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A STUDY OF UPPER GASTRO INTESTINAL BLEEDING BY ENDOSCOPY

Dissertation submitted to

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For the award of the degree of

MASTER OF SURGERY

BY

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CERTIFICATE

This is to certify that the dissertation entitled "A STUDY OF UPPER GI BLEEDING BY ENDOSCOPY" is a bonafide research work carried out by Dr.VVSM KUMAR DONTAMSETTY (USNo.NU14MSGS12) under the guidance of Prof.Dr.BALAKRISHNA N SHETTY in the department of General Surgery of K.S.Hegde Medical Academy. The same is being submitted to the Nitte University in partial fulfilment of the requirements for the award of Master of General Surgery.

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I Dr.VVSM KUMAR DONTAMSETTY (U.S.No.NU14MSGS12) hereby declare that the dissertation work entitled "A STUDY OF UPPER GI BLEEDING BY ENDOSCOPY" is my original work and has been carried out under the guidance of Prof.Dr. BALAKRISHNA N SHETTY, Department of General Surgery, K.S. Hegde Medical Academy is being submitted to the Nitte University in partial fulfillment of the requirements for the award of Master of General Surgery.
I also hereby declare that this work, in part or full, has not been submitted to any other University/ Institution for any Degree/ Diploma.

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Date: Dr.VVSM KUMAR DONTAMSETTY
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LIST OF ABBREVIATIONS

UGIB	Upper Gastrointestinal bleeding
DU	Duodenal Ulcer
EUS	Endoscopic UltraSonography
GI	Gastrointestinal
GV	Gastric Varices
HVPG	Hepatic Venous Pressure Gradient
NSAIDs	Non Steroidal Anti Inflammatory Drug
MW	Mallory weiss tear
GU	Gastric Ulcer
PUD	Peptic ulcer disease

ABSTRACT

Upper gastrointenstinal bleeding (UGIB) is one of the surgical emergencies. The etiological spectrum of gastrointenstinal bleeding is diverse and variable from one geographical area to another. Earlier barium meal used for study of UGIB which has its own drawbacks. Now upper GI scopy used as diagnostic tool.

AIMS AND OBJECTIVES:-

- To compare common etiology of upper gastrointenstional bleeding between Dakshina Kannada, Keralaand Udupi District
- To determine the common etiological factors of upper gastrointestinal bleeding.
- To Establish the site and source of UGIB through endoscopic evalution.

METHODS:- A Prospective study of 100 cases of upper GI bleeding who are admitted in Justice K.S.Hegde hospital between September 2014 to September 2016 and cases are divided into 3 groups

1) Dakshina Kannada 2) Kerala 3) Udupi

RESULTS: A Total of 100 patients who underwent upper GI scopywas studied during the period. The age range of patients was from 18 to 80; Mean age is 50.8 years .Out of 100 patients lesions found in 95% patients.

Majority of patients are presented with haemtemesis (76%).Oesophageal varices secondary to Alcoholic cirrhosis of liver were the most frequent cause of upper GI bleeding followed by PUD (Pepticulcerdisease).

On comparing the 3 groups thereis no significant geographical distribution of etiology of upper GI bleeding (P value - alcoholic cirrhosis of liver were the most frequent cause of upper GI bleeding followed by PUD (Peptic ulcer disease).On comparing the 3 groups there is no significant geographical distribution of etiology of upper GI bleeding (P value -0.211).Cirrhosis is the main etiological factor of upper GI bleed in this study (P value - 0.0001)

CONCLUSION:-

Endoscopy is essential for evaluating of upper GI bleed. In this study endoscopy provided diagnosis in 95% of patients. The most common cause of upper GI bleed was esophagealvarices

There is no significant geographical distribution of etiology of upper GI bleed in population of Dakshina Kannada, Kerala and Udupi

Cirrhosis is the main etiological factor of upper GI bleed in this study

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CHAPTER 1
INTRODUCTION

Upper gastrointestinal bleeding (UGIB) was first described as ground coffee and melena 200 A.D by Galen Claudius[1]. It is an emergency in gastroenterology patients with mortality of 6-13%.

Despite changes in management there is no significantly improved change in mortality seen by gastroenterologist. Its incidence was 50-70000 hospital admissions occur annually in UK.

The incidence for upper gastrointestinal bleeding is 40 to 150 cases/100,000 adults.More than 3,50,000 patients are hosiptalized each year in united states for UGIB[2] and mortality rates are 5-11%.,[3].
Upper gastrointestinal bleeding will present as hematemesis, melena, and hematochezia, or signs and symptoms of iron deficiency anaemia due to occult blood loss.

Definitions: Hematemesis is defined as vomiting fresh red blood4. Coffee ground vomiting is defined as vomiting of altered black blood[4]. Melena is the defined as passage of tarry stools[4].Hematochezia is the defined as passage of red blood per rectum; this is generally seen in lower gastro intestinalbleeding, but also seen in massive upper gastrointestinal bleeding. It represents at least 1000 ml of blood[5].

CAUSES OF UPPER GI BLEEDING

NON VARICEAL BLEEDING	VARICEAL BLEEDING
Peptic ulcer disease 30-50%	Gastroesophagealvarices>90
Mallory Weiss tear 15-20%	Hypertensive portal gastropathy<5
Gastritis or duodenitis 10-15%	Isolated gastric varices ,rare
Esophagitis5-10%	
Av malformation 5%	

Pathophysiology & Clinical features of upper gastrointestinal bleeding (UGIB):

The colour of vomited blood depends on the concentration of hydrochloric acid in the stomach in addition to the duration of its contact with the blood[4]. When vomiting takes place shortly after the onset of bleeding it will appear dark red, and when it stays longer it will appear brown or black. The coffee ground appearance is due to the action of hydrochloric acid on haemoglobin which will change to haematin giving it this characteristic appearance. Hematemesis usually indicates UGIB as a cause, because bleeding distal to the duodenum rarely enters the stomach. Almost all patients with hematemesis have melena, but only half of patient with melena have hematemesis[4]. This is because melena usually occurs in both UGIB and bleeding down to the ascending colon specially when the transit time is sufficiently prolonged[6]. The black colour of melena results from contact of the blood with hydrochloric acid to produce haematin[4]. About 50-60 ml of blood may produce melena for about seven days and a positive test for occult blood for seven days after the stool colour change back to normal4. Positive results for occult blood loss may indicate a serious disease and should be thoroughly investigated[4]. Black stool that is negative for occult blood may result from ingestion of iron, bismuth, or various formulae and should not be mistaken for melena[5]. The manifestations of UGIB depend on the source, rate of bleeding, and underlying or coexistent disease; e.g. a patient with underlying Ischemic heart disease may present with angina or MI after brisk UGIB.

Coexistent heart failure, hypertension, pulmonary disease, renal failure and diabetes mellitus may be aggravated by severe GI bleeding, which may present as shock. Lesser degrees of bleeding may manifest as orthostatic changes in pulse (a change > 10 beat/min) or BP (a drop of ≥ 10mmHg). Orthostatic changes should be interpreted with caution in patients with underlying heart disease or peripheral vascular disease or in those taking drugs known to influence peripheral vascular resistance[4]. UGIB of 60 ml gives only melena. Bleeding of 500 ml is rarely associated with systemic signs; exception are mentioned (elderly, coexistent heart disease or anaemia). Rapid haemorrhage of greater volumes results in decreased venous return to the heart, decreased cardiac output, and increased peripheral resistance due to reflex 13 vasoconstriction. Orthostatic hypotension greater than a change of 10mmHg usually indicate a 20 percent or greater of blood loss. The causes of UGIB are many and are different according to the geographical area In the United Kingdom the commonest cause of again is peptic ulcer causing 30 to 50 percent of all causes of UGIB, but interestingly no cause was identified in 24%[6] of the cases. In Sudan, which is a tropical country, however, bleeding due to oesophageal varices is the commonest cause[7].

Variceal Causes of UGIB

Anatomical & physiological and pathological consideration:

UGIB is one of serious complication for portal hypertension which leads to varices. The portal vein carries blood to liver and formed by the confluence of the superior mesenteric and splenic veins and it is about 8cm in length and has a mean diameter of 10 mm located in right upper quadrant of liver. The superior mesenteric vein receives blood from the small intestine, colon and the head of the pancreas. The splenic vein is formed from several tributaries originating at the splenic hilum, so receiving blood from pancreas and left gastroepiploic and inferior mesenteric vein and the short gastric veins from the fundus of the stomach. Dilated submucosal veins develop due to decompensated liver cause upper GI bleeding[9]. In oesophagus it is seen in distal end. Varices also can seen in other sites like stomach, umbilicus and rectal region. Portal hypertensive gastropathy along with varices they will be gastritis showing cheery red spots and snakeskin like appearance. Chances of rebleeding and blood transfusion more in variceal bleeding. Portal hypertension defined as increase in portal pressure more than 5mm. The portal pressure is determined by flow rate and vascular resistance. The more common abnormality is increased resistance[9].When there is rise in portal pressure, collaterals will form, diverting the portal flow into systemic veins. So in cirrhosis with a severe intrahepatic block only 10% flow will be there through liver ,rest will go to systemic circulation[9]. It is controversial that portal hypertension may decrease. The liver is depends mainly on the hepatic artery for oxygen and nutrient so, the liver is shrunken with impaired ability to regenerate[12]. Almost all collaterals won't lead to bleeding, but the most important are gastroesophageal collaterals, so-called varices. Other collaterals are, caput medusa connecting between paraumbilical and superficial epigastric, rectalvarices connect between superior middle and inferior rectal vein. Splenic collaterals between spleen and renal vein .Similarly, blood from the portal system drains in a retrograde fashion via the left renal vein.

Longstanding state portal hypertension produces not only dilated and torturous veins but also changes in gastric mucosa like vascular ectasia.[10].Such changes can seen in large and small intestine leading to occult gastrointestinal bleeding.

As told before gastroesophageal varices are important collateral. There are two main inflows, from the left gastric vein and from the splenic hilum through the short gastric veins. Oesophageal varices feeding vessel primarily is reversed flow from left gastric varices will form 4 layers named as

intraepithelial veins and superficial venous plexus, the deep intrinsic venous plexus, perforating vein and the adventitial veins. These will form "Cherry red spots"[11]in condition of portal hypertension; this lead to haemorrhage.[11]Gastroesophagealvarices has been classified into zones: The truncal zone, the perforating zone the palisade zone, and the gastric zone. The palisade zone is believed to be the water shed between the portal and systemic system.

Turbulent flow in the veins of the perforating zone with thinning of the muscularis mucosa lead to rupture of varices in this region frequently[12,13,14]. Recurrence of varices due to various communications between venous channels plexus[11]. Ectatic capillaries and venules are seen in the lamina propria and they communicate with deep vessels.[15] Microthrombi and increase in smooth muscle fibres leads to portal hypertensive gastropathy[16].Gross lesions termed as watermelon lesion.

Varix: Is a dilated and tortuous intrinsic vein.
Pathophysiology of gastroesophagealvarices
Increased passive resistance secondary to fibrosis and regenerative nodules; increased hepatic vascular resistance caused by active vasoconstriction by nor epinephrine, endothelin, and other humoral vasoconstrictors; increased portal venous inflow secondary to a hyperdynamic systemic circulation and splanchnic hyperemia.

Cirrhosis accounts for up to 90 percent of the causes of portal hypertension in North America and Europe[17].Most common cause of pre hepatic portal hypertension is portal vein thrombosis[18] This leads to cavernoma formation of portal vein.

In intrahepatic condition they will be cause of portal hypertension in presinusoidal, sinusoidal and post hepatic sinusoidal, common cause of pre sinusoidal is schistomiasis. At sinusoidal level alcohol is most common cause of portal hypertension due to increased resistance.post hepatic includes Budd Chiari syndrome and constructive pericarditis hepatic venous pressure more than 12mm hg is the most important factor to increase the risk of UGIBdeath[19].

Natural history of varices in cirrhosis
1. Development of varices:-
The increase in raise in portovenous pressure lead to formation of varices and gastro esophageal varices formed from intrinsic and extrinsic veins at cardia lead to formation of divergent which leads to flow of portal blood to systemic circulation. this will increase in size with time, if not controlled correctly.

A study of 532 patients with cirrhosis, shows the incidence of varices increases from 12% to 90% over12 years[20]. In a study involving 80 patients followed for 16 months, Cales and Pascal[21], showed 20% of them showed new varices and 42% shows increase in size of varics with time.

The factors which determine varices are hepatic injury and degree of portosystemic shunting.

Bakerand colleagues[22] followed a cohort of 112 patients. They saw regression and absence of esophageal varices depend upon their intake of alchol. This was confirmed in a study done by Dagradi and colleagues[23] who followed a cohort of patients with alcoholic cirrhosis over three years and found out that esophageal varices size will depend on alcohol intake. On the other hand, Cales and Pascal[24] showed that regression of varices occurred in 16% of patient with alcoholic

cirrhosis who continued to imbibe alcohol. This will depend on opening of large portosystemic collaterals.

Risk factor for first variceal bleeding:-

Some risk factors are there in development of varices but they are not clear, known factors are: (i) pressure within the varix, (ii) variceal size (iii) tensionon the variceal wall, and (iv) grade of hepatic injury.

Portal vein pressure:-

In variceal bledding, portal pressure and hepatic venous pressure gradient should be high but they is no direct relationship between them. hepatic pressure gradient will be high with large varices.[23,24]

In a prospective study, where propanolol and placebo compared in reduction of variceal bleeding, Grozzman and colleagues[25] showed that variceal bleeding incidence is reduced if there is portal pressure less than 12mm Hg. This is aim of pharmacological to maintain pressure below 12mm Hg. Variceal size -It depends on La-place's law which state that the tension on the wall of a rigid container is proportional to the radius, so when there is increase in size of varices , here is increase in tension of wall which leads to bleeding . Numerous studies[26,27,28] have shown that the risk of variceal haemorrhage increases with the size of varices.[29,30] . The grades of the varix is as follows:

Grade I - The varices can be depressed by the endoscope.
Grade II - The varices cannot be depressed by the endoscope.
Grade III - The varices are confluent around the circumference of the oesophagus.
Variceal wall -It is depends on La-place's law. When there is increase in size of varix they will be decrease in thickness of wall which leads to bleeding. A study done by Polioand Groszman using an in vito model confirmed it.[13]

The colour of the varices appears white and opaque. Endoscopically "red spots" and "wale" markings were first described by Dagradi. They represent microtelangiectasias lead to bleeding due to increase in tension on variceal wall structure.

Beppu and colleague[29] proved that blue varices or cherry red spots are important predictors of variceal bleeding.

Grade of hepatic injury:-

Both the North Italian Endoscopic Club (NIEC)[31] and the Japanese, Prada[32] shows risk of bleeding depends on 3 factors: Child pugh score, variceal size, and red wale markings.

Child Pugh classification

Number of points

Factors	1	2	3
Bilirubin(mg/dl)	<2	2-3	>3
Albumin(g/dl)	>3.5	2.8-3.5	<2.8
Prothrombin time(sec)	1-3	4-6	>6
Ascites	none	mild	moderate
Encephalopathy	none	minimal	advanced

Common causes of portal hypertension are: Schistosomal periportal fibrosis
Liver cirrhosis

Schistosomiasis:-

Schistosomiasis is the second most prevalent disease, after malaria, in the world. It affects an estimated 200 million people in76 countries and additional 600 million people are at risk of acquiring this infection.[33]

According to the WHO, the global distribution of schistosomiasis has changed in recent years. It has been eradicated from Japan and the Lesser Antilles Islands; transmission has been stopped in Tunisia; and transmission is very low in Morocco, Saudi Arabia, Venezuela, and Puerto Rico[33] Sudan, the infection rate varying from 5 to 23%[38]Elgadal[34] conducted a study which showed schistosoma mansoni has become the dominant infection in the Gezira irrigated areas with prevalence rates upto 80% in local population. Human infections occur in the course of bathing in or wading through contaminated streams, ponds, or irrigation canals.

There are 5 species S.mansoni, S. haematobium, S. intercalatum, S. japonicum, S. mekongi in India, S. haemtobium is found.

Definitive host- man
Intermediate host snail

Egg passed in urine (S.haematobium) feces (S.mansoni, S.Japonicum and S.mekongi) these are Develop into cercariae which release in water and enter human body .All migrate the mesenteric and portal veins except S.hematobium migrate to visceral plexus. About 4 weeks after infection, female worms commence egg lay by a rate of 300 egg/day. For S.mansoni. Eggs not retained in the intestinal wall or excreted in the faeces are carried to the liver; about 50% of all eggs laid are retained in the body. An acute infection syndrome called 'Katayama fever' may occur at this time, with fever, systemic upset and eosinophilia.

Pathogenesis- kind of allergic reaction, granuloma formation (hoepil sign) Ectopic lesion[39]
Pathology colon- Acute mucosa congestion
Chronic fibroobstructive lesion
Liver- enlargement of liver
Chronic- portal hypertension

Symptoms and signs like dermatitis fever allergic reaction, pain abdomen, diarrhoea, eosinophila, hepatosplenomegaly.

Terminal stage like liver cirrhosis, ascites, splenomegaly seen. Ectopic lesion seen in brain and lung

S.haemotobium cause dysuria, haemturia

In acute stage low serum albumin, prolonged prothrombin time, ascites and hepatic encephalopathy. Alcohol abuse and hepatitis type B and C will produce additional effects.

Laboratory investigations include stool test, immunological test, biopsy from rectal region.

Treatment

Praziquantel and oxamniquine are used for treatment of schistomiasis.
Due to lack of oxamniquine against urogenital form of S.haemtobium praziquantel is first option

WHO has developed guidelines for community based treatment in villages for children of young age (School age) are treated.

When more than 20% population affected mass treatment will be implemented Other possible drugs are metrifonateartesunate and mefloquine. The degree of periportal fibrosis is as follows[35]
Grade 1 - Minimal echogenic thickening of the wall in two or more portal radicals with little change in the diameter of main portal vein
Grade 2 – Mild echogenic thickening of the walls of two or more portal radicle mainly peripherally, with little or no thickening of the wall of the main portal vein.The gall bladder wall is thickened.
Grade 3 - Moderate to severe periportal thickening of most portal vein radicals with marked narrowing of central lucency. The thickening is marked at the bifurcation of portal vein and extends to the surface of liver. the wall of main vein thickened from 2-10mm. The gallbladder wall is thickened.
Grade 4 – Marked thickening o the wall of the porta vein radicles with obliteration of central lucency in the peripheral branches forming thick echogenic bands ranging in thickness from 10-20mm, reaching the periphery of the liver .The liver is small and shrunken with irregular surface. The gall bladder greatly thickened.

Splenic vein thrombosis [8]:
Causes:-
1. Acute Pancreatitis(25%)
2. Chronic pancreatitis (65%).
3. Pancreatic carcinoma (18%).
4. Lymphoma.
5. Trauma.
6. Hyper coagulable states.

It leads to left sided portal hypertension. This results in gastrosplenic venous hypertension with superior mesenteric and portal venous pressure normal resulting more in gastric varices than esophageal varices. Left gastroepiploic vein is main collateral because splenic vein drains the spleen and the stomach through the short gastric veins, it causes isolated gastricvarices.

Gastric varices here are diagnosed endoscopically. But when not actively bleeding, Gastricvarices may be difficult to distinguish from benign prominent gastric folds.

Endoscopic ultrasonography (EUS) identifies hypoechoic, tortuous dilated blood vessels in the submucosa are characteristic for gastric varices.

Treatment by splenectomy alone. But acute bleeding can be treated endoscopically; however, rebleeding is the rule, and the mortality rate is as high as 55%.

Portal Vein Thrombosis:-
(PVT)
Portal vein thrombosis was first reported in 1868 by Balfour and Stewart
It is relatively rare condition with incidence of 0.05-0.5%, it is seen in people with cirrhosis of liver with incidence of 5-18%.

Extra hepatic portal vein obstruction is seen in 5-10% population. It may be primarily thrombosis or

caused by malignant obstruction Thrombosis can be done along the full course.

Causes:-

Inherited and acquired disorders of coagulation pathway which leads to hyper-coagulable states[36] like protein C or S deficiency, antithrombin iii deficiency resulting from malnutrition, inflammatory bowel disease, estrogen use, burns, sepsis.

Stasis or mass lesion which includes: cirrhosis of liver (0.6-40% of cirrhotic will develop (PVT)which will increase the risk of bleeding[37].20-60% of tumour of liver will develop PVT due to obstruction of portal vein or its tributaries and leads to bleeding[38].

Congenital anomalies like atrial septal defect 20% may cause portal vein thrombosis.

Inflammation of the portal vein- occurs with appendicitis, diverticulitis, chemical injury due to pancreatitis and primarysclerosing cholangitis with bile leak.

- Diagnosis of portal vein thrombosis is by ultrasound or Doppler imaging.
- Complications like variceal hemorrhage, ascites, portal hypertension seen sometimes it may extend to mesenteric arcades leading to bowel ischemia and infraction.
- Treatment is usually repeated endoscopic therapy or non-selective B-block if no contraindication.

CRYPTOGENIC LIVER CIRRHOSIS

Unknown cause of liver cirrhosis

ROCK ALL SCORE

It is to identify the patients with adverse outcome following acute upper GI bleeding Scoring system includes Clinical criteria (age, co-morbid conditions, signs of shock) Endoscopic findings (diagnosis, stigmata of acute bleeding) It is named after Tim Rockall

Variable	0	1	2	3
Age	<60	60-79	>80	
Shock	No shock	Pulse>100 BP>100 systolic	SBP<100	
Comorbid	Nil		CHF,IHD	Renal Failure Liver Failure Metastatic Cancer
Diagnosis	Mallory Weiss	All other diagnosis	GI malignancy	
Evidence of bleeding	None		Blood, Adherent clot, Spurting vessel	

Non-variceal causes of UGIB

Peptic ulcer disease (PUD):-

GRADE	DESCRIPTION	REBLEEDING RISK
1a	Active pulsatile	High
1b	Active nonpulsatile	High
2a	Nonbleeding visible vessel	High

2b	Adherent clot	Intermediate
2c	With black spot	Low
3	Clean nonbleeding ulcer bed	Low

Peptic ulcer disease is a common cause of acute UGIB, accounting for about 50% of all causes in the western countries. Principally involves promixal duodenum more than gastric ulcers- a two-fold or more difference in most series[40].Approximately 150.000 patients are hospitalized for bleeding ulcers in the United States each year. Incidence of hospitalization and surgery for this ulcers have not decreased since the 1970s,and the mortality rate from bleeding ulcers has remained at about 5% to 110% over the same period[41,42].

Most peptic ulcers arise in lesser curvature, In antral and prepyloric region and in first part of duodenum. Perforation occur more common with duodenal than gastric ulcer.

There are various causes for developing peptic ulcer disease. In that most common are diet. H. pylori, and NSAIDs. Of these factors only NSAIDs appear to be an important risk factor for the development of bleeding ulcer.

The prevalence of H.pylori infection has reduced in recent times. Hosking and co-workers reported that 71% of patients presenting with bleeding duodenal ulcers yielded culture positive for H.pylori, whereas 93% of patient presenting with non-bleeding duodenal ulcers during the same period had H. pylori infection ($p<.01$)(50). Interleukin has emerged as important mediator of inflammation in H.pylori infection

NSAID Ingestion -
NSAIDS ingestion is considered as the most important risk factor identified for the development of bleeding in patients for the development of bleeding in patients in peptic ulcer. A number of epidemiologic studies have demonstrated an increase of complicated ulcer (eg- bleeding, perforation) and overall GI complications in

Patients taking NSAIDS. Shorr[44] and associates found that the relative risk of bleeding ulcers in elderly patients taking NSAIDS in Tennessee is 0.4. In the United Kingdom Langman and colleagues [45reported] a relative risk of 4.5 for peptic ulcer with NSAIDS use.

ANTICOAGULANT THERAPY
Shorr[44] and associates reported that relative risk for a bleeding ulcer in patients taking oral anticoagulants was 3.3. The relative risk when patients were taking both oral anticoagulants and NSAID was 12.7.Ulcers that are located high on the lesser curve of the stomach or the posterior wall of duodenal bulb are more likely to rebleed.

Oesophagitis
The esophagus is rare source for significant haemorrhage accounting for 2% of patients who resented with clinically significant UGIB in one study[46]. It is due to as result of esophagitis. Esophageal inflammation due to GERD by exposing mucosa to acid -pepsin leads to an inflammatory response that can result in chronic blood loss.

Ulceration mostly superficial mucosal ulcerations generally do not bleed acutely and present as anaemia. Other causes of esophageal bleeding include medications, Crohn's disease, and radiation.

Treatment typically includes acid suppressive therapy.Endoscopic control of the haemorrhage usually with electrocoagulationor a heater probe.

Mallory – Weiss Tears:
UGIB secondary to longitudinal mucosal lacerations at the gastroesophageal junctionthat account for about 5% to 15% of cases of UGIB[47].Originaldescription by Mallory and Weiss in 1929.The classic history of vomiting and retching following alcoholic binge. These are typicalantecedent symptoms have been reported in 29% to 86% ofpatients and a history of heavy alcohol use in 30% to 60%.

Haemorrhage from thetear stops spontaneously in 80-90% of cases.Rebleeding occurs in 0% to 5%[48,49] . Mallory weiss tear account for estimated1-15% of UGIB.
Mallory weiss syndrome occurs following gastric contents prolapsed into the esophagus.

Gastritis:
Gastritis is a histologic diagnosis that indicatesinflammation in the gastric mucosa. Acute haemorrhage gastritis iscommonly associated with UGIBdue to intake of aspirin, NSAIDS, stress induced and alcohol.Gastritis is frequently diagnosed as a cause of upper GIhemorrhage[50].
Because of subepithelial hemorrhages and erosions bleeding will occur. Subepithelial hemorrhages and erosionscannot cause major bleeding, in contrast to ulcers, which mayinduce serious bleeding when erodes into arteries below themucosa.

A meta-analysis of prophylaxis for stress induced ulcers[51]indicated that H2 receptor antagonists, antacids, or sucralfate ledto a significant reduction in UGIB. However, no evidence indicates that prophylactictherapy decreases mortality rates.

Neoplasms:
Neoplasms causing upper GI bleed will produce chronic, occultbleeding but rarely produced profuse acute GI bleeding[52].They are primary tumours like adenocarcinomas, stromaltumours, neuroendocrine tumours, lymphoma, or polyps and metastatic from non-GI sources likebreast, melanoma.
Treatment mainly is aimed at surgical resection generally the first choice, if patient is not a candidate for surgery, then endoscopic therapy (injection), angiographic therapy or radiation therapy may be tried [51].

Dieulafoy's lesion:
Dieulafoy's lesion uncommon cause of upper GI bleeding, itaccounts for 1-2% of UGIB.It consists of an abnormally large vessels unlike all other vessels that penetrate the gut wall it retains thelarge caliber of its feeding vessel as it reaches the mucosa[53]. Any tiny erosion or rupture of the vessel into the lumen leads to severe UGIB. Dieulafoy's lesions aredifficult to identify unless actively bleeding or covered by a clot of recent hemorrhage.Earlier, surgical wedge resection was required. Recently indicate that endoscopic thermaltherapy or injection of sclerosant is effective. Unusually found in the proximal stomach.

Gastric antral vascular ectasia (watermelon stomach):

Gastric antral vascular ectasia rare cause of upper GI bleeding. Site is antrum or last part of stomach. It is characterized by linear red streaks running longitudinally in thegastric antrum. Because of the appearance of alternating stripes thelatter has been called watermelon stomach[54]. The lesion consists of collections of dilated venules, often with focal thrombosis, and fibromuscular hyperplasia in the propria.Intervention is required if there is anemia due to iron deficiency, and treatment include endoscopic thermaltherapy or surgical antrectomy[54].

1.1 REVIEW OF LITERATURE

Sohali bhutta et[56]al study done in rawalpindi college shows peptic ulcer was commonest cause of upper gi bleed(34%) followed by varices(21%) .Duodenitis and erosions followed it Channanna et al[57] study done in bellary studied in 150 patients, male and female ratio was 2.5:1, 40-50 age group is most common. Esophageal varices are commonest cause (56%) followed by gastric erosions (13%). Cirrhosis is common cause of UGIB.

Mohamed Abdel-Hay Aubaid [60]et al study done in cairo Egypt studied in 1089 patients .old age group dominant then middle age(71.9%). esophageal varices was commonest cause(60%) followed gastritis and ulcer. common cause of bleeding is congestive gastropathy.

M Uddin Ahmed et al [59]study in rajshashi medical study shows duodenal ulcer was the commonest cause(34%) followed by portal varices and neoplasm William A. Webb [55]et al study in Georgia studied in 125 patients with lesion found in 117, peptic ulcer (74%) was the commonest cause followed by Mallory weiss syndrome (9.8%)and variceal bleeding Gimiga etal [62] study in children shows erosive gastritis (33%) was the commonest cause followed by esophagitis (14%) Hyasinta et al [61]shows esophageal varices is the most common cause(51%) followed by peptic ulcer John n crook [58]et al shows duodenal ulcer(42%) is commonest followed varies

1.2 AIMS AND OBJECTIVES

- To compare common etiology of upper gastrointenstional bleeding between Dakshina Kannada,Keralaand Udupi District.
- To determine the common etiological factors of upper gastrointestinal bleeding.
- To establish the site and source of UGI bleeding through endoscopic evaluation.

CHAPTER 2
METHODOLOGY

Source of Data:

The patient admitted in our hospital wards with the history of any upper gastrointestinal bleed from will be taken up for the study

Study period: October 2014 to October 2017

Sample size: 100 patients are taken for the study and divided into 3 groups Dakshina Kannada, UDUPI and Kerala

Study method: Prospective study

Statistical analysis: Using Fischer exact test

Pvalue of <0.05 was considered to be significant

METHOD OF COLLECTION OF DATA-

- As soon as the patient is admitted a detailed history regarding nature of bleeding whether it has ceased at the time of admission and the time since the onset will be recorded.
- As soon as patient admitted address also will be noted and grouped into 1)Dakshina Kannada 2)Udupi 3)Kerala
- The patients will also be interrogated regarding symptoms of nausea, vomiting, dysphagia, regurgitation, heart burn, abdominal pain, appetite, weight gain or loss and recent changes in bowel habits prior to the bleed.past history of ingestion of drugs over the preceding 48 hrs and frequent ingestion over the preceding months will be enquired about and previous histories of cardiovascular, respiratory, liver diseases will be thoroughly evaluated
- habit of consumption of alcohol by the patient
- Examination of the abdomen for any area of tenderness, palpable masses, ascites and rectal examination will be carried out.
- Based on clinical data obtained a provisional diagnosis will be made.
- These patients will be then submitted to oesophagogastro duodenoscopy using a fibreoptic instrument

INCLUSION CRITERIA

All types of upper GI Bleeds admitted in KSHEMA Hospital

EXCLUSION CRITERIA

Age below 18

Patients who are not willing

CHAPTER 3
RESULTS

TABLE 1: DISTURBTION ACCORDING TO CASES

DISEASE	TOTAL	FREQUENCY
VARICES	48	48%
DUODENAL ULCER	11	11%
GASTRIC ULCER	11	11%
MALLORY WEISS SYNDROME	4	4%
PANGASTRITS	6	6%
NORMAL	5	5%
EROSIONS	12	12%
CA STOMACH	2	2%
TOTAL	100	100%

FIG 1- DISTRIBUTION ACCORDING TO CASES

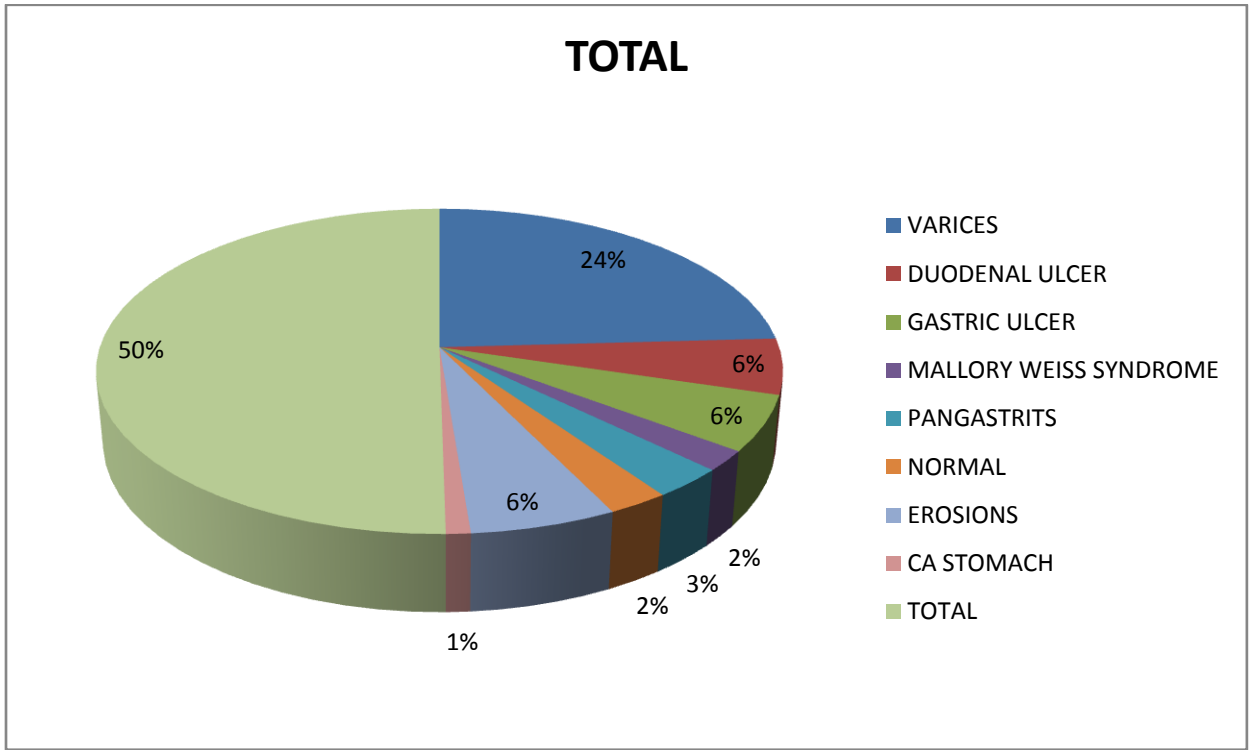

TABLE 2 : DISTRIBUTION OF PATIENTS ACCORDING TO SEX

ETIOLOGY	MALE	FEMALE	TOTAL
VARICES	42	6	48
DUODENAL ULCER	7	4	11
GASTRIC ULCER	10	1	11
MALLORY WEISS SYNDROME	2	2	4
PANGASTRITS	4	2	6
NORMAL	4	1	5
EROSIONS	8	4	12
MALIGNANCY	0	2	2
TOTAL	77	23	100

FIG 2.1 : DISTRIBUTION OF CASES ACCORDING TO SEX (MALE)

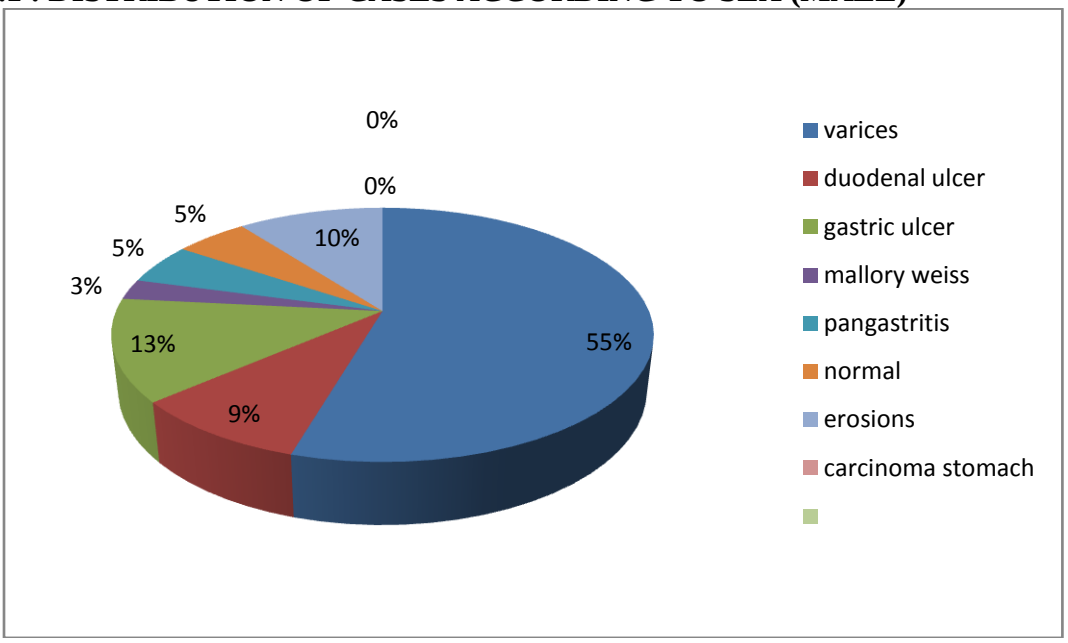

FIG.2.2: DISTRIBUTION OF CASES ACCORDING TO SEX (FEMALE)

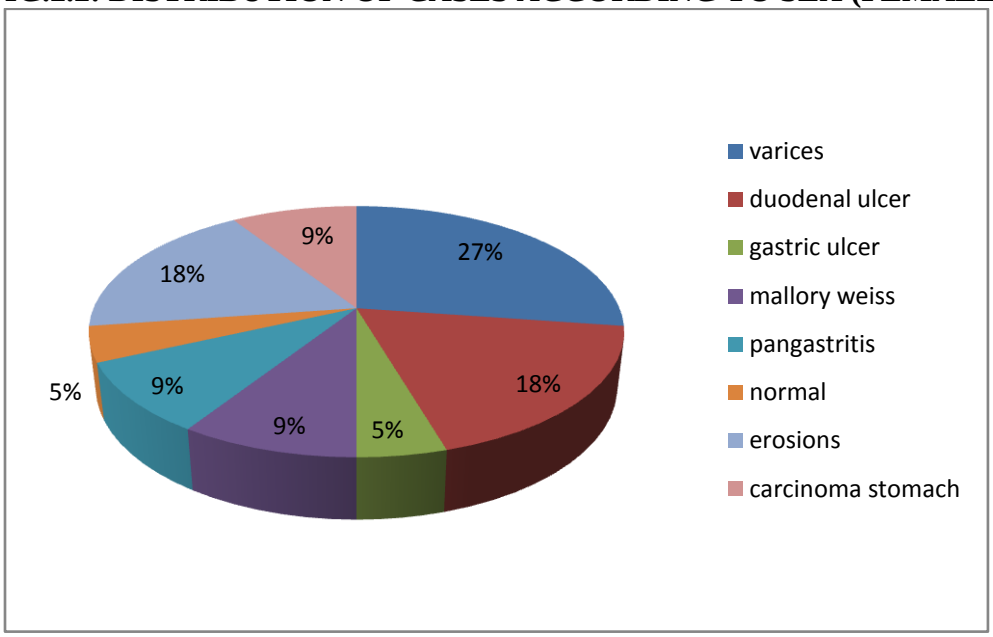

TABLE 3: DISTRIBUTION OF PATIENTS ACCORDING TO PRESENTING SYMPTONS

SYMPTONS	CASES
HEMTEMESIS	76 (76%)
MELENA	24(24%)
HEMATOCHEZIA	0

FIG 3: DISTRIBUTION OF PATIENTS ACCORDING TO PRESENTING SYMPTONS

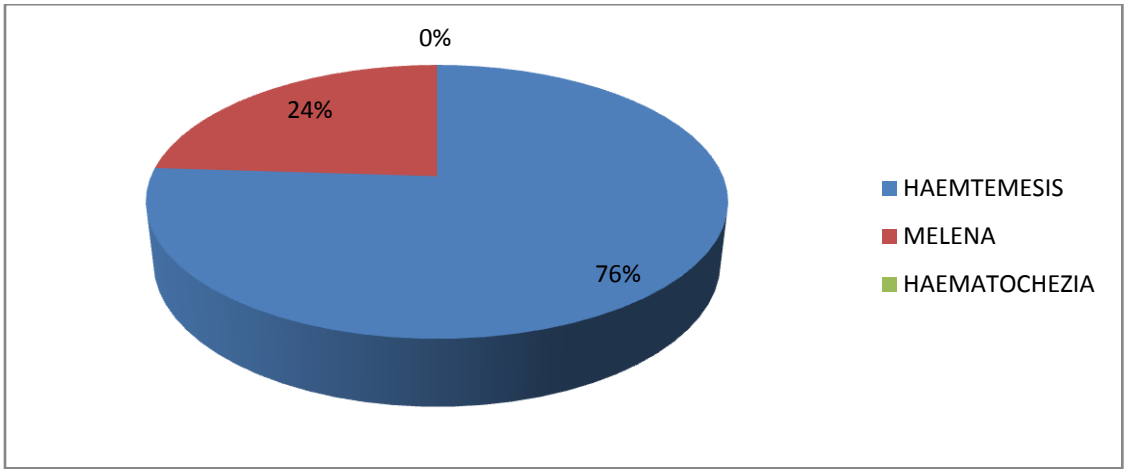

TABLE 4 : DISTRIBUTION OF PATIENTS ACOORDING TO ETIOLOGY OF PORTAL HYPERTENSION

S.NO	ETIOLOGY	%
1	ALCHOLIC CIRRHOSIS	46 95%
2	CRYPTOGENIC	1 0.02%
3	PERIPORTAL FIBROSIS	1 0.02%

FIG 4: DISTRIBUTION OF PATIENTS ACCORDING TO ETIOLOGY OF PORTAL HYPERTENSION

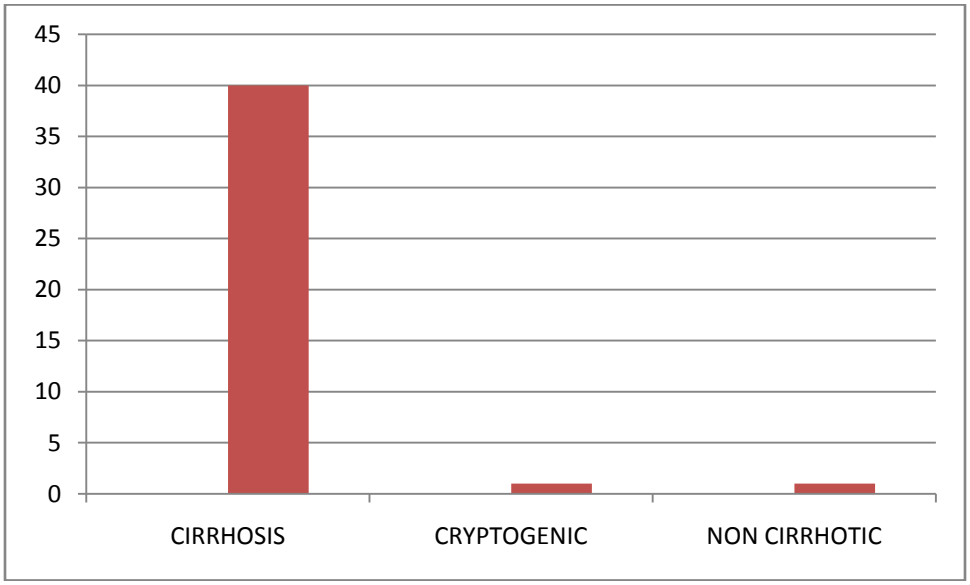

TABLE 5: DISTRIBUTION OF PATIENTS ACCORDING TO AGE

AGE	VARICES	PUD	EROSIONS	PANGASTRITIS	MW	CARCINOMA STOMACH	NORMAL
18-30	-	3	-	1	-	-	1
31-40	12	3	-	1	2	-	2
41-50	9	5	5	-	-	2	-
51-60	16	10	2	3	2	-	1
61-70	10	-	3	1	-	-	1
71-80	1	1	2	-	-	-	-
TOTAL	48	22	12	6	4	2	5

FIG 5: DISTRIBUTION OF CASES ACCORDING TO AGE

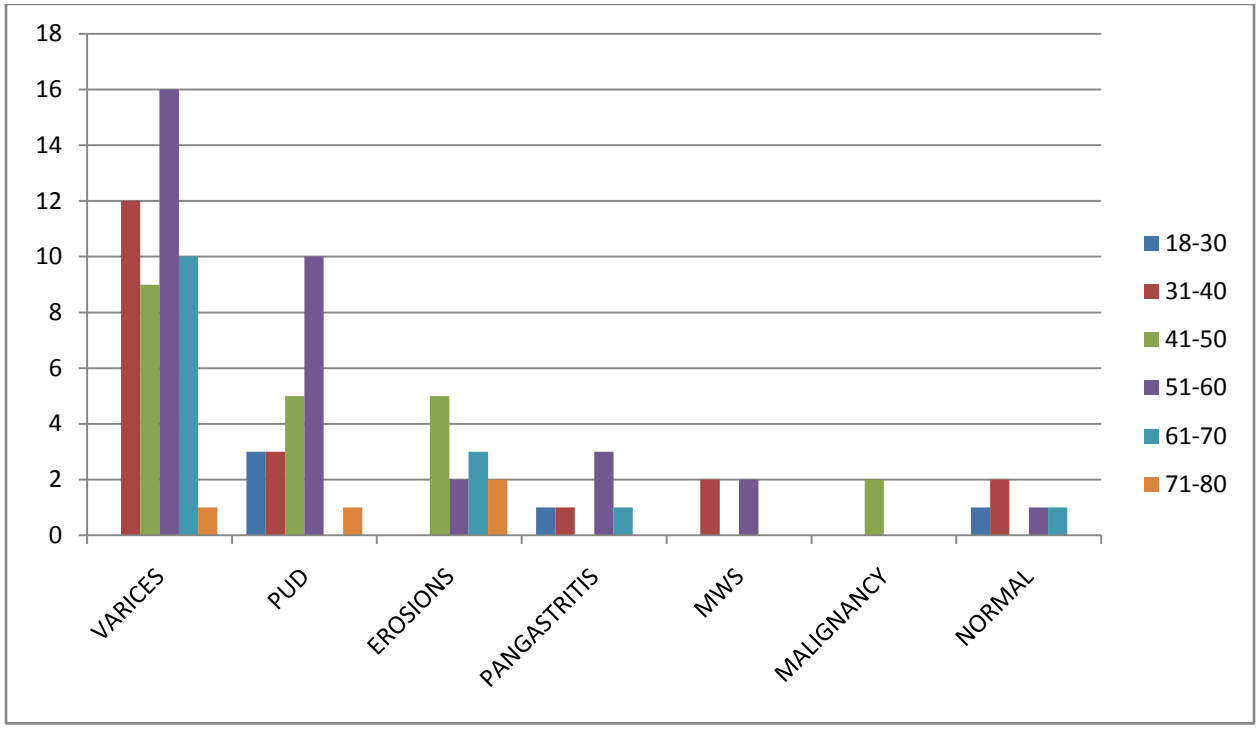

TABLE 6 DISTRIBUTION OF CASES ACOORDING TO EROSIONS AND ULCER ETIOLOGY

EROSIONS ETIOLOGY	CASES
ALCHOL	21
DRUG	1

ULCER ETIOLOGY	CASES
ALCHOL	22
DRUG	0

TABLE 7 : DISTRIBUTION OF CASES ACCORDING TO GEOGRAPHICAL DISTRIBUTION

GEOGRAPHICAL DISTRIBUTION	CASES
DAKSHINA KANNADA	37(37%)
KERALA	38(38%)
UDUPI	25(25%)
TOTAL	100

FIGURE 6: DISTRIBUTION OF CASES ACCORDING TO GEOGRAPHICAL DISTRIBUTION

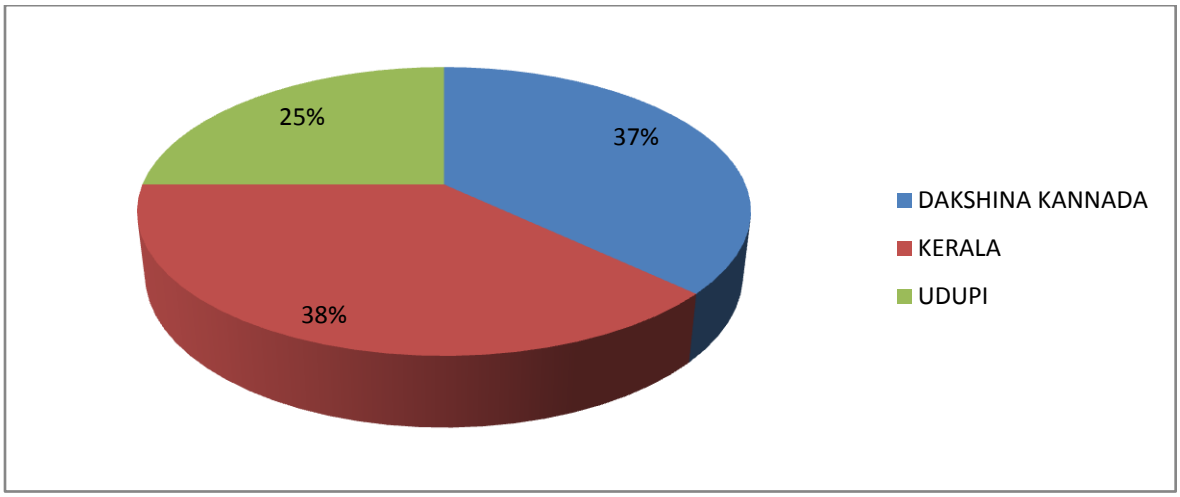

TABLE 8: VARICES GRADING

VARICES	CASES	%
FUNDAL	4	8.5
ESOPHAGEAL	38	83
BOTH	4	8.5

TABLE 9: DISTRIBUTION OF DISEASES ACCORDING TO GEOGRAPHICAL DISTRIBUTION

DISEASE	KERALA	UDUPI	DAKSHINA KANNADA	TOTAL
VARICES	23	9	16	48
PUD	8	6	8	22
EROSIONS	3	4	6	13
PANGASTRITIS	1	3	2	6
MALLORY WEISS SYNDROME	1	0	3	4
MALIGNACY	2	0	0	2
NORMAL	0	3	2	5
TOTAL	38	25	37	100

FISCHERS EXACT TEST P VALUE=0.211 NS

TABLE 10: DISTRIBUTION OF CASES ACCORDING TO CIRRHOSIS

	VARICES PRESENT	ABSENT
CIRRHOSIS PRESENT	46	0
ABSENT	2	52

FISCHERS EXACT TEST P VALUE 0.0001 HS

TABLE 11: DISTRIBUTION OF CASES ACCORDING TO PRSENTING SYMPTON IN KERALA

SYMPTOM	CASES
HAEMTEMESIS	28
MELENA	10
HAEMTOCHEZIA	0

FIGURE 7 –DISTRIBUTION OF CASES ACCORDING TO PRESENTING COMPLAIN IN KERALA

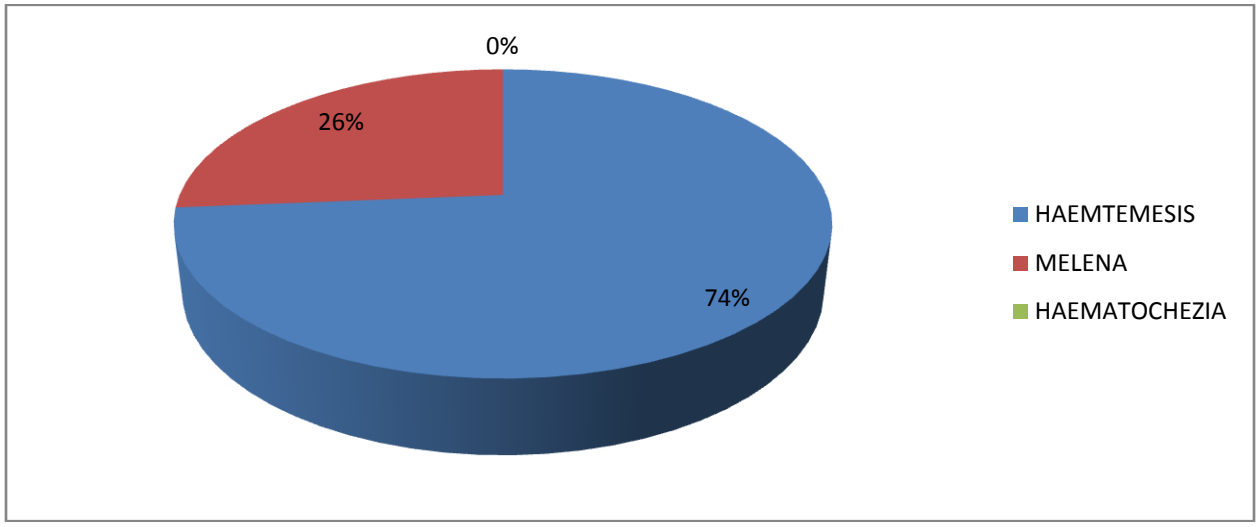

TABLE 12: DISTRIBUTION OF CASES ACCORDING TO ETIOLOGY OF PORTAL HYPERTENSION IN KERALA

ETIOLOGY OF PORTAL HYPERTENSION	CASES
ALCHOLIC CIRRHOSIS	19
CRYPTOGENIC LIVER DISEASE	1
PERIPORTAL FIBROSIS	1

FIGURE 8: DISTRIBUTION OF CASES ACCORDING TO ETIOLOGY OF PORTAL HYPERTENSION IN KERALA

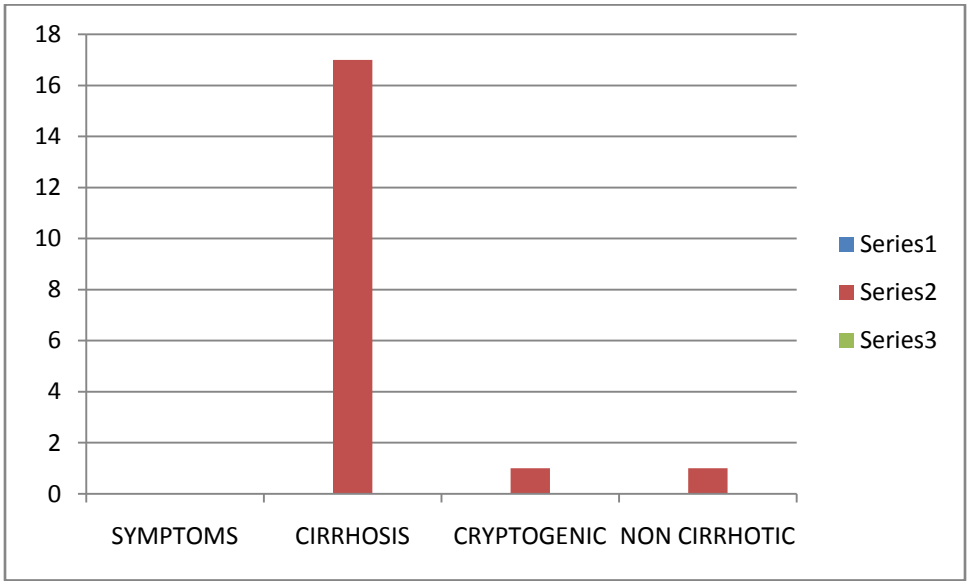

TABLE 13: DISTRIBUTION OF CASES ACCORDING TO SEX IN KERALA

DISEASE	MALE	FEMALE
VARICES	20	3
EROSIONS	2	1
DU	2	3
GU	2	1
MW		
MALIGNANCY	1	1
NORMAL		
PANGASTRITIS		1

FIGURE 9.1: DISTRIBUTION OF CASES ACCORDING TO SEX (MALE) IN KERALA

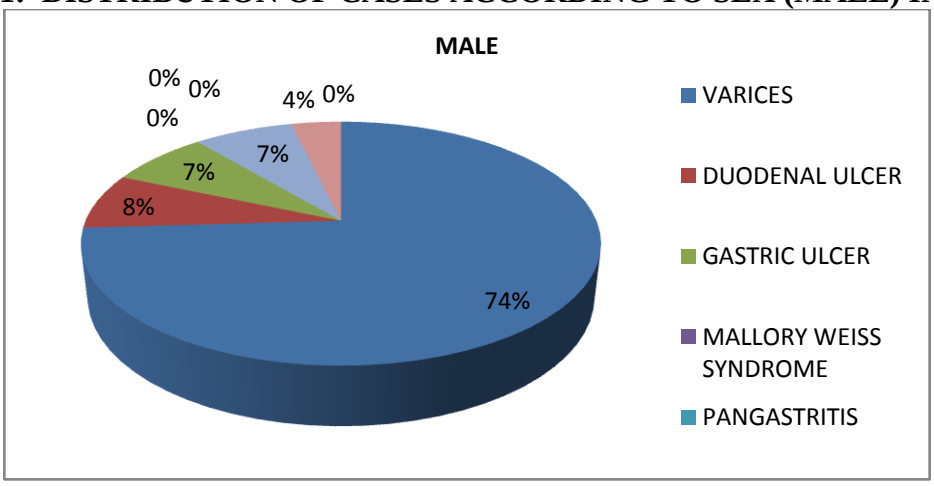

FIGURE 9.2:DISTRIBUTION OF CASES ACOORDING TO SEX (FEMALE) IN KERALA

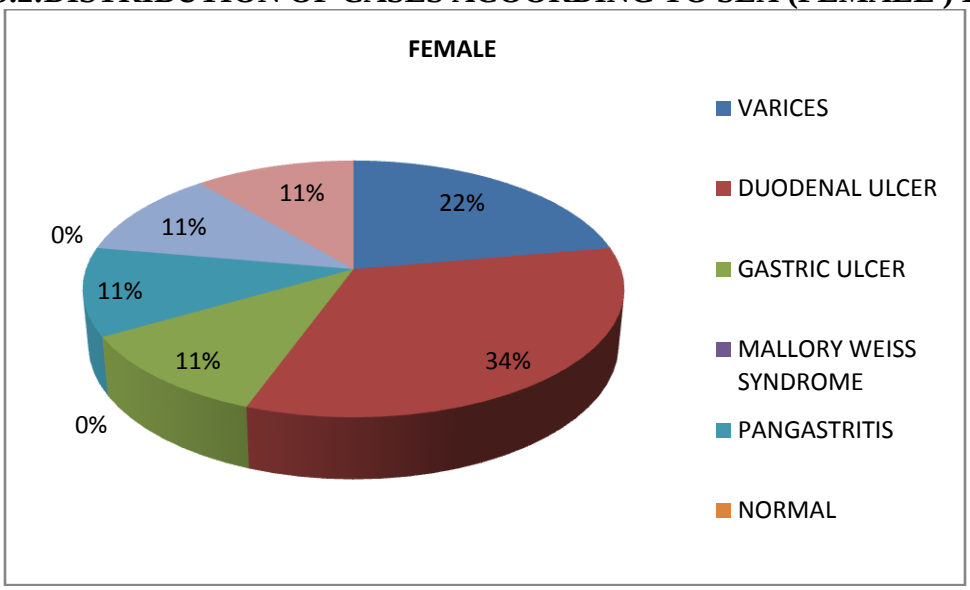

TABLE 14 : DISTRIBUTION OF CASES ACCORDING TO AGE IN KERALA

AGE	VARICES	PUD	EROSIONS	PANGASTRITIS	MW	MALIGNANCY	NORMAL
18-30		-		1	-	-	-
31-40	5	-		-	1	-	-
41-50	4	1		-	-	2	-
51-60	9	3	1	-	-	-	-
61-70	4	4	1	-	-	-	-
71-80	1	-	1	-	-	-	--
	-	-	-	-	-	-	-
TOTAL	23	8	3	1	1	2	-

FIGURE 10: DISTRIBUTION OF CASES ACCORDING TO AGE IN KERALA

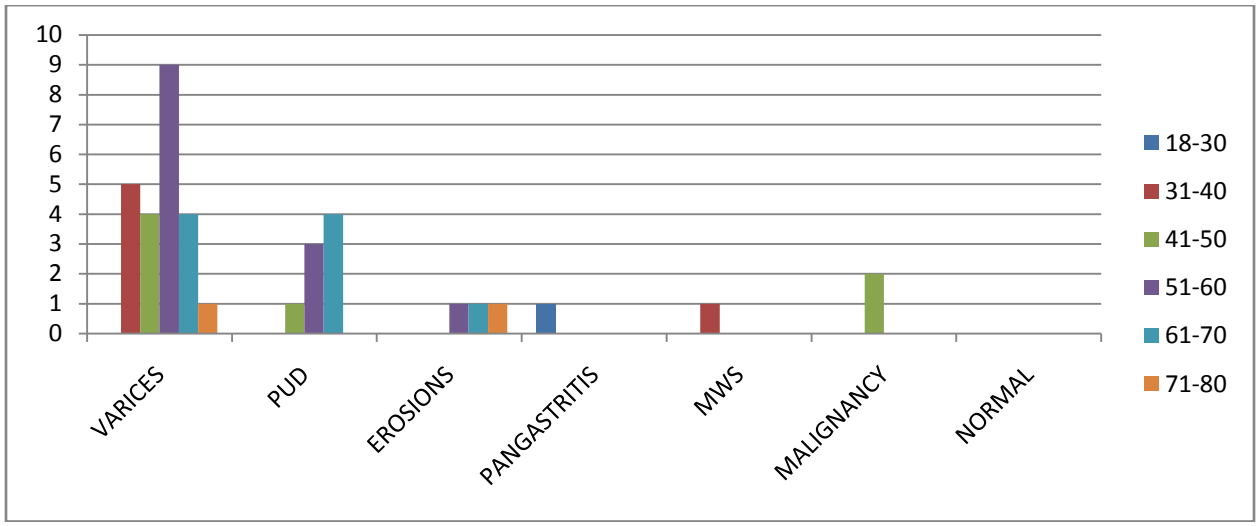

TABLE 15: DISTRIBUTION OF CASES ACCORDING TO EROSIONS AND ULCER ETIOLOGY IN KERALA

EROSIONS ETIOLOGY	CASES
ALCHOL	3
DRUG	1
ULCER ETIOLOGY	**CASES**
ALCOHOL	8
DRUG	0

TABLE 16 : DISTRIBUTION OF CASES ACCORDING TO PRESENTING SYMPTON IN UDUPI

SYMPTONS	CASES
HAEMTEMESIS	19
MELENA	6

FIGURE 11: DISTRIBUTION OF CASES ACCORDING TO PRESNTING SYMPTONS IN UDUPI

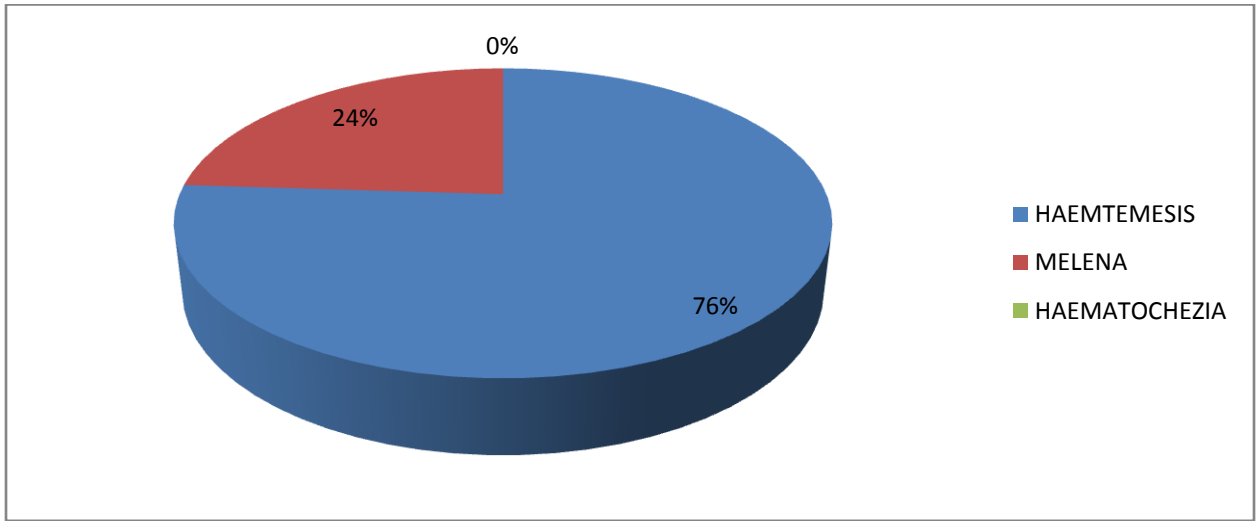

TABLE 17 :DISTRIBUTION OF CASES ACCORDING TO PORTAL HYPERTENSION ETIOLOGY IN UDUPI

ETIOLOGY	CASES
ALCHOLIC CIRRHOSIS	12
CRYPTOGENIC LIVER DISEASE	0
PERIPORTAL FIBROSIS	0

FIGURE 12- DISTRIBUTION OF CASES ACCORDING TO PORTAL HYPERTENSION ETIOLOGY IN UDUPI

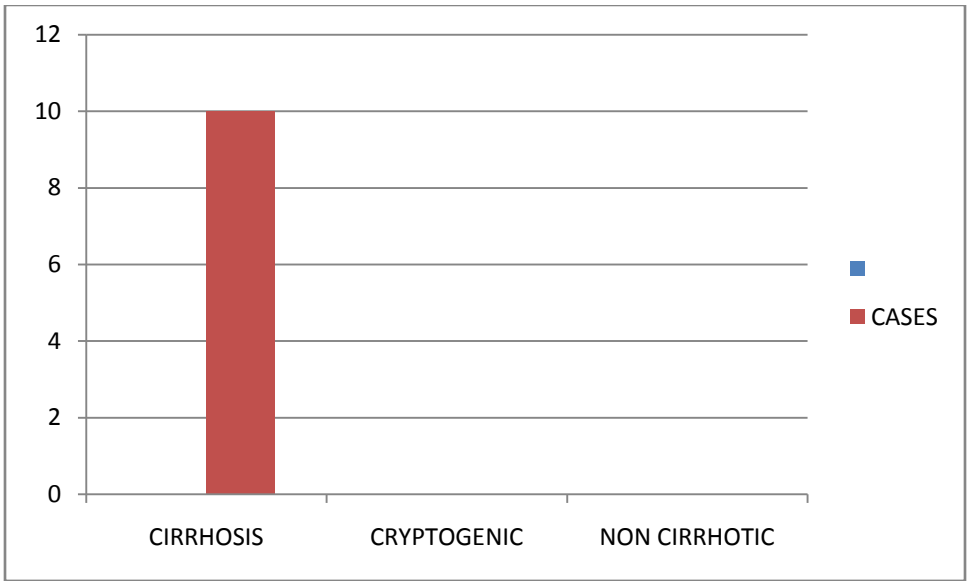

TABLE 18:DISTRIBUTION OF CASES ACCORDING TO SEX IN UDUPI

DISEASE	MALE	FEMALE
VARICES	9	
EROSIONS	2	2
DU		1
GU	5	
MW		
MALIGNANCY		
NORMAL	2	1
PANGASTRITIS	2	1

FIGURE 13.1: DISTRIBUTION OF CASES ACCORDING TO SEX (MALE) IN UDUPI

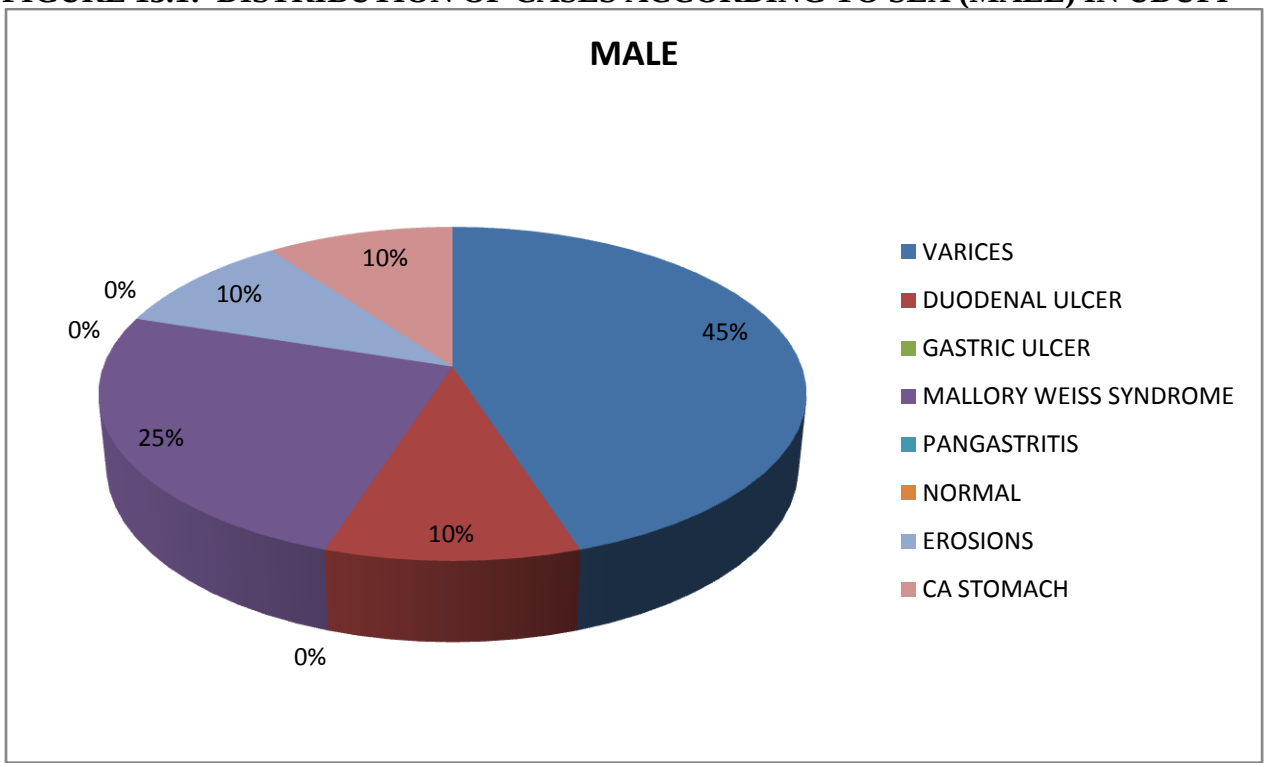

FIGURE 13.2 DISTRIBUTION OF CASES ACCORDING TO SEX(FEMALE) IN UDUPI

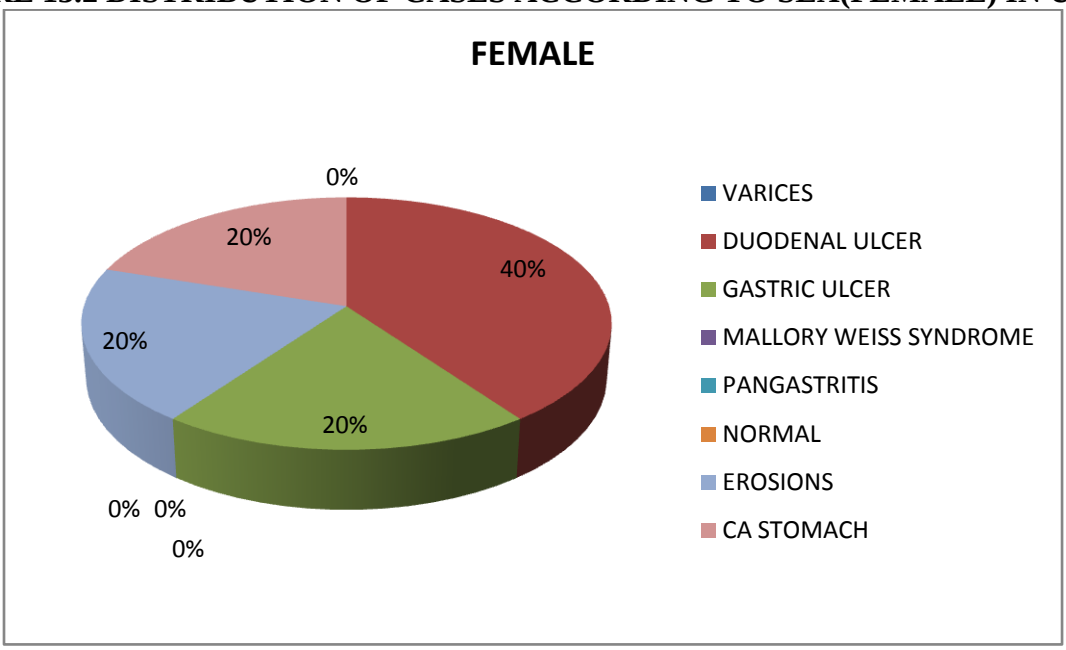

TABLE 19: DISTRIBUTION OF CASES ACCORDING TO AGE IN UDUPI

AGE	VARICES	PUD	EROSIONS	PAN	MW	MALIGNANCY	NORMAL
18-30		**1**					**1**
31-40	3	1		1			1
41-50	2	1	1				
51-60	3	3		1			
61-70	1		2	1			1
71-80			1				
TOTAL	9	6	4	3	0	0	3

FIGURE 14: DISTRIBUTION OF CASES ACCORDING TO AGE IN UDUPI

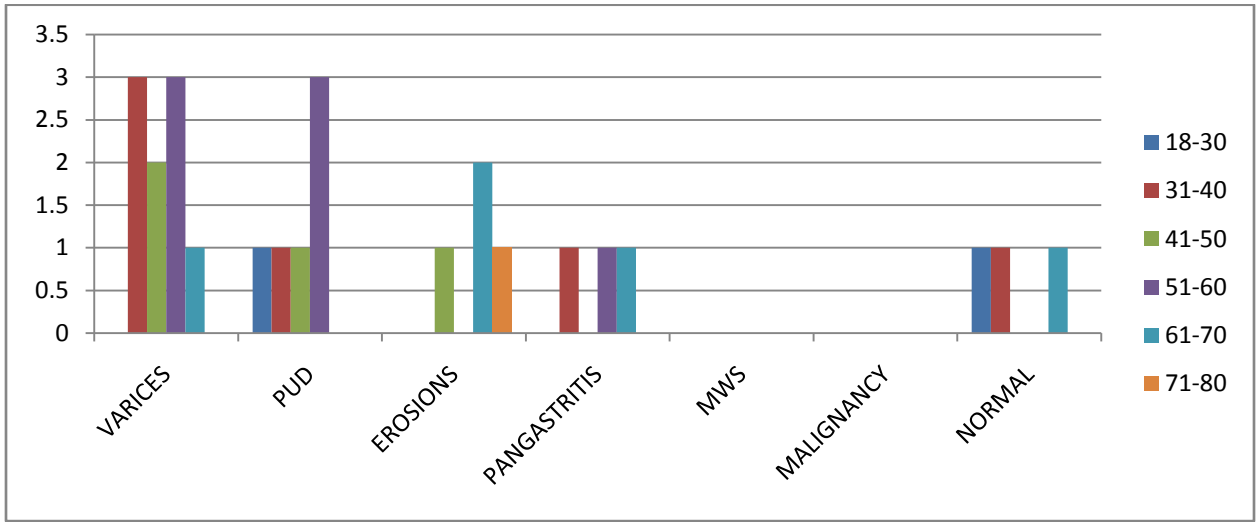

TABLE 20: DISTRIBUTION OF CASES ACCORDING TO ULCER AND ETILOGY IN UDUPI

EROSIONS ETIOLOGY	CASES
ALCHOL	7
DRUG	0

ULCER	CASES
ALCHOL	6
DRUG	0

TABLE 21DISTRIBUTION OF CASES ACCORDING TO PRESENTING COMPLAINT IN DAKSHINA KANNADA

SYMPTONS	CASES
HAEMTEMESIS	28
MELENA	10

FIGURE 15: DISTRIBUTION OF CASES ACCORDING TO PRESENTING COMPLAINT IN DAKSHINA KANNADA

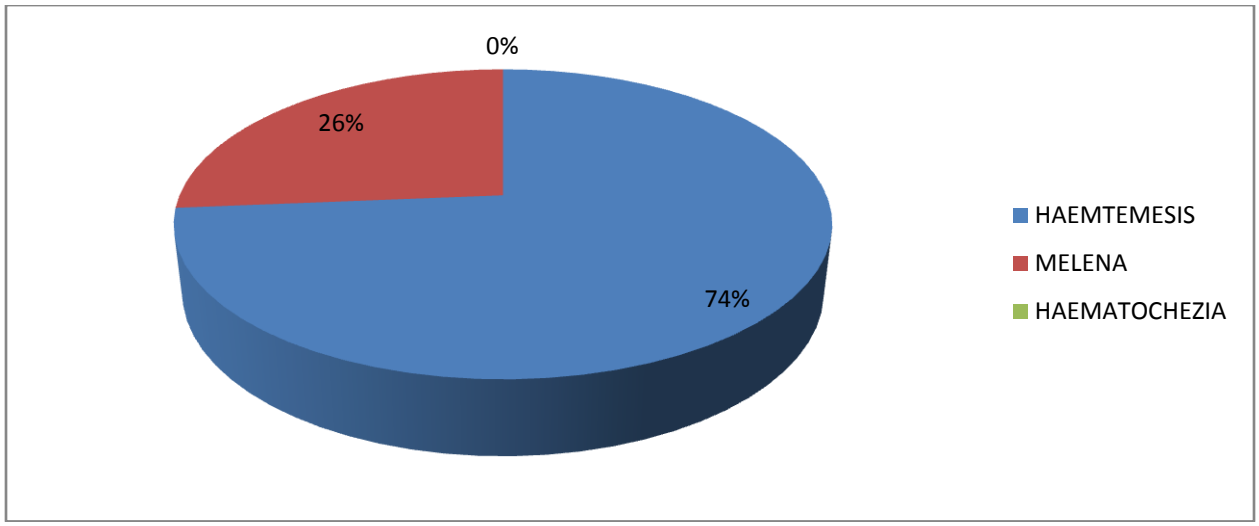

TABLE 22DISTRIBUTION OF CASES ACCORDING TO PORTAL HYPERTENSION ETIOLOGY IN DAKSHINA KANNADA

PORTAL HYPERTENSION ETIOLOGY	CASES
ALCHOLIC CIRRHOSIS	15
CRYPTOGENIC LIVER DISEASE	0
PERIPORTAL FIBROSIS	0

FIGURE 16 DISTRIBUTION OF CASES ACCORDING TO PORTAL HYPERTENSION ETIOLOGY IN DAKSHINA KANNADA

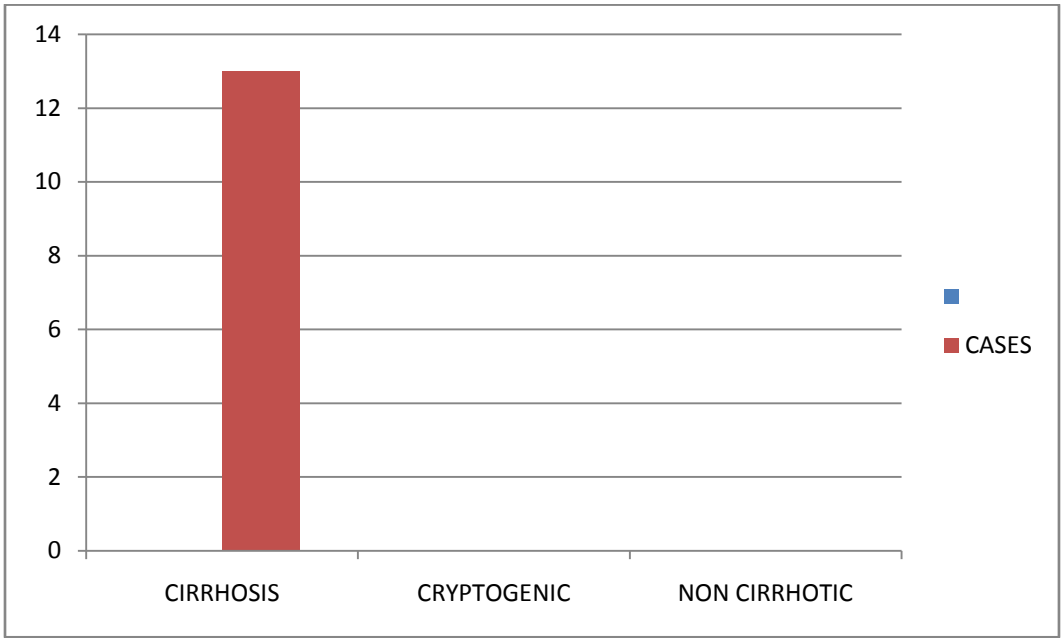

TABLE 23DISTRIBUTION OF CASES ACCORDING TO SEX IN DAKSHINA KANNADA

DISEASE	MALE	FEMALE
VARICES	13	3
DU	4	0
GU	4	0
EROSIONS	4	2
PAN	2	0
MW	2	1
MALIGNANCY	2	0
NORMAL	0	0

FIGURE 17.1: DISTRIBUTION OF CASES ACCORDING TO SEX(MALE) IN DAKSHINA KANNADA

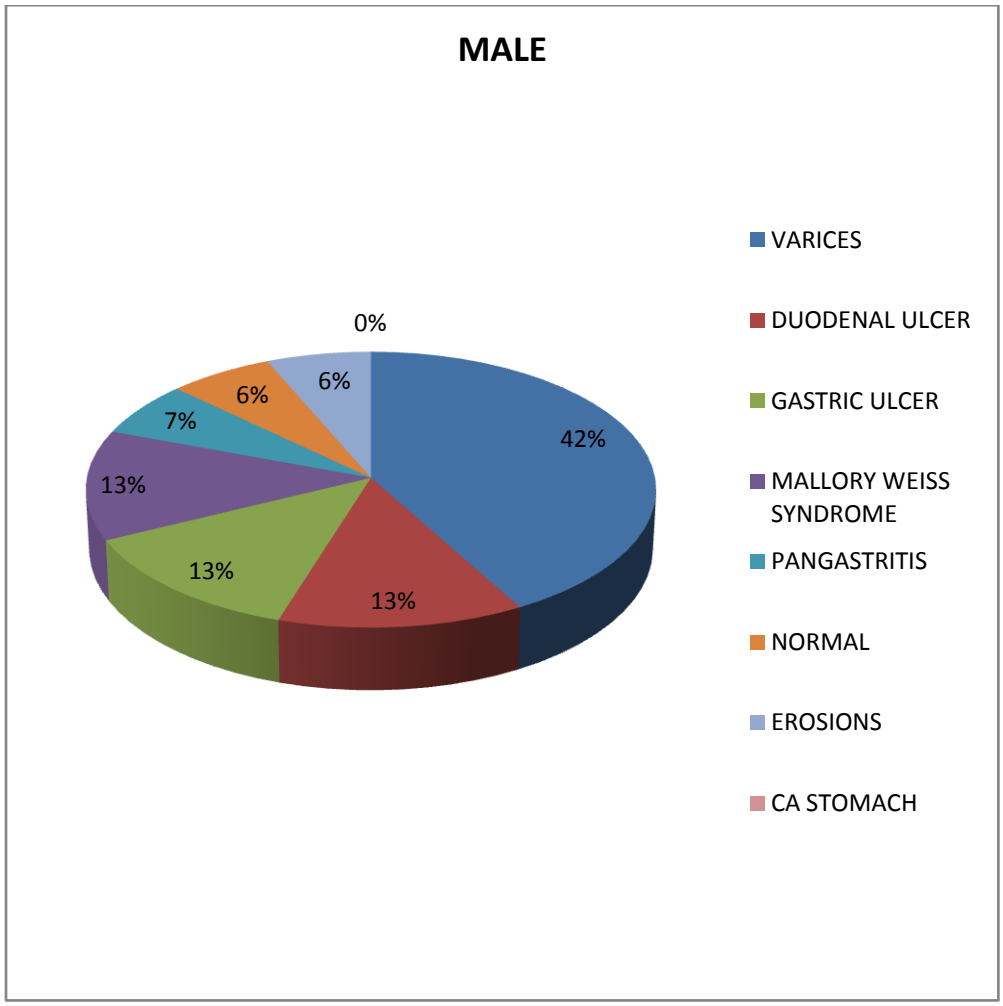

FIGURE 17.2: DISTRIBUTION OF CASES ACCORDING TO SEX(FEMALE) IN DAKSHINA KANNADA

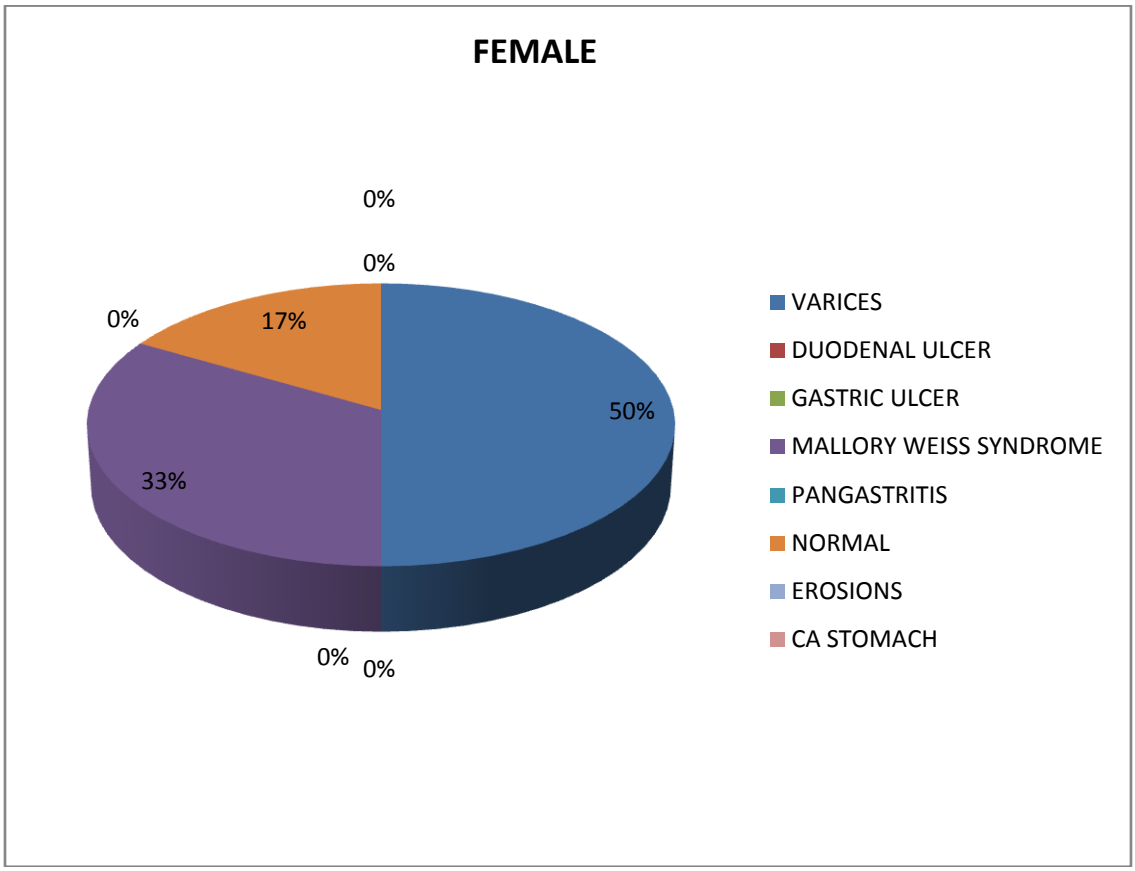

TABLE 24: DISTRIBUTION OF CASES ACCORDING TO AGE IN DAKSHINA KANNADA

AGE	VARICES	PUD	EROSIONS	PANGASTRITIS	MW	MALIGNANCY	NORMAL
18-30	-	-	-	-	-	-	-
31-40	5	2	-	-	1	-	1
41-50	4	1	3	-	-	-	-
51-60	5	1	1	2	2	-	-
61-70	1	3	1	-	-	-	-
71-80	1	1	1	-	-	-	1
TOTAL	16	8	6	2	3	0	2

FIGURE 18: DISTRIBUTION OF CASES ACCORDING TO AGE IN DAKSHINA KANNADA

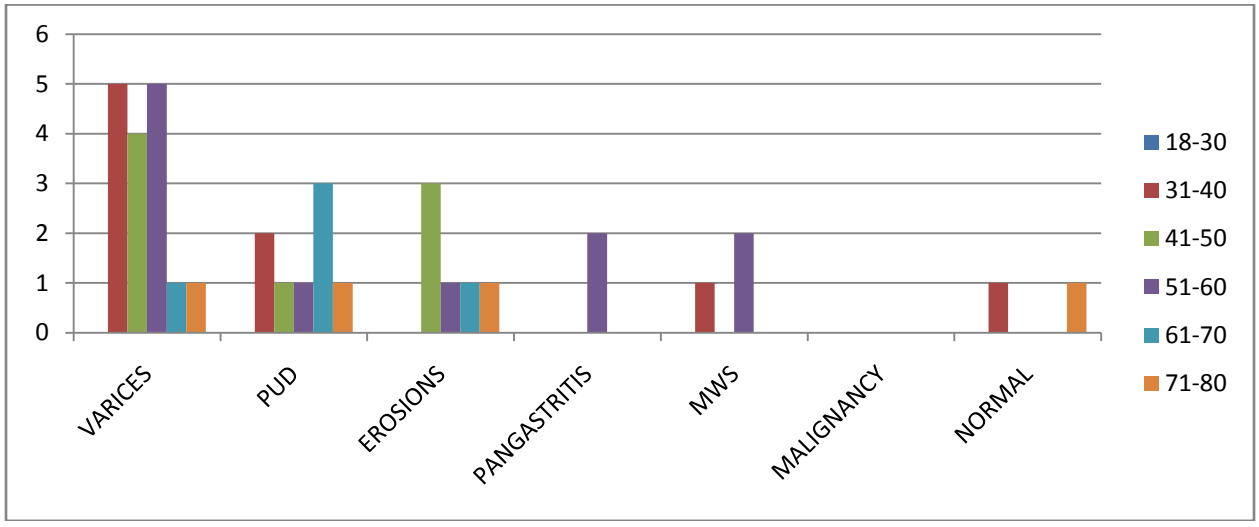

TABLE 25:DISTRIBUTION OF CASES ACCORDING TO ULCER AND EROSIONS ETIOLOGY

EROSIONS ETIOLOGY	CASES
ALCHOL	8
DRUG	0

ULCER ETIOLOGY	CASES
ALCHOL	8
DRUG	0

CHAPTER 4
DISCUSSION

Upper GI bleeding is one of the emergency conditions to admit in surgical intensive care unit. Upper GI scopy is the important procedure to evaluate all kinds of upper GI bleed.It is done by standard gastroenterologist to subside diagnostic error.

CAUSES OF UPPER GI BLEED

Author	Total	Varices	Erosions	Gastric Ulcer	Duodenal ulcer	mw	malignancy	normal
Croock[58]	786	11%	11%	18%	42%	-	-	-
present	100	48	12	11	11	4	2	5

In this study the commonest cause of upper GI bleedwas esophageal varices seen in 48% of the patients and was secondary to cirrhosis with portal hypertension. But study conducted by Croock et al shows the peptic ulcer disease as commonest cause of upper GI bleed, other causes were extra hepatic portal hypertension.

AGE AND SEX DISTRIBUTION

AU Author	Total	Male	Female	Median age
Hyasinta jaka et,al[61]	240	176(73%)	64(26.6%)	31-40
Present study	100	77(77%)	23(23%)	51-60

In the present study consisting of total 100 patients major were presented with upperGI bleed.In this study males were 77%, whereas females were 23%. The median age was 31-40. In study conducted by Hyasinta Jaka et al majority were males 73% and females 26.6% and median age was 51-60 .

In the present study alcohol was commonest precipitating factor for cirrhosis of liver and remained as main etiology of upper GI bleed.
In this study we divided the cases into 3 groups 1) Dakshina Kannada 2) Kerala 3) Udupi and compare the etiological factors between the three groups which shows no significance of geographical distribution between 3 groups.
P value 0.211

In this study cirrhosis of liver is main cause of upper GI bleed(P value-0.0001).

CHAPTER V
CONCLUSION

Endoscopy is essential for evaluating of upper GI bleed.In this study endoscopy provided diagnosis in 96% of patient. The most common cause of upper GI bleed was esophageal varices. There is no geographical distribution of etiology of upper GI bleed in population of Dakshina Kannada, Kerala and Udupi.

Cirrhosis is the main etiological factor of upper GI bleed in this study.

ANNEXURE I – ETHICAL COMMITTEE CERTIFICATE

INSTITUTIONAL ETHICS COMMITTEE
K.S. HEGDE MEDICAL ACADEMY
(Constituent College of Nitte University)
Post Nithyananda Nagar, Deralakatte, Mangalore - 575 018, Karnataka, India.
Phone : 0824-2204490/91/92 Fax : 0824-2204162 E-mail : dean.kshema@nitte.edu.in Website : www.nitte.edu.in

Ref. INST.EC/EC/094/2014-15

Date : 17.09.2014

To

Dr. VVSM Kumar Dontamsetty
Ist year Post graduate
Department of General Surgery
K. S. Hegde Medical Academy,
Deralakatte, Mangalore – 575018

Through the Head of the Department of General Surgery

Ethical Clearance is hereby issued to **Project Titled: "A Study of Upper Gastro Intestinal Bleeding by Endoscopy"** by Dr. VVSM Kumar Dontamsetty, Post Graduate, Department of General Surgery, K. S. Hegde Medical Academy Deralakatte, Mangalore, as discussed and approved by members of the Institutional Ethics Committee during the meeting held on 10th September 2014.

Dr. Sathyanarayana Rao K.N
Member Secretary
Institutional Ethical Committee

Member Secretary
INSTITUTIONAL ETHICS COMMITTEE
K. S. Hegde Medical Academy
NITTE (DEEMED TO BE UNIVERSITY)
NITHYANANDA NAGAR - 575 018

ANNEXURE II – SYNOPSIS

A STUDY OF UPPER GASTRO INTESTINAL BLEEDING BY ENDOSCOPY

MS Student Name: DR.VVSM KUMAR DONTAMSETTY
Name of Guide: DR BALAKRISHNA N SHETTY

Department of General Surgery
KS Hegde Medical Academy
Nitte University
Mangalore – 575018

Month and Year of Submission
October 2014

Name of the candidate: DR VVSM KUMAR DONTAMSETTY

Title of MS research protocol: A STUDY OF UPPER GASTROINTESTINAL BLEEDING BY ENDOSCOPY

Name and designation of the Guide: DR BALAKRISHNA N SHETTY
HEAD OF THE UNIT
GEN SURGERY
KSHEMA HOSPITAL

……..……
Signature of the Candidate
Name

……..……. ……..…….
Name
Signature of the Guide

…….……
Signature of the HOD

……
Signature of the Dean

NEED FOR THE STUDY:

Upper gastrointestinal haemorrhage is one of the important causes ofadmission in Surgical emergency ward. Despite modern techniques of resuscitation, anesthesia and surgery it has a significant mortality.

Earlier years Barium meal examination had been performed as one of the important diagnostic investigation for acute bleeding. It had two major drawbacks. Erosions and small ulcers cannot be picked up. If a lesion is shown it may not be the actual source of the bleeding.

Gastroscopy had been on use for many years by a few advocates as a visual diagnostic approach. But gastric lesions account only for about a half of all bleeding episodes.

Fibreoptic instruments have recently facilitated and extended the range of examinations. The latest generations are highly flexible and maneuverable 'panendoscopes' which allow a complete survey of the esophagus, stomach and duodenum.Remarkable progress in fibreoptic endoscopy during the last two decades has affected the management of many gastrointestinal disorders. Major technical advances include forward viewing endoscopes with complete tip control and sufficient length to permit direct visualization, of mucosal lesions as far distal as the descending duodenum. Our study is aimed at studying the role of upper gastro intestinal endoscopy in gastro intestinal bleeding.

INTRODUCTION

Gastrointenstinal bleeding is defined as the development of sudden blood loss from the GIT leading to haemetemesis,malena, hematochezia[1].

Hemorrhage from the GIT is broadly divided intoBleeding from the upper gastro intestinal tract i.e, proximal to the site of the ligament of TreitzFrom the lower gastro intestinal tract i.e, distal to the ligament of Treitz[(1)].

In patients with UGIB, the most common etiologies are as follows: Peptic ulcer (35%-50%), gastroduodenal erosions (8%-15%), esophagitis (5%-15%), varices(5%-10%), Mallory-Weiss tear (15%), vascular malformations (5%), with other conditions (e.g.Malignancy)

REVIEW OF LITERATURE

- WILLIAM WEBB.MD,FACS.,LINDAMcDANIEL,R N RONNY C.JOHNSONM.D, DOYLE HAYNES, MD FACS..endoscopic evalution of 125 cases of upper gi bleeding on it gastric ulcer is the most common cause..and it is stated that.. Early endoscopy not only help in outcome but also in decrease the longer stay of patients..
- MANDANA RAFEEY,MARYAM SHOARAN,HAMIDEH MAJIDY. Diagnostic endoscopy and clinical characteristics of gastrointenstinal bleeding in children of age 0-18 retrospective analysis and got erosiveesophagitis as common cause and varices less common..
- RC MISRA, A TEWARI,SK JAIN R DEWAN, Study on clinco endoscopic correlation with patients in upper gi bleeding..and found erosive gastritis and esophagitis as common cause

and duodenal ulcer as second cause.endoscopy could make a correct diagnosis in 96%of all cases compared clincal 75%

AIMS AND OBJECTIVES

- To determine the common etiological factors of upper gastrointestinal bleeding.
- To compare the geographical distribution between three groups
  1)Dakshina Kannada
  2)Kerala
  3)Udupi
- To establish the site and source of UGI bleeding through endoscopic evaluation.

Source of data

The patient admitted in our hospital wards with the history of any upper gastrointenstinal bleed from October 2014 to October 2016 will be taken up for the study**.**

Inclusion criteria

All types of upper Gastrointenstinal Bleeds admitted in KSHEMA Hospital

Exclusion criteria

- Age below 18
- High risk patients
- Patients who are not willing

MATERIALS AND METHODS

This is a prospective study to be conducted in the department of General surgery, KSHEMA, Mangalore from October 2014 to October 2016.

Method of collection of data

- As soon as the patient is admitted a detailed history regarding nature of bleeding whether it has ceased at the time of admission and the time since the onset will be recorded and divided into 3 groups according to geographical distribution Dakshina Kannada , Udupi, Kerala
- The patients will also be interrogated regarding symptoms of nausea,vomiting, dysphagia, regurgitation, heart burn, abdominal pain, appetite, weight gain or loss and recent changes in bowel habits prior to the bleed.
- Past history of ingestion of drugs over the preceding 48 hrs and frequent ingestion over the preceding months will be enquired about and previous histories of cardiovascular, respiratory, liver diseases will be thoroughly evaluated
- Habit of consumption of alcohol by the patient
- A detailed examination including patient's mental status general appearance and condition of skin will be done. Pulse rate, BP, JVP, peripheral edema, signs of cardiac failure will also be noted.
- Examination of the abdomen for any area of tenderness, palpable masses, ascites and rectal examination will be carried out.
- Based on clinical data obtained a provisional diagnosis will be made.
- These patients will be then submitted to oesophagogastroduodenoscopy using a fibreoptic instrument

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4. Johnson JH. Endoscopic risk factors for bleeding peptic ulcer. GastrointestEndosc 1990;36(Suppl 5):S16-20.
5. Lin HJ, Hsieh YH, Tseng GY, Perng CL, Chang FY, Lee SD. A prospective, randomized trial of large- versus small-volume endoscopic injection of epinephrine for peptic ulcer bleeding. GastrointestEndosc 2002;55:615
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ANNEXURE 3

CONSENT FORM

I ……..., have understood the information given in the information sheet.The nature, objective, duration and expected effects of the study have been explained tome in ……, a language in which I am conversant. I have beeninformed what I have to do as part of the study. I have had the time and opportunity to enquireabout the study and I have been fully satisfied with the explanations given.

I am ready to participate voluntarily in this study.

I agree to co-operate with the research staff and voluntarily undergo the procedures required in the study.

I understand that I am at liberty to withdraw from this study at any time without justifying my decision to withdraw.

I know that the results from this study may be forwarded to the appropriate authorities, presented in scientific meetings and published.

By signing this consent form, I have not given up any legal rights which I am otherwise entitled to as subject in this study.

I know that I will get a copy of this consent form which is signed and dated.

……
Signature of Subject

…… Date:
Name of Subject

I confirm that I have explained the nature, purpose and expected effects of the study tothe subject whose name is printed above

……
Signature of person providing information

……
Name of person providing information Date:

ANNEXURE IV
PROFORMA

1.Case No. 2. I.P.No.
3. Patient's Name 4. Age
5. Gender – Female 6. Occupation
7. Address

- **CHIEF COMPLAINT:**
- **HISTORY OF PRESENTING ILLNESS**
- **PAST HISTORY**
- **FAMILY HISTORY:**
- **PERSONAL HISTORY:**
- **GENERAL EXAMINATION** :

State of nutrition & built:
PALLOR / ICTERUS / CLUBBING / CYANOSIS / EDEMA / LYMPHADENOPATHY:

- BP : PR:
- RR : SPO2:
- **EXAMINATION OF ABDOMEN** :
- **EXAMINATION OF RESPIRATORY SYSTEM**
- **EXAMINATION OF CARDIOVASCULAR SYSTEM** :
- **EXAMINATION OF CENTRAL NERVOUS SYSTEM** :
- **INVESTIGATIONS**
- Hb
- LFT
- RFT
- SERUM ELECTROLYTES
- COAGULATION PROFILE
- HIV,HBSAG,HCV

UGISCOPY FINDINGS :

ANNEXURE V – MASTER CHART

Sl.No.	IP NUMBER	NAME	AGE/SEX	GEOGRAPHY	SYMPTON	ULTRASOUND FINDINGS	ENDOSCOPY	DISEASE
1	12055247	ANSARI PM	31/F	DAKSHINA KANNADA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
2	13069362	PUSHPA	39/F	DAKSHINA KANNADA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-MALLORY WEISS TEAR	MALLORYWEISSTEAR
3	13073411	NIRANJAN	52/M	DAKSHINA KANNADA	MELENA	USG-FATTY LIVER	UGISCOPY-DUODENAL ULCER	DUODENAL ULCER
4	14004222	ADHAM SAB	60/M	KERALA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
5	14010829	ABDULLA P.M	60/M	KERALA	MELENA	USG- NORMAL	UGISCOPY- GASTRIC ULCER	GASTRIC ULCER
6	14007500	JANAKI	68/F	UDIPI	HAEMTEMESIS	USG-SIMPLE RENAL CYST	UGISCOPY ANTROL EROSIONS	EROSIONS
7	14015744	RAJU	48/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- NORMAL	UGISCOPY- ANTRAL ULCER	GASTRIC ULCER
8	14020441	SHERIN	33/F	KERALA	HAEMTEMESIS	USG- FATTY LIVER	UGISCOPY- ANTRAL ULCER	GASTRIC AND DUODENAL ULCER
9	14039803	PUTTATHAYAMMA	78/F	UDIPI	HAEMTEMESIS	USG-NORMAL	UGISCOPY-GASTRIC EROSIONS	EROSIONS
10	14004729	ABDUL REHMAN	47/M	KERALA	HAEMTEMESIS	USG-FATTY LIVER	UGISCOPY-DUODENAL ULCER	DUODENAL ULCER
11	14045552	CHANDRASEKHAR	27/M	KERALA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-GASTRIC ULCER	GASTRIC ULCER
12	14049162	SHARATH	25/M	DAKSHINA KANNADA	MELENA	USG-NORMAL	UGISCOPY-FUNDAL ULCER	GASTRIC ULCER
13	14052475	SUMITHRA	45/F	DAKSHINA KANNADA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-MULTIPLE GASTRIC EROSIONS	EROSIONS
14	14052345	GOPALAN	52/M	DAKSHINA KANNADA	HAEMTEMESIS	USG-FATTY LIVER	UGISCOPY-GASTRIC EROSIONS	EROSIONS
15	14044568	NAGARAJ K UDASI	38/M	UDIPI	MELENA	USG-FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION (8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
16	15079028	MAHALAKSHMI	52/F	KERALA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTWNSION(8MM)	UGISCOPY ESOPHAGEAL AND FUNDAL VARICES	VARICES

17	14081795	KAMARUNISSA	20/F	KERALA	HAEMTEMESIS	USG- NORMAL	UGISCOPY- PANGASTRITIS	PANGASTRITIS
18	14085796	VENKAPPA SHETTY	76/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- NORMAL	UGISCOPY - DUODENAL ULCER	DUODENAL ULCER
19	14087542	BHARATHI	42/F	KERALA	HAEMTEMESIS	USG-PYLORIC THICKENING	CA STOMACH	CA STOMACH
20	14093966	RAMESH	38/M	UDIPI	HAEMTEMESIS	USG- NORMAL	UGISCOPY- GASTRIC ULCER	GASTRIC ULCER
21	14052015	BABU	66/M	KERALA	MELENA	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY ESOPHAGEAL VARICES	VARICES
22	14041422	RAVICHANDRA	37/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- MILD FATTY LIVER	UGISCOPY- NORMAL	NORMAL
23	14069145	SHREEDHARAN N	67/M	KERALA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
24	14054660	NOORJAHAN	66/M	KERALA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY ESOPHAGEAL VARICES,ANTRAL GASTRITIS	VARICES
25	14019003	SHRIDHAR N GUDIGAR 1	59/M	DAKSHINA KANNADA	MELENA	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
26	14068200	INHAMMA	55/F	KERALA	HAEMTEMESIS	USG- CRYPTOGENIC LIVER DISEASE	UGISCOPY- FUNDAL VARICES	VARICES
27	14076576	BALAKRISHNAN M P	56/M	UDIPI	HAEMTEMESIS	USG- UMBILICAL HERNIA FEATURES OF CLD	UGISCOPY ESOPHAGEAL VARICES	VARICES
28	14076645	SASIDHARAN	42/M	UDIPI	MELENA	USG- FATTY LIVER	UGISCOPY-DUODENAL ULCER	DUODENAL ULCER
29	13037225	NAGESHA RAO	45/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION (8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
30	14045079	UDAY KUMAR	36/M	UDIPI	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
31	14047843	MARIAMMA	51/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
32	14048739	GILBART	36/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(7MM)	UGISCOPY-FUNDAL VARICES	VARICES
33	14049138	BYIJU	35/F	KERALA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-MALLORY WEISS TEAR	MALLORY WEISS TEAR
34	14049417	GIRIJA	50/F	KERALA	MELENA	USG-NORMAL	UGISCOPY-DUODENAL ULCER	DUODENAL ULCER
35	14049923	HARIDAS T	36/F	UDIPI	MELENA	USG-PYLORIC THICKENING	UGISCOPY-PANGASTRITIS	PANGASTRITIS
36	16030814	NAGAPPA LINGA	44/M	KERALA	HAEMTEMESIS	USG-FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
37	14051296	CHIKKAYYA P	55/M	UDIPI	HAEMTEMESIS	USG-NORMAL	UGISCOPY-GASTRIC ULCER	GASTRIC ULCER
38	14051298	PRASHANTH	47/M	UDIPI	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
39	14051	HAFSATH	58/	DAKSHINA	HAEMTE	USG-NORMAL	UGISCOPY-MALLORY WEISS TEAR	MALLORYWEISS TEAR

	459		M	KANNADA	MESIS			
40	16038170	HARINATHAN	55/M	KERALA	MELENA	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY -ESOPHAGEAL VARICES	VARICES
41	13037225	NAGESHA RAO	46/M	DAKSHINA KANNADA	MELENA	USG- FEAURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY ESOPHAGEAL VARICES	VARICES
42	16052504	MAMOJ	33/M	KERALA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(8MM)	UGISCOPY-FUNDAL VARICES	VARICES
43	14044568	NAGARAJ K UDASI	38/M	UDIPI	HAEMTEMESIS	USG- NORMAL	UGISCOPY NORMAL	NORMAL
44	14053665	VEERAPPA GOWDA	51/M	DAKSHINA KANNADA	HAEMTEMESIS	USG NORMAL	UGISCOPY-PANGASTRITIS	PANGASTRITIS
45	14053880	PAVAN KUMAR	19/M	DAKSHINA KANNADA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-GASTRIC ULCER	GASTRIC ULCER
46	16053799	MOHANAN VV	56/M	KERALA	HAEMTEMESIS	USG-FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(9MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
47	14068615	PADHMANABHA	76/M	KERALA	HAEMTEMESIS	USG-PORTAL VEIN DIAMETER 21 MM,RENAL CORTICAL CYST	UGISCOPY VASCULAR ECTASIA	VASCULAR ECTASIA
48	14059398	NASEEMA	27/F	UDIPI	HAEMTEMESIS	USG GRADE 2 RENAL PARENCHYMAL CHANGES	UGISCOPY-NORMAL	NORMAL
49	14056689	BELTHAMMA	80/F	DAKSHINA KANNADA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-GASTRIC EROSIONS	EROSIONS
50	13008807	BHASKARAN P	61/M	UDIPI	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(9MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
51	12041072	SUDHAKARAN	49/M	KERALA	HAEMTEMESIS	USG-FATTY LIVER	UGISCOPY-ESOPHAGEAL VARICES	VARICES
52	13058970	SAJI MATHEW	48/M	KERALA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(7MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
53	14053880	PAVAN KUMAR	60/M	UDIPI	HAEMTEMESIS	USG- NORMAL	UGISCOPY -NORMAL	NORMAL
54	16076757	SOMANATH	54/M	UDIPI	HAEMETEMSIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY-PANGASTRITIS	PANGASTRITIS
55	14059146	SALMAN SAB	52/M	KERALA	HAEMETEMSIS	USG-NORMAL	UGISCOPY-ESOPHAGEAL VARICES	ESOPHAGEAL VARICES
56	13069529	RAGURAMA ALVA	52/M	DAKSHINA KANNADA	HAEMETEMSIS	USG-NORMAL	UGISCOPY-ESOPHAGEAL VARICES	VARICES
57	14055087	VIJAYAN	56/M	DAKSHINA KANNADA	MELENA	USG-NORMAL	UGISCOPY-GASTRIC ULCER	GASTRIC ULCER
58	14060370	ROSHAN D SOUZA	36/M	KERALA	HAEMETEMSIS	USG- FEATURES OF CIRRHOSIS WITHPORTAL HYPERTENSION(8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
59	14035718	KRISHNA NAIR	64/M	KERALA	HAEMETEMSIS	USG FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
60	14011428	CANNY D SOUZA	64/F	KERALA	HAEMETEMSIS	USG-NORMAL	UGISCOPY-GASTRIC EROSIONS	EROSIONS
61	13026817	NANDHESH	36/M	DAKSHINA KANNADA	MELENA	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(8MM)	UGISCOPY-DUODENAL ULCER	DUODENAL ULCER

62	14053665	VEERAPPA GOWDA	51/M	DAKSHINA KANNADA	HAEMETEMSIS	USG-NORMAL	UGISCOPY-MALLORY WEISS TEAR	MALLORYWEISS TEAR
63	14062084	SUDHAKARAN B	50/M	UDIPI	HAEMETEMSIS	USG-NORMAL	UGISCOPY-GASTRIC EROSIONS	EROSIONS
64	16066721	RAJAN K	52/M	DAKSHINA KANNADA	HAEMETEMSIS	USG-NORMAL	UGISCOPY-PANGASTRITIS	PANGASTRITIS
65	13048120	SRINIVAS GATTY	64/M	DAKSHINA KANNADA	MELENA	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(9MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
66	14030814	NAGAPPA LINGA PATAKAR	44/M	DAKSHINA KANNADA	HAEMETEMSIS	USG-FATTY LIVER	UGISCOPY-GASTRIC EROSIONS	EROSIONS
67	14051298	PRASHANTH	47/M	DAKSHINA KANNADA	HAEMETEMSIS	USG-NORMAL	UGISCOPY-GASTRIC EROSIONS	EROSIONS
68	14066436	THOMAS MONTHERO	62/M	KERALA	MELENA	USG -FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
69	12051909	MURALIDHARAN	45/M	KERALA	HAEMTEMESIS	USG-FATTY LIVER	UGISCOPY-GASTRIC EROSIONS	EROSIONS
70	14070978	JOY	57/M	UDIPI	HAEMTEMESIS	USG-NORMAL	UGISCOPY-GASTRIC ULCER	GASTRIC ULCER
71	14006647	UTTAM KUMAR	34/M	UDIPI	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(7MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
72	14035718	KRISHNA NAIR	64/M	KERALA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-ESOPHAGEAL VARICES	VARICES
73	14074233	PRASAD	35/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(9MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
74	14074515	SHANIYAR NARAYAN NAIK	69/M	DAKSHINA KANNADA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-GASTRIC EROSIONS	EROSIONS
75	13026468	VIJAYAN K	53/M	DAKSHINA KANNADA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-ESOPHAGEAL VARICES	VARICES
76	16075161	RAVINDRAN	60/M	UDIPI	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY ESOPHAGEAL VARICES	VARICES
77	14076333	BHASKAR	55/M	UDIPI	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(10MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
78	14055087	VIJAYAN	56/M	KERALA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-GASTRIC EROSIONS	EROSIONS
79	15002185	SURESH V BONGLE	68/M	DAKSHINA KANNADA	HAEMTEMESIS	USG-FATTY LIVER GRADE 2 RENAL PARENCHYMAL CHANGES	UGISCOPY-NORMAL	NORMAL
80	15068166	SHAKUNTALA	43/F	KERALA	HAEMTEMESIS	USG-MILD ASCITES	UGISCOPY-GASTRIC ULCER	GASTRIC ULCER
81	15066159	KUTTI	60/F	KERALA	MELENA	USG-NORMAL	UGISCOPY-DUODENAL ULCER	DUODENAL ULCER
82	15006014	DATTATREYA G BHAT	53/M	KERALA	MELENA	USG-NORMAL	UGISCOPY-DUODENAL ULCER	DUODENAL ULCER
83	15085423	HASANAMBBA	56/F	DAKSHINA KANNADA	MELENA	USG-FATTY LIVER	UGISCOPY-GASTRIC VARICES	VARICES
84	14029	UMESH KULAL	51/	DAKSHINA	MELENA	USG-NORMAL	UGISCOPY-DUODENAL ULCER	DUODENAL ULCER

	442		M	KANNADA				
85	14038085	GANESH NAIK	64/M	KERALA	MELENA	USG-NORMAL	UGISCOPY-DUODENAL ULCER	VARICES
86	14040775	RAMANNA NAIK	56/M	KERALA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(8MM)	UGISCOPY-FUNDAL VARICES	VARICES
87	14072686	RAVI	38/M	KERALA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTWENSION(8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
88	16085805	VENUGOPALA	50/M	UDIPI	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(9MM)	UGISCOPY- ESOPHAGEAL VARICES	VARICES
89	14082636	SHAFI	38/F	DAKSHINA KANNADA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTALHYPERTENSION(9MM)	UGISCOPY- ESOPHAGEAL VARICES	VARICES
90	14085102	UMESH SHETTY	50/M	KERALA	HAEMTEMESIS	USG-PYLORIC THICKENING	UGISCOPY-?CA STOMACH WITH GOO	CA STOMACH
91	16066139	KUTTI	58/F	UDIPI	MELENA	USG-NORMAL	UGISCOPY-DUODENAL ULCER	DUODENAL ULCER
92	14107089	KUNHIKANNAN	59/M	KERALA	HAEMTEMESIS	USG-NORMAL	UGISCOPY-GASTRIC ULCER	GASTRIC ULCER
93	15055134	GANGADHAR	80/M	DAKSHINA KANNADA	HAEMTEMESIS	USG-FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(8MM)	UGISCOPY-	VARICES
94	15020865	MAHESH KUMAR	38/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
95	16081536	DINESH KUMAR	43/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY-ESOPHAGEAL VARICES	VARICES
96	15073538	PADMANABHAN	31/M	KERALA	MELENA	USG- FEATURES OF CIRRHOSIS WITH PORTAL HYPERTENSION(9MM)	UGISCOPY-GASTRIC VARICES	VARICES
97	15078572	KESHAV	54/M	DAKSHINA KANNADA	HAEMTEMESIS	USG- FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY-	VARICES
98	14001551	ASHOK LAKSHMAN NAIK	68/M	UDIPI	MELENA	USG-NORMAL	UGISCOPY-GASTRIC EROSIONS	EROSIONS
99	16045769	KRISHNAN	64/M	KERALA	HAEMTEMESIS	USG- NORMAL	UGISCOPY-PANGASTRITIS	PANGASTRITIS
100	16008471	UNNIKRISHNAN	43/M	KERALA	MELENA	USG-FEATURES OF CIRRHOSISWITH PORTAL HYPERTENSION(8MM)	UGISCOPY- ESOPHAGEAL VARICES	VARICES

ANNEXURE VI – PLAGIARISM CERTIFICATE

K. S. Hegde Medical Academy
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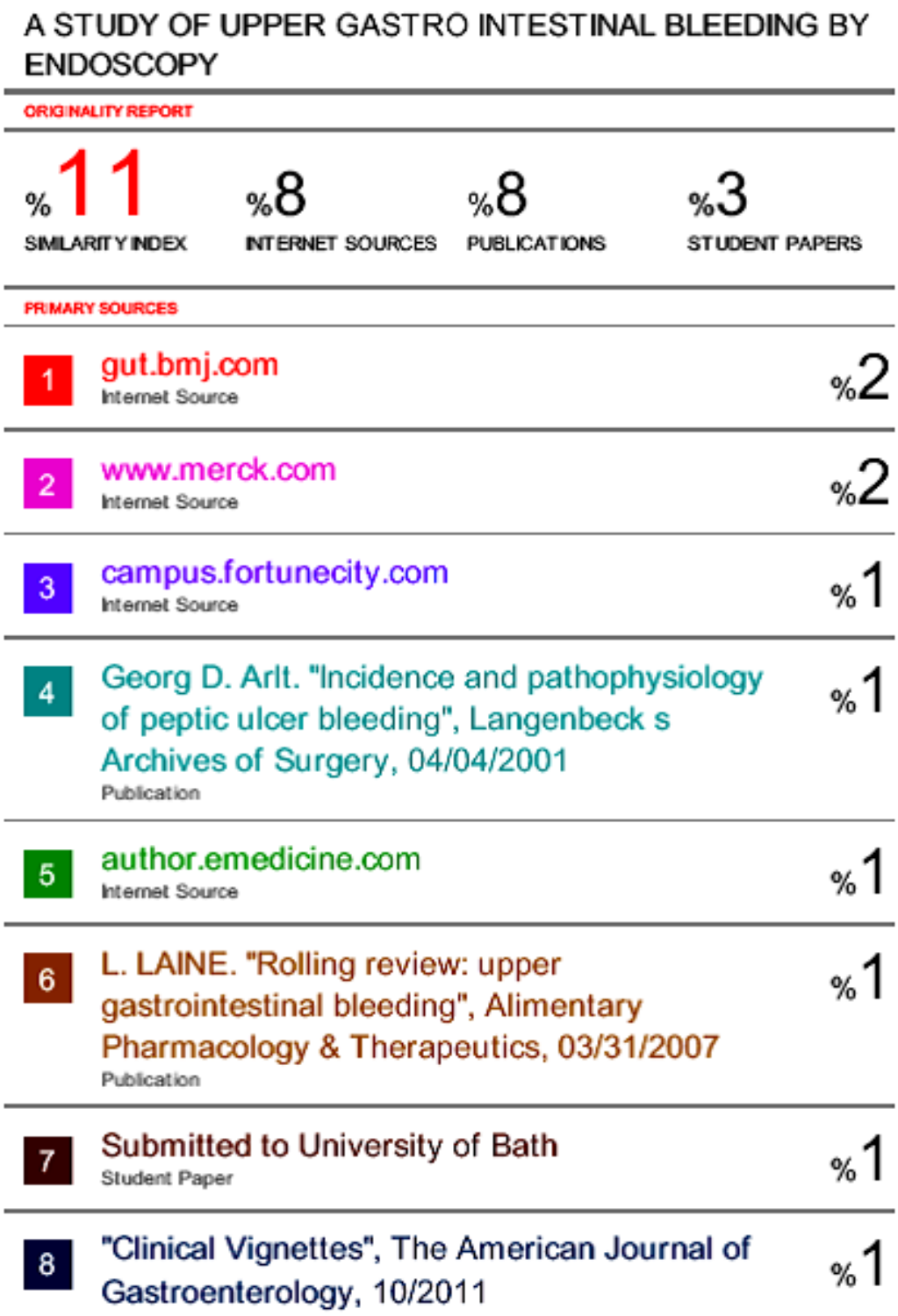

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