BLOOD GASES AFTER ENDOSCOPIC INJECTION SCLEROTHERAPY AND BAND LIGATION OF ESOPHAGEAL VARICES

Thesis

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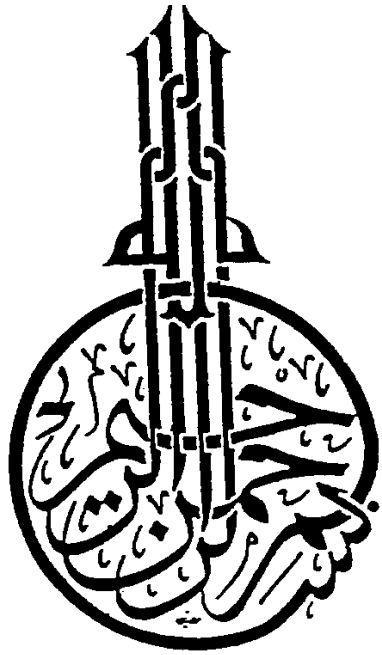
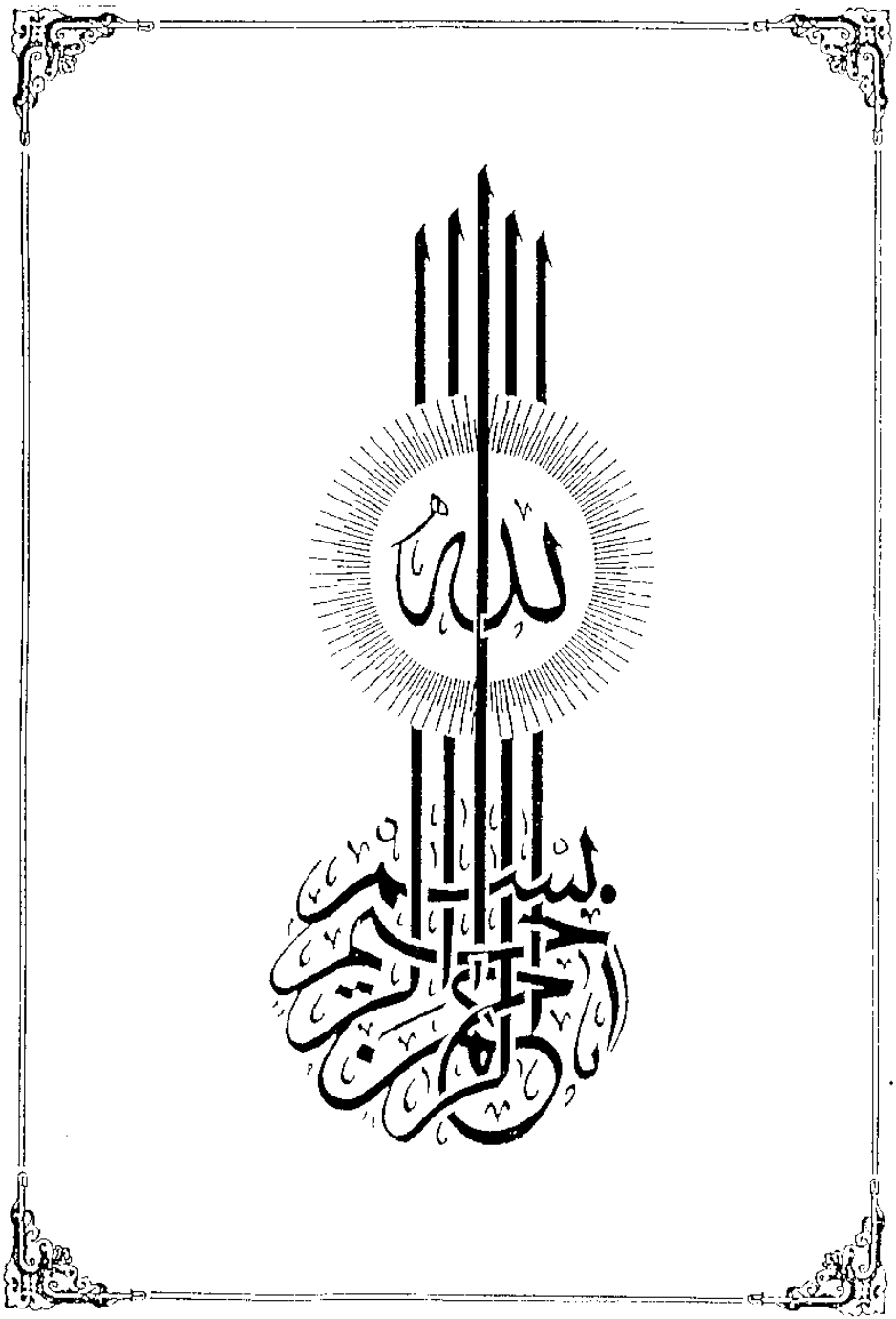


رب إشرح لى صدرى و يسر لى أمرى

و أحلل عقدة من لسانى يفقهوا قولى

((صدق الله العظيم))



CONTENTS

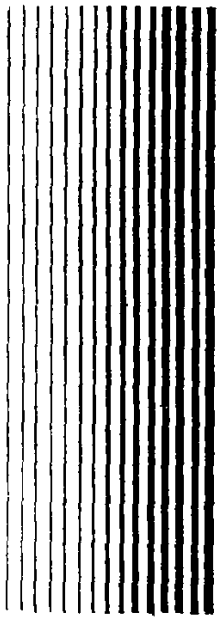
INTRODUCTION	1
AIM OF THE WORK	2
ESOPHAGEAL VARICES	3
PATHOGENESIS OF VARICES FORMATION	4
DIAGNOSIS	13
MANAGEMENT OF VARICEAL BLEEDING	16
INJECTION SCLEROTHERAPY	26
INDICATIONS	27
TECHNIQUE	29
COMPLICATIONS	36
ENDOSCOPIC VARICEAL LIGATION	50
TECHNIQUE	51
COMPLICATIONS	53
BAND LIGATION VERSUS SCLEROTHERAPY	57
ARTERIAL BLOOD GASES	65
UPPER GIT ENDOSCOPY AND BLOOD GASES	71
MATERIAL AND METHODS	72
RESULTS	76
DISCUSSION	92
SUMMARY AND CONCLUSION	103
RECOMMENDATION	105
REFERENCES	106
ARABIC SUMMARY	150

INTRODUCTION

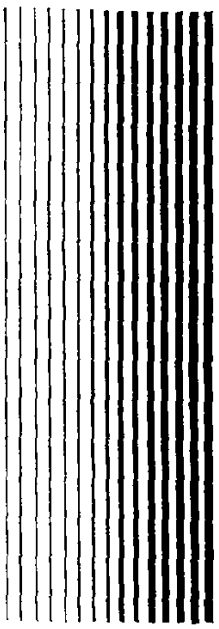
Complications of E.V.L occurs either from the elastic band and its subsequent tissue effects as esophageal ulcerations and stricture or from the use of the overtube as esophageal lacerations , perforation and bleeding ***(Stiegmann et al., 1993)*** .

☯AIM OF THE WORK

The aim of this work is to study the effect of endoscopic sclerotherapy using 5% ethanolamine oleate as sclerosant and band ligation of esophageal varices on blood gases .



REVIEW OF LITERATURE



Esophageal Varices

• Blood supply of the esophagus :

The esophagus is a muscular tube ,25 cm in length . From the cricoid cartilage down to the level of the arch of aorta in the superior mediastinum is supplied by the inferior thyroid arteries , the middle portion by esophageal branches from the descending thoracic aorta , and the lower part by esophageal branches of the left gastric artery *(last , 1973)*

Venous return from the upper part of the esophagus is to the brachiocephalic viens , from the middle part is to the azygos vein and from the lower part is by esophageal tributaries of the left gastric vein Thus , an anastomosis exists in the lower past of the esophagus between portal and systemic venous systems and gives rise to varices in cases of portal hypertension *(Last , 1973)*

There are four layers of veins in the esophagus :

1) Intra - epithelial veins .
2) A superficial venous plexus.
3) Deep intrinsic veins .
4) Adventitial plexus .

The first layer consists of fine vessels running radially within the epithelium of the esophagus and join the superficial venous plexus at right angles immediately below the epithelium.

The deep intrinsic veins lie deep to the superficial venous plexus and constitute three to five main trunks . All esophageal venous channels enlarge , however it is the deep intrinsic veins that become massively enlarged and develop into tortuous variceal channels and displace the superficial venous plexus and lay immediatly below the epithelium . Perforating veins connect the deeper veins with the adventital plexus . *(Kitano et.al. , 1986)*

Varices are usually confined to the lower few centimeters of the

esophagus due to the fact that the veins of the lower 3-5 cms lie chiefly in the lamina propria while the veins of the proximal part are deeply protected in the submucosa but they may develop through the entire length of esophagus *(Spence , 1984)*

It was found that :

52% of varices were present at lower third of the esophagus .
38% were present at lower two thirds of the esophagus .
10% involved the whole esophagus .

(El-Sharkawy et al., 1976)

• Pathogenesis of varices formation :

Many factors which favour the formation of esophagogastric varices are proposed , these include :

I) Portal hypertension . II) Portal systemic collaterals .
III) Hypervolemia . IV) Lower esophageal sphincter .

I - Portal hypertension :

Portal hypertension is due to either :

A) **Increased resistance to blood flow(Backward portal hypertension):**

1) Obstruction of extrahepatic presinusoidal portal venous system :

a) splenic vein thrombosis which can occur as an isolated intity in cases of alcoholic pancreatitis or cancer pancreas . *(Dawson , 1977 and Shwartz , 1979)*

b) portal vein thrombosis *(Sherlock and Dooley , 1993)*

2) Intrahepatic obstruction :

This account for 90% of patients with portal hypertension in most large series . ***Sherlock and Dooley (1993)*** classified intrahepatic obstruction into :

a) Pre-sinusoidal :

- Schistosomiasis .
- Primary biliary cirrhosis .
- Chronic active hepatitis
- Congenital hepatic fibrosis.
- Sarcoidosis
- Toxins : vinyl chloride , arsenic , copper.
- Idiopathic portal hypertension .

b) Sinusoidal :

- Cirrhosis : Viral - alcoholic - primary biliary - autoimmune - metabolic disorders - cryptogenic .
- Non - cirrhotic : acute alcoholic hepatits - cytotoxic drugs - vitamin A intoxication .

c) Post - sinusoidal : - veno - occlusive disease
- Alcoholic central hyaline sclerosis .

3) Post - sinusoidal extrahepatic obstruction ***(Sherlock and Dooley , 1993) :***

1- Hepatic venous obstruction (Budd - chiari syndrome) .
2- Inferior vena caval obstruction .
3- Rise in atrial pressure e.g constrictive pericarditis .

B)Increased blood flow to the liver(forward portal hypertension)

1- Hepatic arterio - portal venous fistula ***(Schwartz , 1979)*** .

2- Splenic arteriovenous fistula :

It is an uncommon entity . Intrasplenic arteriovenous fistula may constitute the pathology of the forward portal hypertension some times reported in cases of Boek's sarcoid , Gaucher disease and myeloid metaplasia ***(Schwartz , 1979).***

3- Idiopathic tropical splenomegaly :

Where splenomegaly may so increase the portal blood flow that the portal radicals within the liver constitute a relative obstruction to the flow and portal hypertension results ***(Banti's syndrome)*** .

Secondary fibrotic changes then occur in the liver around the

portal branches contributing further to the obstruction of the flow *(Dawson , 1977)* .

It is proposed that below a certain level of portal pressure , which would be between 10 and 12 mm Hg over the caval pressure , esophageal varices are usually absent , above this critical level esophageal varices develop , but their size does not depend on the degree of portal hypertension *(Lebrec et al., 1980 and Benhamo , 1982)* and that it had taken months or years for substantial venous enlargement to develop *(Reynolds 1982)* .

II - Portal - systemic collaterals :

The splanchnic portal collaterals are considered to add a further resistence in the portal bed , with consequent dilatation , stagnation and rise of portal pressure *(Khairy ,1960)*

Although collaterals imply portal hypertension , occasionally if the collateral circulation is very extensive portal pressure may fall *(Sherlock and Dooley 1993)*.

The main sites of portal systemic anastomosis according to the frequency are ;

a) At the cardia of stomach & lower esophagus :

Anastomosis occur between left gastric vein , posterior gastric and short gastric veins (portal) with intercostal , diaphragmoesophageal and azygos minor veins (systemic) . Deviation of blood into these channels leads to varices at the lower end of esophagus and funds of stomach .

b) At the anus :

Anastomosis between superior haemorrhoidal vein (Portal) with the middle and inferior haemorrhoidal veins (systemic) . Deviation of blood into these channels lead to rectal varices .

c) In the falciform ligament through the paraumblical veins .

d) Where the abdominal organs are in contact with retroperitoneal tissue or adherent to the abdominal wall e.g veins from the liver to the diaphragm and veins in the lieno - renal ligament and the omentum ***(Sherlock and Dooley 1993)***

These anastomosis may be opened in all the sites and the portal tension would be within normal limits , and if gastroesophageal anastomosis was the only one , varices would be symptomatising and these had been found in 12 % of cases ***(Gheita and Bekheit 1973)*** .

It was found that with a dilated coronary vein , varices were extensive in 84 % of cases ***(Britton 1963)***.

III - Hypervolemia :

In the presence of cirrhosis or bilharzial hepatic fibrosis , there is hypervolemia due to enlargement of the spleen , extensive collateral circulation expanding the vascular bed and the tendency to salt and water retension ***(Hassab, 1964 and Mousa et al., 1964).***

In the presence of increased inflow and obstructed outflow , hypervolemia resulted in congestion of the whole splanchnic area , so with portal hypertension , hypervolemia formed a complicated viscious circle with a further rise in portal pressure ***(Khairy 1966)***.

As a result of stasis , congestion and portal hypervolemia , the submucosal veins of the lower esophagus become varicose and their walls become stretched and thinned out . Atrophy of the connective tissue around them and loss of elastic fibres will follow . The muscularis mucosa is fragmented . The covering epithelium then becomes atrophic or might be absent in some places so that the varices may protrude barely in the lumen of the esophagus ***(Hassab et al., 1968).***

IV - Lower esophageal sphincter :

Submucosal veins may be filled via two pathways , the first

pathway is from opened-up portosystemic submucosal communications as a result of elevated portal pressure above lower esophageal sphincter pressure and perhaps the reason why some cirrhotics had no esophageal varices was their high lower esophageal sphincter pressure *(Miskowiak , 1978)* . The second pathway is from the overfilled periesophageal and epigastric veins which lead to dilatation and incompetence of the valves guarding the perforating veins and consequent backflow of portal blood into the submucosal veins *(Mc Cormack et al., 1983)* . However *Ogle et al., (1978)* found no significant difference in the resting esophageal pressure between cirrhotic patients with varices and cirrhotic patients without varices .

Progressive increase in the size of esophageal varices in cirrhotics who continue to drink alcohol is attributed to its reducing effect on lower esophageal sphincter pressure *(Dagradi , 1972)*. Also it was found that varices become more prominent partly after the administration of anticholinergic drugs which reduce the lower esophageal sphincter pressure *(Dalinka et al., 1972).*

Miskowiak et al ., (1981) proved that lower esophageal sphincter constrictors such as domperidone , metochlopramide and pentagastrin all reduce or even abolish blood flow in the submucosal esophageal varices but not affecting the flow in paraesophageal varices .

The use of metochlopramide as a pharmacological constrictor to the lower esophageal sphincter appeared to offer an effective approach for arresting active bleeding from esophageal varices *(Hosking et al., 1988)* .

• Pathogenesis of variceal bleeding :-

The prediction of when esophageal varices will bleed and why , is still a difficult point *(Sherlock and Dooley 1993)* .

Various theories have been postulated to explain the pathogenesis of variceal bleeding , the two most popular being erosion of esophageal mucosa by acid reflux and rupture due to raised portal

pressur " erosion from without and rupture from within " *(Liebowitz , 1961)*

Factors that may determine the propensity of varices to bleed include:-

a) overlying mucosal changes . b) size of varices .
c) Portal pressure . d) pressure in the varix *(Reynolds , 1982)*

a) overlying mucosal changes :

Varices in the lower third of the esophagus with endoscopically proved erosions will bleed within one year *(Paquet 1979)* on the other hand , *spence (1983)* and his colleagues concluded that a little evidence had been appeared for esophagitis as a major etiological factor in bleeding varices .

Histological examination of esophageal rings removed at transection of bleeding varices revealed dilated intra and subepithelial blood filed channels and had been interpreted as evidence of early esophagitis *(Bessa et al., 1988).*However , another study expressed doubt that the venular dilatation correlates with early esophagitis *(Goldman and Antonioli ,1982)*

These channels have been also found in all patients with variceal bleeding , but in only 50% of non-bleeder *(El-Zayadi et al., 1989)* .

Prolonged venous hypertension may induce an increase in the size of the capillary bed of the lower esophagus and that the increased length of the papillary capillaries had been a manifestation of this rather than of esophagitis and this may indicate increased fragility of intra-epithelial and subepithelial vessels in portal hypertension with subsequent greater tendency to dilatation and rupture *(spence et al., 1984)* .

b) Variceal size:

Lebrec et al . , (1980) reported that variceal haemorrhage is