A COMPARATIVE STUDY OF EFFICACY AND SAFETY OF ENDOSCOPIC BANDING AND SCLEROTHERAPY IN ESOPHAGEAL VARICES

Dissertation submitted in partial fulfillment of regulation for the award of M.S. Degree in General Surgery (Branch I)

THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI

APRIL, 2013.

CERTIFICATE

This is to certify that this dissertation titled ***"A COMPARITIVE STUDY OF SAFETY AND EFFICACY OF ENDOSCOPIC BANDING AND ENDOSCOPIC SCLEROTHERAPY FOR ESOPHAGEAL VARICES "*** submitted to the Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfilment of the requirement for the award of M.S Degree Branch - I (General Surgery) is a bonafide work done by

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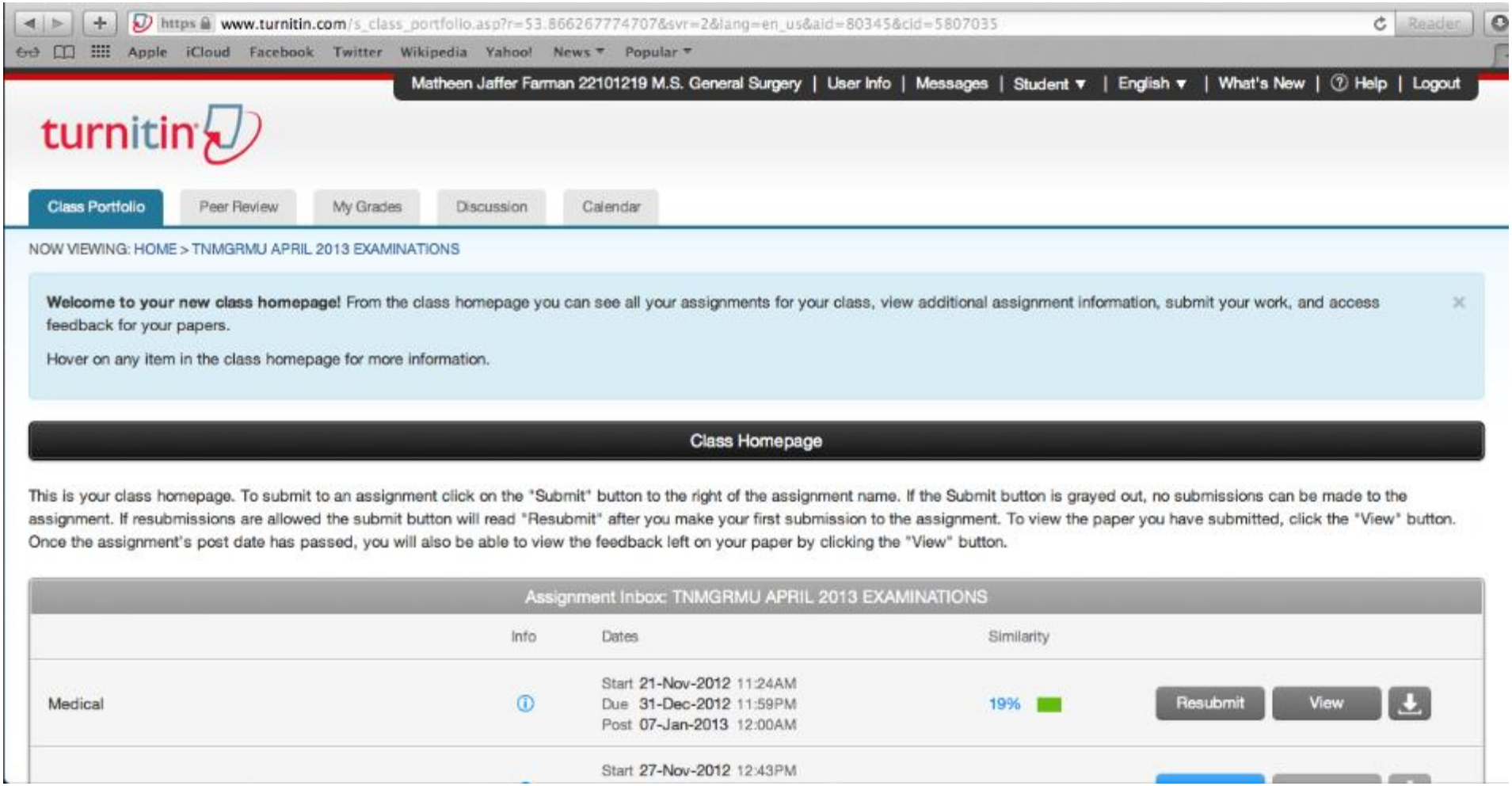
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INTRODUCTION
During the past years the prevalence of cirrhosis and hence portal hypertension has increased. Complications of portal hypertension rank among the leading cause of death in cirrhosis patients. Among these complications, esophageal variceal bleeding remains the most serious outcome. 50-60% of patients with portal hypertension develop cirrhosis and around 30% bleed from esophageal varices. Mortality rate in case of variceal bleeding is
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I solemnly declare that the dissertation titled "A COMPARATIVE STUDY OF EFFICACY AND SAFETY OF ENDOSCOPIC BANDING AND SCLEROTHERAPY IN ESOPHAGEAL VARICES" at Coimbatore Medical College Hospital was done by me from September 2011 to September 2012 under the guidance and supervision of Professor **DR.D.N.RENGANATHAN,M.S.** This dissertation is submitted to The **Tamilnadu Dr. M.G.R. Medical University** towards the partial fulfillment of the requirement for the award of M.S Degree in General Surgery (Branch I).

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ABBREVIATIONS

DSRS	Distal Splenorenal Shunt
EBL	Endoscopic band ligation
EVL	Endoscopic variceal ligation
GIT	Gastro intestinal tract
GEJ	Gastro esophageal junction
HVPG	Hepatic venous pressure gradient
PTFE	Polytetrafluroethylene
STD	Sodium tetradecyl sulphate
TIPS	Transjugular intrahepatic portosystemic shunt

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INTRODUCTION

During the past years the prevalence of cirrhosis and hence portal hypertension has increased. Complications of portal hypertension rank among the leading cause of death in cirrhosis patients. Among these complications, esophageal variceal bleeding remains the most serious outcome. 50-60% of patients with portal hypertension develop cirrhosis and around 30% bleed from esophageal varices. Mortality rate in case of variceal bleeding is around 20-30% and can exceed 50% in some countries. Patients with variceal bleeding for the first episode who had no treatment for it have a 60% risk of rebleeding[1].

Endoscopic treatment modalities widely used are endoscopic sclerotherapy and band ligation for the treatment of acute variceal bleeding and for secondary prophylaxis[2]. Both treatment modalities have their own advantages and disadvantages. Due to the fact that banding therapy has lower rates of complications such as rebleeding, mortality and esophageal stenosis, this method has been considered as the treatment of choice for esophageal varices by some authors[3]. However, a meta-analysis study by Triantos *et al*., have shown that sclerotherapy was superior in efficacy to banding ligation[4]. Also some studies have shown both the methods are equally effective[5].

The different opinions shown in various studies regarding the treatment of choice for esophageal varices, motivated us to do the current study. Thereby in this study we compared the results of endoscopic banding and endoscopic sclerotherapy with 3% sodium tetradecyl suphate in the management of esophageal varices.

2. Aims & objectives

AIM AND OBJECTIVES

To compare the efficacy of endoscopic variceal ligation and endoscopic sclerotherapy in the management of variceal bleeding due to portal hypertension.

3. Review of literature

REVIEW OF LITERATURE

ESOPHAGEAL VARICES:

Esophageal varices are defined as dilated submucosal veins in the lower one third of esophagus. They are due to portal hypertension most often due to cirrhosis of liver. Variceal bleeding is one of the most dreaded complications of portal hypertension and it occurs in nearly 30% of patients with chronic liver disease[6].

Hemorrhage from esophageal varices can be treated by endoscopic, medical and surgical therapy. But therapy by endoscopic methods is the optimal first line treatment for acutely bleeding varices as well as subsequent long term management to prevent rebleeding.

GRADING:

- I – visible veins but not elevated
- II – large and raised veins but not touching each other
- III – raised and tortuous veins almost touching each other
- IV – very large veins filling the entire lumen

ANATOMY OF ESOPHAGUS:

It is a muscular hollow organ of about 25 cm in length extending from pharynx at lower border of cricoid cartilage at the level of C6 vertebra to the stomach at T11 thoracic vertebra. It descends along the vertebral column and mediastinum, piercing the diaphragm ending at the cardiac orifice of the stomach. Esophagus

also presents flexures corresponding to the curvatures in the cervical and thoracic vertebral column which is the narrowest part of the esophagus.

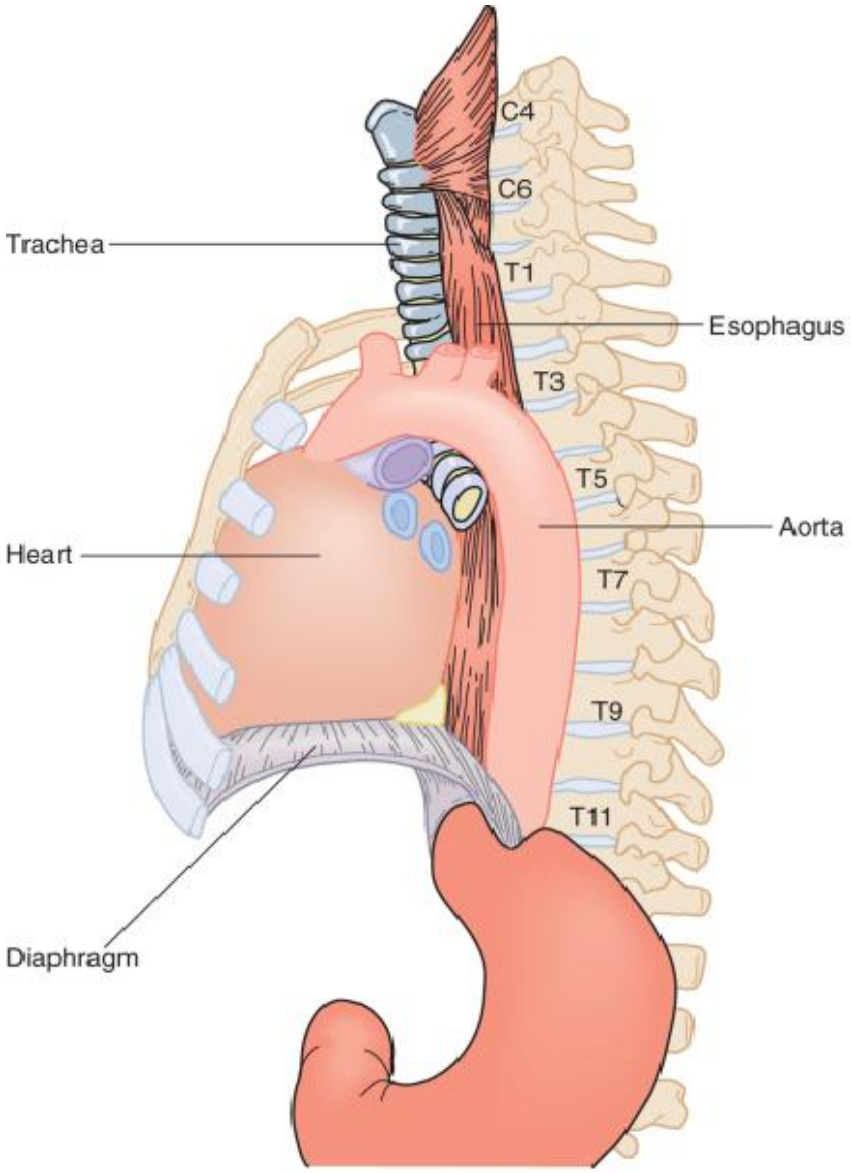


Figure 1: Anatomy of esophagus

RELATIONS:

CERVICAL:

Anterior:

- Trachea
- Thyroid gland

Posterior:

- Vertebral column
- Longus colli muscle

On either side:

- Common carotid artery
- Part of lobes of Thyroid gland
- Recurrent laryngeal nerve

On Left:

- Thoracic duct

THORACIC:

Anterior:

- Trachea
- Aortic arch
- Left bronchus
- Pericardium

Posterior:

- Vertebral column
- Longus colli muscle
- Right Posterior intercostal arteries
- Hemiazygos vein
- Thoracic duct

Left:

- Left Subclavian artery
- Thoracic duct
- Left Pleura
- Left Recurrent laryngeal nerve
- Descending thoracic aorta

Right:

- Right Pleura
- Azygos vein
- Vagus nerve

ABDOMINAL PORTION:

It is about 1.25 cm and situated in the posterior surface of lef lobe of liver in the esophageal groove.

HISTOLOGY:

It consists of four layers

- Mucosa
- Submucosa
- Muscular layer
- Outer fibrous layer
- It lacks serosa

MUCOSA:

- lined with squamous columnar epithelium and has 4 layers

- Epithelium
- Basement membrane
- Lamina propria
- Muscularis mucosa

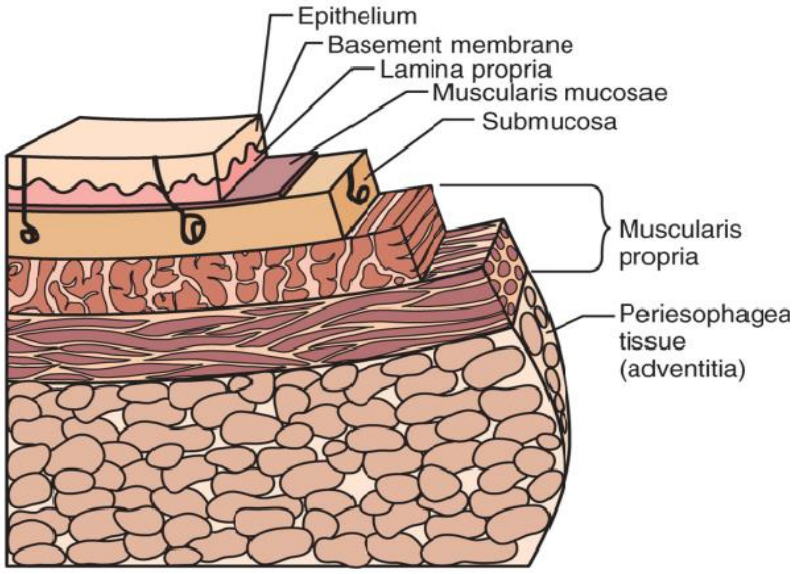


Figure 2: Layers of esophagus

Z LINE:

It is the transition of esophageal mucosa to columnar epithelium in the distal 2 cm of esophagus.

SUBMUCOSA:

It is the red carpet for malignancies since abundant network of vascular and lymphatic structures with Meissner's neural plexus. This is the layer in which dilatation of veins in esophageal varices occurs.

MUSCULARIS PROPRIA:

It consists of 2 layers

- Outer Longitudinal
- Inner Circular

Between these muscle layers, a thin septum consists of network of ganglia called Auerbach's plexus.

GASTRO ESOPHAGEAL JUNCTION:

There are four anatomic points to identify GEJ

- 2 Endoscopic
- 2 External

Endoscopically

- Z line provided patient does not have Barret's esophagus

- Transition from smooth esophageal lining to rugae of stomach

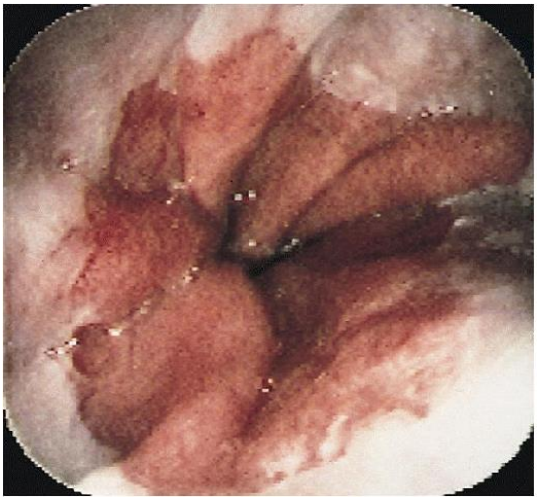


Figure 3:Gastro esophageal junction on endoscopy

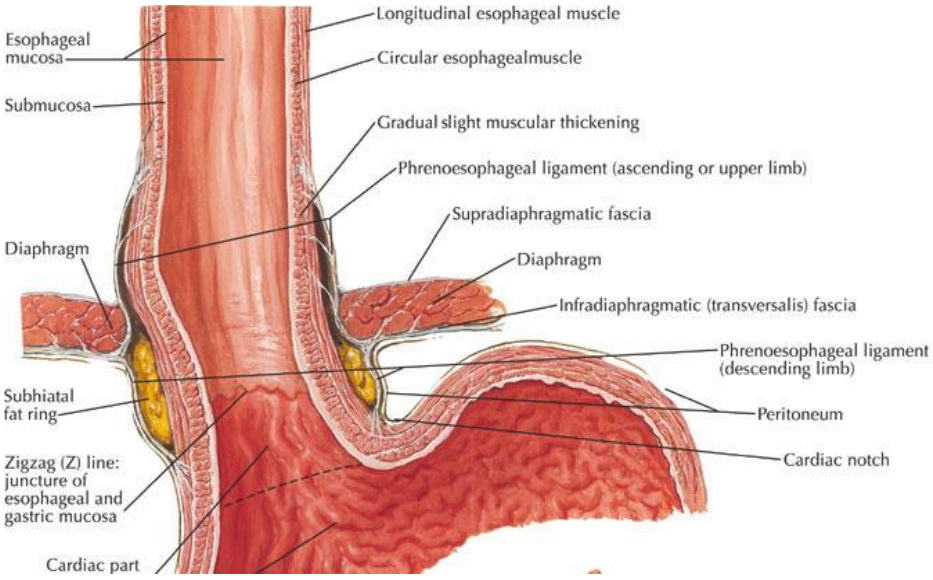


Figure 4: Gastro esophageal junction

Externally

- Collar of Helvetius (Willis Loop)

It is the region where the circular muscle fibre of the esophagus joins the oblique fibre of the stomach

- Gastro esophageal fat pad

BLOOD SUPPLY:

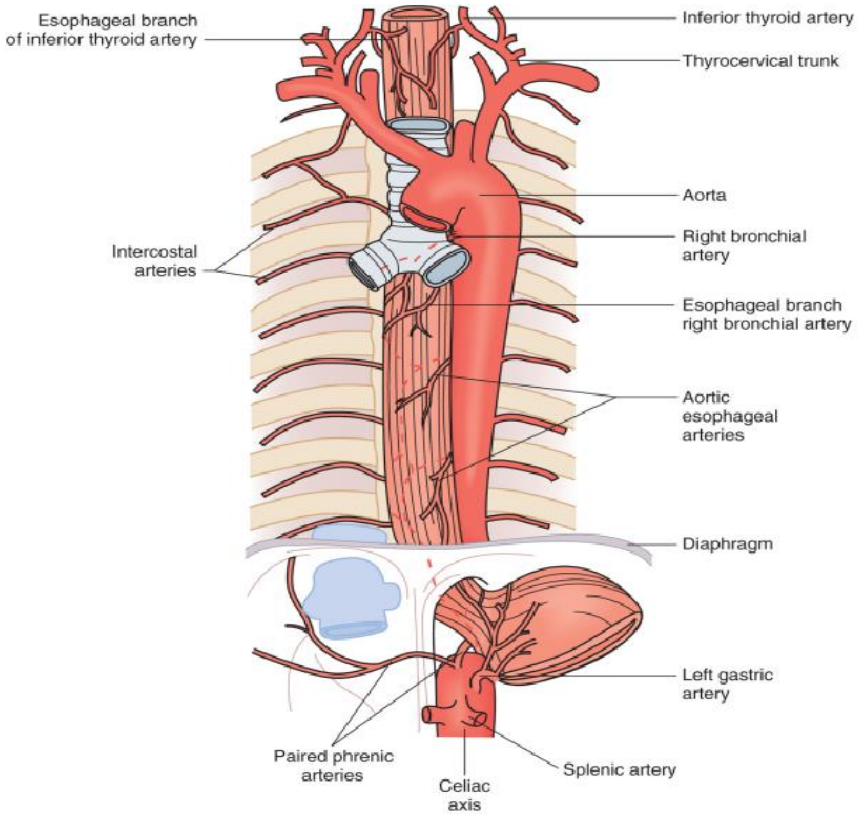


Figure 5: Arterial supply of esophagus

Arterial supply:

- Cervical – Inferior thyroid artery
- Thoracic – 4-6 Esophageal artery from Aorta

  Esophageal branch from Right and Left

  Bronchial arteries
- Abdominal – Left Gastric artery

  Inferior Phrenic artery

The arteries end in a fine capillary network before penetrating the muscular wall. After penetrating the muscularis propria, capillary network continues throughout the length of the esophagus within the submucosal layer.

Venous drainage:

The venous drainage corresponds to the arteries. Rich submucous venous plexus is the first bin for venous drainage. In Cervical esophagus, submucous venous plexus drain Inferior Thyroid vein which in turn into Left Subclavian and Right brachiocephalic vein

In Thoracic esophagus, the submucous venous plexus joins with more superficial esophageal venous plexus and venae comitantes and envelop the esophagus. These plexus in turn drain into Hemiazygos and Azygos veins.

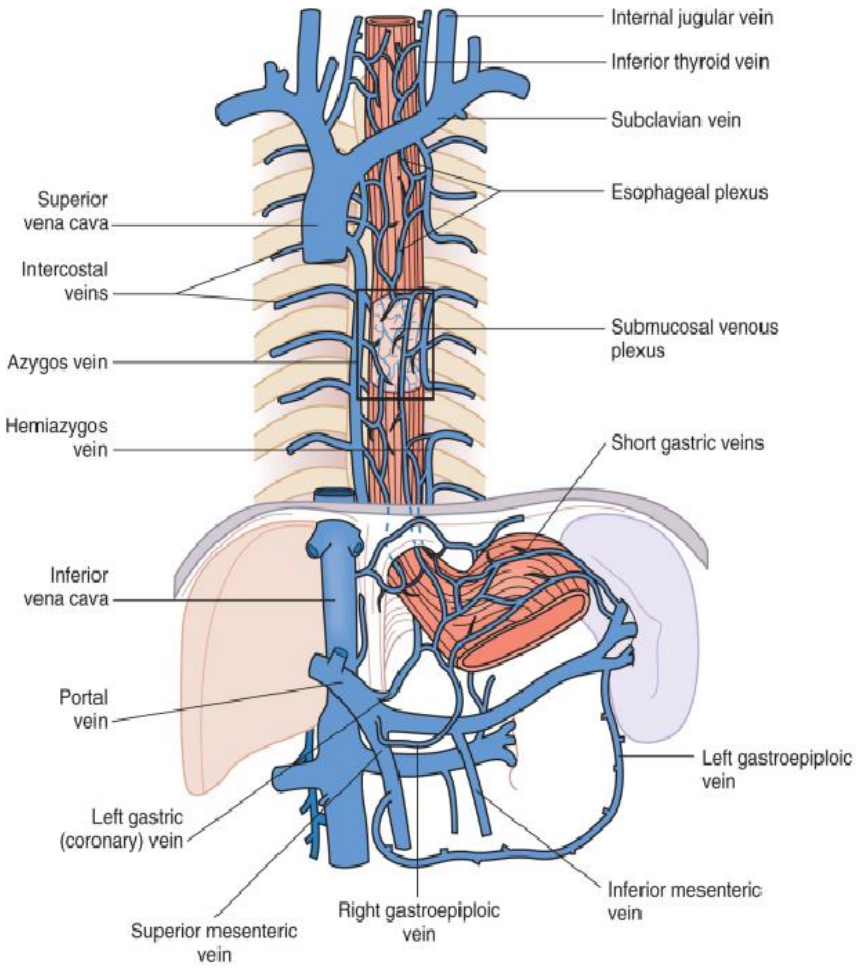


Figure 6: Venous drainage of esophagus

Abdominal esophageal veins drain into both systemic and portal venous system through the right phrenic vein and left gastric vein (coronary and short gastric vein respectively).

LYMPHATICS:

It consists of two interconnecting lymphatic plexus arising from the submucous and muscularis layer. In the upper two-third of esophagus, the lymphatics flow upwards and in lower one-third of esophagus, lymphatics flow downwards.

NERVE SUPPLY:

Esophagus is innervated by both vagus and sympathetic trunk. They both form a plexus which contains a group of ganglions between the muscular layers and also in the submucous layer.

EMBRYOLOGY:

The development of esophagus begins in the third week of gestation and fetus takes its first swallow by fourteenth week.

- Formation of gut
- Molecular regulation of the gut
- Differentiation of the endoderm.

FORMATION OF GUT:

Cephalocaudal and lateral folding of embryo occurs. Thus the endoderm lined yolk sac cavity is incorporated into embryo forming primitive gut.

It is a blind ending tube consisting of foregut, midgut and hind gut. Foregut gives rise to esophagus extending from the pharyngeal tube up to the liver outgrowth.

MOLECULAR REGULATION:

Differentiation depends on the reciprocal interaction between the endoderm of the gut and the surrounding splanchnic mesoderm through HOX code.

DIFFERENTIATION OF ENDODERM:

Epithelium becomes two to five cells thick and remains stratified columnar epithelium till sixth to eighth week of gestation. By tenth week the stratified columnar becomes ciliated and by tweflth week epithelium becomes completely ciliated. Again during fourth and fifth month of gestation stratified squamous epithelium replaces ciliated columnar epithelium.

MUSCULAR DEVELOPMENT:

The other part of esophagus is formed from mesoderm. By sixth week it is surrounded by undifferentiated mesoderm and a circular layer of myoblast. Longitudinal and circular muscle fibre appears.

Smooth muscle that forms the lower two-third of esophagus arises from splanchnic plexus and it is supplied by splanchnic plexus. By twelfth and fifteenth week, the striated muscle that forms the upper esophagus arises from branchial arches. Muscular proliferation is at its high limits during the eleventh and twelfth week. Hence the longitudinal muscles are well defined.

PORTAL HYERTENSION:

It is defined as the increase in the hepatic venous pressure gradient > 5mm Hg.

It may be due to

- Increased resistance to blood flow through liver

- Increased splanchnic blood circulation due to vasodilatation[7]

Symptoms of portal hypertension occurs once the pressure rises above 10mm Hg

CAUSES OF PORTAL HYPERTENSION:

PRE HEPATIC[8]:

- Portal vein thrombosis
  - Congenital
  - Sepsis
  - Trauma
- Splenic vein thrombosis
- Massive splenomegaly (Banti's syndrome)
- Malignant occlusion

HEPATIC:

- PRE SINUSOIDAL
  - Schistosomiasis
  - Congenital hepatic fibrosis
- SINUSOIDAL
  - Cirrhosis
  - Alcoholic hepatitis

- POST SINUSOIDAL
    - Hepatic sinusoidal obstruction (veno-occlusive syndrome)

POST HEPATIC:

- Budd Chiari syndrome
- Venoocclusive disease
- Inferior Vena caval webs
- Cardiac causes
    - Restrictive cardiomyopathy
    - Severe congestive heart failure
    - Constrictive pericarditis

Portal vein drains deoxygenated blood from stomach, intestine, spleen, pancreas, and gall bladder. It is formed by superior mesentric vein and splenic vein. Superior mesentric vein drains entire small bowel, ascending colon and a part of descending colon and head of pancreas. Inferior mesentric vein joins splenic vein and hence draining the transverse colon and part of descending colon and upper two thirds of rectum. Thus portal vein receives blood supply from entire gastrointestinal tract.

Patients with a pressure gradient of >12mm Hg are at high risk for variceal bleeding.

The major complications of portal hypertension are

- massive upper GI bleeding due to ruptyred esophageal varices and portal hypertension gastropathy
- Ascites
- Hepatorenal syndrome
- Hepatic encephalopathy.

CLINICAL COURSE OF VARICEAL BLEEDING:

Portal hypertension causes porto- systemic collaterals development, among which esophageal and gastric varices are troublesome, because their rupture causes variceal hemorrhage, which is most lethal complications of cirrhosis.

90% of cirrhotic patients develop esophageal varices in their lifetime and 30% of these will bleed[9]. When cirrhosis is diagnosed, about 30%-40% of compensated patients have varices and 60% of decompensated have varices. Once cirrhosis is diagnosed, the expected incidence of new varices is 5% per year[10].

In course, varices increase in size before they eventually rupture and bleed. Rates of progression of varices range from 5% to 30% per year[10]. This variability is due to inter-observer variability, selection of patients, Decompensated cirrhosis (Child B/C), alcoholic etiology, and the presence of red spots in the esophageal varices.

Once varices have been diagnosed, the annual incidence of variceal bleeding is 10%-15% in non-selected patients. The most important factors are variceal size, severity of liver dysfunction and red wale markings[11]. The North Italian Endoscopy Club (NIEC) index allows the classifications of patients into different groups with a predicted 1-year bleeding risk. According to the NIEC index, patients with small varices and advanced liver disease are at higher risk of early bleeding. The probability of bleeding within 1 year in Child-Pugh class A patients with large varices and red signs is 24%. But in Child-Pugh C patients with small varices and no red signs, the incidence of bleeding within 1 year is 20%[12].

Variceal size is the most useful predictor for variceal bleeding. The risk of bleeding is very low (1-2%) in patients without varices, and increases to 5% per year patients with small varices, and increases to 15% per year in patients with medium or large varices at diagnosis. Red sign is another predictor of variceal bleeding[13]. Variceal size and red signs denote the variceal wall tension (radius, wall thickness), which is the factor determining variceal rupture. Studies have shown that variceal bleeding occurs if the HVPG reaches a threshold value of 12 mmHg. If the HVPG is reduced (below 12 mmHg or by > 20% of the baseline levels), there is a marked reduction in the risk of bleeding, development of ascites, spontaneous bacterial peritonitis and death[12].

Variceal bleeding is the second most common cause of mortality among the cirrhotic patients. In cirrhotic patients, variceal bleeding causes 70% of all upper digestive bleeding[14]. Mortality from variceal bleeding has greatly decreased from 42% to 6-12% over the last two decades according to the Graham and Smith study in 1981[7]. This is due to implementation of effective endoscopic and pharmacological therapies, transjugular intrahepatic portosystemic shunt (TIPS) and improved general medical care. Death occurring within 6 wk from hospital admission for variceal bleeding should be considered as a bleeding-related death[15].

Immediate mortality from uncontrolled bleeding is about 4% to 8%. Prehospital mortality from variceal bleeding is around 3%[13]. Other causes for mortality are due to infection, kidney failure, hepatic encephalopathy, poor liver function, severe portal hypertension with HVPG > 20 mmHg, and active bleeding at endoscopy[14].

MANAGEMENT OF VARICES:

- **Pharmacological**
- **Decompressive shunts**
- **Devascularisation procedure**
- **Endoscopic therapy**
- **Liver transplantation**

PHARMACOLOGICAL THERAPY:

- Non cardioselective beta blockers
- Somatostatin analogue
- Vasopressin, terlipressin

NON CARDIOSELETIVE BETA BLOCKERS:

- Propanolol

- Nadolol

These drugs were introduced by Lebrec and his colleagues in early 1980s to reduce portal hypertension which is the mainstay of prophylactic therapy.

Advantages:

It plays a major role in preventing the initial bleed, managing acute variceal bleed and also a first line in preventing the rebleeding.

Disadvantages:

It has limited use in patients with heart disease, kidney disease, asthma and other lung disease, diabetes and other drug allergies.

SOMATOSTATIN ANALOGUE (OCTREOTIDE):

These are synthetic analogues of somatostatin which act by inhibiting the release of vasodilatory hormones and also causing splanchnic vasoconstriction which in turn lowers the portal blood flow thereby decreasing bleeding and preventing rebleeding.

VASOPRESSIN AND TERLIPRESSIN:

These drugs are used in acute variceal bleeding by reducing the portal pressure. Vasopressin has significant side effects with systemic vasoconstriction. So it is largely replaced by terlipressin.

DECOMPRESSIVE SHUNTS:

Decompression is mostly used as a second line and is reserved only who rebleed after endoscopic therapy and beta blockers. Surgical shunts are of 3 categories

- Total shunt
- Partial shunt
- Selective shunt

TOTAL SHUNT:

- Classic end to side porto caval shunt
- Side to side porto caval shunt

Shunt size need to be at least 10mm in diameter. Both are effective in controlling varices but especially end to side shunt is more effective than side to side shunt while only the latter is effective in controlling ascites[16].

DISADVANTAGE:

These shunts are associated with increased incidence of Hepatic encephalopathy.

PARTIAL SHUNT:

Shunt size is about 8mm. Polytetrafluroethylene (PTFE) interposition the grafts between portal vein and inferior venacava found to be greater than 90% control of varices and maintain portal perfusion.

SELECTIVE SHUNTS:

Distal Splenorenal Shunt (DSRS):

It is the anastamoses between splenic vein with the left renal vein after its dysjunction with the superior mesentric vein[17]. Control of bleeding and portal perfusion is maintained in more than 95% of indviduals. Incidence of hepatic encephalopathy after the shunt is around 15%.

Transjugular intrahepatic porto systemic shunt (TIPS):

TIPS was described by Rosch in 1969 but only in 1982 it was first used in humans by Dr.Ronald Colapinto. It became successful only with development of endovascular stents in 1985. From 1988 the procedure has widely being accepted and preferred method for treating portal hypertension refractory to medical treatment. Hence TIPS widely replaced the surgical portocaval shunt. TIPS is the puncture of internal jugular vein, passage of catheter into one of the major hepatic vein(usually right) through the right atrium followed by transparenchymal liver puncture to cannulate the portal vein.

Intraparenchymal tract is dilated and is stented with an expandable metal stent. Pressures are measured before and after keeping the stent and the goal is to attain a pressure difference of less than 10mm Hg between portal vein and right atrium. The success rate is high with less morbidity.

Disadvantage of TIPS is its thrombosis and restenosis which necessitates frequent repeat procedures and monitoring. The early thrombosis is related to bile duct puncture since the bile is extremely thrombogenic, occlusion occurs within the first 24 hrs.

DEVASCULARISATION PROCEDURES:

These operative procedures take care of the variceal bleeding by interrupting the inflow to the varices. The effiacy depends on the aggressiveness of the procedure. This is popularized in Japan by Sugiura, and in Egypt by Hassab and in Mexico by Orozco. The components of this procedure include splenectomy, gastric and esophageal devascularisation and occasionally esophageal transection. The advantage is that they do not reduce portal hypertension and hence maintaining portal perfusion of the cirrhotic liver. The disadvantage is relentless recollateralization of varices across the esophagus and stomach with risk of rebleeding.

LIVER TRANSPLANTATION:

Variceal bleeding per se is not an indication for liver transplantation while the associated ascites and encephalopathy are indicators of end stage liver disease and for liver transplantation. The timing of transplant is by the severity of the underlying liver disease.

ENDOSCOPIC THERAPY:

- SCLEROTHERAPY
- BANDING
- TISSUE ADHESIVES
- ENDOLOOPS
- BALLOON TAMPONADE
- CYANOACRYLATE GLUE INJECTION

ENDOSCOPIC SCLEROTHERPY:

HISTORICAL ASPECTS:

Endoscopic sclerotherapy for esophageal varices was first reported by two Swedish surgeons Crafoord and Freckner in 1939 in 19yr old female using Quinine as sclerosant every alternate day for one month till the varices obliterated. Then in 1940, a thoracic surgeon Moensch at Mayo clinic reported the second case of sclerotherapy. However endoscopic sclerotherapy largely ignored until 1970s, when Johnston and Rodgers of Ireland reported their fifteen years with

sclerotherapy[6]. Further studies in late 1970s and 1980s established the efficacy of injection sclerotherapy. In the above studies they have been compared with other treatment modalities of esophageal varices.

SCLEROSING AGENTS:

Sclerosing agents were actually used in 1920s for varicose veins of lower limb. The choice of sclerosants depends on the number of considerations including the efficacy of the agent, injection technique, safety profile, availability and cost.

The sclerosing agents available are

SYNTHETIC PRODUCTS

- Sodium tetradecyl sulphate
- Polidocanol

FATTY ACID DERIVATIVES

- Sodium morrhuate
- Ethonalamine oleate

OTHER AGENTS

- 3% phenol in water
- 5% phenol in oil
- Absolute alcohol

FACTORS INFLUENCING SCLEROTHERAPY:

A number of factors influence the effect of sclerotherapy on esophageal varices which includes choice of endoscope, injection sites, timing of the injection, amount and type of the sclerosant used and the clinical condition of the patient.

MECHANISM OF ACTION:

The mechanisms of action of these agents are poorly defined but the effects involve more than simple initiation of the clotting process of intimal injury. Autopsy studies showed that the thrombosis of the submucosal vessels occur within the first 24 hours along with tissue necrosis even in the absence of extravasation of these agents, while superficial or deep ulceration occurs after seven days. Submucosal fibrosis was seen after one month of sclerotherapy.

These extravasation effects may be responsible for the long time success of sclerotherapy with the development of fibrosis preventing the formation of new variceal channels in the adjacent mucosa. Hence, procedures which are directed only at the varices often fail because of subsequent ligation of collaterals. Sclerotherapy achieves hemostasis through a tamponade effect and also by induction of local thrombosis followed by sclerosis due to sclerosant.

SODIUM MORRHUATE:

Sodium morrhuate, a sodium salt of the fatty acid in cod liver oil was first described in 1933. It is available as 5% solution. It is being less irritating to the adjacent tissues than phenol and quinine mixtures which were in use at that time. Studies shows rebleeding rate was 17%, ulcerations were seen in 23%, fever in 28% and pleural effusion in 7% and esophagopleural fistula in 4%. Although sodium morrhuate appears to be an effective sclerosing agent the incidence of deep post sclerosis ulceration and other serious complications is clearly a restricting factor in its use.

ETHANOLAMINE OLEATE:

Ethanolamine oleate is derived from oleic acid and is similar in physical properties to sodium morrhuate. It is also available in 5% solution. Johnston and Rodgers used 5% ethanolamine oleate in their experience of 15 years reported rebleeding rate of 7% and a mortality of 18%.

The most common complications were pyrexia which was seen in 39% and retrosternal discomfort which was seen in 30% of patients. Even though ethanolamine oleate enjoys a good reputation as a sclerosing agent, the data available at present would not appear to give this drug a clear advantage in either safety or efficacy over other agents.

ALCOHOL:

The advantage of alcohol is its easy availability and economy. The success rate in controlling the variceal bleed was 92% with a rebleed rate of 32%. There is a higher complication rate with an intravariceal injection of absolute alcohol most commonly ulcerations. Though alcohol may appear to be an effective sclerosing agent, the higher incidence of severe retrosternal pain, dysphagia, ulcers and stricture is clearly a restricting factor in its use.

PHENOL:

Supe in 1994 used 3% aqueous phenol for sclerotherapy of esophageal variceal bleeding. Preobliteration variceal bleeding appeared in 15% of the patients. Complications such as esophageal ulceration, stricture and perforation were observed in 32%, 4.5% and 1% of the patients respectively. Though it is cheap and freely available, because of the high complication rate, use of phenol as a sclerosing agent was given up.

POLIDOCANOL:

Hydroxypolyethyoxydodecan (HPD) or polidocanol is commercially available as aethoxysclerol.It is a synthetic product available in uniform lots. It is marketed as 0.5, 1, 2, 3% solutions. Paquet has concluded by his study that 1% polidocanol was associated with decreased rate of complications. Deep ulcerations were seen in 11 patients out of 640 patients, while superficial ulceration occurred in 30 patients, pleural effusion in 14 patients. Minor complications like retrosternal pain were observed in 15% of the patients. Sorensen *et al.*, described a higher rate of esophageal stricture (59%) with the use of 3% polidocanol especially when more treatment sessions and greater amount of scerosants were used[18].

SODIUM TETRADECYL SULPHATE:

Sodium tetradecyl sulphate was first suggested as a sclerosing agent in1946. Reiner noted that the agents in use at that time, such as sodium morrhuate, were soaps of naturally occuring oils and allergic reaction did occur ranging from rash to anaphylaxis. Surface activity of the fatty acid anions of the soap was believed to be the physical activity responsible for thrombosis. This activity was enhanced in this synthetic anionic detergent, sodium tetradecyl sulphate.

Hence being a detergent based chemical, its action is on the lipid molecules in the cells of the vein wall which results in the destruction of the internal lining of the vein and eventual sclerosis of the vein. It is available in the concentrations of 0.2%, 0.5%, 1.0%, 1.5%, 3.0% solution.

Sodium tetradecyl sulphate occurs as white waxy solid. Sotradecol is a sterile non pyrogenic solution of sodium tetra decyl sulphate which is used as a sclerosing agent. These drugs are widely used for varicose veins.

ADVERSE EFFECTS:

Local reactions such as pain, ulceration are common at the site of injection. Allergic reactions like asthma, hay fever and anaphylactic shock were reported. Systemic reactions reported are headache, nausea and vomiting.

Six deaths have been reported among which anaphylactic shock accounts for four patients, one is due to asthma and the last is due to its concomitant use with anti-ovulatory agent.

Blenkinsopp showed that 3% STD was efficacious than 1% but damage to arterial wall was seen at both 1% and 3% concentrations but at a lesser incidence compared to other sclerosants. This is of interest because the bleeding from deep ulcerations following sclerotherapy was to be related to arterial damage rather due

to portal hypertension. Post sclerotherapy ulcerations were found to be superficial ulcerations and not considered a drawback. Thus sodium tetradecyl sulphate can be considered as one of the potent sclerosing agent at present.

TECHNIQUE:

There is no accepted standard technique for sclerotherapy injections. One disparity lies between paravariceal and intravariceal injection

Intravariceal technique:

It is also known as Anglo-American method. Sclerosants are directly injected into the varices. All visible varices are injected with 1-2ml of sclerosant directly then with 1-2ml 1cm below the bleeding site. Then 1 ml of sclerosant is injected at gastroesophaeal junction along all the varices. Even though the varices are present more proximally injections are placed up to 10cm from gastroesophageal junction in 3-5 cm intervals unless a more proximal bleeding site is identified since the sclerosant can escape from varix into the azygos system and then into pulmonary circulation. Total volume of sclerosant should never exceed 20 ml per session or 5 ml per varix.

Paravariceal technique:

It is also known as European approach. In this technique sclerosnt is injected into the adjacent submucosa of the visible varices. At first sclerosant is injected at the gastroesophageal junction and it is repeated circumferentially up to 10 cm proximally in a spiral fashion. The advantage is that it controls bleeding by causing subsequent inflammation and fibrosis around the vessel wall while preserving vessel patency allowing for portal decompression and also preventing the formation of collateral vessels.

Sarin *et al*., used a transparent teflon injector with a needle for injection sclerotherapy. If after puncturing the varices, blood could be seen to flow up into the teflon injector, it was taken as intravariceal injection. He concluded that intravariceal sclerotherapy was superior to paravariceal sclerotherapy in the control of active bleeding and for total variceal obliteration but paravariceal injection is associated with low recurrence rate[19]. Hence a combination of intravariceal and paravariceal injection is superior to the above two.

COMPLICATIONS OF SCLEROTHERAPY:

EARLY

-low grade fever

-dysphagia

-retrosternal pain

-pleural effusion

-chest radiographic changes

DELAYED

-mucosal ulcerations

-perforation

-esophageal strictures

-acute respiratory distress syndrome

-mediastinitis

-pneumothorax

-pericarditis

-fistulas

-esophageal motility disorder

-mesenteric venous thrombosis

-bacteremia

Sclerotherapy is associated with a wide range of complications ranging from transient pyrexia to esophageal perforation resulting in death. Complications following sclerotherapy depends on a number of factors namely nature of sclerosant used, amount of sclerosant, injection site, concentration of the drug and the time interval between the sessions. Minor complications like fever, retrosternal pain and dysphagia occurs so frequently that these are considered as side effects and not as a complication.

ESOPHAGEAL COMPLICATIONS:

Esophageal ulceration occurs frequently following emergency sclerotherapy. Sarles included the most superficial ulcers too and arrived at a 63% incidence of ulceration with 1% sodium tetradecyl sulphate. Conversely, Soderlund included only the ulcers that were associated with bleeding or were deep enough to prevent further sclerotherapy and reported an incidence of 20%. Jasperson reported esophageal ulceration in 78% and ulcerogenic bleeding in 14% following sclerotherapy with 1% poidocanol. In the presence of deep ulcers, further injection should be deferred to prevent esophageal perforation.

Esophageal perforation is the most dreaded complication and has an incidence of 1-7%. These patients may be managed nonoperatively with either enteral feeding or parenteral hyperalimentation and a course of intravenous antibiotics. Sarles, using 1.5% sodium tetradecyl sulphate, noted esophageal perforation in 0.4% of patients, while Jasperson reported a 2% incidence of perforation with the use of 1% polidocanol.

The incidence of esophageal strictures ranges from 0.8%, as reported by Johnson with the use of ethanolamine oleate to 52% with the use of 3% polidocanol. Sorenson's high rate of esophageal

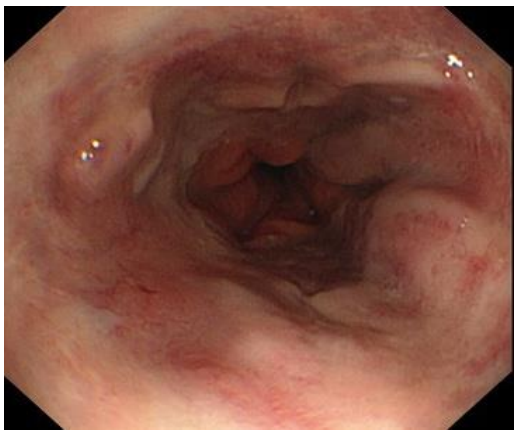
strictures was attributed to the frequency and amount of sclerosant used.

Transient substernal pain may occur in 50% of patients following sclerotherapy. The pain is due to inflammatory mediastinitis or esophageal spasm. Chronic dysphagia following sclerotherapy may be due to distal esophageal strictures but 4-5% is due to impaired motility. Esophageal manometric studies show no decrease in esophageal sphincter pressure but show marked abnormalities in esophageal peristalsis.

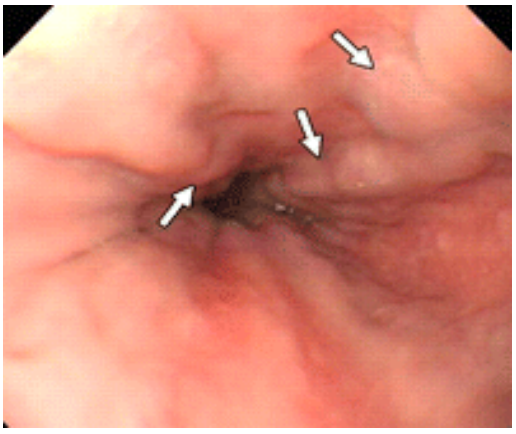
Esophageal carcinoma has been reported in one case after undergoing nine sessions of sclerotherapy with 3% polidocanol.

REGIONAL COMPLICATIONS:

Pulmonary complications due to sclerotherapy range from asymptomatic changes on X-ray to pleural effusion, pneumonia and ARDS. Hughes reported 60% in incidence of pleural effusion or pulmonary infiltrates following sclerotherapy. Pleural effusion resolves spontaneously. Pneumonia occurs due to aspiration. The development of ARDS occurs with the use of sodium morrhuate. Monroe discovered that sodium morrhuate caused transient pulmonary hypertension associated with increased flow of protein poor lymph.



GRADE I



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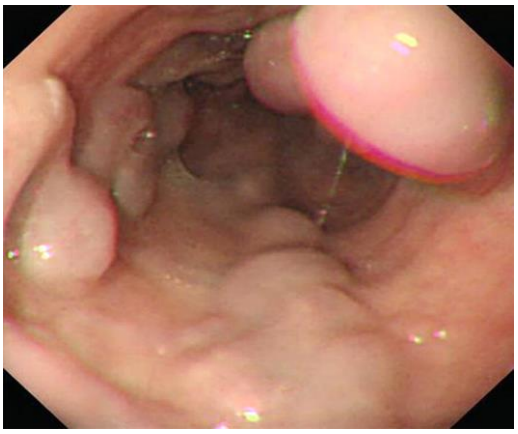
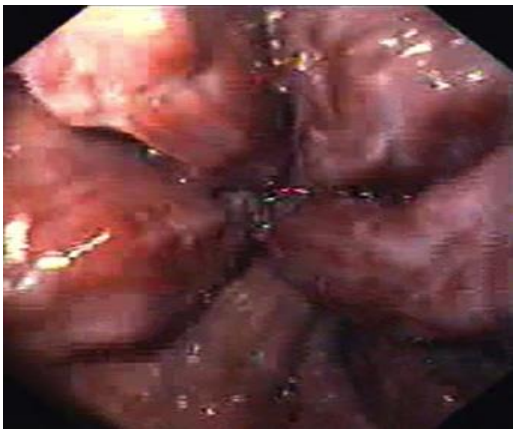


GRADE III



GRADE IV

SEPTIC COMPLICATIONS:

20-40% of patients had fever lasting for 24-48 hours following sclerotherapy. When fever lasts more than 2 days, the diagnosis of sepsis should be made. Transient bacteremia was reported in 16% of patients. Pneumococcus, streptococcus and staphylococcus were the commonest organisms isolated. The risk of sepsis due to sclerotherapy was not related to the amount of sclerosant used, the number of sessions, or the cause of the liver disease. Snady demonstrated a threefold increase in the incidence of bacteremia if the needle was inserted up to 6-8 mm instead of 3-4 mm. The incidence of clinically evident sepsis after sclerotherapy is low, so antibiotic prophylaxis is not needed. But Lange and Durack advised endocarditis prophylaxis for the patients undergoing sclerotherapy those who have significant valvular heart disease.

Other septic complications such as brain abscess, spontaneous bacterial peritonitis, perinephric abscess, purulent meningitis have also been reported. Other complications such as gastric ulcers, bleeding duodenal varices, colonic varices, portal vein thrombosis and mesenteric vein thrombosis have also been reported.

ENDOSCOPIC VARICEL LIGATION:

Endoscopic variceal ligation was first introduced to esophageal varices in 1989 by Stiegmann and Goff. This technique is an adaptation of the similar banding ligation of internal hemorrhoids. In contrast to sclerotherapy where chemical action is used, in variceal ligation obliteration of the varices is caused by mechanical strangulation with rubber bands. Because of its action on the suctioned, entrapped varices, the reaction is usually limited to the superficial esophageal mucosa.

Endoscopic variceal ligation is the placement of the rubber rings over the variceal column which is then sucked into a plastic cylinder which is attached to the tip of the endoscope.

Previously single shot ligation was used. Multiple shot devices have replaced the previous one because of its simplicity and rapidity and also over tube is not required and hence preventing its serious complications related to its use. And also new transparent caps are available which improve the visibility (old caps reduce the visibility by 30%). Several commercial multiband devices are available for EBL. Multiband devices have 4-10 bands.

<u>TECHNIQUE:</u>

Diagnostic endoscopy is performed and varices are identified. The distance is measured from the mouth by the markings in the endoscope. The endoscope is withdrawn and is loaded with ligation devices. Device is firmly attached to the scope and placed in neutral mode. Endoscopy with the loading devices is passed which needs little experience. Slight flexion of the neck, gentle and constant advancement of scope with a slight torque of the shaft right and left with visualization of the pharynx would guide it. After intubation the device is kept in forward only mode. Once varix is identified, the tip is pointed towards it and continuous suction is applied to the varices till it is filled in the cap. Smooth movement left and right will help it. Once the red out sign appears, band can be fixed.

Usually the procedure is performed by starting from gastroesophageal junction and proceeding upwards in a spiral fashion to avoid circumferential placement at same level which would increase risks of stricture.

In case of active bleeding the visual field is restricted due to the cylinder attachment which makes the technique difficult to perform and thus requiring active flushing with water and suction repeatedly.

The rubber band should be delivered at a point on the varices but if it is missed, banding of the normal mucosa is not harmful compared to the sclerosant injection, which may cause a serious side effects.

If the bleeding point is not identified, then a multiple banding device can be used to place multiple bands at the gastroesophageal junction checking that no subcardial prolongation occurs. This might reduce torrential bleeding and then the band can be fixed upward.

After the application of rubber bands over the esophageal varices, the ligated tissues with the rubber bands will fall off within 10 days. The variceal sloughing causes shallow esophageal ulcers at the ligated sites while the esophageal varices reduce in size. Though the ligation induced ulcers have a greater surface area, they are shallower and hence heal more quickly than that are caused by sclerotherapy. Liquid diet is started for the first 12 hours and then patient is advised to have soft foods. A recent study tells that patient who received pantaprazole after elective EVL found to have smaller post-banding ulcers than the other patients who received placebo therapy on follow up endoscopy. But the symptoms and ulcer number remained the same.

Eradication of the esophageal varices requires 2-3 sessions of endoscopic variceal ligation. De Franchis and Primignani conducted a meta-analysis in 1999 included 13 articles in which the mean number of sessions to obliterate varices was reduced from 5.4 in patients receiving sclerotherapy to 3.6 in patients receiving endoscopic variceal ligation. Both the time interval between the sessions and the

number of bands placed in each session should be noted to improve the efficacy of banding. Varices is said to be obliterated if they either disappear or unable to grasp. Eradication can be obtained in about 90% of subjects although recurrence is common. The major disadvantage is higher incidence of recurrent varices. But these is recurrent varices can be treated by ligation. Moreover recurrent varices do not increase the chance of rebleeding and do not cause endoscopic difficulties. A study from Japan described that EVL performed once in two months is better than that is performed in two weeks regarding variceal occurrence. Because the rebleeding rate is significantly reduced who received endoscopic therapy at early and who achieves variceal obliteration in a shorter period. The incidence of bacteremia and infectious sequelae are less in EVL compared to sclerotherapy. Endoscopic band ligation is an alternate to sclerotherapy with less complications but there are the below list of complications.

COMPLICATIONS:

- Esophageal ulceration
- Esophageal perforation (due to trauma of over tube)
- Transient dysphagia
- Retrosternal pain
- Esophageal strictures

- Gastropathy
- Ulcer bleeding
- Bacteremia

The advantage of EVL is the low rate of treatment induced complications. This is because the quantity of tissue ligated is limited by the design of the device resulting in fewer complications involving the esophageal wall[20].

Complications of EVL are either due to the ligation procedure or from the use of the overtube. Retrosternal pain, transient dysphagia occurs frequently in the immediate post ligation period and is considered as side effects rather than a complication.

ESOPHAGEAL ULCERATION:

The band ligations usually produce small ulcerations and rarely produce symptoms. They present as mucosal defects at the site of application of bands.

Gimson *et al.*, have reported esophageal ulcerations in 36 out of 54 patients who had banding. 23 of them had small ulcerations (size <5mm) and 13 of them were large (size >5mm)[21]. Laine *et al*., has reported esophageal ulcerations producing rebleeding in 5% of patients who had undergone ligation. Steigmann has reported bleeding from ulcers in 11% of patients following ligation.

Young *et al.*, compared the ulcerations produced by ligation and sclerotherapy by means of scored ERCP cannula to measure the length, width and depth of ulcers in a randomized trial. Esophageal ligation produced shallow circular ulcerations with large surface area that resolved in 14.4 days[22]. Sclerotherapy produced linear, deep ulcerations with a smaller surface area that resolved in 20.9 days.

Van Vlierberghe *et al.*, reported early rebleeding after ligation in patients with Child-Pugh class C cirrhosis. This was attributed to the impaired clotting function as a result of liver disease and the greater size of the ulcers due to ligation.

ESOPHAGEAL STRICTURES:

Esophageal ulcerations leading onto strictures are less common following ligation than with sclerotherapy. Laine *et al*., in a meta-analysis of 7 randomized trials involving 547 patients found esophageal strictures in 7 patients. Another study by Laine *et al.*, demonstrated a significant reduction in stricture formation in ligation (none) when compared to sclerotherapy (33%). Low rates of stricture formation have been reported by Baronicin due to ligation (11%) when compared to sclerotherapy. Steigmann and Sarin also reported a lower incidence of stricture formation following ligation (2% and 0%)[23].

ALTERATION OF ESOPHAGEAL MOTILITY:

In a study by Berner *et al*., 75% of patients had reported a transient dysphagia which lasted up to 24-72 hours after the procedure. This is due to the engorged banded varices. In a study conducted by Ming-Chih Hou *et al*., he compared the alteration of esophageal motility following sclerotherapy and ligation. He found that ligation produced a little change at 1 month or 3 months after eradication, while sclerotherapy produced a significant prolongation of transit time for 1month after eradication which was reversible and improved after 3 months.

SYSTEMIC COMPLICATIONS:

Risk of bacteremia following ligation is 3-6% compared to sclerotherapy which is 5-53%. It is associated with fewer episodes of infectious sequelae such as spontaneous bacterial peritonitis or pneumonia.

Gin-Ho *et al*., reported that infectious sequelae due to sclerotherapy is about 18% compared to 1.8% in ligation. This is because the mechanical strangulation of varices during ligation obliterates the submucosal channels which diminish the entrance of

bacteria into the blood stream. In a meta-analysis of 7 trials conducted by Laine *et al*., 6 out of 524 patients had pulmonary infection and 5 had bacterial peritonitis which was significantly lower comparing sclerotherapy[3].

OVERTUBE ASSOCIATED COMPLICATIONS:

In the past during endoscopic ligation using a single shot ligator the necessary repeated esophageal intubation with the scope is facilitated by a flexible plastic overtube passed over the endoscope. Majority of complications reported following ligation are associated with the use of oertube. Overtube injury to pharynx and proximal esophagus transient vocal cord paralysis, cricopharyngeal perforation, proximal esophageal perforation, varix rupture and free esophageal perforation have also been reported.

Mucosal injury has been reported in 72% of treatment sessions in one study. Massive bleeding has been reported distal to the overtube, probably due to blockage in venous outflow by the tube.

Esophageal perforations occurred with over the endoscope placement technique because of the large gap between the endoscope and the overtube which entrapped esophageal mucosa during the process of sliding the overtube over the endoscope during its insertion[24]. Since the development of multiband ligator, not a single esophageal perforation has been reported.

Banding is also associated with food impaction resulting from a combination of lumen obstruction by banded varices and distal esophageal spasm.

OTHER ENDOSCOPIC OPTIONS:

TISSUE ADHESIVES (VARICEAL OBTURATION):

N-butyl-2-cyanoacrylate and isobutyl-2-cyanoacrylate have been used in the control of esophageal and gastric varices with control of bleeding in 90% of cases. Tissue adhesives were first used by Lunderquist in 1978 in treatment of varices.

Cyanoacrylate is a hydrophilic tissue adhesive with a consistency similar to water. This when added to blood rapidly polymerizes forming a solid cast of the injected vessels which results in rapid hemostasis of active bleeding and prevents recurrence of bleeding. It has to be diluted with lipid based contrast agent to the dilution of about 2:1 ratio to delay the instantaneous polymerization within the injection syringe and needle.

Complications are due to embolisation of the glue producing cerebral stroke, pulmonary embolism, portal vein thrombosis, splenic infarction, retrogastric abscess and visceral fistula. Chances of damage to endoscope are high due to clogging of the accessory channel. There is also danger of eye injury due to accidental spraying of the cyanoacrylate.

ENDOLOOPS:

Endoloops are detachable nylon snares initially developed to control post polypectomy bleeding. This technique has been applied for control of bleeding from esophageal varices and reports suggest it as a safe and effective as banding or sclerotherapy. Pontecarvo and Pesce invented the detachable snare, the 'safety snare' in 1986.

4. Materials & methods:

MATERIALS AND METHODS

The study was conducted in the Department of General Surgery in collaboration with the Department of Medical Gastroenterology and

Department of Medicine, Coimbatore Medical College Hospital from September 2011 to August 2012. This study was approved by the ethical committee of Coimbatore Medical College Hospital.

STUDY POPULATION:

50 patients with portal hypertension who were admitted during the study period of September 2011 to August 2012 in medicine and surgery wards, with the complaints of hematemesis and/or malena, who had Grade 3 and 4 varices without gastric varices and other causes of upper GI bleeding in upper GI endoscopy were included in this study.

RANDOMISATION:

Every alternate patients presenting with above history is divided into 2 groups. One group is treated with esophageal banding and other group is treated with 3% Sodium tetradecyl sulphate after getting informed and written consent from the patient.

INCLUSION CRITERIA:

- Age 21-70 years
- Both sex

- Patients complaining with hematemesis and/or malena
- Grade III and IV esophageal varices
- Due to Portal Hypertension

EXCLUSION CRITERIA:

- Age < 21 and > 70
- Non portal hypertension causes of upper GI bleeding
- Grade I and II varices
- Gastric or combined Gastric and Esophageal varices
- Presence of Hepatic Encephalopathy, Hepatorenal syndrome and life expectancy less than 48 hours
- Prior history of endoscopic treatment and shunt operation for varices
- Patients with positive serology for Hepatitis B (HbsAg) and C viruses (anti HCV)

PROCEDURE:

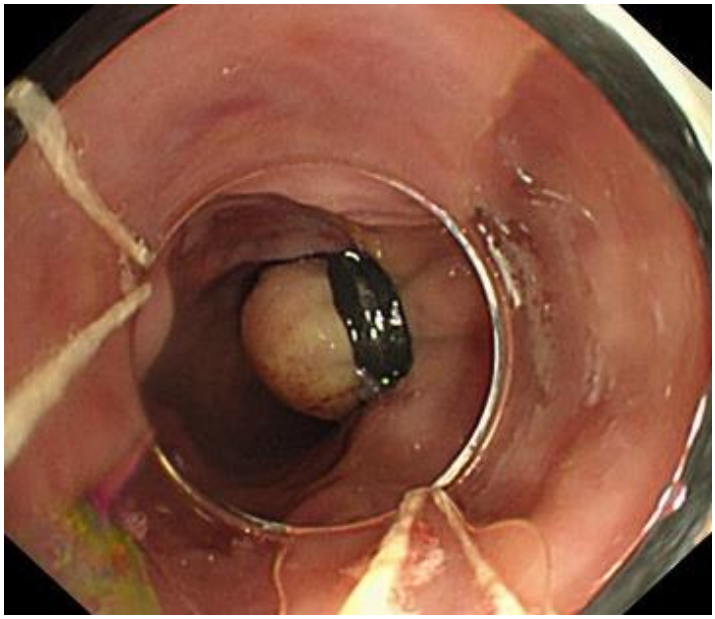
Informed consent is obtained from the patient about the procedure. Patient is kept in NPO for 6 hours. Xylocaine spray is applied over the posterior pharyngeal wall. Diagnostic endoscopy is performed. Presence of the culprit grade III and IV varices are identified and confirmed.

GROUP I:

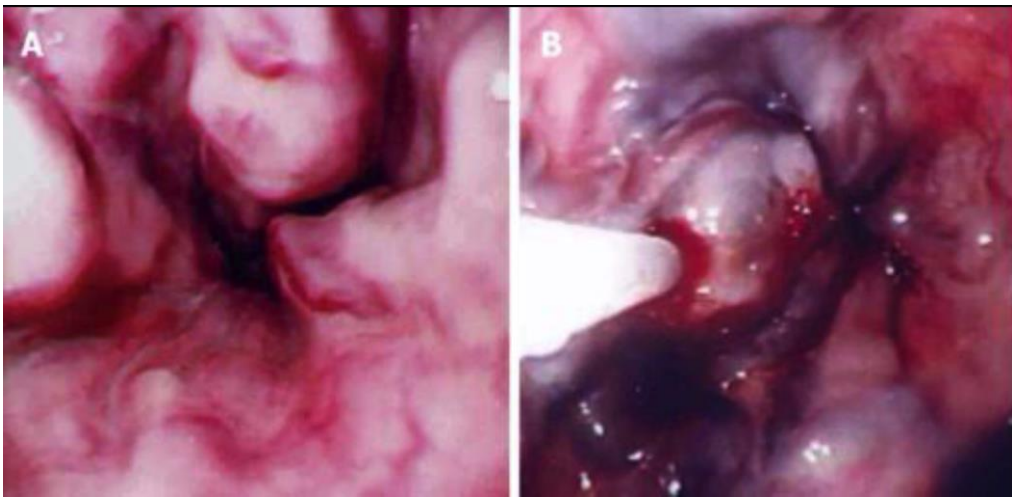
They are subjected to esophageal variceal banding. Diagnostic endoscopy is performed and varices are identified. The distance is measured from the mouth by the markings in the endoscope. The endoscope is withdrawn and is loaded with ligation devices. Device is firmly attached to the scope and placed in neutral mode. Endoscopy with the loading devices is passed. After intubation the device is kept in forward only mode. Once varix is identified, the tip is pointed towards it and continuous suction is applied to the varices till it is filled in the cap. Once the red out sign appears, band can be fixed starting from gastroesophageal junction and proceeding upwards in a spiral fashion.

GROUP II:

They are subjected to endoscopic sclerotherapy treatment. Diagnostic endoscopy is done and varices are identified. All visible varices are injected with 1-2ml of 3% sodium tetradecyl sulphate below the bleeding site directly into the varices and the colour change is noted to confirm. Then the adjacent submucosa of the varices is injected with 1ml of the sclerosant carefully upto 10cm from Gastroesophageal junction proximally in a spiral fashion. Care is



BANDING



SCLEROTHERAPY

taken that not more than 20ml is injected in a single session to a patient.

Three sessions were planned for every patient in an interval of 3 weeks. Thereafter patients were reviewed once in a month for a period of three months. For each session the number of bands and the amount of sclerosant used are recorded. After the procedure all the patients are treated with beta blockers.

During each visit, patients were assessed for complications such as retrosternal pain, esophageal ulcers, strictures, pleural effusion and mediastinitis.

Esophageal ulcers are defined as depression in the mucosal surface with an overlying injury exudate. They were classified as superficial if shallow and less than 2cm in diameter and as deep if more than 2cm with shaggy border and a greyish necrotic base. Chest x-ray was taken if the patients had persistent pain for detection of pleural effusion or mediastinitis if symptoms warranted.

Fever was defined as oral temperature more than 99 degree F in the first 24 hours following therapy.

Dysphagia was defined as difficulty in swallowing food and was graded into 4 grades.

- Grade I – Able to swallow both solid and liquid foods but with difficulty

- Grade II – Able to swallow liquid foods but not solid foods
- Grade III – Not able to swallow both solid liquid and solid foods
- Grade IV – Absolute dysphagia including inability to swallow saliva

Esophageal strictures was diagnosed if the patients repoted with dysphagia and had evidence of narrowing by endoscopy and barium swallow. The efficacy of treatment was assessed in terms of

- Eradication of varices
- Number of sessions for variceal eradication
- Variceal recurrence
- Rebleeding episodes prior to eradication
- Associated complications

Variceal eradication was defined as the absence of visible variceal channels in the distal 5cm of esophagus or presence of only mucosal tags[25].

Variceal recurrence was defined as reemergence of variceal columns following previous complete eradication.

Control of active hemorrhage was defined as absence of clinically detectable upper GI bleeding for 48 hours after endoscopic variceal ligation was performed for active bleeding[25].

Failure of therapy was defined as recurrent variceal bleeding after three endoscopic treatment sessions or during the course of therapy.

STATISTICAL ANALYSIS:

GraphPad Instat software was used for statistical analysis. Fisher's exact test was used for comparing the outcomes of endoscopic band ligation therapy and sclerotherapy. A p value of < 0.05 was considered statistically significant.

5. Results:

RESULTS

In the study population, 29 males and 21 females have undergone treatment for varices and the majority were in the age group of 31-50 years.

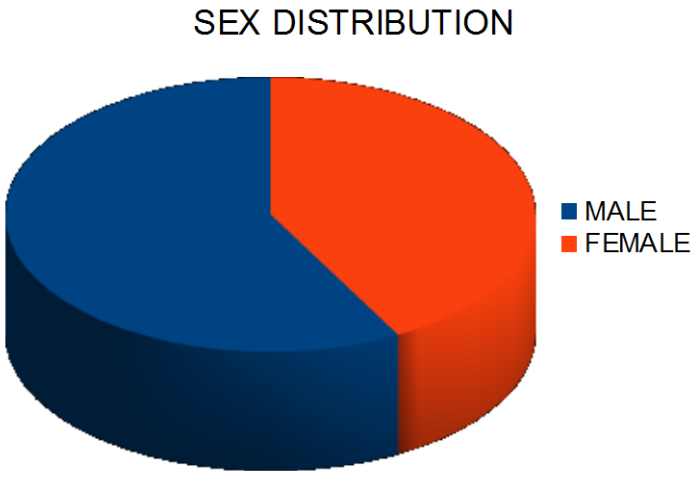


Figure 7: Sex distribution of the study population

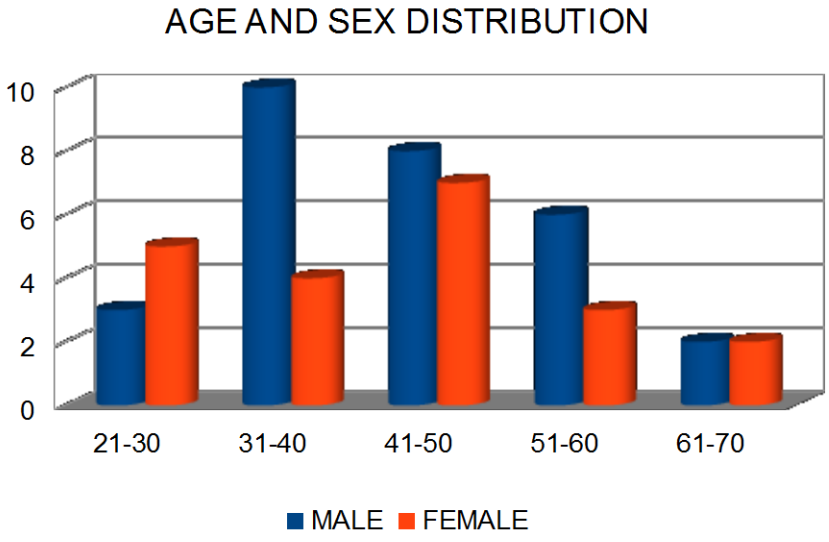


Figure 8: Age & sex distribution of the study population

AGE & GRADE DISTRIBUTION:

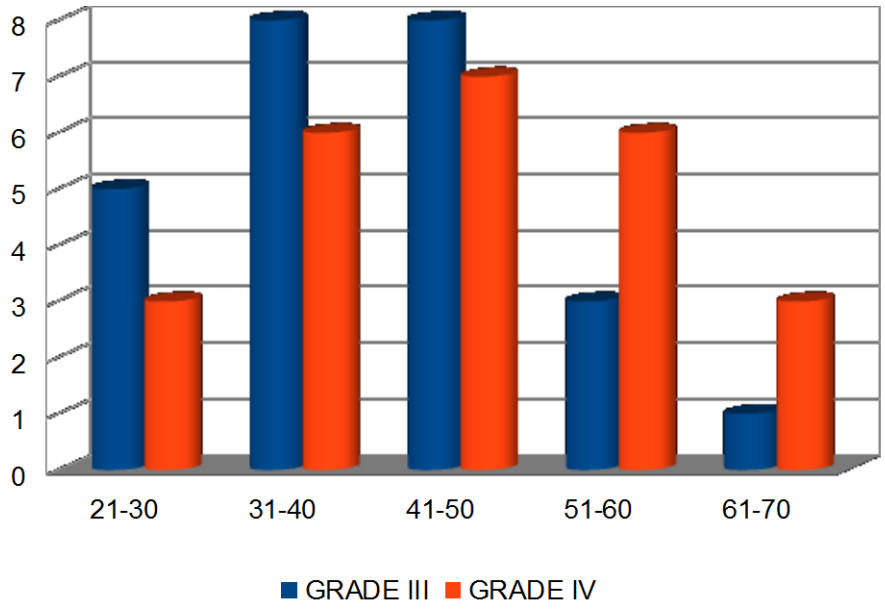


Figure 9: Age & variceal grade distribution of the study population

As the age progresses grade IV varices was more common than grade III at presentation.

Study group randomization:

Table 1: Banding & sclerotherapy in bleeding varices

ACTIVELY BLEEDING VARICES	BANDING		SCLEROTHERAPY		TOTAL
	GRADE III	GRADE IV	GRADE III	GRADE IV	
+	6	4	5	7	22
-	7	8	7	6	28
TOTAL	13	12	12	13	50

22 cases presented with active bleeding during the procedure and the remaining 28 patients had no signs of bleeding. Active bleeding was more common among male patients.

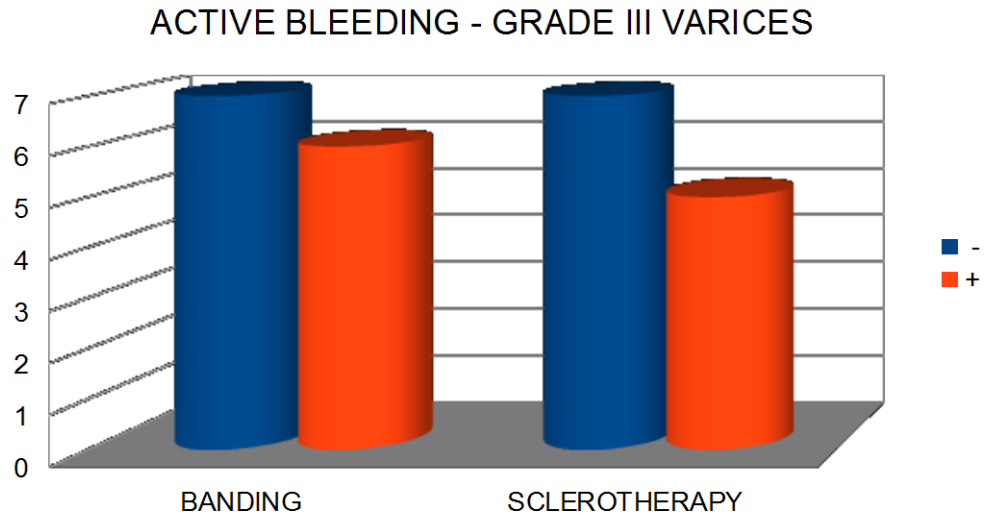


Figure 10: Randomization of actively bleeding Grade III varices for banding & sclerotherpy

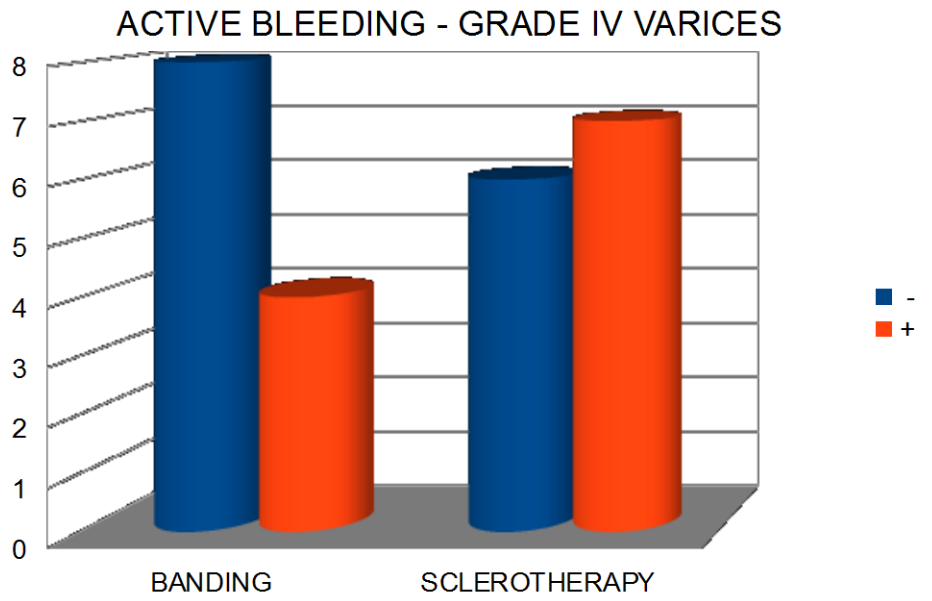


Figure 11: Randomization of actively bleeding Grade IV varices for banding & sclerotherpy

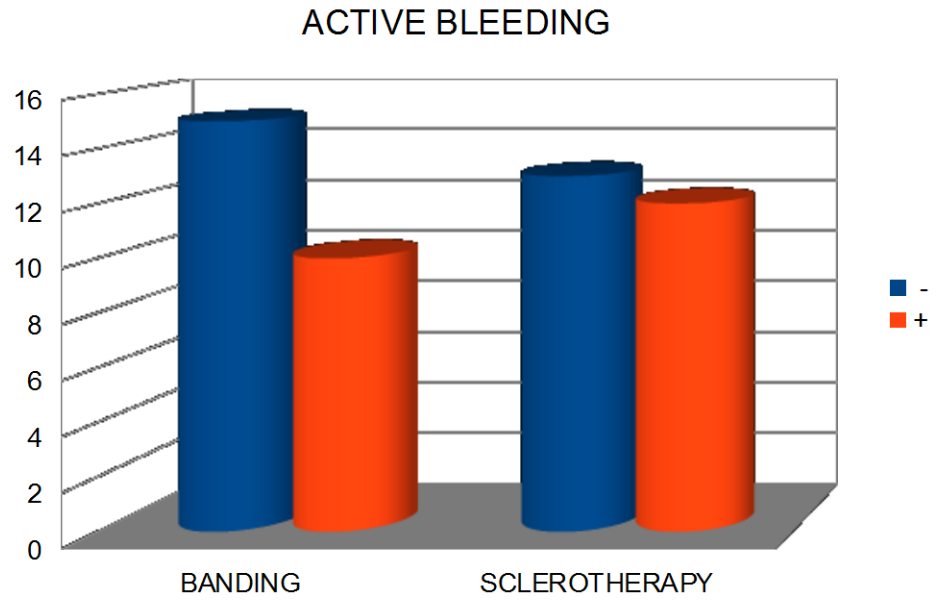


Figure 12: Randomization of actively bleeding varices for banding & sclerotherpy

NUMBER OF BANDS & AMOUNT OF SCLEROTHERAPY USED

Table 2: Number of bands and amount of sclerotherapy used

GRADE	ACTIVE BLEED	TOTAL NO. OF BANDS USED	TOTAL AMT. OF SCLEROSANT(ml)
III	+	52	168
	-	42	120
IV	+	44	196
	-	46	120
TOTAL		194	532

94 bands were used for grade III varices and 90 bands were used for grade IV varices.188 ml of sclerosant is used for grade III varices and 216 ml of sclerosant used for grade IV varices

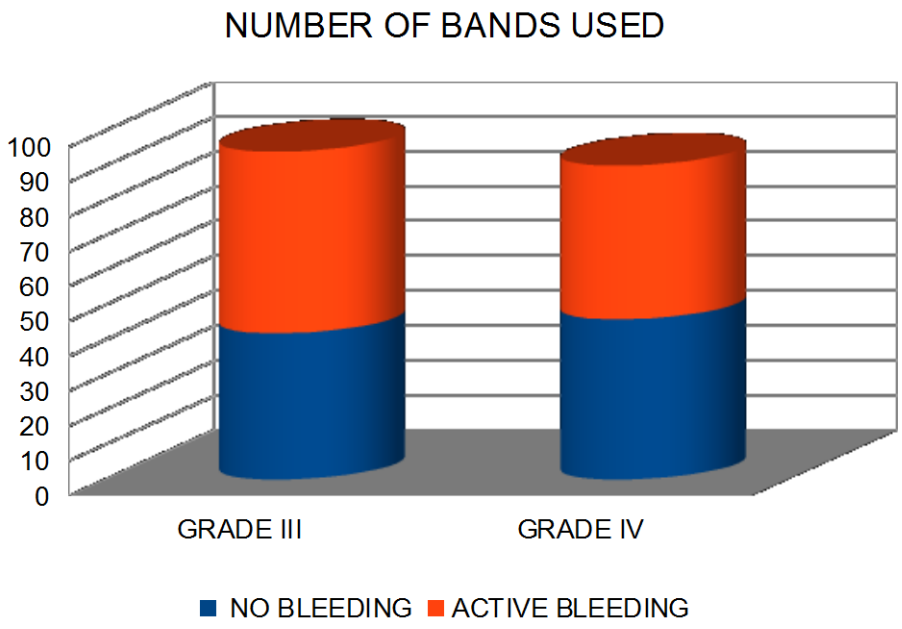


Figure 13: Number of bands used

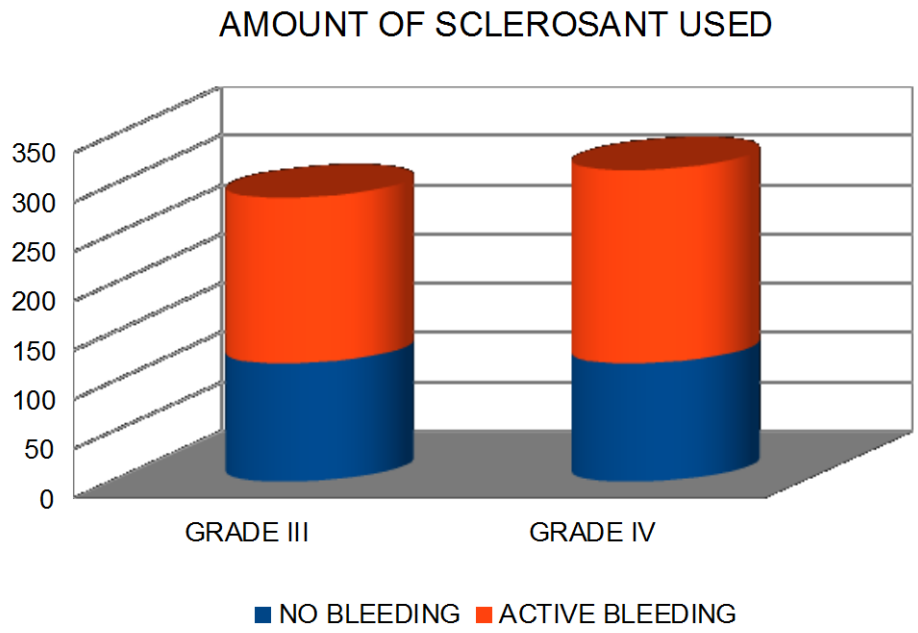


Figure 14: Amount of sclerosant used

In case of active bleeding the number of bands and the amount of sclerotherapy required are found to be more compared to non-bleeding varices.

COMPLICATIONS:

Table 3: Complications of banding & sclerotherapy

COMPLICATIONS	BANDING				SCLEROTHERAPY			
	MALE		FEMALE		MALE		FEMALE	
		%		%		%		%
RETROSTERNAL PAIN	3	10	3	15	5	16.5	6	30
ODYNOPHAGIA	1	3.3	3	15	4	13.2	8	40
FEVER	1	3.3	1	5	5	16.5	6	30
TACHYCARDIA	0	0	2	10	3	10	5	25
ESOPHAGEAL ULCER	3	10	3	15	9	29.7	4	20
STRICTURE	0	0	0	0	1	3.3	0	0
REBLEEDING	2	6.6	1	5	1	3.3	0	0
FAILURE	0	0	0	0	2	6.6	3	15

Females had more complications and frequency of hospital visits after procedure than males

RETROSTERNAL PAIN:

Table 4: Comparison of banding vs sclerotherapy and retrosternal pain

RETROSTERNAL PAIN	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	6	11	17
ABSENT	19	14	33
TOTAL	25	25	50

Though retrosternal pain was seen in more number of cases who underwent sclerotherapy, the association was not statistically significant (p value 0.2321).

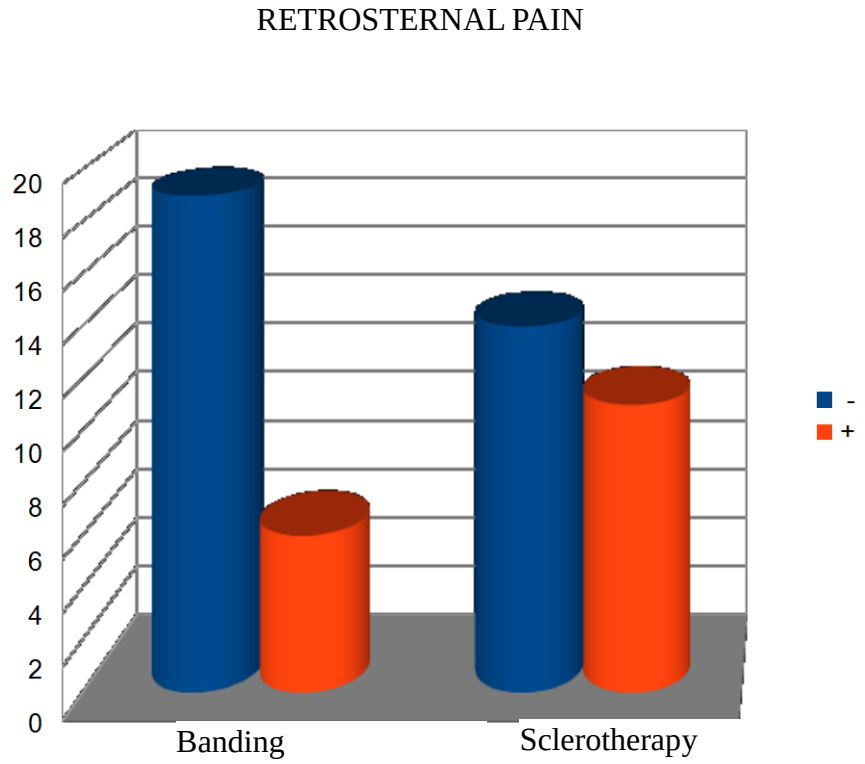


Figure 15: Comparison of banding vs sclerotherapy and retrosternal pain

ODYNOPHAGIA:

Table 5: Comparison of banding vs sclerotherapy and odynophagia

ODYNOPHAGIA	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	4	12	16
ABSENT	21	13	34
TOTAL	25	25	50

Twelve patients in the sclerotherapy group suffered odynophagia. However, there is no statistically significant difference between the two groups (p value 0.0322).

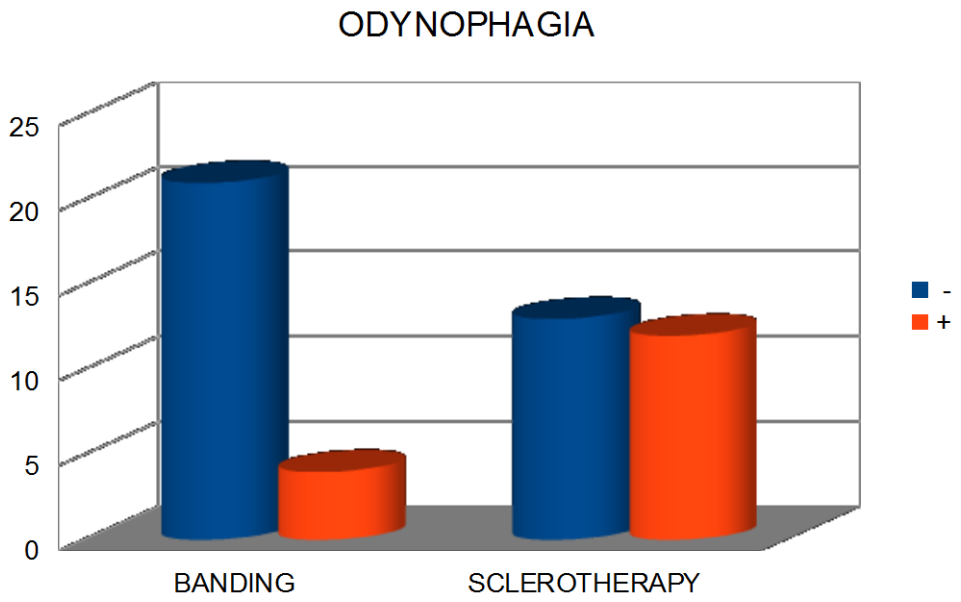


Figure 16: Comparison of banding vs sclerotherapy and odynophagia

FEVER:

Table 6: Comparison of banding vs sclerotherapy and fever

FEVER	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	2	11	13
ABSENT	23	14	47
TOTAL	25	25	50

Most of the patients who received sclerotherapy had fever, while only two patients in the banding group had fever and this difference was statistically significant (p value 0.0083).

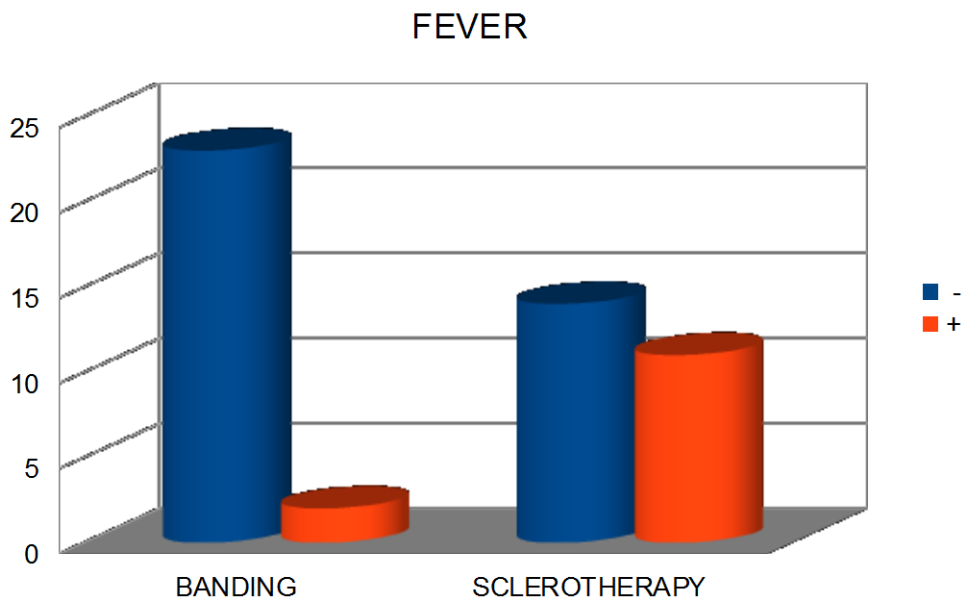


Figure 17: Comparison of banding vs sclerotherapy and fever

TACHYCARDIA:

Table 7: Comparison of banding vs sclerotherapy and tachycardia

TACHYCARDIA	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	2	8	10
ABSENT	23	17	40
TOTAL	25	25	50

The two treatment groups did not have a statistically significant difference when tachycardia among two groups was compared (p value 0.0738).

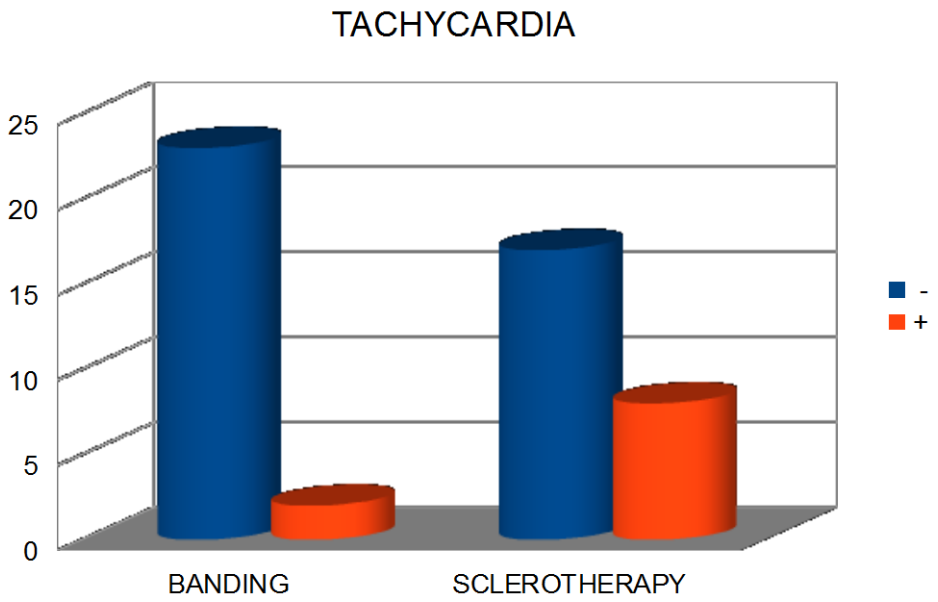


Figure 18: Comparison of banding vs sclerotherapy and tachycardia

ESOPHAGEAL ULCER

Table 8: Comparison of banding vs sclerotherapy and esophageal ulcer

ESOPHAGEAL ULCER	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	6	13	19
ABSENT	19	12	31
TOTAL	25	25	50

Though esophageal ulcer was seen in more number of cases who underwent sclerotherapy, the association was not statistically significant (p value 0.0792)

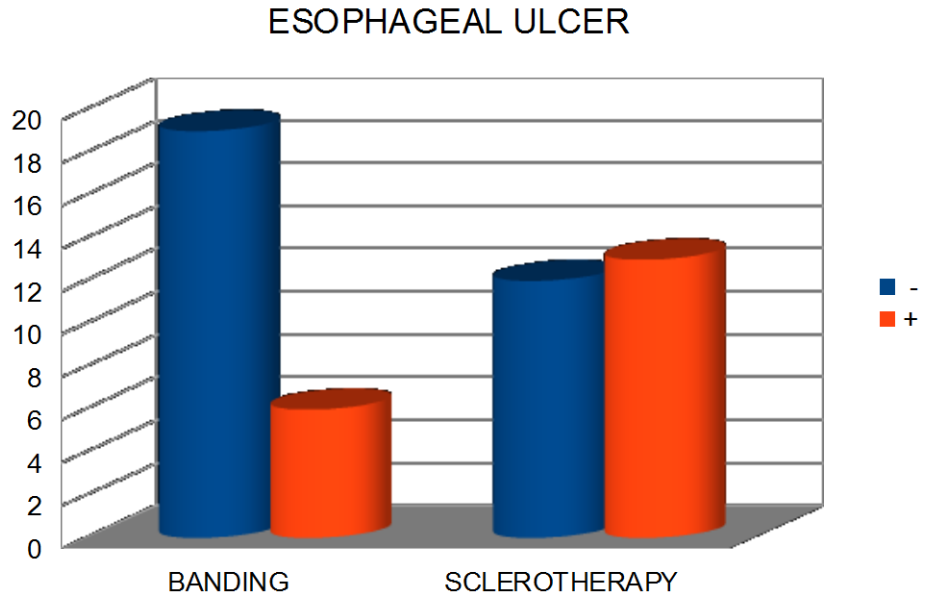


Figure 19: Comparison of banding vs sclerotherapy and esophageal ulcer

ESOPHAGEAL STRICTURE:

Table 9: Comparison of banding vs sclerotherapy and esophageal stricture

ESOPHAGEAL STRICTURE	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	0	1	1
ABSENT	25	24	49
TOTAL	25	25	50

Esophageal stricture was observed only among the sclerotherapy group, however it is not a statistically significant difference (p value 0.3124)

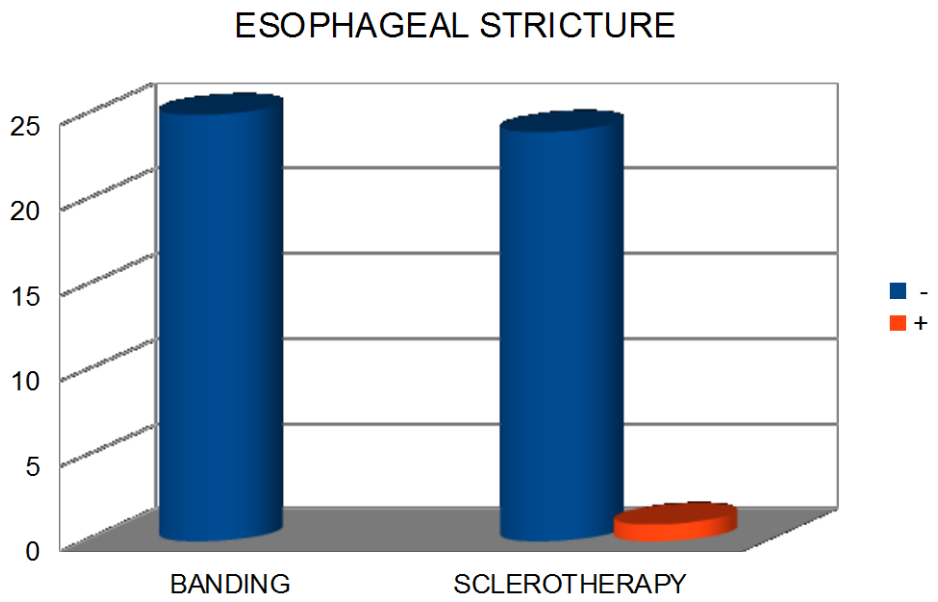


Figure 20: Comparison of banding vs sclerotherapy and esophageal stricture

REBLEEDING

Table 10: Comparison of banding vs sclerotherapy and rebleeding

REBLEEDING	BANDING	SCLEROTHERAPY	TOTAL
PRESENT	3	1	4
ABSENT	22	24	46
TOTAL	25	25	50

Banding therapy was associated with bleeding in 2 patients more than the sclerotherapy group. However, this difference was not statistically significant (p value 0.6022).

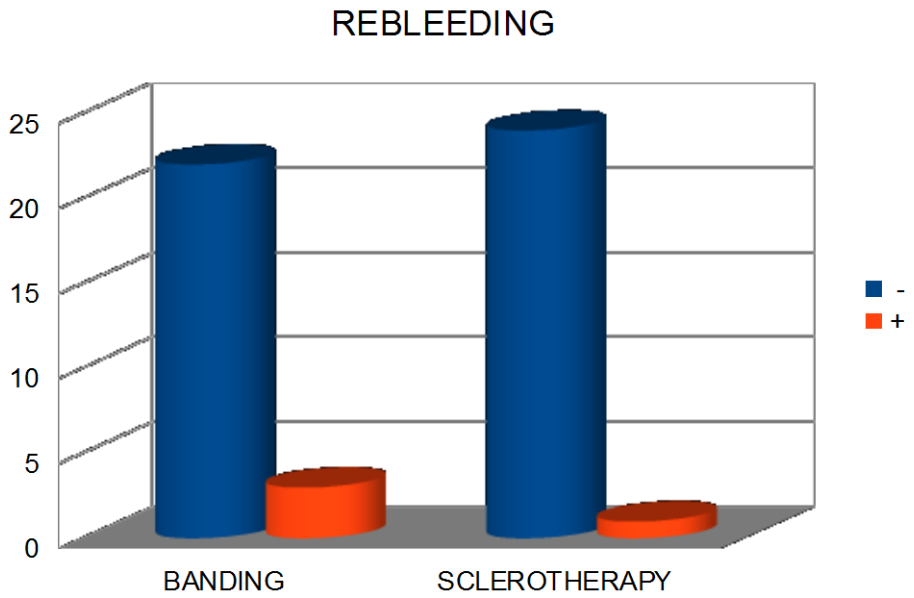


Figure 21: Comparison of banding vs sclerotherapy and rebleeding

FAILURE:

Table 11: Comparison of banding vs sclerotherapy and failure

FAILURE	BANDING	SCLEROTHERAPY	TOTAL
+	0	6	6
-	25	19	44
TOTAL	25	25	50

Sclerotherapy was more commonly associated with treatment failure than banding (p value 0.0223).

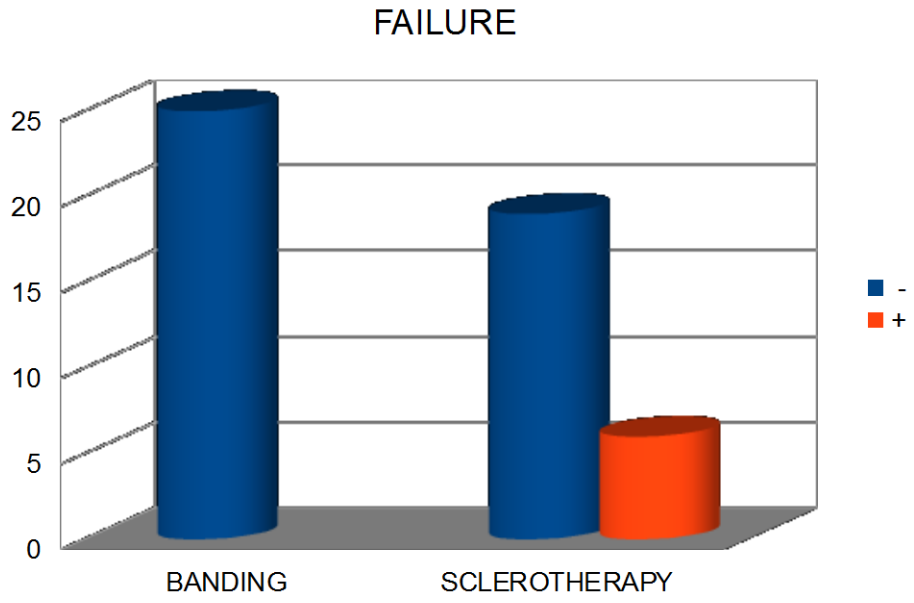


Figure 22: Comparison of banding vs sclerotherapy and failure

6. Discussion:

DISCUSSION

The improvement in the results of the treatment of the variceal bleeding might be attributed to better clinical management of the above patients. Although in most of the studies performed, sclerotherapy is found to be inferior to band ligation for primary and secondary prophylaxis for variceal bleeding and also with lot of complications compared to banding some studies suggest that both are equally efficacious in the treatment of the esophageal varices. In 1986, Steigmann and his colleagues introduced band ligation which acts by mechanical action by causing strangulation of the variceal cord resulting in necrosis and scar formation 7-10 days later. The differences in the technique are provided by number of bands used. Upto 10 bands can be used in a single session. Since this procedure is easy to perform, results are often reproducible without variations. The difficult is that ligation of small varices is tedious.

But in case of variceal injection of sclerotherapy, which was the first endoscopic treatment used approximately 50 years before the band ligation, there are numerous variations including the type of sclerosant, sclerosing technique, concentration of sclerosing agent, injected volume and location of the sclerosant (intravariceal and paravariceal is combined) which is the reason for heterogenous results of sclerotherapy presented in different publication. And also this

technique requires more experience and significant skill of the endoscopist and hence this technique is more operator dependent technique rather than banding.

Hence the concept of combining ligation with sclerotherapy by employing ligation when the varices are large and converting to sclerotherapy when the varices become smaller has been put forward to maximize the benefits of both the techniques and minimize the complications associated with each other. But most of the studies which compared ligation and sclerotherapy with ligation alone showed no greater benefits[26–28].

A total of 50 patients included in the present study, 60% of the patients are in the age group of 31-50 years. Cirrhosis was the most common cause for portal hypertension. This was followed by non-cirrhotic portal fibrosis and extra hepatic portal vein obstruction. In the present study, only the patients who had either grade III or grade IV varices at presentation are included. Since the patients with grade I and grade II varices were not considered for ligation because of the technical difficulty in banding.

Most of the patient had cirrhosis has their etiology while the others had extrahepatic portal venous obstruction.

CONTROL OF BLEEDING:

A meta-analysis is done comparing the use of band ligation and 3% sodium tetradecyl sulphate and was published in 2006 and consists a total of 12 studies and consists a total of 1310 patients. The efficacy of endoscopic band ligation for initial hematemesis was found to be an average of 97% while that of the endoscopic sclerotherapy was found to be an average of 95%. Despite the better results obtained in the control of bleeding in band ligation than sclerotherapy, there is no difference in the mortality noted. In the present study, the efficacy of banding is 96% and that of sclerotherapy is 88%. This excellent control of variceal bleeding is comparable to other reports has been mentioned by many authors that during active bleeding, presence of fresh blood and blood clots obscures the vision leading to difficulty in banding. In this study about 22 patients had active bleeding. In that 10 were subjected to banding and 12 were subjected to sclerotherapy. About 1 patient had rebleeding in banding and 3 patients had rebleeding who were subjected to sclerotherapy.

VARICEAL ERADICATION:

Several studies on ligation have reported successful eradication of varices in 55-93% of patients. Steigmann *et al*., reported an eradication rate of 51% with ligation with a median of 5 treatment sessions and 14 ligation per patient. Laine *et al*., in their study of 38

patients who underwent ligation reported 59% of eradication with a median of 4 treatment session at an average of 3.9 bands at each session. Gimson *et al*., reported an eradication rate of 70% in patients who underwent ligation with a median of 3.4 endoscopic sessions[21]. Sarles *et al*., reported 28% obliteration rate[29]. In another study, variceal obliteration was achieved in 54% of patients who were treated with sodium tetradecyl sulphate. Despite these old studies, new studies such as Bhargava *et al*., reported eradication in 87% of patients at a median of 6 endoscopic sessions and also showed 88% eradication rate in use of sodium tetradecyl sulphate[30].

The King's college study reported a satisfactory eradication of esophageal varices by the use of banding with less complication than sclerotherapy although much of the complications are strictures. A study conducted by Grimson and Ramage *et al*., with an aim to find whether endoscopic variceal ligation is more effective in eradicating varices than sclerotherapy showed that both the techniques were effective in controlling the bleeding (92% for banding and 91% for sclerotherapy). Variceal obliteration was not achieved in some patients in each group (3% in banding and 6% in sclerotherapy). Though there was no significant difference between the above two techniques in eradication, ligation achieved more quickly than sclerotherapy. Thus in newer studies, the efficacy of both band ligation and sclerotherapy

in eradicating the varices have increased a lot. This might be due to the advancement in the instrumentations and devices such as use of multiband ligating devices rather than single band ligating devices. In the present study of about 50 patients, 25 were treated by endoscopic variceal banding and other 25 patients by endoscopic sclerotherapy (3% sodium tetradecyl sulphate), the eradication of varices by banding was 100% while that of sclerotherapy was only 80%. As the most of the previous studies, the present study also suggests that the endoscopic variceal banding is superior to sclerotherapy in the eradication of varices.

COMPLICATIONS:

Most of the studies suggest that the main advantage of ligation over sclerotherapy is the low rate of complication. Laine *et al*., have reported complication in 24% of patients who had ligation. AlTraif *et al*., have reported a complication rate of 60% using sclerotherapy[28]. In the present study, the complications were found in 30 of 50 patients among which the majority were esophageal ulceration, retrosternal pain, odynophagia, fever and tachycardia. Similar observations were made out in most of the studies. The patients who had active bleeding during the procedure had more complication rate.

ESOPHAGEAL ULCER:

Esophageal ulcer was the commonest complication following sclerotherapy in most of other studies. The occurrence of post sclerotherapy ulceration was attributed to the higher volume of sclerosant per session, shorter interval between sclerotherapy sessions, higher concentration of sclerosant and nature of sclerosant used. In case of banding large superficial ulcerations are common due to necrosis. Esophageal ulceration was reported in 36% of patients who had undergone ligation by Gimson *et al*., in the study of 54 patients[21]. Korula *et al*., has also shown similar reports- 70% of patient who had sclerotherapy. Blenkinsopp *et al*., showed that diluting sodium tetradecyl sulphate from 3% to 1% reduce the rate of ulceration with only a minimal decrease in efficacy. Westaby *et al*., compared the effect of sclerotherapy at one weekly interval and at three weekly interval and found that ulceration were common in one weekly interval. In present study, the esophageal ulceration is found to be the most common complication of both banding and sclerotherapy. 6 out of 25 patients had esophageal ulceration who underwent variceal banding while 13 patients of 25 had esophageal ulceration who underwent sclerotherapy. Thus 25% of patients who had banding and about 50% of patients who had sclerotherapy developed esophageal

ulceration. All ulceration were found to be superficial without bleeding.

The higher incidence of ulceration in the sodium tetradecyl sulphate group was probabaly due to concentration used (3%) and also due to ulcerogenic property of sodium tetradecyl sulphate.

RETROSTERNAL PAIN:

Transient retrosternal pain following sclerotherapy can be due to mediastinitis and due to esophagitis. Korula *et al.,* reported an incidence of 24% with 1.5% sodium tetradecyl sulphate. In a study done by Lebski *et al*., the banding is associated with about 86% of patients who underwent the treatment[31]. These results were compared to our study in which about 40% of patients developed retrosternal pain among which 6 out of 25 patients who underwent banding and 11 of 25 patients who underwent sclerotherapy had retrosternal pain.

ODYNOPHAGIA:

In a study by Berner *et al*., 75% of patients had reported a transient dysphagia which lasted upto 24-72 hours after the procedure. This is due to the engorged banded varices. Bargava *et al.,* noted that dysphagia significantly common with sclerotherapy with sodium tetradecyl sulphate[30]. In the present study also, odynophagia is more commonly seen in sodium tetradecyl sulphate group than banding group. The difference was statistically signifcant. Edema and

inflammation around the ulcer contributes to the narrowing of esophagus. This explains why dysphagia is more common in use of sclerotherapy because of its ulcerogenic property. Hence around 4 patients of 25 who underwent banding and 12 of 25 patients who underwent sclerotherapy had dysphagia with the significant 'P' value of < 0.05.

FEVER:

In most of studies, fever lasting for 24-48 hours after sclerotherapy and banding occurred in 20-40% of patients. Fever usually subsided spontaneously. In the present study, fever was seen in 8% of patients who underwent banding and 44% of patients who underwent sclerotherapy with significant 'P' value. Most of the other studies have shown similar report with present study.

TACHYCARDIA:

Tachycardia following banding and sclerotherapy could be due to febrile spikes or anxiety by the procedure. Kumar *et al*., reported tachycardia in 48% of patient with 3% sodium tetradecyl sulphate. In the present study 32% of patients who underwent sclerotherapy had tachycardia while only 8% of patients who underwent banding had tachycardia.

ESOPHAGEAL STRICTURE:

Esophageal strictures are due to healing of deep esophageal ulceration. Bargava *et al*., reported an incidence of 27% of strictures with 1.5% sodium tetradecyl sulphate[30]. Sorensen used 3% sodium tetradecyl sulphate and reported strictures in 35% of patients. He attributed this higher rate to frequent sclerotherapy sessions. Most of other studies reported stricture rate ranging from 1-20%. Laine *et al*., in a meta-analysis of 7 randomized trials involving 547 patients found esophageal strictures in 7 patients[3]. Another study by Laine *et al*., demonstrated a significant reduction in stricture formation in ligation (none) when compared to sclerotherapy (33%)[32]. Low rates of stricture formation have been reported by Baronicin *et al*., due to ligation (11%) when compared to sclerotherapy. Steigmann and Sarin also reported a lower incidence of stricture formation following ligation (2% and 0%)[19]. In the present study, only one patient with stricture has been noted who underwent sclerotherapy four months after the procedure. No patients were found to develop stricture following banding. This is probably due to proper banding technique in a spiral fashion and restricting the sclerotherapy to proximal 10cm of gastroesophageal junction.

7.Summary:

SUMMARY

This study is conducted prospectively on a total of 50 patients with bleeding esophageal varies from September 2011 to August 2012 with the prime aim of evaluating the efficacy and safety of endoscopic variceal banding and endoscopic sclerotherapy.

1. In this study cirrhosis is the most common etiology of portal hypertension accounting for 90% of the study population.

2. 60% of study population were in 31-50 years of age group.

3. In this study more than 70% of old age people presented grade IV than grade III varices.

4. In this study about 45% of patients had signs of active bleeding.

5. In actively bleeding varices, sclerotherapy has a little added advantage over banding because of the technical difficulty of the banding due to obscured field.

6. In this study about 194 bands and 532 ml of sclerosant were used with a mean of 7.76 bands and 21.2 ml of sclerosant per person respectively.

7. In actively bleeding varices the mean number of bands and the amount of sclerotherapy is more compared to that of mean of number of bands and amount of sclerotherapy in non-bleeding patients.

8. In this study more number and percentage of females had complications compared to males though the study population of females is less.

9. 24% of patients had retrosternal pain in banding while 44% of patients in sclerotherapy had retrosternal pain.

10.16% of patients with banding and 48% of patients with sclerotherapy had odynophagia.

11.22% of patients with banding and 44% of patients with sclerotherapy had fever.

12. 32% of patients with sclerotherapy had tachycardia. No patients with banding complained of tachycardia.

13. 24% of patients with banding and 52% of patients with sclerotherapy had esophageal ulceration.

14. 4% of patients with sclerotherapy developed stricture.No patients with banding developed stricture over the follow up period.

15. 12% of patients with banding and 3% of patients with sclerotherapy had rebleeding during and after the procedures.

16. 20% of patients with sclerotherapy had recurrences of varices while no recurrence was seen in banding for the follow up period.

17. 'P' value is significant in complications such as odynophagia, fever and recurrence.

8. Conclusion:

CONCLUSION

- Both banding and 3% sodium tetradecyl sulphate are equally effective in controlling acute variceal hemorrhage among which sclerotherapy has a small advantage and also in preventing rebleeding.
- Both banding and sclerotherpy are effective in eradicating varices but banding is more efficacious
- Both banding and sclerotherapy have their side effects but sclerotherapy has more frequent and dreaded complications.
- Hence banding is superior to sclerotherapy both in efficacy and safety

9. Limitations of the study:

LIMITATION OF THE STUDY

The limitations of this study are:

- The numbers of patients studied were small.
- The period of follow-up of patients was short.
- The compliance of patients for follow-up was poor.
- Non-availability of bands lead to exclusion of some patients.

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11: Annexure

S.NO	NAME	Age	Sex	IP.NO	DIAGNOSIS	GRADE	BLEEDING	NO. OF BANDS PER SESSION			RETRO STERNAL PAIN	ODYNOPHAGIA	FEVER	TACHYCARDIA	ULCER	STRICTURE	REBLEEDING	FAILURE
								I	II	III								
1	Srinivasan	50	M	52652	Cirrhosis	III	+	6	3	1	+	+			+			
2	Neelamani	55	F	57521	Cirrhosis	III	-	5	1	-								
3	Manoranjitham	22	F	59715	EHPVO	IV	-	5	1	-								
4	Ganesh Moorthy	48	M	49443	Cirrhosis	III	+	6	2	1								
5	Chikuthayammal	48	F	60189	Cirrhosis	IV	+	6	3	2	+		+	+	+			
6	Ramathal	48	F	54168	Cirrhosis	IV	+	6	4	1								
7	Kasi	51	M	53238	Cirrhosis	IV	-	5	1	-								
8	Bannari	42	F	11712	Cirrhosis	III	-	5	1	-								
9	Karpagam	28	F	26310	Cirrhosis	III	+	6	2	0								
10	Kalyani	24	F	31521	EHPVO	III	-	5	1	-								
11	Kulalmani	69	F	35761	Cirrhosis	IV	-	5	1	-								
12	Palanisamy	52	M	28633	Cirrhosis	IV	-	5	1	-								
13	Rajendran	42	M	48723	Cirrhosis	III	+	6	2	-								
14	Alvikutty	40	M	55875	Cirrhosis	IV	+	6	4	1	+				+		+	
15	Kumar	40	M	54429	Cirrhosis	III	-	5	1	-								
16	Sivagami	50	F	68952	Cirrhosis	IV	+	6	3	2	+			+	+		+	
17	Kalimuthu	35	M	22986	Cirrhosis	III	-	5	1	-								
18	Raman	55	M	51489	Cirrhosis	IV	-	5	-	-	+		+		+			
19	Neelamani	40	F	53935	Cirrhosis	III	+	5	4	-	+				+			
20	Ravikumar	53	M	38512	Cirrhosis	IV	-	5	-	-								
21	Jaganathan	36	M	32116	Cirrhosis	III	-	5	1	-								
22	Vimal	22	M	44874	EHPVO	IV	-	5	1	-								
23	Vinoth	38	M	42023	Cirrhosis	IV	-	5	1	-								
24	Karthikeyan	26	M	39965	EHPVO	IV	+	6	2	1							+	
25	Lakshmi	55	F	46713	Cirrhosis	III	-	5	1	-		+						

S.NO	NAME	Age	Sex	IP.NO	DIAGNOSIS	GRADE	BLEEDING	3% STD PER SESSION			RETRO STERNAL PAIN	ODYNOPHAGIA	FEVER	TACHYCARDIA	ULCER	STRICTURE	REBLEEDING	FAILURE
								I	II	III								
1	Rajan	65	M	52815	Cirrhosis	III	+	16	9	8	+		+		+			
2	Ramathal	65	F	57643	Cirrhosis	IV	-	13	8	5	+	+	+	+				
3	Chandran	41	M	59812	Cirrhosis	IV	-	10	8	6					+			
4	Zubair	40	M	50617	Cirrhosis	III	+	15	11	8	+	+			+			
5	Ganesh	39	M	61256	Cirrhosis	IV	+	10	10	8								
6	Palani	58	M	55431	Cirrhosis	IV	-	11	7	4								
7	Rukmani	49	F	54521	Cirrhosis	IV	+	15	8	5	+	+	+	+	+			+
8	Muniyandi	52	M	12604	Cirrhosis	III	-	11	5	2	+	+	+	+				
9	Lakshmi	60	F	27213	Cirrhosis	IV	+	16	7	4	+	+	+	+				
10	Poongothai	33	F	32698	Cirrhosis	III	-	10	3	0		+	+	+				
11	Natraj	43	M	36845	Cirrhosis	IV	+	17	9	6	+	+			+			
12	Sundaram	50	M	29119	Cirrhosis	IV	-	11	7	6					+			
13	Sugapriya	24	F	49760	EHPVO	III	+	16	10	9		+			+			
14	Venugopal	65	M	56734	Cirrhosis	IV	+	13	10	5			+	+	+	+		
15	Sarojini	40	F	55926	Cirrhosis	III	-	10	2	0	+	+						
16	Selval	74	F	69183	Cirrhosis	III	-	8	2	3								+
17	Poovathal	50	F	23615	Cirrhosis	IV	-	10	6	4	+	+	+	+				+
18	Chittisekar	33	M	52740	Cirrhosis	IV	+	14	9	3					+			
19	Eswari	40	F	54291	Cirrhosis	III	-	9	5	1			+					
20	Jeyalaksmi	46	F	41572	Cirrhosis	III	-	12	5	3	+	+						
21	Ravi	48	M	34797	Cirrhosis	III	+	15	10	9	+	+			+		+	+
22	Natraj	52	M	45102	Cirrhosis	IV	+	14	12	5			+	+	+			+
23	Niwaz	26	M	43681	EHPVO	IV	-	11	3	1			+					
24	Vasanth	36	M	52739	Cirrhosis	IV	+	10	11	7					+			
25	Thenmozhi	28	F	34065	EHPVO	III	-	10	3	1					+			

ANNEXURE-1

PROFORMA

Serial Number:

Name:
Age : Sex :
Hospital No: Ward :
Address :

D.O.A D.O.D D.O.S

I. PRESENTING COMPLAINTS

Hemetemesis:
Malena:
Jaundice:
Fever:
Abdominal pain:
Abdominal distension:
Altered sensorium:
Pedal edema:
Drug intake:

II. PAST HISTORY

Hemetemesis:
Malena:
Jaundice:
Alcoholism:
Hypertension:

PREVIOUS TREATMENT

III. EXAMINATION

Built and Nourishment: Icterus:
Mental status: Cyanosis:
Pallor: Clubbing:
Lymphadenopathy:
E/o Liver failure:

PR: Temp: RR: BP:

P/A:

Distension: Abdominal veins:

Liver: Spleen:

Ascitis:

Respiratory system:

CVS:

IV. INVESTIGATION:

Hb:	Platelet count:
TC:	Peripherel smear:
DC:	
Blood Urea:	Blood Sugar:
Na/K:	HbsAg:
Sr.Bilirubin:	SGPT:
SGOT	Sr. Alk PO4:

USG of Abdomen:

TREATMENT

Date:
Grade of varices:
No. of columns:
No. Of bands applied:
Amount of sclerosant injected:

COMPLICATIONS:

Retrosternal Pain:

Odynophagia:

Fever:

Tachycardia:

Esophageal ulcer:

Esophageal perforation:

Pleural effusion:

Mediastinitis:

Rebleed:

FOLLOW UP

SESSION	1	2	3	FOLLOW UP	
DATE					
GRADE					
NO. OF COLUMNS					
NO. OF BANDS APPLIED					
AMOUNT OF SCLEROSANT					
COMPLICATIONS					

CONSENT FORM

It has been explained to me in my mother tongue and I completely understand my condition, its related complications and the treatment options available. I have been explained in detail regarding this study- "***A COMPARATIVE STUDY OF EFFICACY AND SAFETY OF ENDOSCOPIC BANDING AND SCLEROTHERAPY IN ESOPHAGEAL VARICES*** ". I hereby give my consent to participate in the above mentioned study.

DATE:

PLACE:

SIGNATURE OF THE RELATIVE
WITH NAME :

SIGNATURE OF THE PATIENT
WITH NAME :

SIGNATURE OF THE WITNESS
WITH NAME :