TREATMENT OF PORTAL HYPERTENSION

ESSAY

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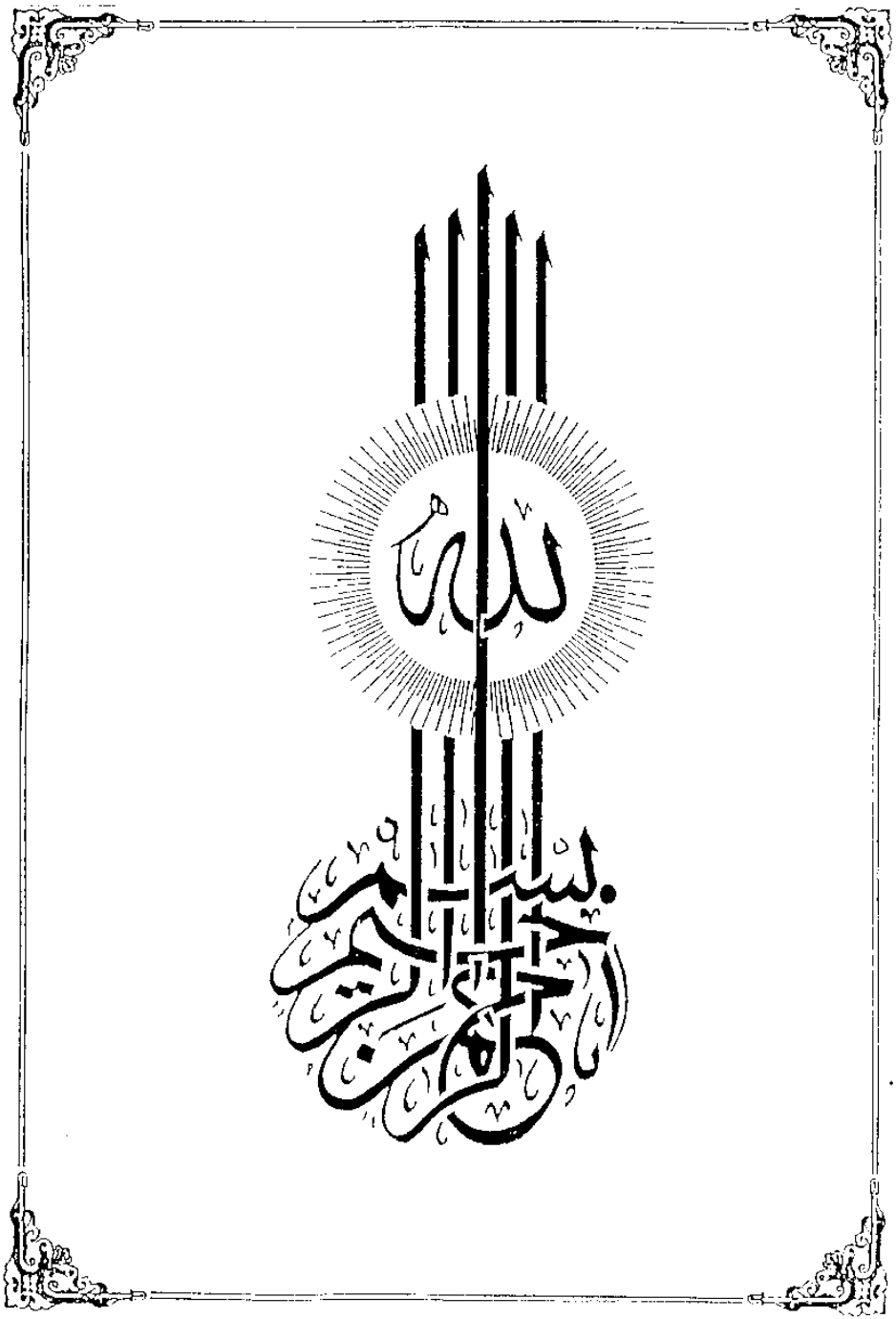

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INTRODUCTION

INTRODUCTION

The treatment of portal hypertension merits a special note and effort among surgeons. The prevalence of the condition in Egypt and the frequent occurrence of varices abroad have attracted the attention of investigators and workers in this field. This work is a sort of a summary of the huge volume of knowledge concerning bleeding oesophageal varices.

The leading Egyptian surgeons efforts are especially referred to.

Treatment of portal hypertension

Managment of acutely bleeding varicer

TREATMENT OF PORTAL HYPERTENSION

The term Portal Hypertension denotes pathologic state with an elevated pressure in the portal venous system.

The diseases in which portal hypertension is significant feature vary from simple vein obstruction to complex entities in which multiple abnormalities are involved, such as cirrhosis of the liver.

One physiologic feature tends to bring these diverse problems together: the development of extraphepatic pathways from the portal to the systemic venous circulation.

There are several regions in which these connections are present and become enlarged in the patient with portal hypertension.

By far, the most significant are those of the portal azygous system which lead to development of gastrooesophageal varices (Warren et al., 1983).

Surgical decompression of portal venous system was performed on an experimental basis by (Nicoli Eck, 1877).

Then Whipple et al (1945) demonstrated the feasibility of shunting procedure.

Management of Acutely Bleeding Varices

Conservative Treatment:

A. Emergency action:

This aims at rapid stabilization of the circulation and establishment of the diagnosis.

- *Reassurance of the patient and complete bed rest.*

Start one or more intravenous infusions with large needles using an immediately available volume expander such as saline, plasma or dextran.

Sedation: *Patient with liver disease is oversensitive to analgesics, hypnotics and sedatives because most of these drugs are detoxified or conjugated in the liver. Morphine is contraindicated. Pethidine or barbiturates may be used in the restless patient: Paraldehyde is the least dangerous, and it is the only one recommended in decompensated patient (Ibrahim et al., 1969).*

- **Oxygen therapy:** *Oxygen inhalation helps in combating the adverse effects of oligaemic shock and also suppresses the reflex dyspnoea following severe bleeding. This minimizes diaphragmatic contractions which are thought to be an important factor in causation and persistence of variceal haemorrhage (Khairy, 1960).*

Vitamin K: *In severe liver disease, when its synthetic function fails, plasma level of both Vit. K-dependent factors and factor V are reduced. So a variety of haemostatic abnormalities can occur. Accordingly Vit. K_1 is administered, fresh blood, fresh frozen plasma, platelet concentrates can be attempted to arrest bleeding (Roberts & Cederbaum, 1972).*

A nasogastric tube is placed in the stomach. The stomach is lavaged with iced saline until the returns are clear. The tube is placed on suction with intermittent lavage with iced saline to remove

blood clots (Wilson, 1973).

Draw blood for determination of: *blood typing, cross matching, haemoglobin and haematocrit as soon as feasible before haemodilution.*

Request an hourly initial blood pressure chart, pulse chart, central venous pressure as its measurement may be of great value to determine accurately the replacement of blood volume, (Jonson, 1964).

Perfect history may attract attention to the causative agent of bleeding. History of bilharziasis or excessive alcohol is searched for. Many stigmata of portal hypertension may be found as enlarged liver or spleen or both, ascites, spider angiomata. The presence of any three of these is strong presumptive evidence of liver disease and portal hypertension (Warren, 1964).

Arrange for emergency endoscopy and emergency barium swallow.

Blood Transfusion:

Reliable intravenous line should be established at once and start blood transfusion without delay.

Blood should be fresh for two reasons:

* *First to correct deficiencies of clotting factors in patients with liver disease.*
* *Second, to avoid ammonia load that banked blood provides it (Rizkallah, 1981).*

According to Wilson, 1973, the objective of blood transfusion is to relieve shock and restore the haematocrit to 35% and haemoglobin to 12 gm/100 ml.

The volume of blood required to accomplish this must be estimated empirically. If moderate shock is present (B.P. 70-90, pulse rate 110-130 and there are clinical signs of hypovolaemia), transfusion equivalent to 25% of normal blood volume will be required for resuscitation. If shock is severe with B.P. below 70, the initial volume of blood transfusion required may be 40-50% of normal blood volume (Wilson, 1973).

The patient should be resuscitated rapidly and comletely. Poor response usually means continued bleeding or inadequate replacement.

When the patient is out of shock, blood transfusion may be slowed and total volume of blood to be given is thendetermined by the course of the disease.

The ideal line for blood transfusion is the central venous line for many reasons (Maingot, 1974):

* *It enables us a rapid transfusion sufficient to bring the C.V.P. to normal.*
* *It enables us to record the C.V.P. as it is a good indicator to the state of venous reservoir and venous return.*
* *It helps in accurate titration of blood loss.*
* *It is an early excellent indication of recurrent bleeding and detection of overtransfusion and congestive heart failure.*

The supraclavicular subclavian vein technique is one that can be readily acquired, using a large size intracatheter aiming to have the tip in the superior vena cava, this catheter is connected to a simple manometer (Maingot, 1974). Normal value ranges between

1-5 cm H_2O. Therefore, if the C.V.P. is low the venous return should be suplemented by intravenous infusion. Any sustained rise of pressure to maximum demands immediate cessation of transfusion.

Determination of adequate replacement: This is very important for making a decision for surgical interference. This can be obtained when these data return to normal values and become sustained:

* Blood pressure and pulse rate.
* Central venous pressure.
* Haematocrit index.
* Haemoglobin.

B. Prevention of Hepatic Coma:

In all patients receiving conservative treatment, or after surgical intervention, measures should be taken to prevent and treat hepatic coma:

Blood protein represents the most significant source of nitrogen. So blood accumulating in the gastrointestinal tract must be evacuated by repeated enemata and laxatives which may be introduced through Sengataken tube (Ibrahim et al., 1969). Pitressin given intravenously to control bleeding has a cathartic effect.

Non-absorbed antibiotics are used with the aim of inhibiting the intestinal flora and lowering the incidence of ammonia intoxication. Neomycin is the best drug. It is given in a dose of 1 gm/4 hours. If a Sengataken tube has been inserted, the content of a Neomycin capsule is added to some water and given through the tube (Ibrahim et al., 1969).

Glutamic acid which combines with ammonia to form glutamine is used once there are signs of impending coma, 25-125 gms of glutamic acid are given daily in divided doses by mouth. For I.V. injection a 25-gm capsule of sodium glutamate is added to one litre of 5% glucose. L-Arginine is superior to sodium glutamate in reducing elevated levels of blood ammonia (Ibrahim et al., 1969).

Lactulose: *This is a synthetic non-absorbable disaccharide unaffected by the intestinal lactase. It is effective clinically and its action is complex. It provides a substrate which favours the lactobacillus, lowers the gut pH and has a purgative effect. It is given in a dose of 10-30 ml t.d.s. It is particularly safe for long term use in chronic hepatic encephalopathy (Sherlock, 1977).*

Urease Inhibitors: *Blood in the gastrointestinal tract and its degradation leads to urea production. Urease acts on this urea and ammonia is produced and absorbed to the blood. Acetohydroxymate (urease inhibitor) proved to lower blood ammonia (Fishbein et al., 1965 and Summerskill et al., 1967).*

Levodopa: *In hepatic encephalopathy, false neurotransmitters closely resembling dopamine, may accumulate in the brain and diminish the response to dopamine. The beneficial action of levodopa may depend on the replinishment of dopamine in the brain (Sherlock, 1977). Levodopa may be used as it has an arousal effect in patients suffering from hepatic coma. Improvement is dramatic but not always constant. (Fisher & James, 1972).*

Active Measures to Control Bleeding

Use of Posterior Pituitary Extract (Vasopressin):

Lowers the mesenteric blood flow and hence the portal pressure by constricting mesenteric arterioles.

Oesophagoscopy performed during the phase of pitressin administration recorded a blanching of mucosa with loss of turgidity and wrinkling of mucous membrane overlying the varices. The effect lasts for 3/4 - 1 hour, a sufficient time for a clot to form at the site of variceal rupture (Ibrahim et al., 1969).

Vasopressin is given as a peripheral intravenous infusion, twenty units diluted in 200 ml of 5% glucose at a rate of 1 unit/mn. The only contraindication for its use is coronary ischaemia as it is a coronary vasoconstrictor.

Recently, Pierre et al, (1984) combines vasopressin with nitroprusside. It was found that nitroprusside inhibited the undesirable cardiovascular side-effects of vasopressin, while maintaining the therapeutic benefits of portal pressure reductions in patients with liver cirrhosis. However, the drug combination deteriorates pulmonary gas exchange which contributes to a reduction in O_2 delivery to the tissues.

Noeije et al. (1983) combined vasopressin with isosorbide dinitrate: 5 mg of isosorbide dinitrate sublingually combined with 0.4 U/min. intravenously.It was found that isosorbide dinitrate reduces effectively portal hypertension in patients with liver cirrhosis, but also decreases oxygen delivery to the tissues as a consequence of fall in cardiac