyloric or lesser curvature filling defects with narrowing of lumen, delayed or incomplete emptying of contrast material from stomach.

Jergens *et al.* (1992) observed that positive contrast radiography demonstrated focal filling defects along the gastric margins as suggestive of gastric ulceration.

Kantrowitz and Biller (1992) described that double contrast procedure, in which the stomach is distended with air following coating of stomach with barium improves sensitivity in detecting ulcers.

2.6.2 Radiography of gastrointestinal obstruction

According to Gibbs and Pearson (1973), radiological signs of obstruction of small intestine were (a) dilatation of the proximal intestine, often with excessive gas accumulation (b) multiple horizontal interface indicating gas-capped fluid levels on lateral erect films. (c) Partial or total obstruction to the flow of ingesta (and contrast medium) down the intestine. (d) With some lesions, persistent displacement of intestinal loops.

Wilson and Burt (1974) reported that patients with intussusception had the appearance of partial obstruction on plain radiographs, with varying degree of mechanical ileus and gas accumulation proximal to the obstructed bowel. Barium contrast showed space-occupying intussusception having a lucent appearance and often an abrupt end to the contrasted loop of the bowel.

Stomach, duodenum, cecum and colon occupied relatively constant locations in the abdomen, while the normal small intestine not only propelled its contents but shifts within the limits of its mesenteric attachments. With obstruction, not only the movement of intestinal contents slowed or stopped, but also the intestinal tract tends to occupy the same position in sequential radiographs. This phenomenon termed fixation was an important sign of obstruction (Kleine, 1985b).

Kantrowitz and Biller (1992) opined that intestinal obstruction could be identified by gas and fluid dilatation of the small bowel.

Jayaprakash *et al.* (1999) reported that survey abdominal radiographs of linear foreign body intestinal obstruction in a dog revealed distension and gathering of the small intestine

with tapered enteric gas bubbles and on contrast radiography with barium meal, plicated appearance of the intestine.

Jose (2001) observed that plain radiography was useful for identifying obstruction in the alimentary tract caused by radiopaque materials. While, contrast radiography using barium meal revealed seats of obstructions due to radiolucent foreign bodies and anatomical alterations.

2.6.3 Radiography of hepatobiliary system

O'Brien (1978) opined that hepatic fibrosis and obstruction of the venacava as the common cause of ascites in dogs and radiographically this results in generalized ill defined abdominal density.

Kleine (1985a) described radiographic density of liver as water density and that it is clearly seen in the lateral view, except when abdominal contrast is greatly diminished.

Burk and Ackerman (1996) cited that liver appears small in hypotrophic cirrhosis or fibrosis and portosystemic shunts.

2.6.4 Radiography of kidneys

Kleine (1985c) opined that in uremia contrast medium is excreted through liver and small bowel mucosa when excretory urography is performed. The nephrogram phase may be delayed in cases of inadequate renal perfusion, hydronephrosis, stenosis of renal artery or impaired renal function and nephrogram persisted in renal failure, hydronephrosis and obstruction of the renal vein.

Burk and Ackerman (1996) reported that kidneys appear unilaterally or bilaterally small, regular and in excretory urography renal opacification during nephrogram phase was usually less than normal in cases with chronic interstitial nephritis.

2.7 Clinical pathology

2.7.1 Haematology

Wilson and Burt (1974) and Weaver (1977) observed neutrophilic leucocytosis in cases associated with intestinal intussusceptions.

Jain (1986) postulated the reference range of haematological parameters for normal dogs:

Erythrocytes	5.5–8.5 x 10^6cells per microlitre
Haemoglobin	12 – 18grams per decilitre
Packed cell volume	37 - 55 per cent
Leucocytes	6000-17000 cells per microlitre
Neutrophils	3000-11,500 cells per microlitre
Bands	0 – 300 cells per microlitre
Lymphocytes	1000-4800 cells per microlitre
Monocytes	150-1350 cells per microlitre
Eosinophils	100-1250 cells per microlitre
Basophils	rare

McCaw *et al.* (1989) stated that anemia was common in chronic renal failure and was normocytic, normochromic, non-regenerative anemia.

Rutgers (1996) reported marked regenerative anemia in case of liver disease and moderate non-regenerative anemia in case of chronic liver disease with low packed cell volume.

Jose (2001) reported leucocytosis and lymphocytosis in upper alimentary tract obstruction and normal leukocyte count with neutrophilia in lower alimentary tract obstructions. Packed cell volume and total erythrocyte count was low in both cases.

2.7.2 Serum biochemistry

2.7.2.1 Alanine Amino Transferase (ALT)

Weaver (1977) recorded elevated ALT in dogs associated with intestinal intussusception.

Sevelius (1995) reported normal to mildly increased concentrations of ALT in cases of liver cirrhosis and hepatitis and markedly elevated ALT levels in cases with chronic cholangiohepatitis.

Kaneko *et al.* (1997) postulated ALT concentration in serum of normal dogs as 21 – 102 units per litre.

Jose (2001) observed elevated ALT in dogs with lower alimentary tract obstruction and normal levels in upper alimentary tract obstructions.

2.7.2.2 Alkaline Phosphatase (ALP)

Dunn (1992) opined that in liver disease ALP concentration in serum increased along with ALT concentration.

Sevelius (1995) recorded moderate to marked elevation of ALP in cases of liver cirrhosis and hepatitis and marked elevation in chronic cholangiohepatitis.

Cornelius (1997) opined that elevated ALP values are seen in cholestatic hepatobiliary diseases, glucocorticoid therapy and hyperadrenocorticism.

Kaneko *et al.* (1997) postulated normal value of alkaline phosphatase in dogs to range from 20 to 156 units per litre.

2.7.2.3 Serum proteins

Weaver (1977) observed low plasma albumin and globulin in dogs with intestinal intussusceptions.

Cornelius (1979) and Dunn (1992) reported increase in serum globulins in dogs with advanced liver disease.

Hypoalbuminemia was the common feature of liver cirrhosis and chronic progressive hepatitis (Cornelius, 1979, Dunn, 1992 and Sevelius, 1995).

Benjamin (1985) cited total protein and albumin values (in grams per decilitre) in normal dogs as 5.3 – 7.3 (mean 6.3) and 3.1 – 4.0 (3.56) respectively.

Hypoproteinemia and hypoalbuminemia was recorded by Jergens *et al.* (1992) in idiopathic inflammatory bowel diseases associated with gastroduodenal ulceration – erosion in dogs and cats.

2.7.2.4 Urea nitrogen

Weaver (1977) reported elevated blood urea in cases with intestinal intussusceptions.

Kantrowitz *et al.* (1988) observed elevated blood urea nitrogen in a case of acute renal failure due to ethylene glycol toxicity in a dog.

McCaw *et al.* (1989) cited that urea nitrogen value can be elevated due to non-renal causes and in most cases it will not exceed 50 milligrams/decilitre. It was also stated that dehydration and reduced renal perfusion could rarely elevate urea nitrogen levels above 120 milligrams/decilitre.

Srinivasan *et al.* (1993) recorded lower value of blood urea in cases with chronic renal insufficiency and attributed it to low protein diets in the ration of dogs studied.

Kaneko *et al.* (1997) postulated normal serum urea nitrogen in dogs as 10 – 28 milligrams per decilitre.

2.7.2.5 Creatinine

Benjamin (1985) stated normal values of creatinine in dogs to range from 1-2 milligram per decilitre while Kaneko *et al.* (1997) cited normal value as 0.5 – 1.5 milligram per decilitre.

Kantrowitz *et al.* (1988) reported creatinine value of 6.7 milligram per decilitre in a case of acute renal failure due to ethylene glycol toxicity.

Srinivasan *et al.* (1993) recorded serum creatinine values of 4.56 ± 0.72 milligrams per decilitre in dogs with chronic renal insufficiency and 4.63 ± 1.29 milligrams per decilitre in dogs with acute renal insufficiency.

2.7.2.6 Bicarbonate

When significant quantity of hydrogen and chloride is lost as in vomiting due to pyloric outflow obstruction, plasma bicarbonate levels raises causing metabolic alkalosis (Twedt, 1983).

Bellenger *et al.* (1990) recorded elevated bicarbonate values with range of 40.3 – 46.2 millimoles per litre in cases with chronic hypertrophic pyloric gastropathy.

Kaneko *et al.* (1997) stated that the normal reference range for serum bicarbonate as 18-24 milliequivalents per litre.

Evans *et al.* (1994) reported increased bicarbonate concentration in dogs with linear foreign bodies in the gastrointestinal tract..

Greco (1998) cited that acid bases status can be reflected by serum bicarbonate concentrations. It was also stated that hyperbicarbonatemia (>30 milliequivalents per litre) is associated with metabolic alkalosis and hypobicarbonatemia (<10 milliequivalents per litre) is indicative of metabolic acidosis.

2.7.2.7 Sodium and potassium

Twedt (1983) opined that gastric vomiting would result in loss of water containing sodium, chloride, hydrogen and potassium and hypokalemia as the most frequent electrolyte abnormality observed in the vomiting patient.

Benjamin (1985) recorded normal serum sodium as 143 (range 137-149) milliequivalents per litre and potassium 4.4 (3.7–5.8) milliequivalents per litre.

Jergens *et al.* (1992) observed hypokalemia in dogs with gastroduodenal ulceration – erosion having vomiting.

Gastric foreign bodies that caused pyloric obstruction resulted in hyponatremia and hypokalemia (Moore, 1992).

Richter (1992) stated that vomiting of gastric contents caused hypernatremia, hypokalemia and alkalosis, while gastric or proximal duodenal obstruction resulted in hyponatremia, hypochloremia, hypokalemia and metabolic alkalosis.

Evans *et al.* (1994) reported hypokalemia and hyponatremia in 31 per cent and 22 per cent respectively, of dogs associated with gastrointestinal linear foreign bodies.

Hyponatremia with hypervolemia was seen in dogs with cirrhosis, nephritic syndrome and hypoalbuminemia and such patients had ascites or oedema (DiBartola, 1998). Dogs with vomiting, renal failure and intestinal obstruction had hypovolemic hypernatremia (Marks and Taboada, 1998).

Hyperkalemia associated with increased total body potassium is seen in dogs with oliguric or anuric renal failure and hypoadrenocorticism; Hypokalemia resulting from decreased total body potassium seen in dogs with gastric vomiting. Hypokalemia was the uncommon manifestation of renal failure in dogs than in cats (Phillips and Polzin, 1998).

2.7.3 Histopathology

2.7.3.1 Stomach

Happe *et al.* (1981) and Walter *et al.* (1985) reported mucosal foveolar and glandular hyperplasia, cystic glandular dilatation, superficial ulcerations, and various cellular infiltrates

as the microscopic picture in obstructive lesions of chronic hypertrophic pyloric gastropathy.

2.7.3.2 Liver

Obwolo and French (1988) observed loss of lobular pattern and rare portal areas, hepatocytes grouped in nodular structures separated by connective tissue and diffuse fatty changes in cases with hepatic cirrhosis.

Rutgers (1996) described total loss of normal liver architecture and marked fibrosis surrounding small groups of hepatocytes in liver cirrhosis.

2.7.3.3 Kidney

Smith *et al.* (1974) suggested that infiltration with plasma cells and lymphocyte was the significant finding in interstitial nephritis. Interstitial fibrosis and thickness of Bowmans capsule were believed to be long term effects.

Histopathological lesion in chronic interstitial nephritis was suggested by Sastry (1983). There was increased fibrous tissue proliferation with atrophy and disappearance of

tubules. Some tubules showed cystic dilation with granular and hyaline casts in them. Infiltration with lymphocytes and plasma cells was noted. Calcification of glomeruli was reported to occur in a few areas.

Maxie (1993) stated that mononuclear cell infiltration, interstitial fibrosis and generalised tubular atrophy were the significant lesions in chronic interstitial nephritis. Similar findings were reported by Vegad and Katiyar (1998).

Materials and Methods

3. MATERIALS AND METHODS

The study was conducted in the Department of Clinical Medicine, College of Veterinary and Animal Sciences, Mannuthy over a period of two semesters from November 2000 to August 2001. Dogs presented to Veterinary College Hospital, Mannuthy were selected and utilised for the study.

3.1 Design of the study

The study consisted of apparently healthy animals as control and clinical cases.

3.2 Selection of animals

3.2.1 Control

Six apparently healthy dogs that were brought to the hospitals for general check-up and routine vaccination were selected as the control for studying normal parameters.

3.2.2 Clinical cases

A total of 1800 dogs were presented to the medical unit of Veterinary College Hospital, Mannuthy during the period of study, of which 302 dogs had history of vomiting. Of the 302 dogs,

143 had gastroenteritis, and were excluded from the study. Dogs found positive of gastrointestinal parasites by faecal examination were also excluded from the study. Of the remaining dogs those, which responded to treatment with antiemetic were not considered for further study. Twenty dogs that did not respond to such therapy were selected for detailed study.

The selected clinical cases of dogs were subjected to detailed study and all parameters under study were analysed. The twenty animals under detailed study were grouped based on the type of affection as follows:

Group I (n=6) - Consisted of dogs with gastritis

Group II (n=6) - Consisted of dogs with gastrointestinal tract obstructions

Group III (n=4) - Consisted of dogs with renal disorders

Group IV (n=4) - Consisted of dogs with hepatic and uterine disorders (two had cirrhosis, one had cholangitis, and one had pyometra). Since the dogs with liver affections had wide variations in the data, they were not subjected to statistical analysis and data were presented as such.

3.3 Parameters studied

History and clinical signs

Physical examination

Hydration status

Abdominal ultrasonography

Abdominal radiography – survey and contrast

Clinical pathology

Hematology

Hemoglobin (Hb) concentration

Volume of Packed Red Cells (VPRC)

Total Erythrocyte Count (TEC)

Total Leucocyte Count (TLC)

Differential Leucocyte Counts (DLC)

Serum biochemistry

Alanine Amino Transferase (ALT)

Alkaline Phosphotase (ALP)

Total Protein

Albumin

Albumin – Globulin (A/G) ratio

Serum urea nitrogen

Creatinine

Sodium

Potassium

Bicarbonate

Histopathology of gastric mucosal polyp biopsy, liver and kidney in selected cases.

3.4 Outline of study

3.4.1 Signalment and anamnesis

Age, breed and sex of the patients were noted. Present and past history, frequency of vomiting and colour and consistency of the vomitus were recorded in the proforma prepared (Appendix).

3.4.2 Clinical examination

General clinical examination of all animals under study was carried out as described by Kelly (1974).

3.4.3 Hydration status

Hydration status was assessed by estimating capillary refill time (CRT), degree of sunken eye balls and skin turgor and estimating volume of packed red cells (VPRC) as suggested by Greco (1998).

3.4.4 Ultrasonography

3.4.4.1 Equipment

Ultrasound scanning of abdomen was carried out using "L&T Symphony 4.0 ultrasound scanner" having 3.5, 5.0 and 7.5 MHz mechanical sector transducer. Permanent records of the scanning was made by recording in a Video Cassette Recorder

(VCR) and still pictures were taken from the monitor after freezing the picture.

3.4.4.2 Ultrasound scanning procedure

The hair on the ventral abdomen was clipped and the animal was placed on dorsal recumbency(Plate 1) for scanning. Occasionally the animal was put on lateral recumbency or made to stand to facilitate visualization of the organ of interest. Coupling gel was applied liberally to the skin to displace air before scanning.

The transducer was placed on the skin and moved cranial to caudal abdomen and from left to right side to make scans in longitudinal and transverse planes of the entire abdomen. The echo patterns were observed on the monitor. Absence of echo produced black dots (referred as anechoic) and return of a strong echo as white dots (referred as hyperechoic). Echos of intermediate intensity were represented on the monitor as gray shades of dots. Stomach was visualized by instilling water @ 15 millilitres per kilogram body weight of the patient to displace gas using a stomach tube. No special patient preparation was done to evaluate liver and kidney. The animal's position was altered to displace gas and to aid better visualisation of these organs when required.

Multiple sweeps of these organs in both longitudinal and transverse planes were taken to assess and characterise any lesions as per Nyland and Mattoon (1995). Measurements were obtained using the inbuilt electronic callipers wherever required.

3.5 Radiography

Radiography of abdomen was carried out using Seimens Heliphos X – Ray machine with exposure factors and animals position varying with the animal and condition of the patient.

3.5.1 Plain radiography

Plain lateral/ventro-dorsal radiographs of abdomen were taken in all selected cases.

3.5.2 Contrast radiography

3.5.2.1 Gastrointestinal barium series

Micropulverized barium sulphate* was used for contrast radiographic studies of the gastrointestinal tract. After

* Microbar® 96 per cent w/v, *M/S* Eskay fine chemicals, Mumbai

administration of 50-100 ml of barium suspension orally the radiographs were taken at 30 minutes interval until the lesion was identified or completely emptied, as described by Dennis (1992).

3.5.2.2 Intravenous pyelography

A mixture of sodium and meglumine salts of diatrizoate* was used for intravenous pyelographic studies. Low volume rapid infusion technique without abdominal compression was carried out by injecting contrast agent @ 850 milligrams iodine per kilogram body weight as rapidly as possible into the cephalic vein. Ventro-dorsal radiographs were taken at 10 seconds, 1 minute, 3 – 5 minutes and 15 minutes post injection of contrast agent as described by Ticer (1984).

3.6 Clinical pathology

3.6.1 Collection of clinical materials

Blood was collected in clean and dry vials by puncturing the saphenous/cephalic vein. Sodium citrate was used as the anticoagulant. Blood smear was prepared on a clean and dry glass slide with a drop of blood collected from the ear tip.

* Trazograf 76 per cent 20 ml amp.

In a clean and dry tube blood was collected without anticoagulant and serum was separated for determination of serum biochemistry.

Tissue specimens were collected during surgery or post mortem and samples were fixed in 10 per cent neutral buffered formalin.

3.6.2 Examination of clinical material

3.6.2.1 Haematology

Haematological parameters viz., Hb, VPRC, TEC, TLC and DLC were estimated as described by Schalm *et al.* (1975).

3.6.2.2 Serum biochemistry

Biochemical analyses were carried out using photometer 5110 (Boehringer Manheim) under standard conditions of operation. Commercially available reagent kits were used for the assay.

Alanine amino transferase was estimated by the method described by Reitman and Frankel (1957)[1]. Alkaline phosphatase was determined by the method described by Bowers and McComb (1975)[2].

Serum total protein was determined by modified Biurets method (Gornall *et al.*, 1949)[1]. Serum albumin was estimated by bromocresol green dye binding method described by Doumas (1971)[1].

Serum urea nitrogen were estimated by diacetyl monoxime method (Marsh *et al.*, 1965)[1].

Serum creatinine was estimated by Jaffe's alkaline picrate method (Slot, 1965) [1].

Serum bicarbonate was estimated by phosphoenol pyruvate carboxylase method cited by Tiezt (1976)[3].

Serum sodium and potassium were analysed by emission flame photometry as described by Oser (1971).

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1 *M/S* Qualigens Diagnostics, Mumbai
2 M/S Nicholas Piramal, Mumbai
3 Erba test kit, *M/S* Trans Asia Biomedicals Limited, Daman

3.6.2.3 Histopathology

The tissue after complete fixation was processed by routine paraffin embedding techniques as described by Sheehan and Hrapchak (1980). The sections were stained with Haematoxylin and Eosin as per techniques followed by Bancroft and Cook (1984) for evaluation of the histological changes.

3.7 Statistical analysis

Data obtained were analysed statistically as per Snedecor and Cochran (1994). The means of all the groups were compared with that of the control using analysis of variance (ANOVA).

Results

4. RESULTS

The selected dogs were subjected to detailed examination and all parameters under study viz., signalment, history, physical examination, hydration status, ultrasonography, radiography, haematology, serum biochemistry and wherever possible histopathology were carried out. The data obtained were analysed statistically wherever applicable. Qualitative data were presented as such.

The twenty dogs under detailed study were grouped based on the type of affection into four groups:

Group I (n=6) - Consisted of dogs with gastritis

Group II (n=6) - Consisted of dogs with gastrointestinal tract obstructions

Group III(n=4) - Consisted of dogs with renal disorders

Group IV (n=4) - Consisted of dogs with hepatic and uterine disorders. Of the dogs in this group, two had cirrhosis, one had cholangitis, and one had pyometra. Since the dogs with liver affections had wide variations in the data, they were not subjected to statistical analysis and data were presented as such.

4.1 Occurrence

In the present study the occurrence of vomiting in dogs was recorded as 16.78 per cent, of which 47.35 per cent had gastroenteritis.

4.2 Colour and consistency of vomitus

Group I

Colour and consistency of vomitus varied in this group, yellowish watery in three dogs, frothy white in two dogs and yellowish to brownish colour with blood clots in one.

Group II

In dogs with intestinal intussusception and those with stone as the intestinal foreign body, vomitus was watery and yellowish.

Dogs with pyloric stenosis had frothy or watery white vomitus and were mixed with food.

Group III

Dogs of this group had watery and yellowish vomitus with blood tinge in one animal.

Group IV

Dogs with cirrhosis and pyometra had watery yellowish vomitus while dog with cholangitis had watery vomitus mixed with blood.

4.3 Frequency of vomiting

Group I

Dogs of this group vomited two to seven times per day and duration of illness ranged from two to four days. Vomiting was seen mostly after food or water intake.

Group II

Dogs with intussusception vomited two to three times daily and period of illness ranged from three days in one dog to seven days in another dog.

Vomiting in dogs with pyloric stenosis was seen immediately to 30 minutes post feeding with the period of illness of nearly one month in both the dogs.

Dogs with stone as intestinal foreign body vomited two to seven times a day with the period of illness of one week in each dog.

Group III

Dogs of this group vomited once to thrice daily and duration of illness ranged from five to fourteen days.

Group IV

Dogs with cirrhosis had vomiting occasionally i.e., once in two to three days. Dog with cholangitis had vomiting two to three times daily. Dog with pyometra vomited immediately after food intake.

4.4 Physical examination

Group I

In dogs of this group abdomen appeared tucked up and on palpation abdominal muscles were held tensely. Pain in the epigastric area was evident in four of the six dogs examined.

Dogs of this group had varying degrees of dehydration as evident from elevated capillary refill time (2-5 seconds), skin turgor and sunken eyeballs (Table 1).

Group II

Abdominal palpation in dogs with intussusception revealed tubular mass intra-abdominally with animal evincing pain on palpation at that area.

In dogs with stone as the intestinal foreign body, a round hard mass was palpabale in mid-abdomen.

In one dog with pyloric stenosis there was marked distention of the left flank and on palpation appeared as a fluid filled structure.

In five of the six dogs in this group dehydration was evident. Elevated capillary refill time (CRT)(2-4 seconds), skin turgor and sunken eyeballs of varying degrees were observed (Table 1).

Group III

No abnormalities were detected on abdominal palpation.

Dehydration in three of the four dogs was evident by elevated CRT (2-3 seconds), skin turgor and sunken eyeballs of varying degrees (Table 1).

Group IV

In two dogs with cirrhosis abdomen was markedly distended with fluid. Fluid thrill was appreciable on tactile percussion in one and in the other, abdominal palpation did not give any clue of fluid in the abdominal cavity.

A tubular mass in the posterior abdomen was palpable in the dog with pyometra.

4.5 Ultrasonography

Group I

No abnormalities were detected ultrasonographically in this group. The stomach wall thickness measurements in dogs of this group ranged from 4.3 millimetres to 5.6 millimetres (Plate 2).

Group II

Ultrasound scanning detected five of the six cases with gastrointestinal tract obstruction.

In a dog with intussusception a picture of hyperechoic centre with a single hypoechoic rim surrounding it in cross section was obtained (Plate 3). While in another dog the typical concentric ring appearance on cross section was obtained (Plate 4). In both the cases a multi-laminar appearance was appreciable on longitudinal section (Plate 5).

In two dogs with stone as the intestinal foreign body, ultrasound produced curvy linear hyperechoic area with posterior acoustic shadowing (Plate 8).

In one dog with pyloric stenosis, stomach wall showed generalised thickening, measuring 24.7 mm (Plate 10) and there was circumferential thickening of pylorus with a small lumen (Plate 11). Ultrasound scanning did not give any clue to obstruction/stenosis in pylorus in another dog.

Group III

Normal nephrosonographic architecture of a central bright echo complex corresponding to renal sinus and peripelvic

fat, a hypoechoic region surrounding the pelvis representing renal medulla and an outer zone of intermediate echogenicity of the renal cortex was not appreciable in all dogs in this group (Plate 14). The corticomedullary junction was indistinguishable. In three of the four cases the parenchyma was hypoechoic, while in the other, right kidney appeared hyperechoic than liver parenchyma (Plate 15).

Group IV

In two dogs with cirrhosis, ultrasound showed that liver lobes were nodular with lobes floating in the ascitic fluid (Plate 16). In both these dogs, gall bladder appeared distended with thickened wall (Plates 17 & 18).

In a dog with cholangitis, liver parenchyma had same echointensity as that of the cortex of the kidney (Plate 19) and gall bladder was markedly distended (Plate 20).

In a dog with pyometra, uterus was visualised ultrasonographically as two round anechoic areas with echogenic debris dorsal to the urinary bladder (Plate 21).

4.6 Radiography

4.6.1 Plain radiography

Group I and III

Plain radiographs did not give any clue about the abnormalities.

Group II

Plain radiographs detected the cause of obstruction in two of the six dogs in this group. Radio-opaque foreign bodies (stone) were detected in both these dogs (Plate 9).

In dogs with intussusceptions, plain radiographs showed gas filled distended intestinal loops (Plate 6).

In one dog with pyloric stenosis, stomach was filled with fluid and gas while in the other dog stomach appeared to be distended with gas (Plate 12).

Group IV

In dogs with cirrhosis and ascites, entire abdomen showed uniform density and had a ground glass appearance.

An irregular soft tissue density suggestive of enlarged uterine horns was seen in the dog with pyometra in posterior abdomen (Plate 22).

4.6.2 Contrast radiography

Gastrointestinal barium series could detect obstruction in dogs with intussusceptions. Flow of barium sulphate was obstructed one and a half hours after administration in front of a lucent appearing mass (Plate 7).

There was no gastric emptying of barium in dogs with pyloric obstruction (Plate 13) even after one hour of administration.

Intravenous pyelography was performed in one dog with renal problem. There was no opacification of kidney or excretion of the contrast agent even after 15 minutes of administration.

4.7 Clinical pathology

4.7.1 Hematology

The mean ± SE of haematological values of control and clinical cases are given in Table 2.

4.7.1.1 Haemoglobin

There was no statistically significant difference in the mean value of haemoglobin of groups I and II with that of the control dogs, but the mean values of haemoglobin were slightly higher than control.

The mean value of hemoglobin in group III was significantly lower.

Hemoglobin in the two dogs with cirrhosis was 16.6 and 14.0 grams per decilitre, 14 grams per decilitre in the dog with cholangitis and 16 grams per decilitre in the dog with pyometra.

4.7.1.2 Volume of packed red cells (VPRC)

VPRC was significantly higher than control dogs in groups I and II. There was no statistically significant difference between means of group III and the control dogs, but the mean value of VPRC in group III was less than control dogs.

VPRC was 38 and 34 per cent in the two dogs with cirrhosis, and 38 per cent each in the dogs with cholangitis and pyometra.

4.7.1.3 Total Erythrocyte Count (TEC)

There was no significant difference in the mean value of TEC of groups I and II with that of the control dogs, but the mean value of TEC of both these groups were lower than that of the control dogs.

The mean value of TEC of group III dogs was significantly lower than that of the control dogs.

In the two dogs with cirrhosis, TEC was 6.28 and 4.68 million cells per microlitre, in the dog with cholangitis 4.22 million cells per microlitre and in the dog with pyometra 6.23 million cells per microlitre.

4.7.1.4 Total Leucocyte Count (TLC)

Dogs of group II had mean value of TLC significantly higher while there was no statistically significant difference in the mean value of TLC of groups I and III with that of the control.

In dogs with cirrhosis, TLC was higher than the control dogs. The values were 21550 and 18500 cells per micro litre respectively.

Leucocytosis was evident in the dogs with cholangitis and pyometra with values of 15350 and 25800 cells per microlitre respectively.

4.7.1.5 Differential Leucocyte Counts (DLC)

The mean value of neutrophils in group II dogs were significantly higher while there was no statistically significant difference in the means of groups I and III with that of the control dogs.

Dogs with cirrhosis had neutrophils higher than the control dogs (18533 and 15170 cells per microlitre respectively).

Neutrophilia was seen in the dogs with cholangitis and pyometra (13047.5 and 21156 neutrophils per microlitre). The dog with pyometra had 4231.2 immature neutrophils out of the 21156 neutrophils per micro litre of blood indicating left shift.

There was no statistically significant difference between the means of lymphocytes, monocytes and eosinophils of the various groups with that of the control dogs.

Dogs with cirrhosis, cholangitis, and pyometra had lymphocytes, monocytes and eosinophils within the normal range.

4.7.2 Serum biochemistry

The mean ± SE of serum biochemical values of control and clinical cases are given in Table 3.

4.7.2.1 Alanine Amino Transferase (ALT)

There was no statistically significant difference in the mean value of ALT of groups I, II, and III with that of the control dogs.

ALT was 270 and 10 units per litre respectively in the dogs with cirrhosis, 44 units per litre in the dog with cholangitis and 32 units per litre in the dog with pyometra.

4.7.2.2 Alkaline Phosphotase (ALP)

There was no statistically significant difference in the mean values of ALP in groups I, II, and III with that of the control dogs.

The two dogs with cirrhosis had ALP values of 200 and 520 units per litre. In the dog with cholangitis it was 6468 units per litre and 10 units per litre in the case of pyometra.

4.7.2.3 Total protein

There was no statistically significant difference in the mean values of total protein in groups I, II, and III with that of the control groups of dogs.

Total proteins in dogs with cirrhosis were 6.83 and 5.68 grams per decilitre respectively. In the dog with cholangitis it was 6.4 grams per decilitre and 6.0 grams per decilitre in the dog with pyometra.

4.7.2.4 Albumin

There was no statistically significant difference in groups I, II, and III with that of the control dogs in mean values of serum albumin.

Serum albumin was low at one gram per decilitre each in the dogs with cirrhosis. It was found to be within the normal

range in the dogs with cholangitis and pyometra with values of 2.1 and 3.0 grams per decilitre.

4.7.2.5 Albumin – globulin (A/G) ratio

There was no statistically significant difference in the mean values of A/G ratio in the dogs of groups I, II, and III with that of the control group of dogs.

A/G ratio was low in the two dogs with cirrhosis at 0.17 and 0.21 respectively. A/G ratio in dogs with cholangitis and pyometra was found to be in the normal range with values of 0.45 and 1.0 respectively.

4.7.2.6 Serum urea nitrogen (SUN)

There was no statistically significant difference between the mean value of SUN of group I and control.

The mean value of SUN in groups II and III were found to be significantly higher than control dogs with values of 47.83 ± 10.18 and 106.63 ± 5.70 milligrams per decilitre respectively.

Dogs with cirrhosis had SUN of 34 milligram per decilitre each and dogs with cholangitis and pyometra had 32.22 and 25 milligram per decilitre respectively.

4.7.2.7 Creatinine

The mean value of creatinine in groups I and II had no statistically significant difference with that of the control group of dogs.

The mean value of creatinine in dogs of group III was 10.00 ± 2.20 milligrams per decilitre and was found to be significantly higher than dogs in the control group.

Dogs with cirrhosis had creatinine value of 0.9 milligrams per decilitre each and dogs with cholangitis and pyometra had creatinine values of 0.2 and 0.3 milligrams per decilitre respectively.

4.7.2.8 Serum sodium

There was no statistically significant difference between means of serum sodium level of groups I, II, III and

control dogs. The mean values of serum sodium of all the three groups were less than the mean value of the control.

Serum sodium was 134.41 and 196.57 milliequivalents per litre respectively in dogs with cirrhosis and 189.70 and 173.99 milliequivalents in dogs with cholangitis and pyometra respectively.

4.7.2.9 Serum potassium

The mean ± SE values of serum potassium recorded in groups I, II and III were 3.80 ± 0.23, 3.68 ± 0.39 and 6.94 ± 0.57 milliequivalents per litre respectively. The mean value of potassium in group III was significantly more than that of the control dogs and significantly lower in the dogs of groups I and II.

Serum potassium levels was low in dogs with cholangitis and pyometra with values of 3.27 and 3.79 milliequivalents per litre respectively and was within the normal range in dogs with cirrhosis (4.80 and 5.36 milliequivalents per litre respectively).

4.7.2.10 Bicarbonate

The mean values of serum bicarbonate in groups I and II were significantly elevated compared to control group dogs (129.00 ± 13.19 and 116.833 ± 12.34 milliequivalents per litre respectively).

There was no statistically significant difference between the mean value of serum bicarbonate in group III and control dogs.

The serum bicarbonate in the dogs with cirrhosis was 53 and 48 milliequivalents per litre respectively. It was 39 milliequivalents per litre in the dog with cholangitis and was high in the dog with pyometra with a value of 124 milliequivalents per litre.

4.7.3 Histopathology

Histopathology was carried out in one dog with pyloric stenosis, one dog with cirrhosis and three dogs with renal disorders.

Histopathology of mucosal polyp biopsy obtained in a dog with pyloric stenosis showed hyperplasia of the villi, proliferation of goblet cells, infiltration of inflammatory cells and focal areas of hemorrhages (Plate 23).

Fibrous tissue proliferations in parenchyma, severe vacuolar degeneration, moderate fatty changes in the centrilobular areas of hepatocytes with loss of hepatic architecture were the histological features in liver of the dog with cirrhosis (Plate 24).

Chronic interstitial nephritis was noted histopathologically in the three dogs with renal disorders. The histological changes observed were severe fibrous tissue proliferation and infiltration of mononuclear cells, vacuolar degeneration of the tubules, calcification of glomerular and tubular basement membranes in two dogs (Plates 25 & 26).

Table 1: Assessment of hydration status (mean ± SE)

	Volume of packed red cells (VPRC) %	Capillary refill time (CRT) in seconds	Skin turgor in seconds	Degree of sunken eye balls	Degree of dehydration (%)
Controls	34.00 ± 1.184	<1.00 to 1.00	<1.00 to 1.00	0	0
Group I	41.833 ± 2.993	3.17 ± 1.20	4.83 ± 1.97	++	7
Group II	41.50 ± 2.499	2.67 ± 1.00	3.33 ± 1.35	++	5
Group III	31.250 ± 1.435	3.25 ± 1.54	4.50 ± 2.29	++	7

Table 2: Hematology of dogs – control and clinical cases (mean ± SE)

Parameters	Control	Group I	Group II	Group III
Hemoglobin (g/dl)	14.833 ± 0.478	17.250 ± 1.139	16.017 ± 1.139	10.425 ± 0.625**
Volume of Packed Red Cells (%)	34.000 ± 1.184	41.833 ± 2.993*	41.500 ± 2.499*	31.250 ± 1.435
Total erythrocyte count (million cells/µl)	6.457 ± 0.318	6.297 ± 0.135	5.803 ± 0.351	3.360 ± 0.195**
Total Leucocyte Count (Cells/µl)	10916.667± 853.17	11468.333± 720.93	18746.667± 1853.33**	11850.00 ±603.89
Neutrophils (cells/µl)	7715.083 ± 659.10	8542.883 ± 362.32	14751.067 ± 1867.88**	8911.100 ± 704.55
Lymphocytes (cells/µl)	2911.00 ± 206.38	2742.033 ± 447.46	3551.050 ± 488.28	2763.300 ± 462.57
Monocytes (cells/µl)	225.58 ± 100.27	141.91 ± 74.19	298.50 ± 121.85	94.16 ± 93.63
Eosinophils (cells/µl)	65.0 ± 37.78	41.5 ± 29.36	135.67 ± 88.63	86.50 ± 51.69

* Significant ($P < 0.05$)

** Highly significant ($P < 0.01$)

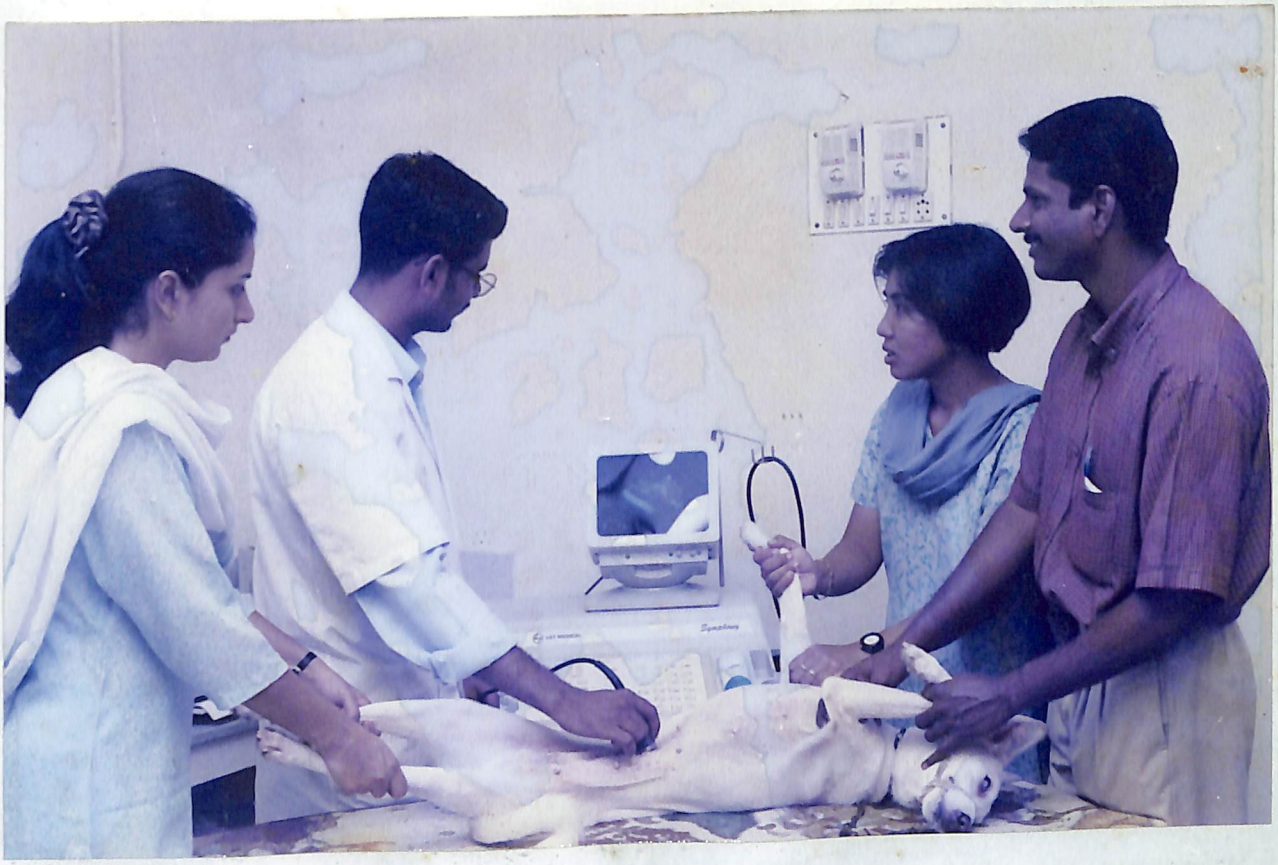
Table 3: Serum Biochemical values of dogs – control and clinical cases (mean ± SE)

Parameters	Control	Group I	Group II	Group III
Alanine Amino Transferase(U/L)	25.00 ± 2.31	23.00 ± 3.35	21.83 ± 4.14	30.50 ± 3.12
Alkaline Phosphatase(U/L)	114.50 ± 2.74	41.67 ± 11.89	87.83 ± 23.15	48.50 ± 9.75
Total protein (g/dL)	6.16 ± 0.16	5.80 ± 0.16	5.50 ± 0.34	6.00 ± 0.41
Albumin (g/dL)	2.33 ± 0.21	3.00 ± 0.00	2.67 ± 0.21	2.75 ± 0.25
Albumin – globulin ratio	0.65 ± 0.17	1.10 ± 0.15	1.02 ± 0.16	1.25 ± 0.16
Serum urea nitrogen (mg/dL)	18.00 ± 1.315	29.238 ± 5.13	47.83 ± 10.18**	106.63 ± 5.70**
Creatinine (mg/dL)	1.067 ± 0.249	0.450 ± 0.127	0.733 ± 0.147	10.000 ± 2.20**
Sodium (mEq/L)	163.12 ± 4.88	144.51 ± 15.47	135.18 ± 13.13	148.62 ± 13.32
Potassium (mEq/L)	5.32 ± 0.29	3.80 ± 0.23**	3.68 ± 0.39**	6.94 ± 0.57
Bicarbonate (mEq/L)	33.50 ± 2.56	129.00 ± 13.19**	116.833 ± 12.34**	26.375 ± 0.745

** Highly significant ($P<0.01$)

PLATE 1
Ultrasound scanning in progress

PLATE 2
Ultrasonographic appearance of stomach distended with water. The different layers of wall are appreciable. L = lumen, stomach wall measures 4.3 mm between the callipers (+).



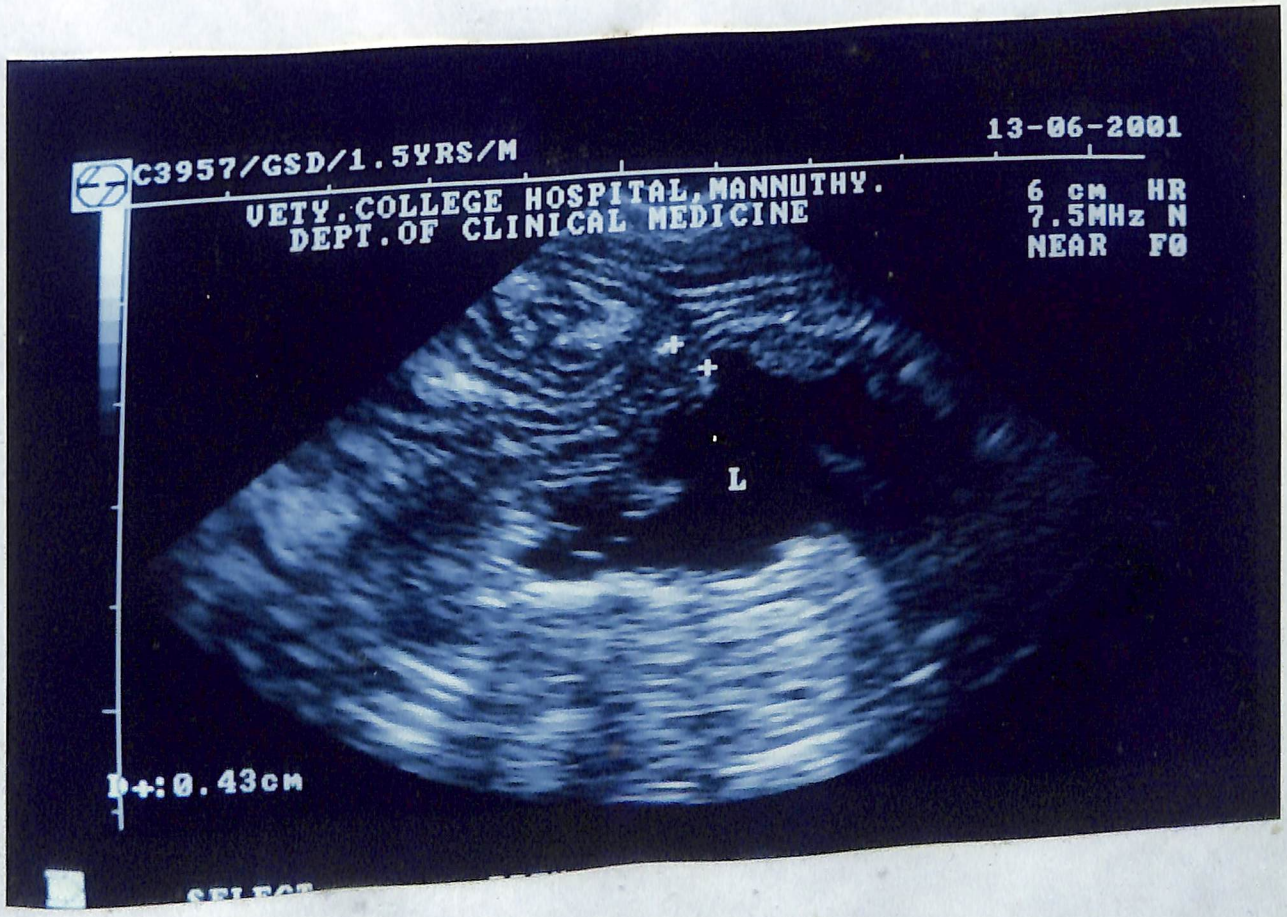
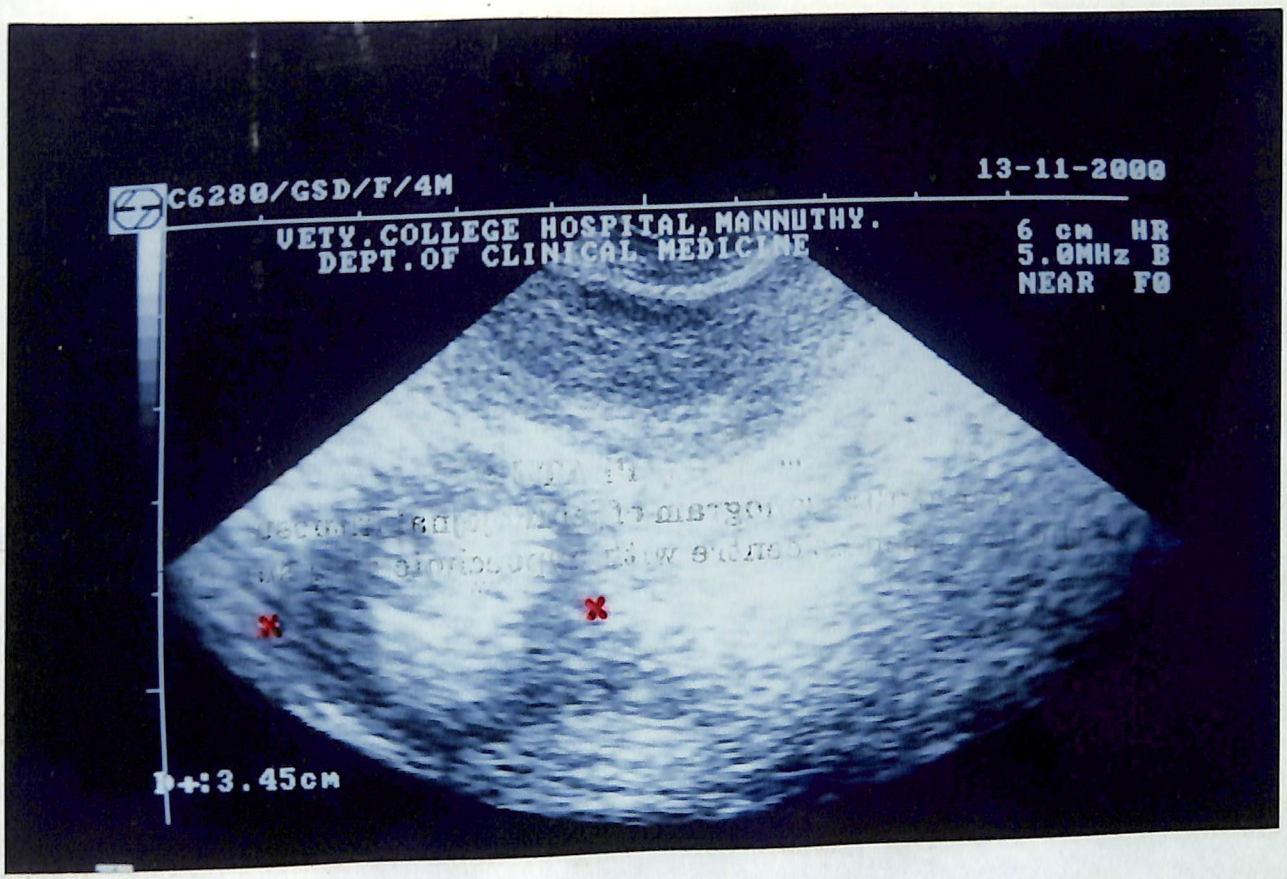

C3957/GSD/1.5YRS/M
13-06-2001
VETY.COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
6 cm HR
7.5MHz N
NEAR FO
L
D+:0.43cm

PLATE 3

Transverse ultrasonogram of jejunojejnal intussusception. Note the hyperechoic centre with hypoechoic ring surrounding it.

PLATE 4

Transverse scan of ileocolic intussusception showing concentric hyperechoic and hypoechoic ring appearance.


C6280/GSD/F/4M
13-11-2000
VETY.COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
6 cm HR
5.0MHz B
NEAR FO
D+:3.45cm

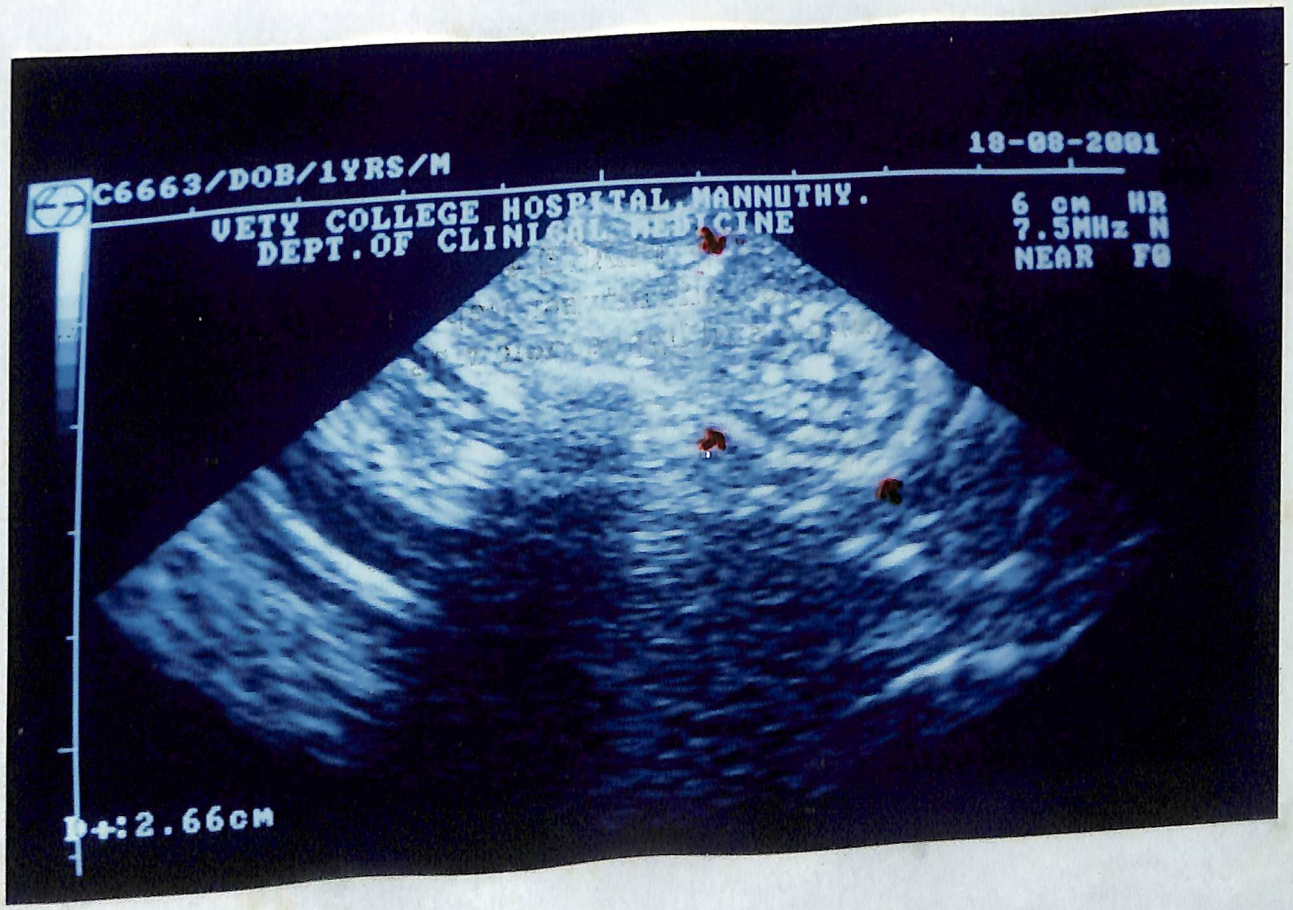
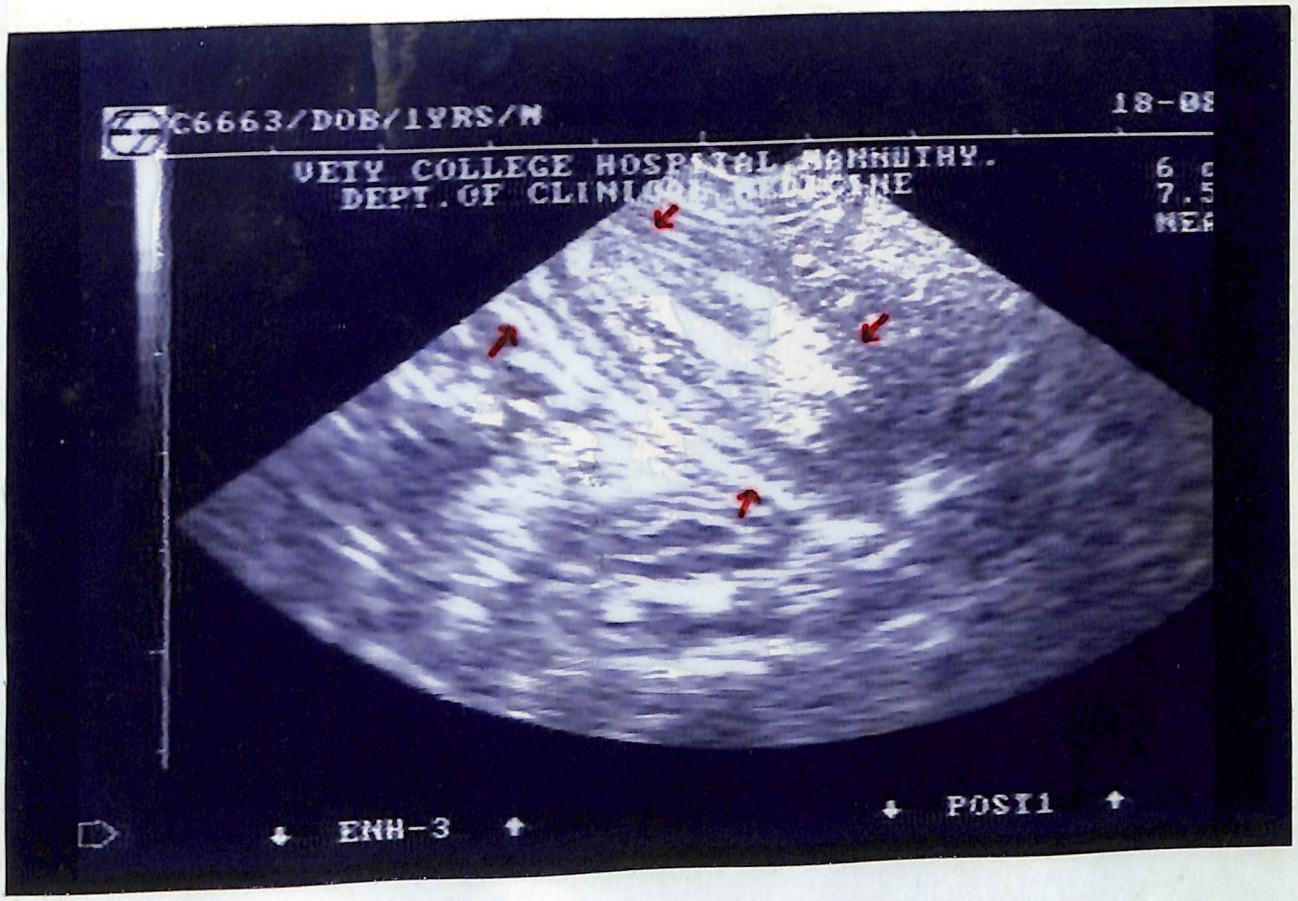

C6663/DOB/1YRS/M
18-08-2001
VETY COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
6 cm HR
7.5MHz N
NEAR FO
D+:2.66cm

PLATE 5

Multilaminar appearance (between arrows) – longitudinal ultrasonogram of ileocolic intussusception represented in plate 4

PLATE 6

Skiagram of lateral radiograph showing gas filled dilated intestinal loops (between arrows) from a dog with intussusception


C6663/DOB/1YRS/M
18-08
ENH-3
POSI1

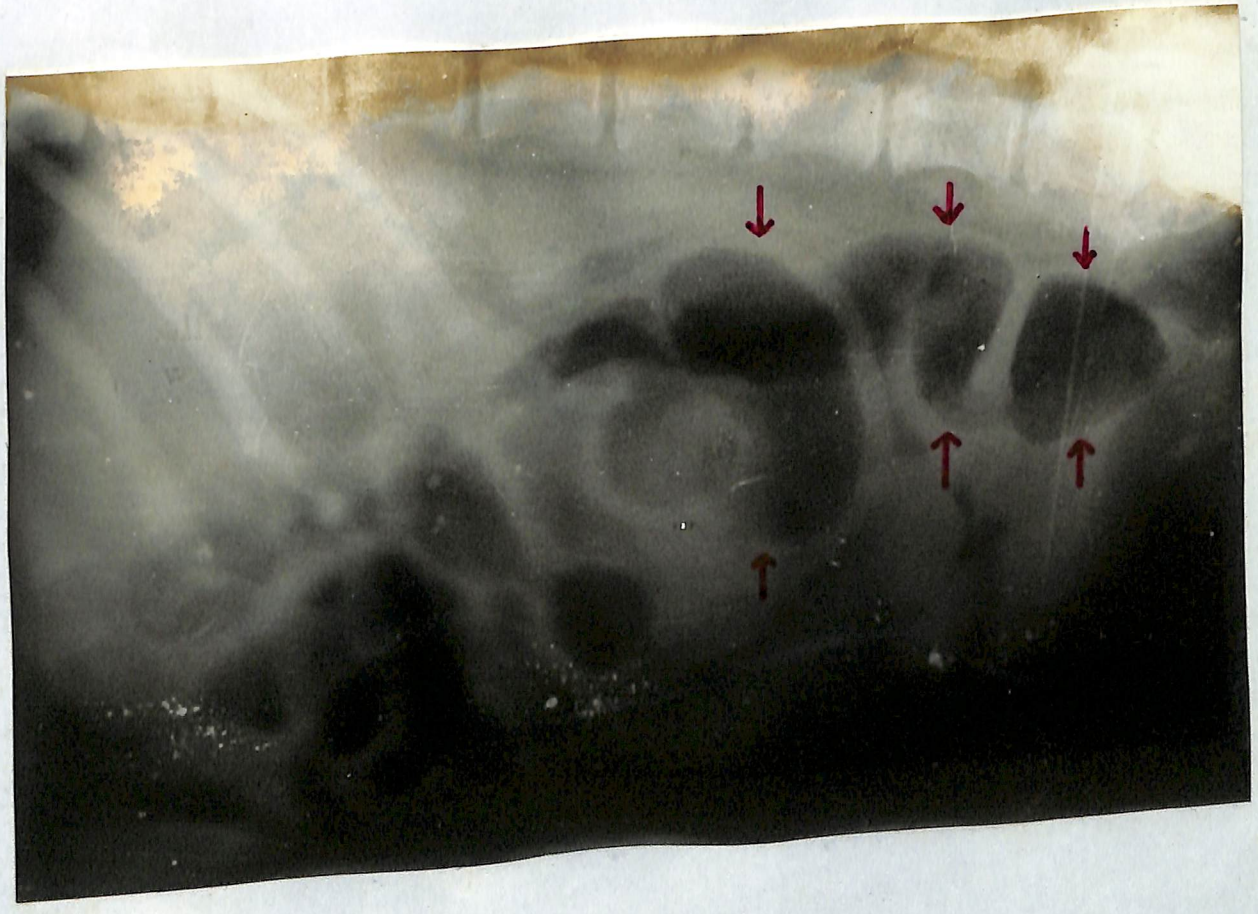
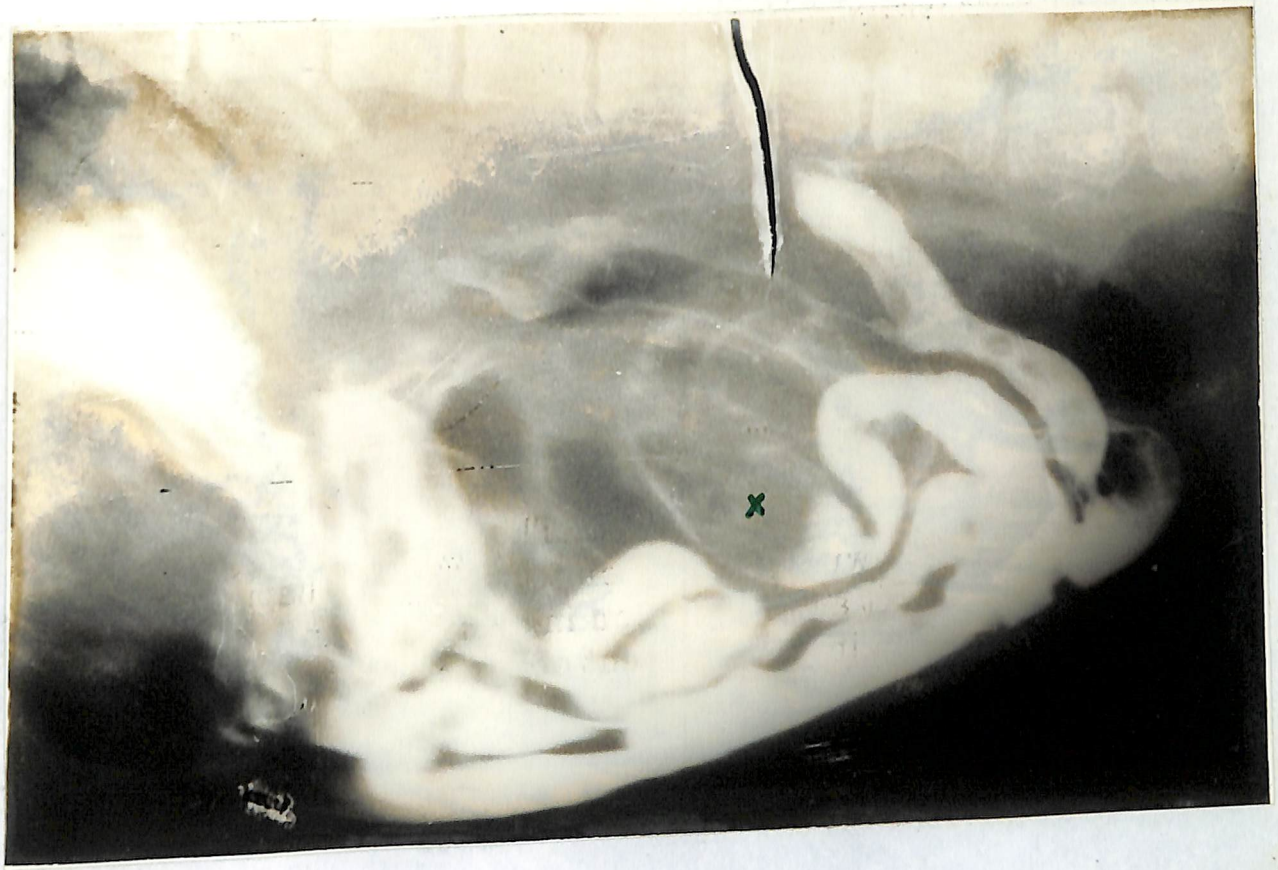


PLATE 7

Skiagram showing obstruction to the flow of barium, one and a half hour post administration in a dog with intussusception. X indicates lucent obstructing mass.

PLATE 8

Curvy linear hyperechoic area between '+' – ultrasonogram of intestinal foreign body (stone). S-acoustic shadowing



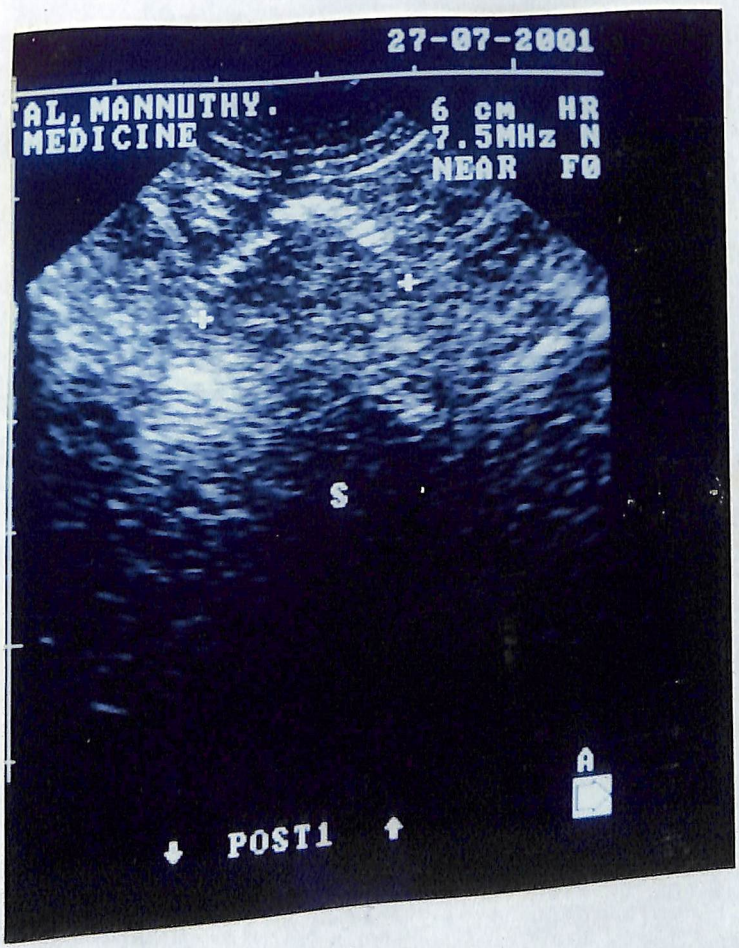
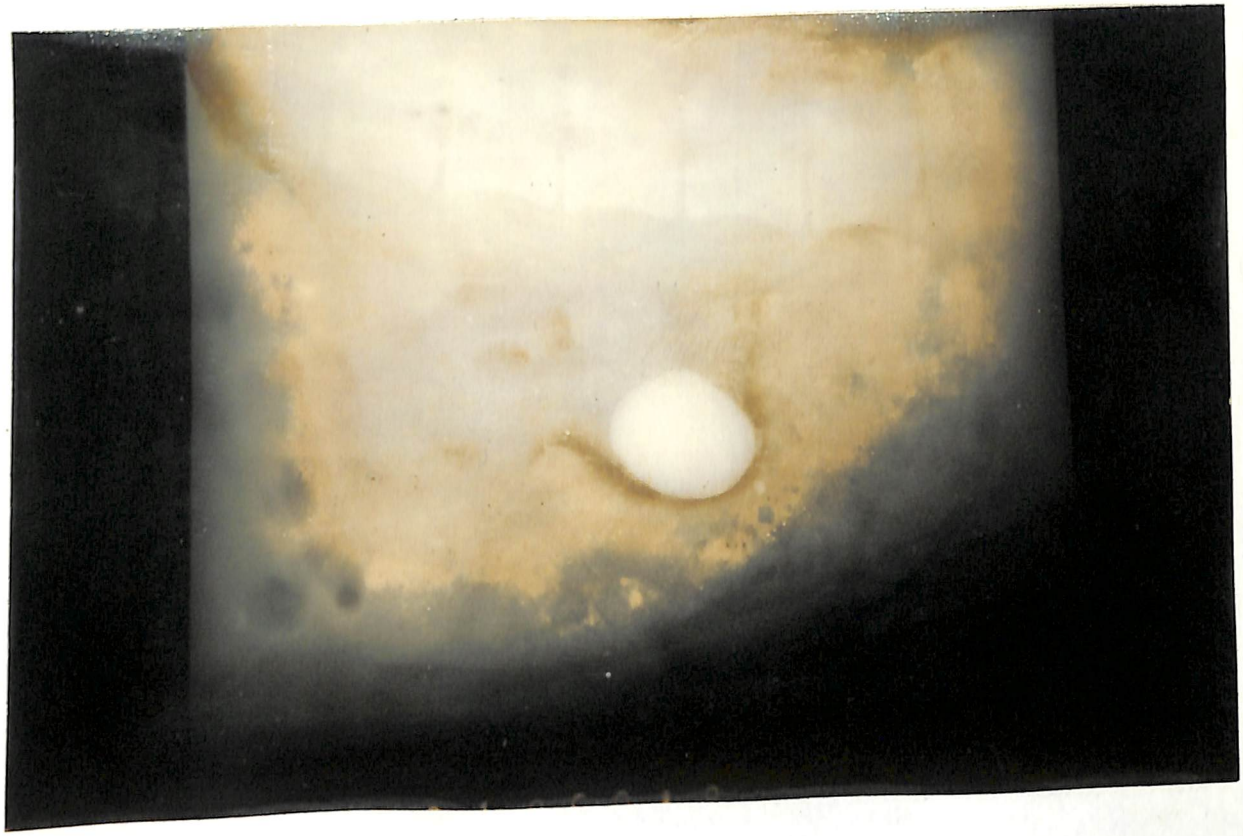

27-07-2001
AL,MANNUTHY.
MEDICINE
6 cm HR
7.5MHz N
NEAR F0
S
A
POST1

PLATE 9
Skiagram showing radio-opaque foreign body in mid-abdomen

PLATE 10
Ultrasonogram showing distended fluid filled stomach (ST).
Stomach wall measures 24.7 mm (between arrows)



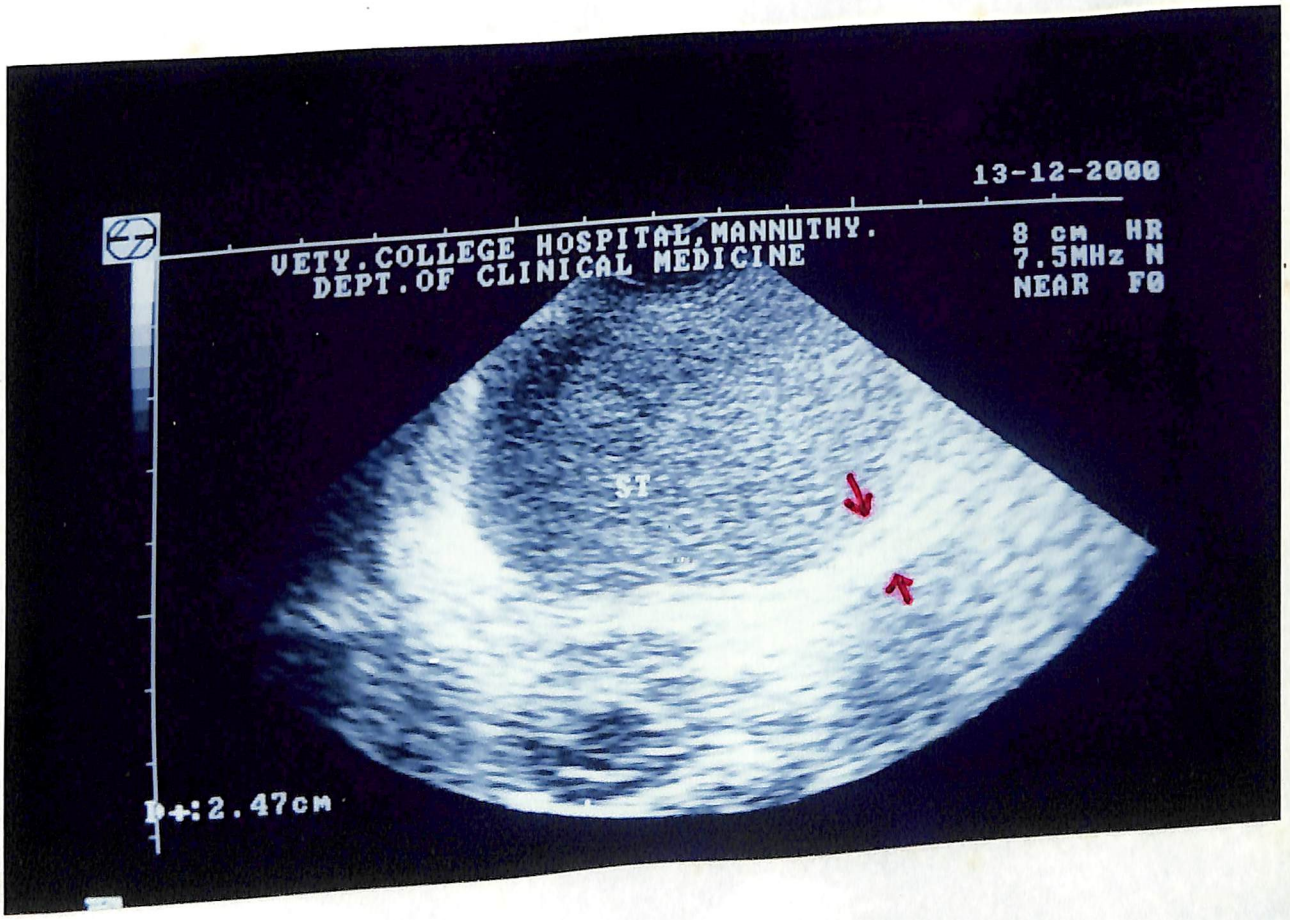
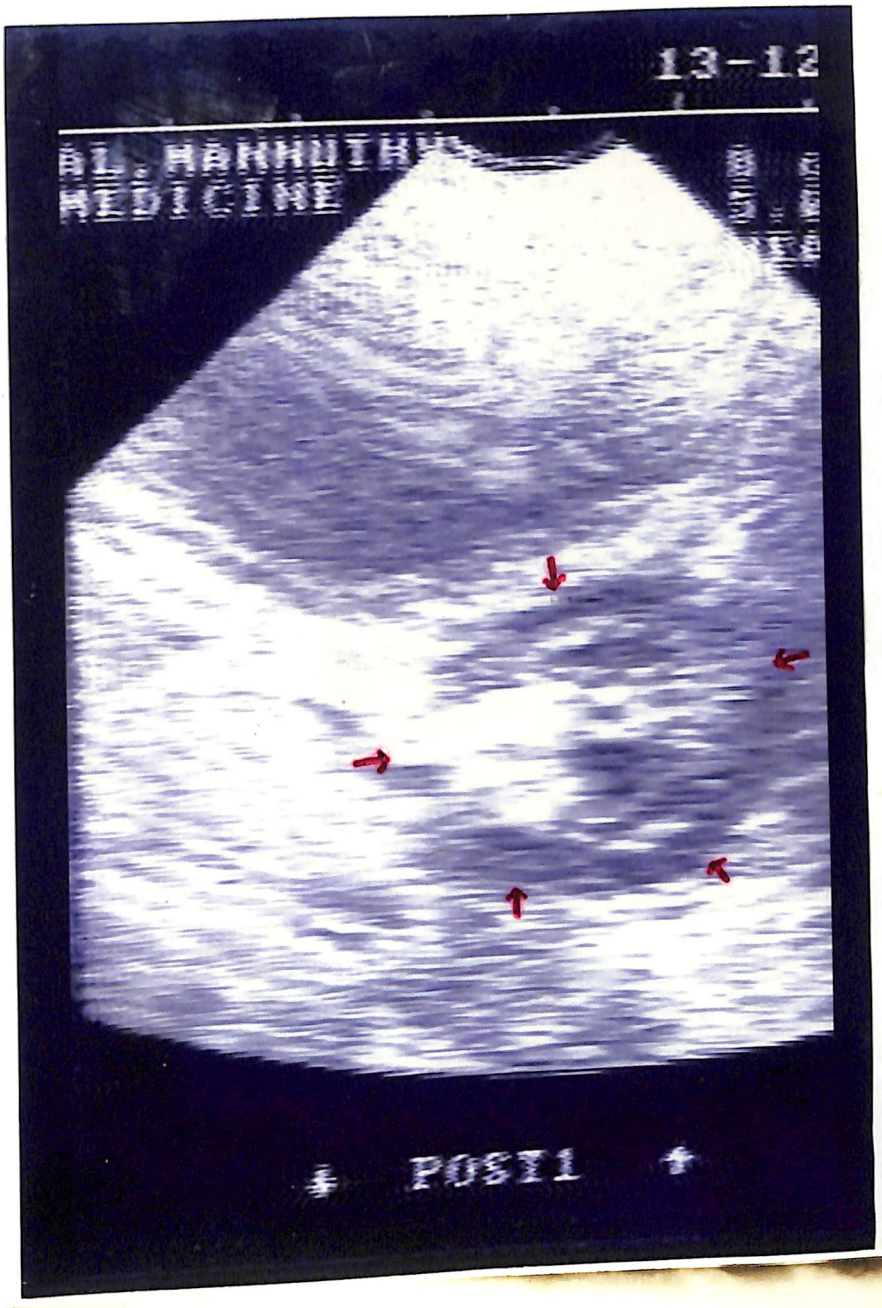

13-12-2000
VETY.COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
8 cm HR
7.5MHz N
NEAR F0
ST
D+:2.47cm

PLATE 11

Transverse scan of pylorus shows circumferential thickening of the pyloric wall (arrows) and small lumen (L)

PLATE 12

Skiagram of the lateral radiograph showing distended gas filled stomach (between arrows)


13-12
AL HAMMUTHY
MEDICINE
POST1

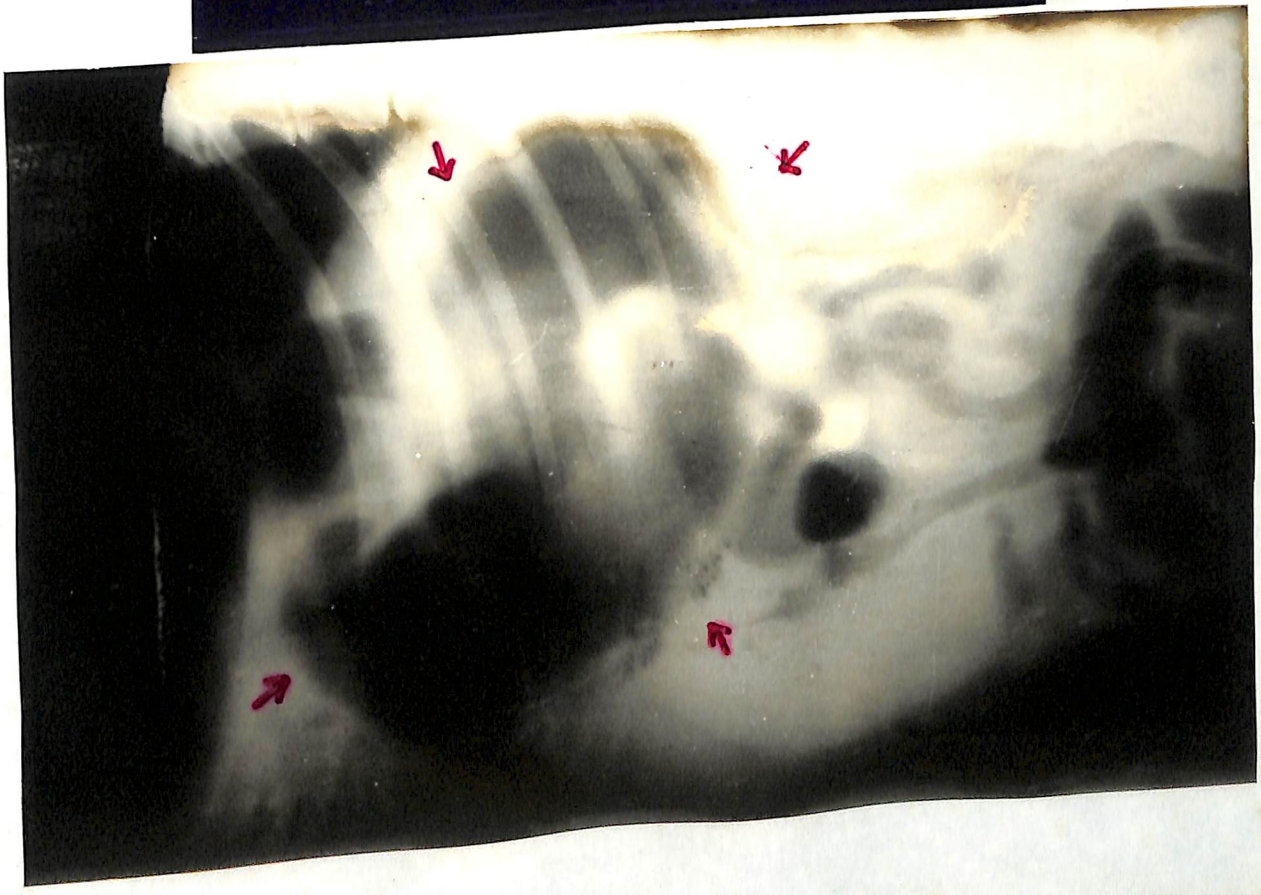
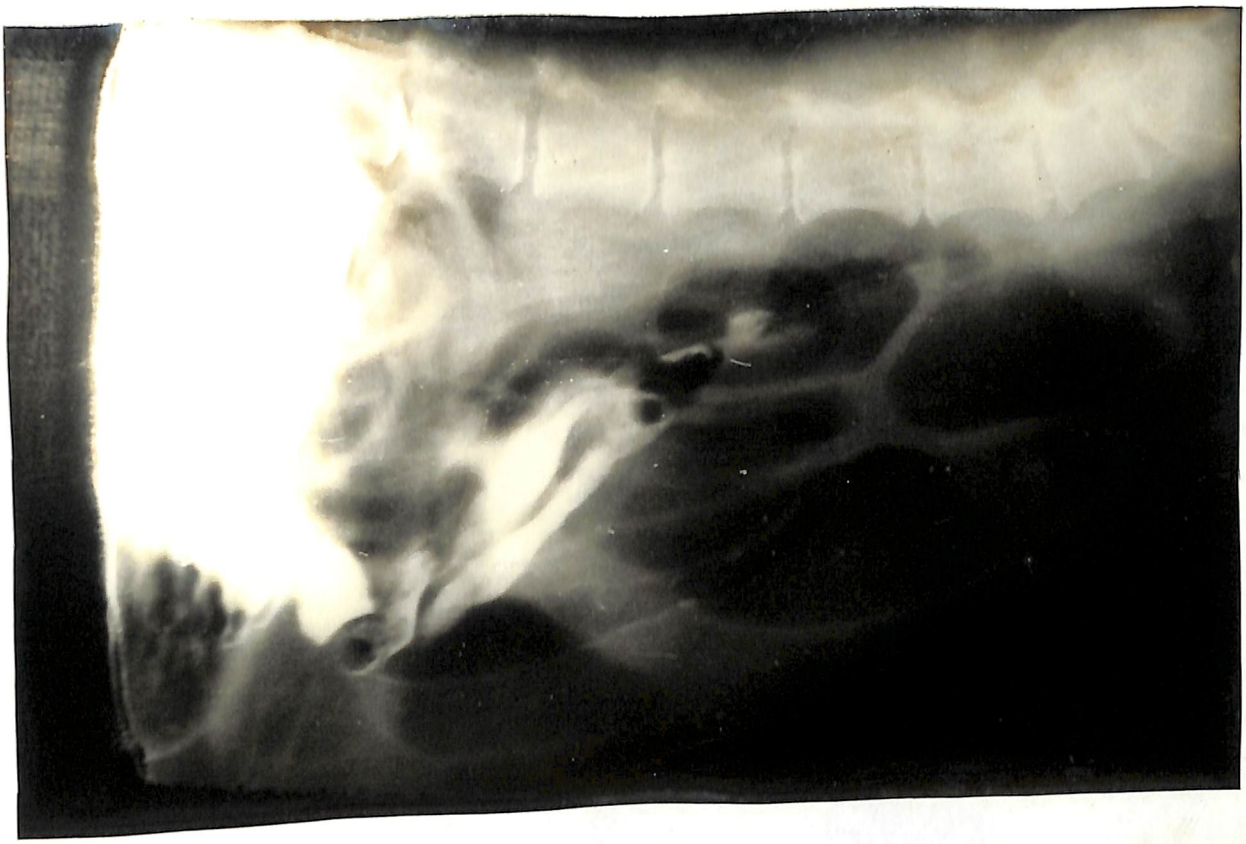


PLATE 13

Skiagram showing delayed emptying of barium from stomach one hour post administration. A small quantity of barium as entered the duodenum

PLATE 14

Ultrasonograms of left kidney of the dog with chronic interstitial nephritis. Note the corticomedullary junction is indistinguishable and normal nephrosonographic architecture is not appreciable



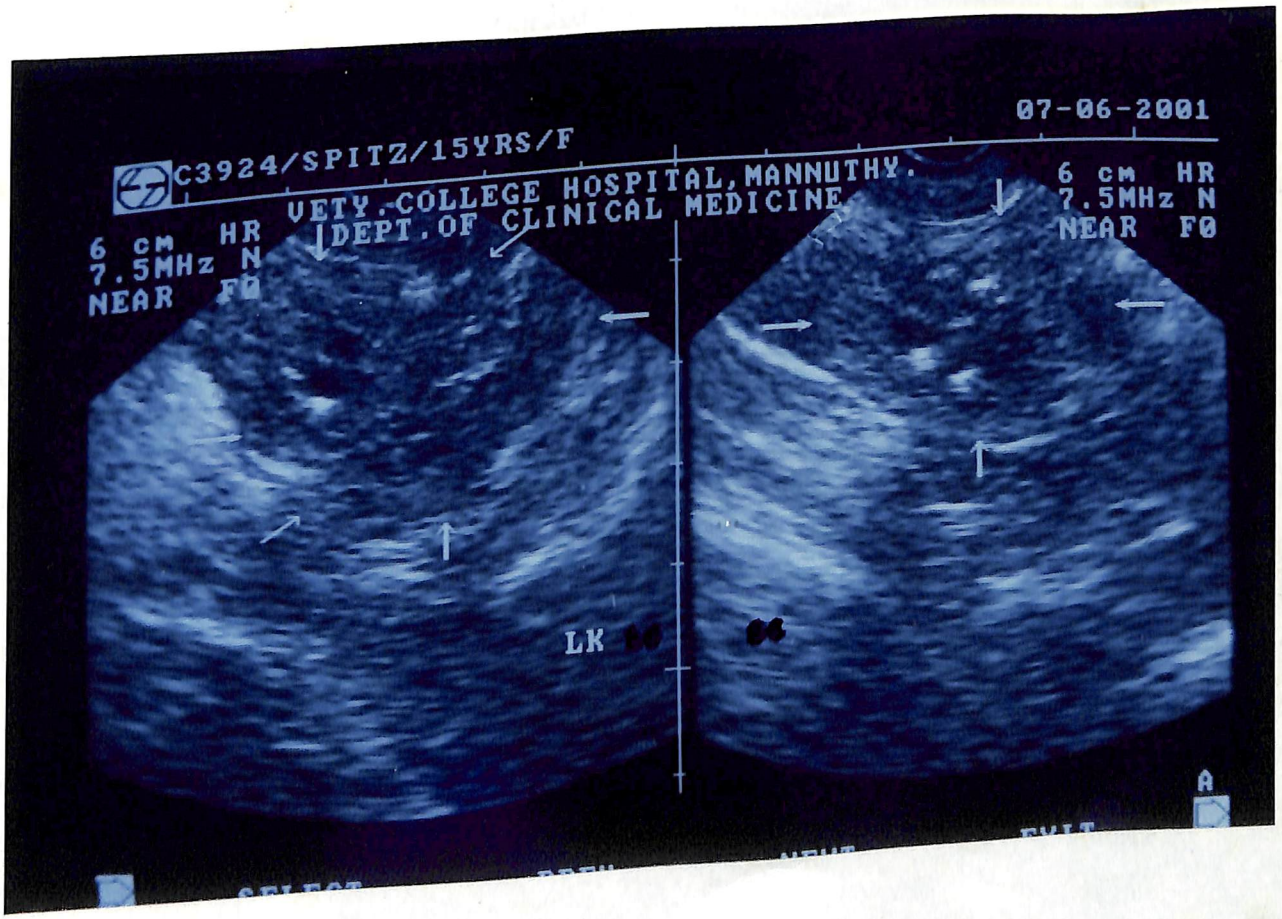
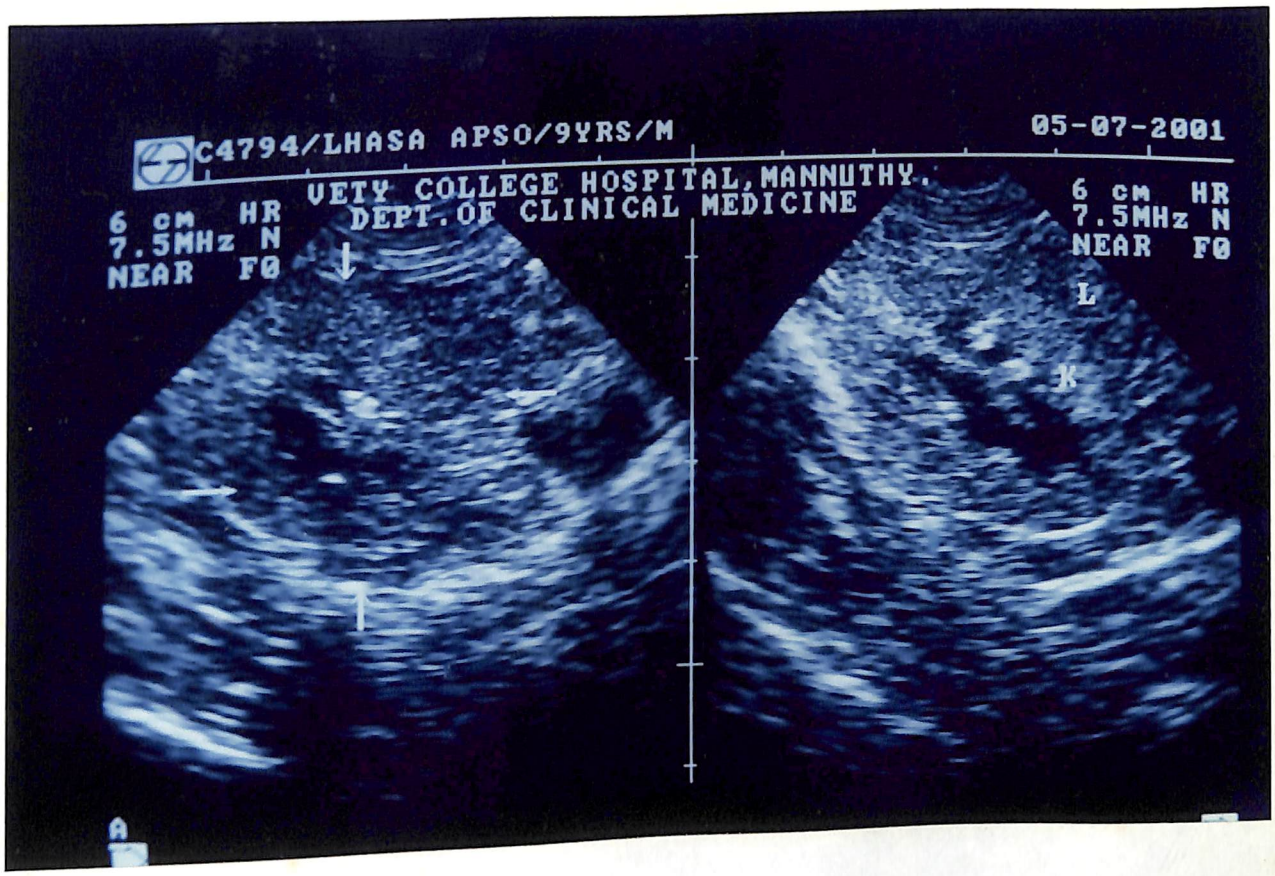

07-06-2001
C3924/SPITZ/15YRS/F
VETY.COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
6 cm HR
7.5MHz N
NEAR F0
6 cm HR
7.5MHz N
NEAR F0
LK
A
EXIT

PLATE 15

Nephrosonogram of dog with chronic interstitial nephritis. The normal architecture of kidney is not appreciable - note that the echointensity of cortex is more than that of liver parenchyma.

PLATE 16

Sonogram showing liver lobe floating in ascitic fluid (PF). Note the nodular and irregular contour of the liver.


C4794/LHASA APSO/9YRS/M
05-07-2001
VETY COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
6 cm HR
7.5MHz N
NEAR F0
6 cm HR
7.5MHz N
NEAR F0
L
K
A

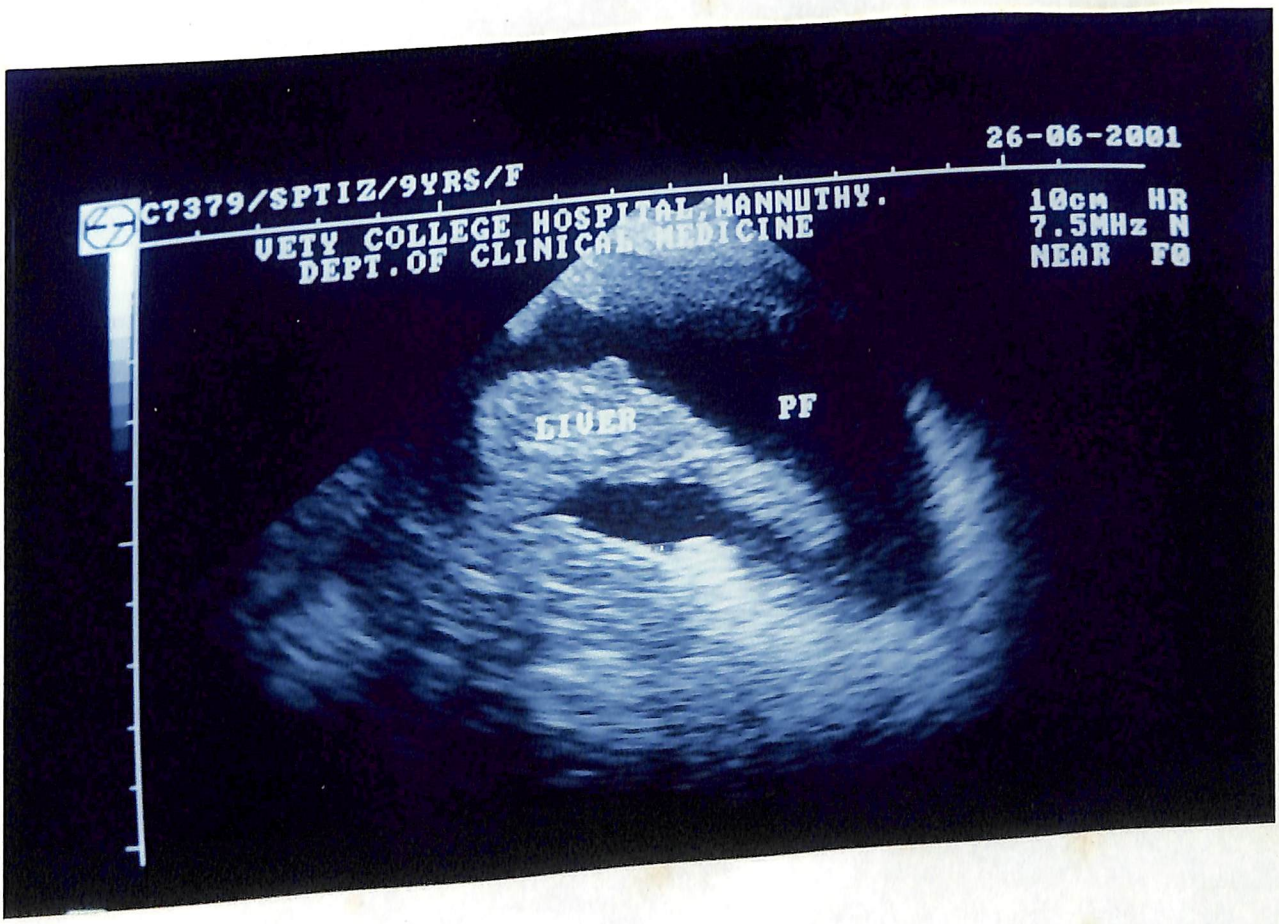
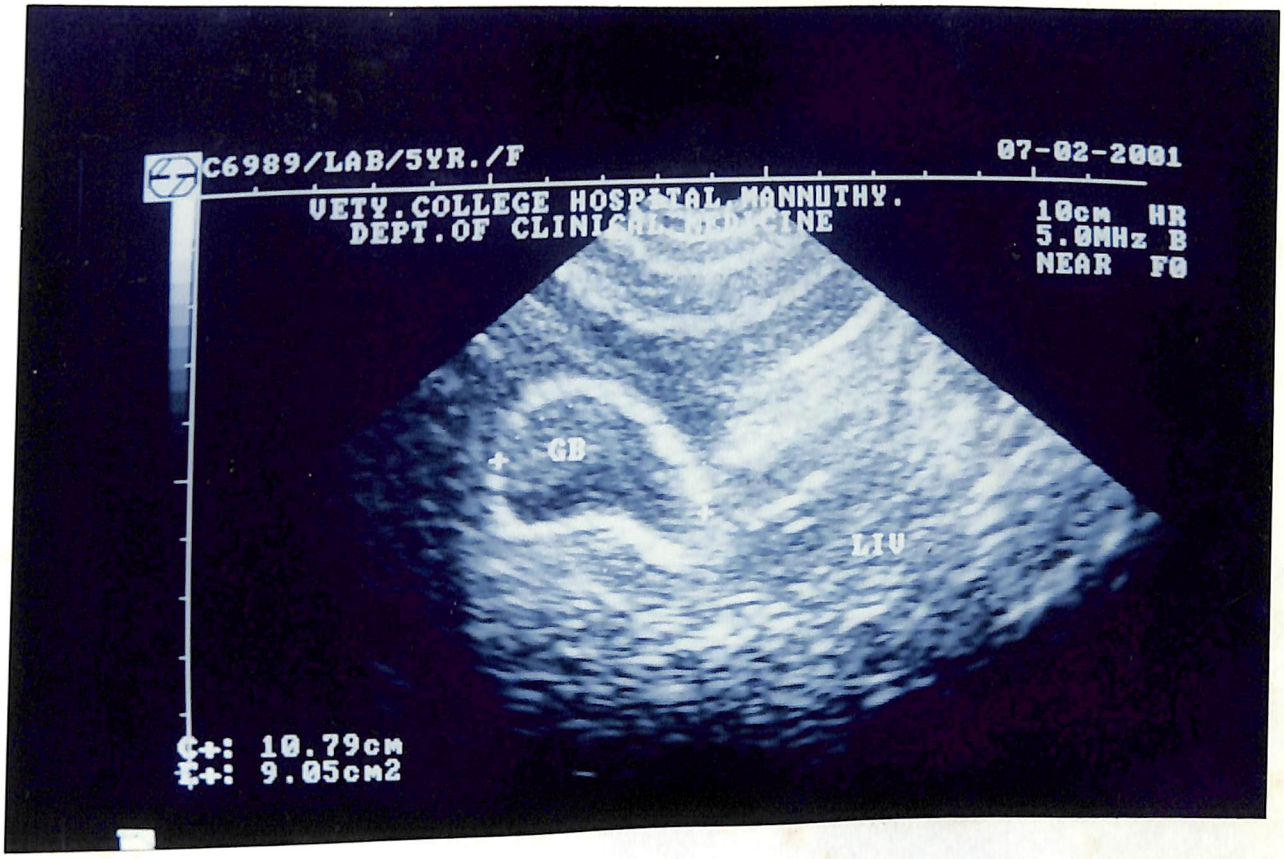

C7379/SPTIZ/9YRS/F
26-06-2001
VETY COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
10cm HR
7.5MHz N
NEAR F0
LIVER
PF

PLATE 17

Gall bladder (GB) in a dog with cirrhosis. Note the thickened wall and increased echointensity of the liver parenchyma

PLATE 18

Sonogram showing distended gall bladder (GB) with thickened wall and nodular liver lobes floating in ascitic fluid (PF)


C6989/LAB/5YR./F
07-02-2001
VETY.COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
10cm HR
5.0MHz B
NEAR F0
GB
LIV
C+: 10.79cm
E+: 9.05cm2

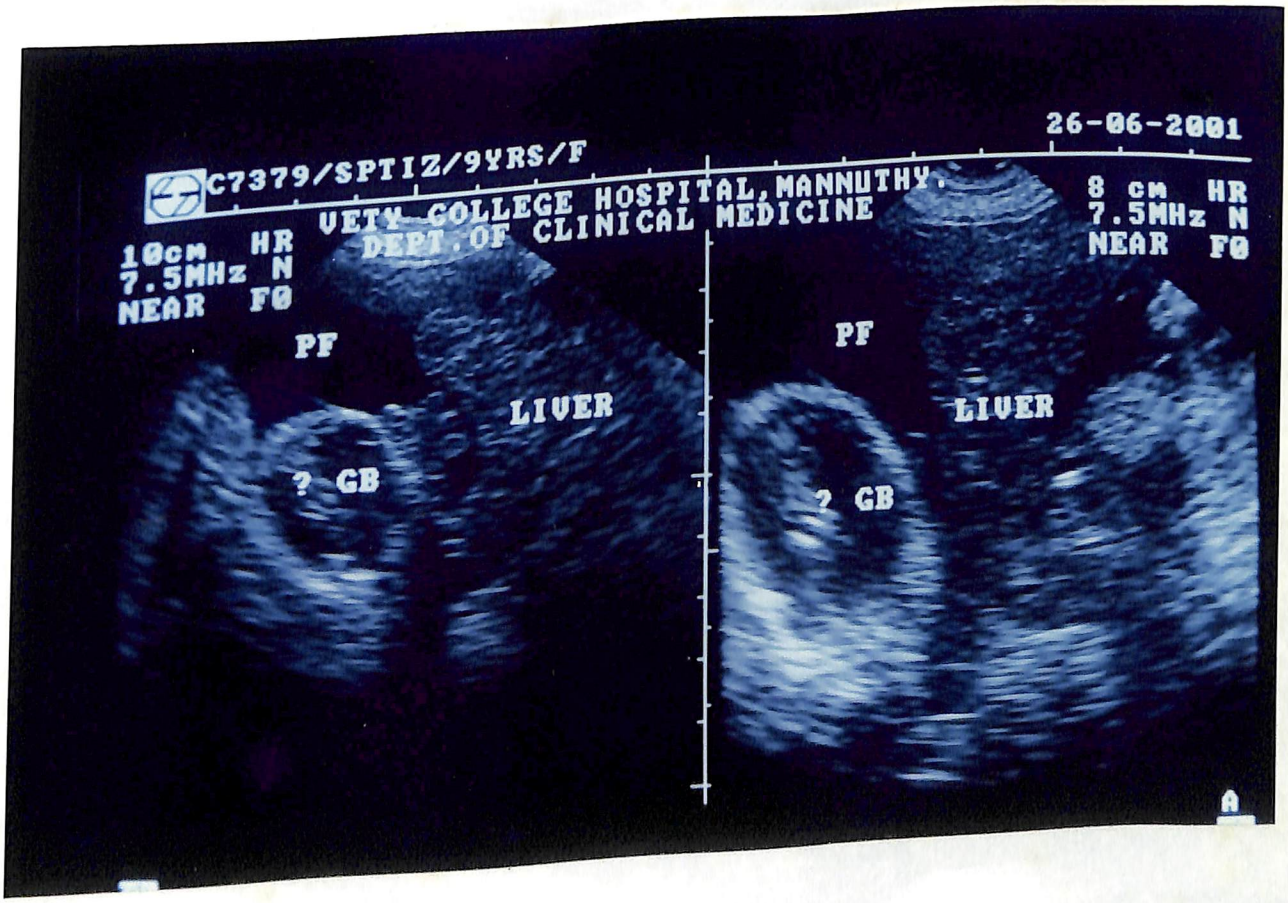
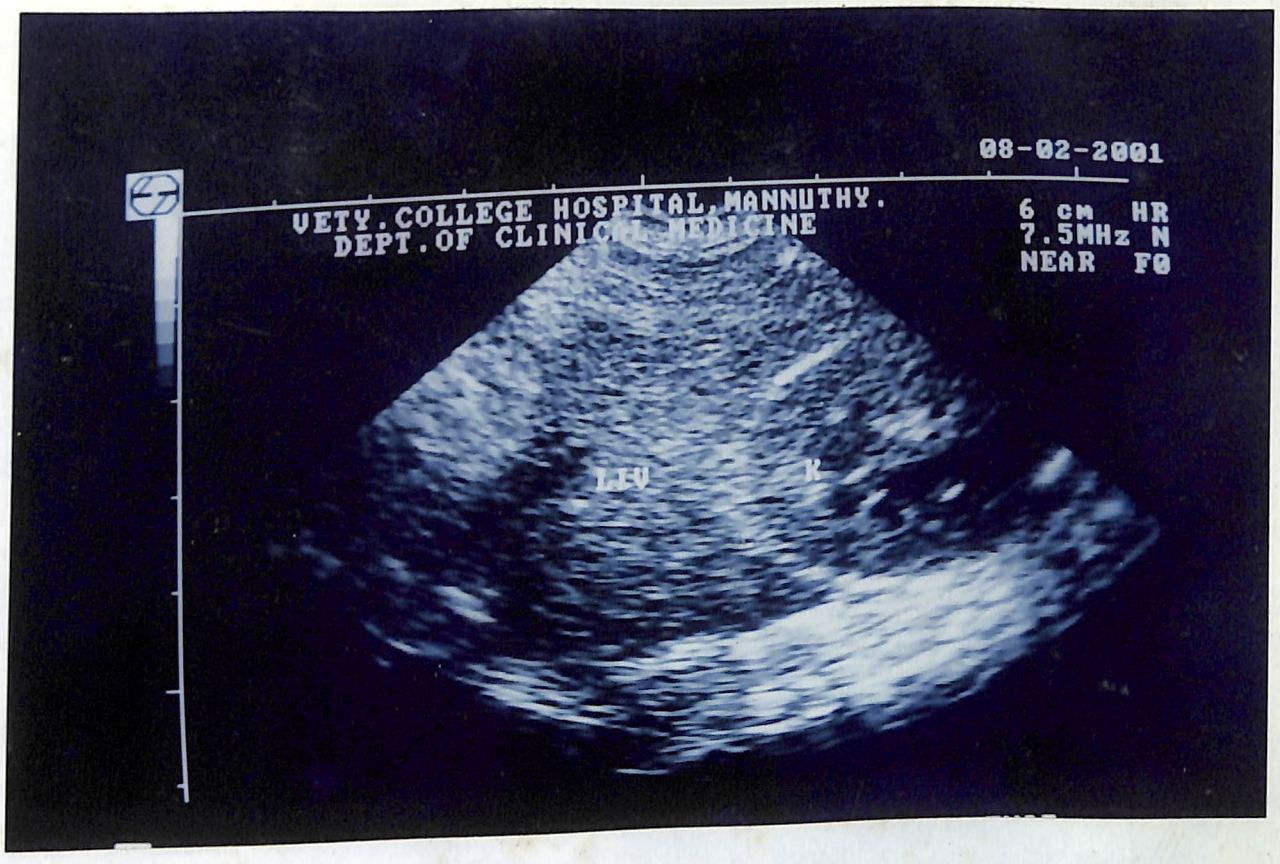

C7379/SPTIZ/9YRS/F
26-06-2001
VETY.COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
10cm HR
7.5MHz N
NEAR F0
8 cm HR
7.5MHz N
NEAR F0
PF
LIVER
? GB
PF
LIVER
? GB

PLATE 19

Sonogram showing liver and right kidney at same scanning depth and settings. Note that the liver parenchyma has same echointensity of the cortex of the kidney

PLATE 20

Sonogram showing marked distension of gall bladder (GB)


08-02-2001
VETY.COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
6 cm HR
7.5MHz N
NEAR F0
LIV
K

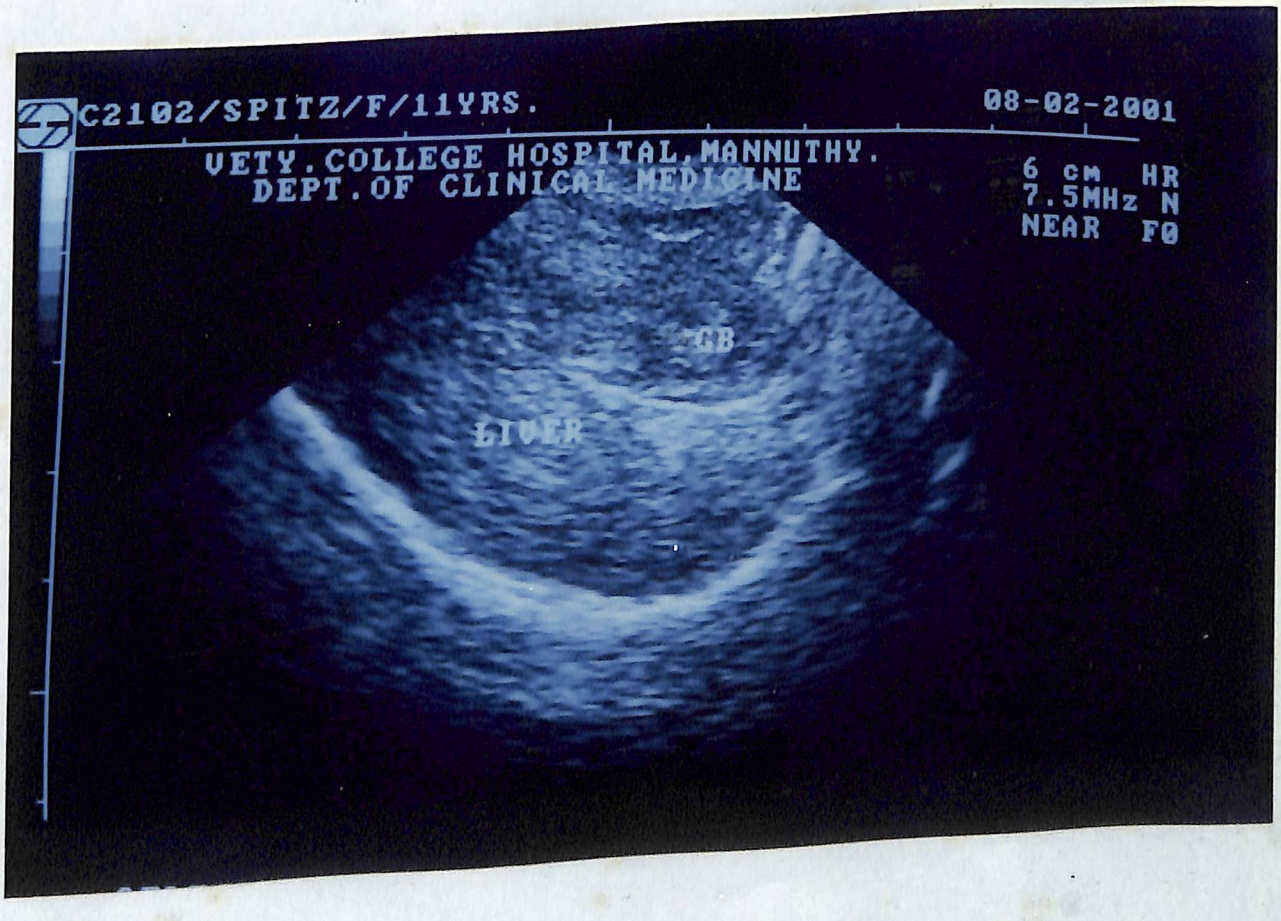
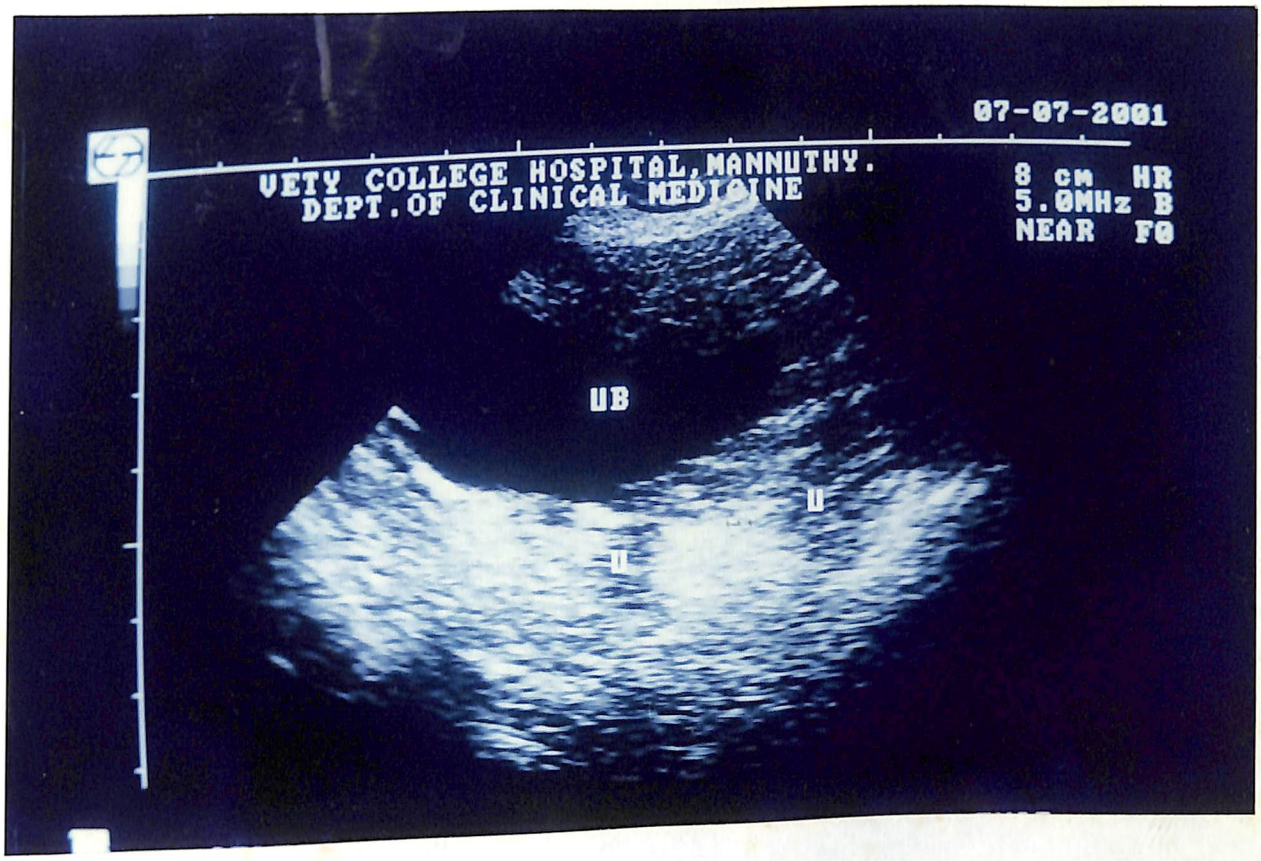

C2102/SPITZ/F/11YRS.
08-02-2001
VETY.COLLEGE HOSPITAL,MANNUTHY.
DEPT.OF CLINICAL MEDICINE
6 cm HR
7.5MHz N
NEAR F0
GB
LIVER

PLATE 21

Sonogram showing uterine horns (U) enlarged and filled with echogenic debris. UB=urinary bladder.

PLATE 22

Skiagram showing irregular soft tissue density (between arrows) suggestive of enlarged uterine horns.


07-07-2001
VETY COLLEGE HOSPITAL, MANNUTHY.
DEPT. OF CLINICAL MEDICINE
8 cm HR
5.0MHz B
NEAR FO
UB
U
U

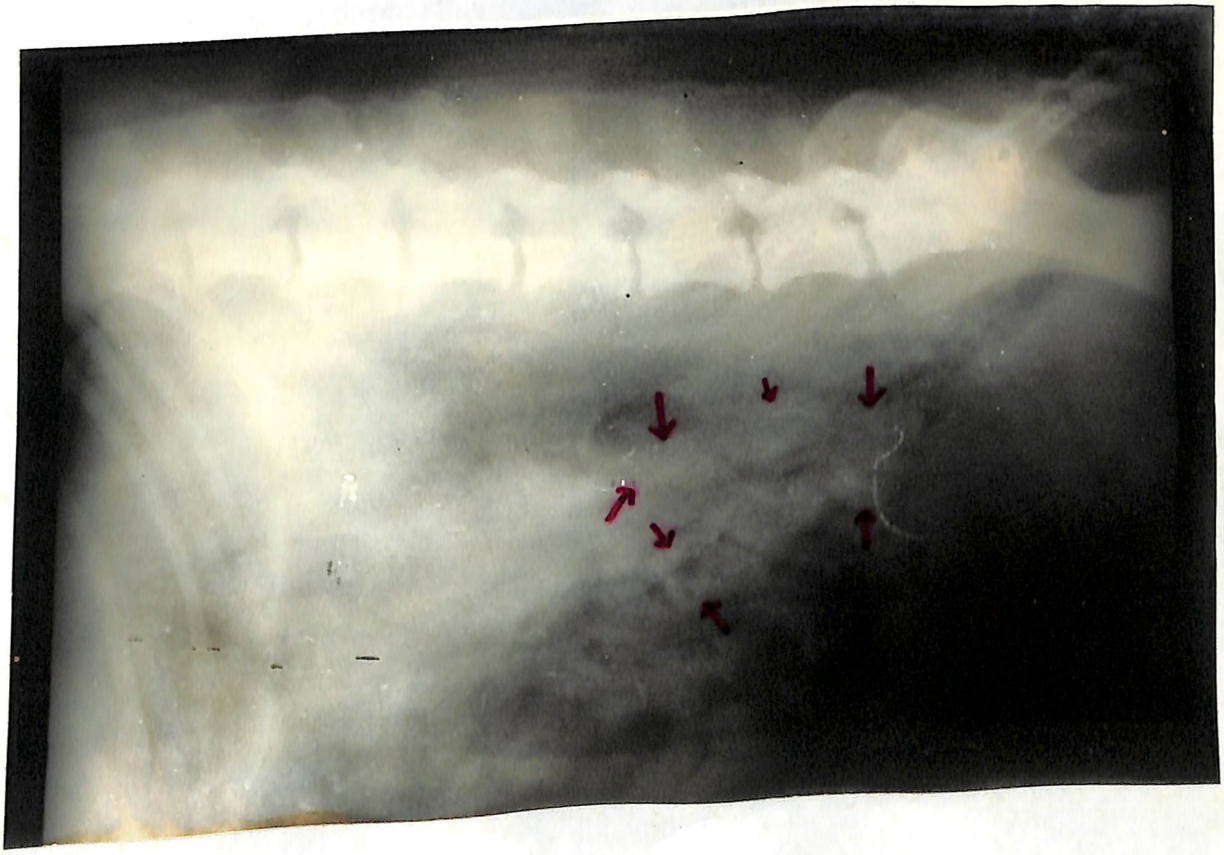
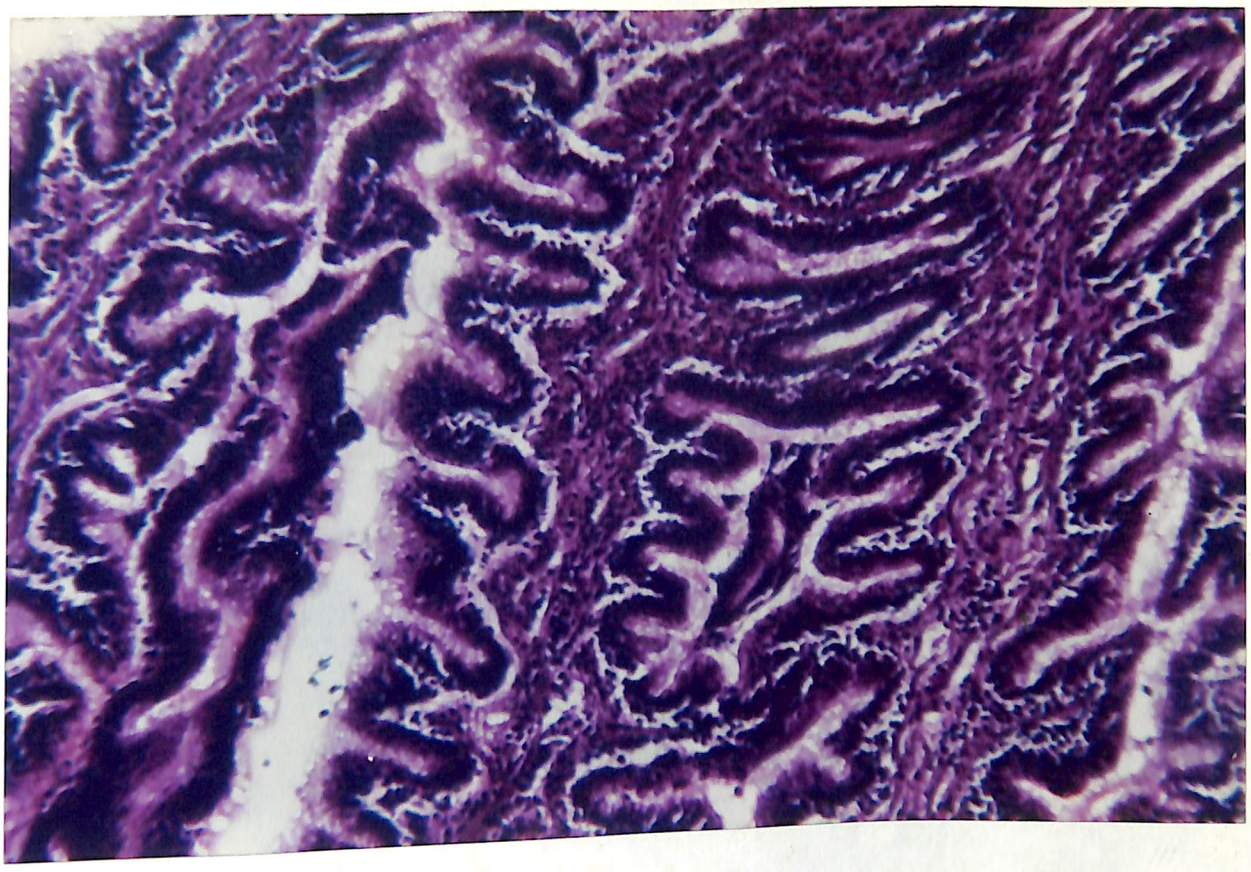


PLATE 23
Stomach: goblet cell proliferation, villar hypertrophy and infilteration. H&E, x 250

PLATE 24
Liver: cirrhosis: fibrous tissue proliferation, vacuolar degeneration of hepatocytes and infilteration with mononuclear cells. H&E, x 250



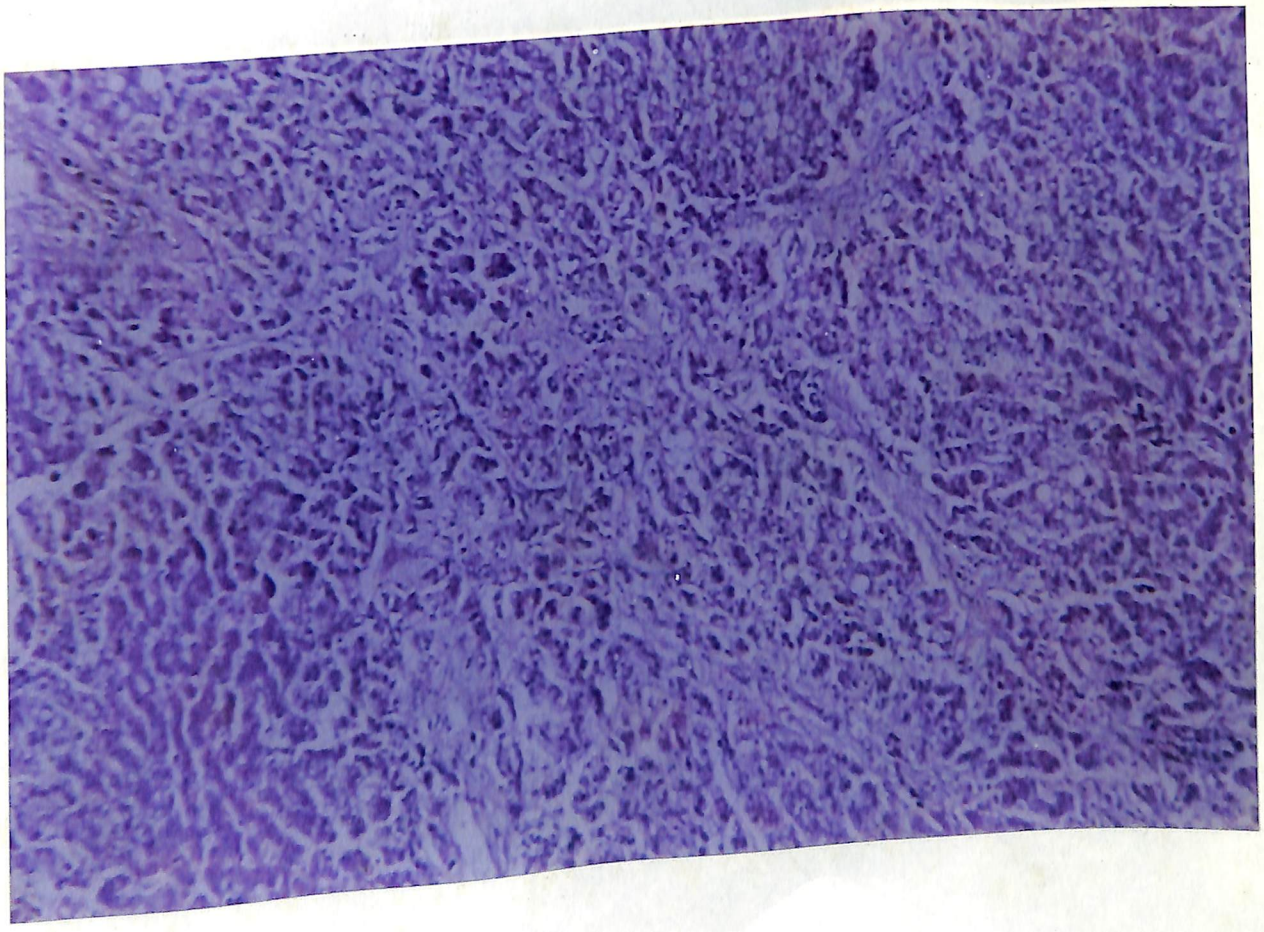
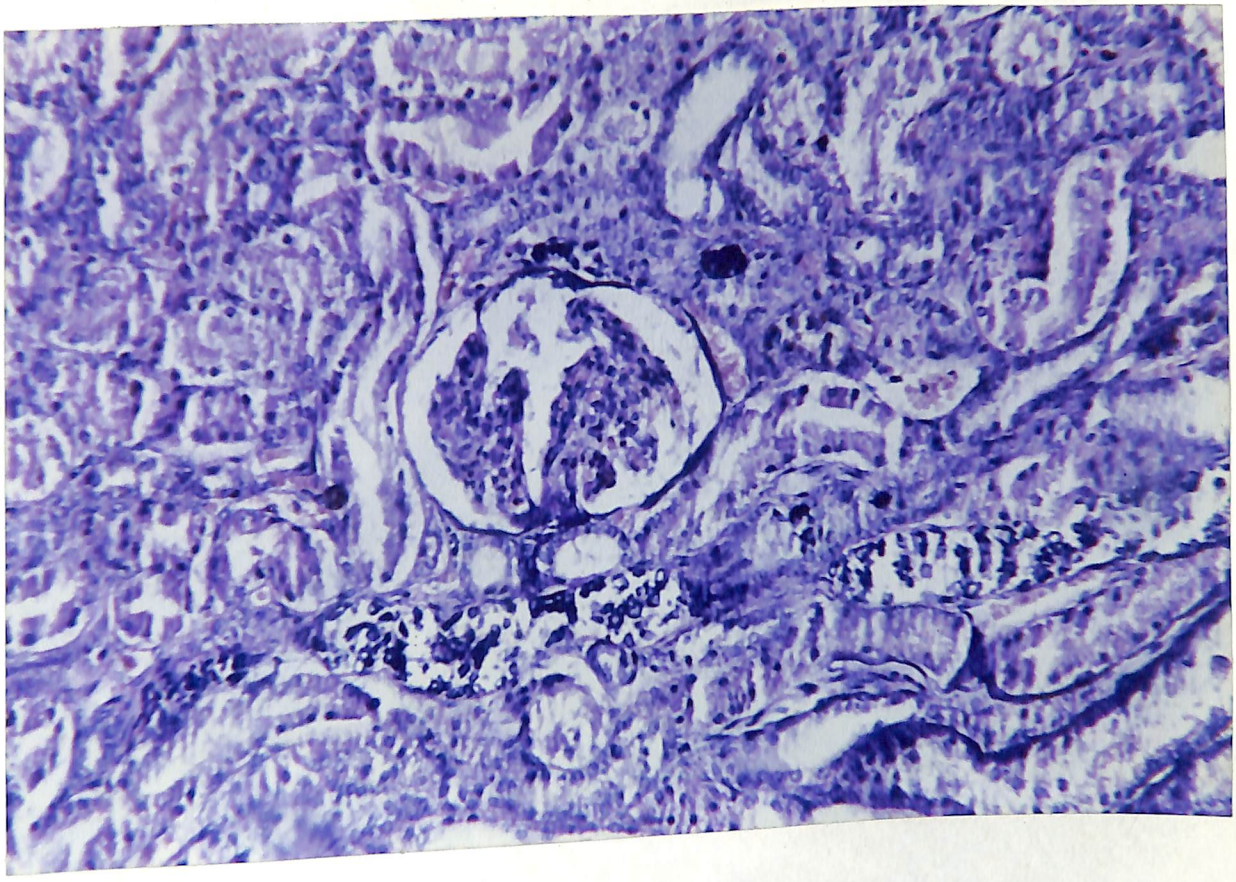


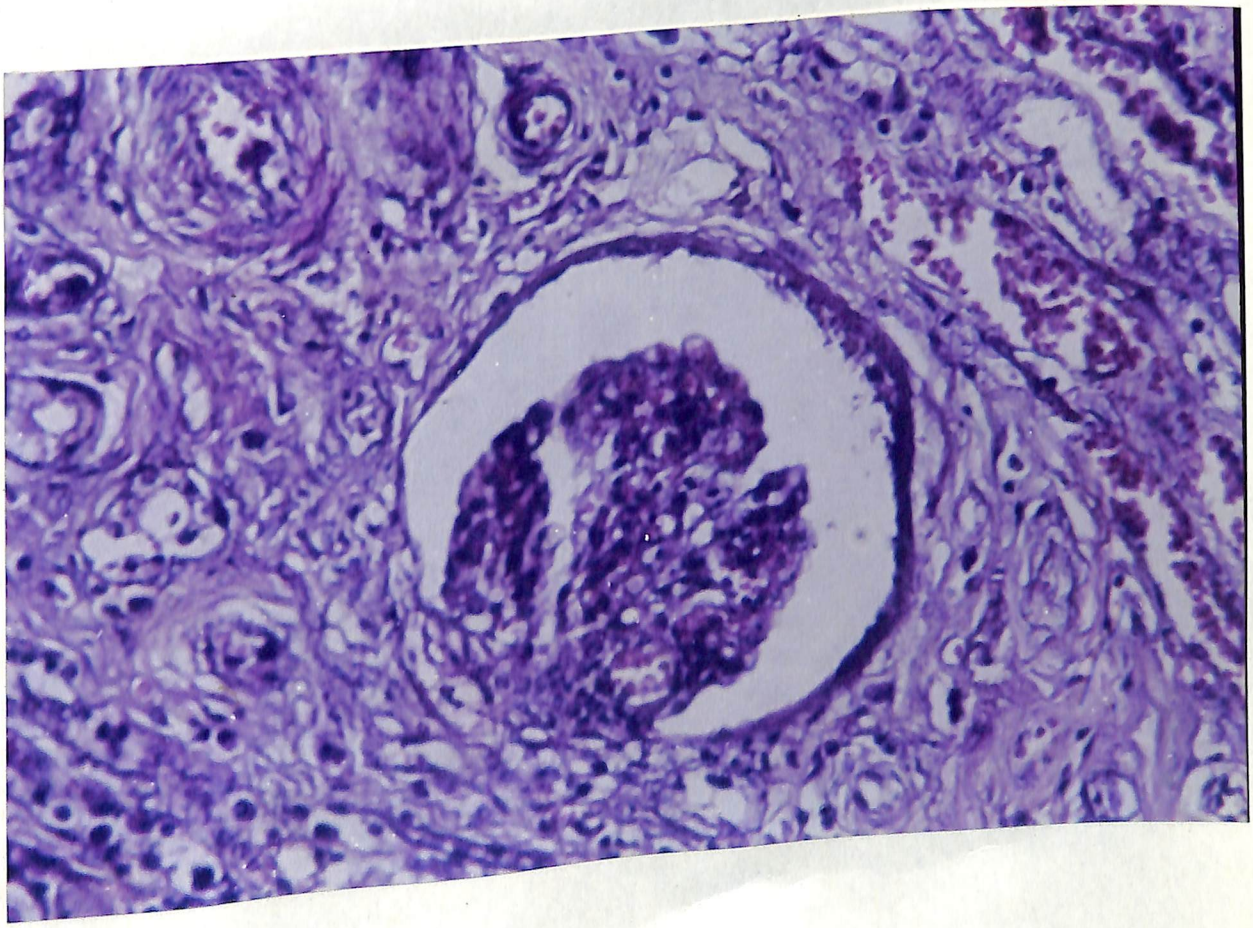
PLATE 25

Kidney: Chronic interstitial nephritis: calcification of the glomerular basement membrane and tubules. H&E, x 250

PLATE 26

Kidney: Chronic interstitial nephritis: fibrous tissue proliferation, dilatation of Bowmans space and thickening of the parietal layer of glomerulus. H&E, x 400





Discussion

5. DISCUSSION

During the period of the study, 302 dogs with history of vomiting were presented to the medical unit of Veterinary College Hospital, Mannuthy. Of these, dogs with gastroenteritis and those found positive for gastrointestinal parasites by faecal examination were excluded from the study. Of the remaining dogs those that responded to treatment with antiemetic were not considered for study. Leib (1992) stated that acute vomiting may be self-limiting or life-threatening. It was also stated that self-limiting disorders need only minimal diagnostic testing and simple supportive therapy and continuation or progression of clinical signs in these dogs after appropriate symptomatic therapy suggested possibility of life-threatening disorders. Twenty dogs which did not respond to one day therapy were selected for detailed examination and all parameters under study were carried out.

5.1 Colour and consistency of vomitus

Most of the dogs under study had bile stained watery vomitus. Dogs with pyloric stenosis had frothy or watery white vomitus. With gastric outflow obstruction bile staining of vomitus is usually absent (Johnson *et al.*, 2000).

Jose (2001) observed that the dogs with upper alimentary tract obstruction had vomitus that was frothy or white in colour and in the lower alimentary tract obstruction was frothy or yellow in colour with partially digested food.

Dogs with intestinal intussusceptions, intestinal foreign bodies, renal failure, gastritis, cirrhosis and pyometra had patent pylorus to permit the reflux of bile while in the dogs with pyloric stenosis bile reflex was absent resulting in frothy white vomitus.

5.2 Frequency of vomiting

In this study, dogs with gastritis had vomiting two to seven times per day and mostly occurred after feeding or drinking. Leib (1997a) suggested that vomiting occurring immediately after eating might indicate gastritis or duodenitis.

Dogs with intussusceptions and intestinal foreign bodies vomited two to seven times a day and with pyloric stenosis vomiting was associated with food intake (immediately to 30 minutes post feeding).

Pearson (1979) opined that predominant clinical sign of pyloric stenosis in dog was vomiting with variable frequency (several times each day to once or twice a week).

Jose (2001) observed in upper alimentary tract obstruction vomiting with frequency four times a day and in lower alimentary tract obstruction three times or less a day.

Dogs with renal failure and cirrhosis had no association with food intake and had vomiting two to three times daily to once/twice every one to two days. Leib (1997a) stated that an inconsistent relation between vomiting and eating might be associated with systemic disorders.

5.3 Physical Examination

In four of the six dogs with gastritis there was pain in the epigastric area on abdominal palpation. These animals held their abdominal muscles tensed and abdomen appeared tucked up. This could be due to the pain in the gastric area due to gastritis. The physical examination findings according to Gorman (1998) in primary gastric disease were usually non-specific such as cranial abdominal pain, distension or mass, or the presence of melena on digital rectal examination.

Abdominal palpation in dogs with intestinal intussusception revealed painful tubular mass. Similar observation was made by Kantrowitz *et al.* (1988) who detected a painful tubular mass in the left cranioventral abdomen in a dog with jejunal intussusception. But Wilson and Burt (1974) and Weaver (1977) reported that in intestinal intussusception abdominal palpation revealed an elongated mass and minimal pain on palpation.

In the dogs with stone as the intestinal foreign body, abdominal palpation easily detected a round hard mass, which was later confirmed by ultrasonography and radiography.

In one dog with pyloric stenosis there was marked distension of the left flank and on palpation appeared as a fluid filled structure. Johnson *et al.* (2000) reported that an enlarged fluid or food filled stomach caused abdominal distension due to pyloric outflow obstructions.

In two dogs with cirrhosis abdomen was markedly distended with fluid. According to Sevelius (1995) ascites was the most common clinical finding in chronic hepatitis associated with cirrhosis and chronic progressive hepatitis. Fluid thrill was appreciable on tactile percussion in one and in the other abdominal

palpation did not give any clue of fluid in the abdominal cavity. This may be the result of the animal holding its abdominal muscles tensely. Ultrasonography helped to detect ascitic fluid in this dog.

A tubular mass was palpable in posterior abdomen in the dog with pyometra. But Gorman (1998) cited that abdominal palpation in dogs with pyometra revealed pain in the acute phase (one to two days) only and there were no other findings on palpation. Contradicting this report but similar to the findings of this study Purswell(1997) reported that mildly enlarged uterus is palpable in dogs with pyometra.

5.4 Hydration status

In all dogs of group I, five of the six dogs in the group II, and three of the four dogs in group III had varying degrees of dehydration as evidenced by elevated CRT, skin turgor and sunken eyeballs of varying degrees (Table 1). This is in agreement with Twedt (1983) who opined that vomiting caused fluid depletion and that dehydration increased with each vomiting due to the inability to take sufficient oral maintenance fluids.

Greco (1998) suggested that elevated CRT, sunken eyeballs, dryness of mucous membranes and elevated VPRC and total plasma protein concentration as indicative of dehydration. Dogs of group I and group II had statistically significant elevation in VPRC. There was no significant difference between the means of VPRC of group III and control and further the mean value of group III was less than that of control. Even though dehydration in group III was evident by other signs, VPRC was not elevated. It could be because of the anemic status of these animals as suggested by low hemoglobin and TEC values.

5.5 Ultrasonography

Group I

No abnormalities were detected in the stomach ultrasonographically in this group and stomach wall thickness measurements ranged from 4.3 to 5.6 millimetres (Plate 2). Penninck *et al.* (1989) suggested that pathological thickening should be suspected when the stomach wall measures more than six to seven millimeters.

Physical or chemical or biological irritants damaged the gastric mucosal barrier and increased its permeability to luminal acid. Acid caused further mucosal, subepithelial and

vascular damage and stimulated mucosal mast cells to degranulate. Release of histamine stimulates further gastric acid secretion. A self-perpetuating cycle occurs wherein increased permeability to acid leads to further epithelial damage and increased permeability. These changes stimulate efferent neurons of vagal and sympathetic pathways to trigger vomiting center in the medulla and cause the animal to vomit (Leib, 1997a).

These dogs were diagnosed to be affected with gastritis based on the response to routine treatment for gastritis.

Group II

Ultrasonography was done in all the dogs of this group and ultrasound could detect cause of obstruction in five of the six dogs. In the remaining one dog with pyloric obstruction/stenosis, obstruction was not detected, but was confirmed by contrast radiography. Intraluminal gas posed a hindrance to imaging in this case (Plate 12).

Tidwell and Penninck (1992) reported that ultrasonography confirmed presence and location of foreign material within the gastrointestinal tract when radiographic findings were unclear.

In dogs with intussusception, one dog produced a picture of a hyperechoic centre with a single hypoechoic rim surrounding it in cross section (Plate 3) while in the other dog multiple concentric ring appearance in cross section (Plate 4) was obtained. Penninck *et al.* (1990); Lamb and Mantis (1998) and Prathaban *et al.* (2001) reported multiple concentric hyperechoic and hypoechoic rings as the ultrasonographic feature of intestinal intussusceptions in dogs. The finding in the second dog agreed with this but the feature recorded in the first dog was contradictory. Finding similar to that of the first dog was reported in humans. Fleischer *et al.* (1980) reported a second ultrasongraphic pattern in humans as a dense echogenic centre surrounded by a single hypoechoic ring.

A curvy linear hyperechoic area with posterior acoustic shadowing was recorded in dogs with stone as intestinal foreign body (Plate 8). Penninck *et al.* (1990) stated that sonographic appearance of gastric foreign objects was variable depending on the physical properties of the objects. Tidwell and Penninck (1992) opined that acoustic pattern arising from each foreign body varied depending on its physical properties and interaction with ultrasound beam.

In one dog with pyloric stenosis the stomach wall had thickness of 24.7 millimeters (Plate 10) and there was circumferential thickening of pylorus with small lumen (Plate 11). This is in concurrence with reports of Penninck *et al.* (1990). The generalized thickening of stomach wall may be attributed to chronic gastritis associated with this condition (Johnson *et al.*, 2000). This condition was diagnosed as chronic hypertrophic pyloric gastropathy based on the histopathological findings.

Vomiting in these dogs with gastrointestinal obstructions may be attributed to frank obstruction of the bowel or due to reflex vagal simulation from the viscera involved (Wilson and Burt, 1974).

The diagnosis in this group was confirmed by recovery of obstructive lesion or foreign body at surgery.

Group III

Normal kidney has three distinct regions, first is of a central bright echo complex corresponding to renal sinus and peripelvic fat, second a hypoechoic region surrounding the pelvis representing renal medulla and lastly an outer zone of

intermediate echogenisity of the renal cortex (Nyland and Mattoon, 1995).

In all dogs in this group the normal nephrosonographic architecture was not appreciable with indistinguishable corticomedullary junction (Plate 14). These findings corroborate with Bhadwal and Mirakhur (2000) who reported that dogs suffering from chronic renal failure had small kidneys with loss of architectural details and hyperechoic periphery with anechoic centre.

In one of the four dogs, the parenchyma was hyperechoic than liver parenchyma (Plate 15). Similar findings were cited by Kantrowitz *et al.* (1988) and was suggested that it was due to fibrosis and scarring of tissue. A diagnosis of chronic interstitial nephritis was made in this group dogs based on histopathological examination.

Group IV

Diez- Bru (1994) reported diffuse heterogenous hyperechogenicity of the parenchyma and the nodular contour of the liver as the sonographic feature in cirrhosis. This is similar to the findings recorded in this study in dog with cirrhosis (Plate 16).

Histopathology carried out in one of these dogs confirmed the diagnosis of cirrhosis.

Nyland and Mattoon (1995) cited that normal liver was equal or slightly more echogenic than the cortex of the right kidney at the same scanning depth and instrument gain settings. In the dog with cholangitis liver parenchyma had same echointensity of the cortex of right kidney (Plate 19). Gall bladder was markedly distended in this dog (Plate 20). Diez-Bru *et al.* (1997) opined that it was not the distension of gall bladder but dilation of cystic duct together with distension of the gall bladder as the sign of biliary obstruction. In this dog cystic duct was not appreciable and this may be because of the obliteration of the duct canaliculi due to inflammatory products (Sastry, 1983).

Anechoic fluid filled uterine horns with echogenic debris dorsal and cranial to urinary bladder was recorded in dogs with pyometra (Plate 21) and was in agreement with the findings of Vijayakumar *et al.* (1998).

5.6 Radiography

5.6.1 Plain radiography

In groups I and III, plain radiography did not give any clue of abnormalities. Carlson (1967) opined that radiographic

demonstration of gastritis was nearly impossible, because of great variability in the normal pattern of the stomach mucosa. It was also suggested that changes in the kidney were rarely great enough to be demonstrated radiographically in dogs with nephritis.

Group II

Plain radiographs could detect the cause of obstruction in two of the six dogs with the gastrointestinal tract obstruction. Radio-opaque foreign bodies were detected in both these dogs (Plate 9). Carlson (1967) and Jose (2001) suggested that radio-opaque foreign objects were easily seen on plain radiographs.

Dogs with intussusceptions produced characteristic shadow consisting of gas filled dilated stomach and intestines (Plate 6) similar to observations made by Wilson and Burt (1974) and Kantrowitz and Biller (1992).

One of the dog with pyloric stenosis had stomach filled with fluid and gas while in another dog, stomach appeared to be distended with gas (Plate 12). This is in agreement with Carlson (1967); Pearson (1970) and Gibbs and Pearson (1973) all of whom suggested that dog with pyloric stenosis had stomach nearly always filled with fluid and gas.

Radio-opaque foreign bodies (stones) were easily detected on plain radiography but gave only clue of obstruction in dogs with intussusceptions and pyloric stenosis.

Group IV

Dogs with cirrhosis and ascites produced radiographic picture of uniform density of abdomen. This finding is corroborated by O'Brien (1978) who stated that ascites produced radiographicaly ill- defined abdominal density.

An irregular soft tissue density suggestive of enlarged uterine horns was seen in the dog with pyometra (Plate 22). Carlson (1967) cited that pyometra was visualised as multiple homogenous, ground-glass appearing masses in the posterior ventral abdomen.

5.6.2 Contrast radiography

Gastrointestinal barium series could detect obstruction in four dogs. In dogs with intussusceptions flow of barium sulphate was obstructed one and a half hours after administration in front of a lucent appearing mass (Plate 7) similar to findings reported by Wilson and Burt (1974).

There was no gastric emptying of barium in dogs with pyloric stenosis even after one hour of administration of barium sulphate (Plate 13). According to Pearson (1970), stomach begins to empty within 30 minutes and if there is delay for an hour or more there is probably some degree of pyloric stenosis or pylorospasm.

There was no opacification of kidneys or excretion of contrast agent even after 15 minutes and this finding is in agreement with Burk and Ackerman (1996) who reported that renal opacification during nephrogram phase of excretory urography was usually less than normal in dogs with chronic interstitial nephritis. Carlson (1967) reported that there was no excretion of contrast medium even after one hour in a dog with chronic interstitial nephritis.

5.7 Clinical pathology

5.7.1 Hematology

5.7.1.1 Hemoglobin

There was no significant difference between the means of groups I and II with that of the control but the mean values of hemoglobin was slightly higher than control dogs. This finding

might have resulted because of the dehydration prevailing in dogs of these groups.

The mean value of hemoglobin in group III was significantly lower than control dogs. This finding is corroborated by statements of Benjamin (1985) and McCaw *et al.* (1989) who suggested that dogs with chronic renal failure have non-regenerative anemia.

Rutgers (1996) reported moderate non-regenerative anemia in dogs with chronic liver disease but in the dogs with cirrhosis in this study hemoglobin values were within the normal range.

5.7.1.2 Volume of Packed Red Cells (VPRC)

VPRC was significantly higher than control dogs in groups I and II. This elevation might be the result of dehydration and hence hemoconcentration (Greco, 1998).

The mean value of VPRC in group III was less than control, but there was no statistically significant difference. Even though dehydration was evident in these dogs, VPRC might have

not elevated because of the anemic status of these animals with chronic renal failure (Benjamin, 1985 and McCaw *et al.*, 1989).

VPRC in dogs with cirrhosis was 38 and 34 per cent respectively and were within the normal range. Rutgers (1996) reported moderate non-regenerative anemia in dogs with chronic liver disease and it was also stated that such dogs have low packed cell volume.

5.7.1.3 Total erythrocyte count (TEC)

Jose (2001) reported low TEC values in dogs with alimentary obstruction. In this study the dogs of groups I and II had mean value of TEC values lower than control dogs but there was no statistically significant difference.

The mean value of TEC of group III was significantly lower than control dogs and this finding was in concurrence with the reports of Benjamin (1985) and McCaw *et al.* (1989).

5.7.1.4 Leucogram

Dogs of groups I and III had leucograms within the normal range and there was no statistically significant difference

between these groups and control dogs. This may be because of non-infectious cause resulting in these conditions.

Dogs of group II had mean value of total leucocytes count (TLC) and neutrophils significantly higher than control dogs. These findings were in agreement with Wilson and Burt (1974) and Weaver (1977) who reported leucocytosis with neutrophilia in dogs with intussusception, and Jose (2001) who observed leucocytosis in dogs with upper alimentary tract obstructions.

In dogs with cirrhosis there was leucocytosis with neutrophilia. Leucograms recorded in dogs with gastrointestinal obstructions and cirrhosis might be attributed to stress. Coles (1986) suggested that total leucocytes response in stress is often great in dogs.

Leucocytosis with neutrophilia was evident in dogs with cholangitis and pyometra. Leucogram showed marked neutrophilic shift to left. This could be a response to inflammatory process in these dogs. Similar reports were made by Benjamin (1985) and Purswell (1997).

5.7.2 Serum biochemistry

5.7.2.1 Alanine Amino Transferase (ALT)

There was no statistically significant difference in the mean value of ALT of groups I, II, and III with that of the control dogs. Jose (2001) recorded normal values of ALT in upper alimentary tract obstruction.

ALT was 270 and 10 units per litre in the two dogs with cirrhosis. Sevelius (1995) reported normal to mildly increased concentrations of ALT in dogs with liver cirrhosis. In this study one dog had elevated ALT and other was within the normal reference range.

Sevelius (1995) reported markedly elevated ALT levels in dogs with chronic cholangiohepatitis. In dog with cholangitis since only biliary canaliculi were involved and liver parenchyma was found to be not affected, ALT values were within the normal range.

5.7.2.2 Alkaline phosphotase (ALP)

There was no statistically significant difference in the mean value of ALP in groups I, II and III with that of control dogs.

Benjamin (1985) reported increased ALP values in dogs with gastrointestinal lesion and renal disease and that it was an inconsistent finding in dogs with renal disease.

Sevelius (1995) recorded moderate to marked elevation of ALP in dogs with liver cirrhosis. Milne (1985) reported that increases in ALP values of upto 10 times normal indicated the existence of hepatic lesions and with values of upto 100 times normal in extraheptic biliary obstruction. The value of ALP obtained in dogs with cirrhosis and cholangitis in this study agreed with the above reports.

In dog with cholangitis, ALP value of 6468 units per litre was recorded. This was about 60 times more than that of the control dogs. This high value of ALP might have resulted from inflammation of biliary ducts and subsequent obstruction of the biliary duct due to the inflammatory products. A very high total and direct (conjugated) bilirubin value of 29.9 and 23.4 milligrams per decilitre was recorded (Normal values according to Benjamin, 1985 are 0.25 ± 0.1 and 0.14 milligrams per decilitre respectively) suggestive of post-hepatic cholestasis and considering the inflammatory leucogram recorded in this dog, the case was diagnosed as cholangitis.

Vomiting in these animals with hepatic cirrhosis according to Leib (1997b) was due to chronic gastritis or presence of duodenal ulcer. It was also reported that abnormal metabolism of gastrointestinal peptides or stimulation of the chemoreceptor trigger zone by circulating toxins not metabolised by liver as the additional reason.

Dogs with cholangitis had marked distension of the gall bladder. Gall bladder and larger bile ducts have rich sympathetic innervations and dilatation of these may trigger efferent stimulus to vomiting centre causing vomiting (Rothuizen and Meyer, 2000).

5.7.2.3 Total Protein

There was no statistically significant difference in the mean values of total protein in groups I, II and III with that of the control dogs. Weaver (1977) observed low plasma albumin and globulins in dogs with intestinal intussusceptions. Benjamin (1985) cited total protein values in normal dogs as 5.3 to 7.3 grams per decilitre.

Total serum proteins in dogs with cirrhosis and cholangitis were within the normal range. According to Benjamin

(1985) total serum protein concentrations is usually of little value in assessment of liver function or disease. It was also stated that hypoproteinemia is usually the result of hypoalbuminemia. In this study, dogs with cirrhosis had hypoalbuminemia but total serum protein was within the normal range as a result of increased globulins as evident by low albumin – globulins ratio.

5.7.2.4 Albumin

There was no statistically significant difference in groups I, II and III with that of the control dogs in the mean value of serum albumin. The mean ± SE recorded in the diseased groups were 3.00±0.00, 2.67±0.21, and 2.75±0.25 respectively. Benjamin (1985) cited serum albumin levels of normal dogs as 2.30 to 3.20 grams per decilitre.

Serum albumin was low at one gram per decilitre each in dogs with cirrhosis. This finding is corroborated by reports of Cornelius (1979), Dunn (1992) and Sevelius (1995) who stated that hypoalbuminemia was the common feature of liver cirrhosis.

5.7.2.5 Albumin – globulin ratio (A/G ratio)

The mean ± SE values of A/G ratio in control and groups I, II and III recorded were 0.65 ± 0.17, 1.10 ± 0.15,

1.02±0.16, and 1.25±0.16 respectively but, there was no statistically significant difference between control and that of groups I, II and III dogs. Benjamin (1985) reported A/G ratio of 0.59-1.11 as normal values in dogs.

Dogs with cirrhosis had low A/G ratio of 0.17 and 0.21 respectively. Benjamin (1985) opined that low A/G ratio is either due to relative or absolute increase in globulins and/or a decrease in albumin levels. Cornelius (1979) and Dunn (1992) reported increase in serum globulins in dogs with advanced liver disease. The dogs with cirrhosis had low albumin and increased globulins in the present study and was in agreement with the above authors.

5.7.2.6 Serum urea nitrogen (SUN)

There was no statistically significant difference between the means of group I and control dogs but, the mean value was greater than control dogs. This might be corroborated by statement of McCaw *et al.* (1989), who reported that blood urea nitrogen (BUN) rarely exceeded 50 milligrams per decilitre as a result of non-renal causes. This elevation in SUN could therefore be attributed to dehydration in these animals.

The mean ± SE of group II was 47.83 ± 10.18 and was significantly elevated than control dogs. Weaver (1977) reported elevated blood urea in dogs with intestinal intussusceptions. This elevation recorded in SUN values in group II might be result of dehydrated status of these animals in concurrence with MaCaw *et al.* (1989).

The mean ± SE of group III was 106 ± 5.70 and was statistically elevated and is in concurrence with McCaw *et al.* (1989) who reported elevated blood urea nitrogen in dogs with renal failure.

5.7.2.7 Creatinine

The mean value of creatinine in groups I and II was 0.45±0.127 and 0.733±0.147 milligrams per decilitre respectively and in dogs with cirrhosis were 0.9 milligram per decilitre each. In dogs with cholangitis and pyometra it was found to be 0.2 and 0.3 milligram per decilitre respectively. Benjamin (1985) reported normal values of creatinine in dogs to range from one to two milligrams per decilitre and Kaneko *et al.* (1997) cited normal values of creatinine as 0.5 to 1.5 milligrams per decilitre.

The mean value of creatinine in dogs of group III was significantly elevated and was in concurrence with McCaw *et al.* (1989) and Srinivasan *et al.* (1993) who reported elevated creatinine values in dogs with chronic renal insufficiency.

The elevations of SUN and creatinine recorded in dogs of group III indicated uremic status of these animals, which might be the reason for vomiting in these dogs. Vomiting in dogs with renal failure, according to Schulman and Krawiec (2000) was due to the prevailing hypokalemia and acidosis or uremic toxemia triggering chemoreceptor trigger zone. It was also suggested that hypergastrinemia which resulted from lack of metabolism of gastrin hormone by kidney, initiates a vicious cycle to produce hydrochloric acid and as a result produce gastritis and therefore vomiting in these animals.

5.7.2.8 Serum sodium

There was no statistically significant difference in the mean values of serum sodium level of groups I, II and III with that of the control dogs but the mean value of these dogs was less than control dogs. This finding is corroborated by Twedt (1983) and Moore (1992) who stated that animals with gastric vomiting have hyponatremia. It is contradictory to reports of Richter (1992) who

stated that vomiting of gastric contents caused hypernatremia and Marks and Taboada (1998) who opined that dogs with vomiting, renal failure and intestinal obstruction have hypovolemic hypernatremia.

5.7.2.9 Serum potassium

Serum potassium levels were significantly lowered in groups I and II and could be corroborated by reports of Twedt (1983); Richter (1992) and Phillips and Polzin (1998) who stated hypokalemia as the most frequent electrolyte abnormality in vomiting patients. This was due to loss of potassium rich gastric fluid by vomiting.

The mean value of potassium in group III was significantly elevated than control dogs. This findings is in concurrence with Phillips and Polzin (1998) who suggested that hyperkalemia is associated with oliguria or anuric renal failure and oliguria or anuria is the sign in dogs with end stage chronic renal failure (Mikiciuk *et al.*, 1989).

5.7.2.10 Serum bicarbonate

The mean values of serum bicarbonate were significantly elevated in groups I and II and is in agreement with

Twedt (1983) and Evans *et al.* (1994) who reported metabolic alkalosis in dogs with gastric vomiting and those with gastrointestinal foreign body obstructions.

The mean value of serum bicarbonate was less than control in group III but was not statistically significant. This may be the result of metabolic acidosis present in these animals with chronic renal failure (Mikiciuk *et al.*, 1989).

5.7.3 Histopathology

Histopathology of mucosal polyp biopsy obtained in a dog with pyloric stenosis showed hyperplasia of the villi, proliferations of goblet cells, infiltration of inflammatory cells and focal areas of haemorrhages (Plate 23). This is in agreement with Happe *et al.* (1981) and Walter *et al.* (1985) who reported similar findings in obstructive lesions of chronic hypertropic pyloric gastropathy.

Fibrous tissue proliferations in parenchyma, severe vacuolar degeneration, and moderate fatty changes with loss of hepatic architecture were the histological features observed in dogs

with cirrhosis (Plate 24). Obwolo and French (1988) reported loss of lobular pattern and rare portal areas, hepatocytes grouped in nodular structures separated by connective tissue and diffuse fatty changes in cases with hepatic cirrhosis. Rutgers (1996) reported total loss of normal liver architecture histologically in hepatic cirrhosis.

Smith *et al.* (1974); Sastry (1983); Maxie (1993) and Vegad and Katiyar (1998) stated that mononuclear cell infiltration, interstitial fibrosis and generalised tubular atrophy were the significant histopathological findings in chronic interstitial nephritis. Similar lesions were recorded in the kidney of three dogs with renal failure, which were examined histologically (Plates 25 & 26).

Summary

6. SUMMARY

The study "Investigations on etio-pathology of vomiting in dogs" was conducted to evaluate ultrasonography and radiography as diagnostic tools in vomiting dogs; to assess hydration status, electrolyte and acid-base balance in vomiting dogs and to correlate clinico-pathologic findings with ultrasonographic and radiographic changes. The study included 20 animals with vomiting as clinical cases and six apparently healthy dogs as control to study normal parameters.

The following parameters were studied:

History and clinical signs

Physical examination

Hydration status

Ultrasonography

Radiography-plain and contrast

Haematology

Serum biochemistry

Histopathology

Based on the diagnosis the clinical cases were divided into four groups:

Group I : Consisted of six dogs with gastritis.

Group II : Consisted of six dogs with gastrointestinal tract obstructions.

Group III : Consisted of four dogs with renal disorders.

Group IV : Consisted of four dogs with hepatic and uterine disorders. Among these dogs two had cirrhosis, one had cholangitis, and one had pyometra. Since the dogs with liver affections had wide variations in the data, they were not grouped and subjected to statistical analysis and data were presented as such.

Most of the dogs under study had bile stained watery vomitus with frequency of two to seven times per day in dogs with gastritis and gastrointestinal (GI) obstruction. While dogs with renal and liver affections had variable frequency of vomiting, dogs with pyloric stenosis vomited frothy or watery white vomitus and were associated with food intake.

Physical examination findings in dogs with gastritis and renal disorder were vague and non-specific. Mass lesions or foreign body as the cause of obstruction were detected in dogs with GI obstructions.

Physical examination findings such as elevated capillary refill time (CRT), skin turgor and degree of sunken eye balls were helpful to assess dehydration. Estimation of volume of packed red cells (VPRC) was beneficial to assess dehydration unless the dogs were anemic as in renal failure. Total protein assay was inconsistent to give any clue of dehydration.

Ultrasonography couldn't identify any lesions in dogs with gastritis, but was found to be useful to detect GI obstruction due to foreign body, intussusception and pyloric stenosis and to characterise lesions in the parenchymal organs such as liver and kidney.

Plain radiographs could not identify any abnormalities in dogs with gastritis, hepatic and chronic interstitial nephritis. While it could identify radio-opaque foreign bodies, it gave clue of obstruction in dogs with pyloric stenosis and intussusception, where it showed a characteristic gas filled stomach and intestines with varying degrees of radio-density.

Contrast radiography could identify sites of obstruction in dogs with pyloric stenosis and intussusception.

Stress leucogram were recorded in dogs with GI obstructions and cirrhosis; inflammatory leucograms in dogs with cholangitis and pyometra and anemic hemogram in dogs with renal failure.

Alkaline phosphotase was moderately elevated in dogs with cirrhosis (200 and 520 units per litre respectively), while it was markedly elevated in dogs with cholangitis (6468 units per litre).

Total protein values in the two dogs with cirrhosis were 6.83 and 5.68 grams per decilitre and was found to be not of value in assessment of liver function. Serum albumin values of one gram per decilitre each and albumin-globulin ratio of 0.17 and 0.21 in the dogs with cirrhosis, suggesting hypoalbuminemia and low albumin – globulin ratio and were found to be of value in assessment of liver function.

The mean values of serum urea nitrogen and creatinine were significantly elevated in dogs with renal failure with values of 106.63 ± 5.70 and 10.00 ± 2.20 milligrams per decilitre respectively.

Serum potassium in groups of dogs with gastritis and GI obstruction were 3.80±0.23 and 3.68±0.39 milliequivalents per litre respectively, suggesting hypokalemia as the significant electrolyte abnormality recorded in these dogs with vomiting. Hyperkalemia with value of 6.94 ± 0.57 milliequivalents per litre were recorded in dogs with vomiting due to renal disorders.

The mean values of serum sodium recorded in the groups I, II and III were 144.51±15.47, 135.18±13.13 and 148.62±13.32 milliequivalents per litre respectively. There was no statistically significant difference with control.

Alkalosis was evident in all dogs except those with renal failure by hyperbicarbonatemia, with serum bicarbonate values of 129.00±13.19, 116.833±12.34 and 26.375±0.745 milliequivalents per litre respectively in groups I, II and III.

From this study it was concluded that:

1. Ultrasonography could detect GI obstructions due to foreign bodies, intussusception and pyloric stenosis. While plain radiography could give clue of obstruction in non-radiopaque GI obstructions, contrast radiography could detect sites of obstruction.

2. Ultrasonography and radiography was not of much use to assess any pathologic changes in the stomach in gastritis of dogs.

3. Ultrasound scanning was better than radiography in characterising the lesions in parenchymal organ such as liver and kidney.

4. Ultrasonography together with clinico-pathologic findings provided accurate diagnosis in hepatic and renal disorders.

5. The gastritis recorded in this study might be due to non-infectious causes as evidenced by absence of haematological changes.

6. Hypokalemia was the significant electrolyte abnormality in vomiting dogs.

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INVESTIGATIONS ON ETIO-PATHOLOGY OF VOMITING IN DOGS

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ABSTRACT OF THE THESIS

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ABSTRACT

The study "**INVESTIGATIONS ON ETIO-PATHOLOGY OF VOMITING IN DOGS**" was conducted in 20 dogs to evaluate ultrasonography and radiography as diagnostic tools in vomiting dogs; to assess hydration status, electrolyte and acid-base balance in vomiting dogs and to correlate clinico-pathologic findings with radiographic and ultrasonographic changes. Various parameters such as history, physical examination, hydration status, ultrasonography, radiography-plain and contrast, haematology, serum biochemistry, and wherever possible histopathology were studied.

Most of the dogs under study had bile stained watery vomitus but dogs with pyloric stenosis had frothy or watery white vomitus. The frequency of vomiting in dogs with gastritis and gastrointestinal (GI) obstruction was two to seven times per day, it was variable in dogs with hepatic and renal disorders, but was associated with food intake in dogs with pyloric stenosis.

Physical examination was found useful in dogs with GI obstruction, while it was non-specific in dogs with gastritis and renal disorders.

Capillary refill time (CRT) and degree of sunken eye balls were helpful to assess dehydration. Estimation of volume of packed red cells (VPRC) was found beneficial to assess dehydration unless the dogs are anemic.

Ultrasonography could not identify any lesions in dogs with gastritis, but was useful to detect GI obstructions due to pyloric stenosis, intussusception and foreign body and to characterise lesions in the parenchymal organs like liver and kidney.

While plain radiographs could give indication to possible non-radiopaque GI obstructions, contrast radiography was required to confirm.

Radiography could not identify any lesions in dogs with gastritis, hepatic and chronic intestitial nephritis.

Hypokalemia with metabolic alkalosis was the significant electrolyte and acid-base derangement in dogs with vomiting due to gastritis and GI obstructions.

APPENDIX

PROFORMA FOR CLINICAL CASE STUDY

INVESTIGATIONS ON ETIO-PATHOLOGY VOMITING IN DOGS

Case No: Date:

Name and Address of owner:

Breed: Sex: Age:

Vaccination:DHLPPi Rabies

Deworming Status:

Last de-wormed--------

Feeding:

Frequency of feeding:

Type of feed:

Quantity:

Any change in type of feed: Yes/No

HISTORY:

Present:

Number of times vomited: consistency of vomitus:

Colour of vomitus: Odour of vomitus:

pH of vomitus:

Past:

CLINICAL EXAMINATION

1. General appearance
2. Behaviour
3. Expression
4. Bodily condition
5. Appearance of abdomen
6. Eating and drinking
7. Defecation
8. Micturition
9. Rate of respiration
10. Character of respiration
11. Mucous membrane
12. Pulse
13. Temperature

PHYSICAL EXAMINATION OF ABDOMEN

ASSESSMENT OF HYDRATION STATUS

Capillary Refill Time (CRT)---sec. Skin turgor--sec.

Sunken Eyes: Barely visible --- + --- +++ ---- ++++ ---

LAB EXAMINATION

1. Fecal Examination
2. Complete Blood Count
   - Hb : ------ g%
   - TEC: ------millions/cu.mm
   - TLC: ------ cells/cu/mm
   - DLC:- N: ----% L:-----% M:----% E:----% B:---%
3. VPRC ------%
4. Serum Biochemistry
   1. ALT (SGPT) --------U/L
   2. ALP ------U/L
   3. Total protein -----g%
   4. Albumin -------g%
   5. Albumin globulin ratio----
   6. Urea nitrogen-----mg%
   7. Creatinine------mg%
   8. Sodium--------mEq/L
   9. Potassium -----mEq/L
   10. Bicarbonate: -------mEq/L

SPECIAL EXAMINATION

1. Survey Radiography
2. Contrast Series
3. Ultrasound Changes