سامية محمد مصطفى

شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

سامية محمد مصطفى

شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

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جامعة عين شمس

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قسم

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بالرسالة صفحات لم ترد بالأصل

Assiut University
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Sclerotherapy versus propranolol in prophylaxis of oesophageal varices bleeding

Thesis

Submitted for partial fulfillment for Master Degree in Tropical Medicine and Gastroenterology

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2001

ACKNOWLEDGEMENT

First and most of all thanks to GOD, Greater of all for completing this work.

I would like to express may sincere thanks and deepest gratitude to professor Dr. Mohammed Adawy Nafeh, Professor of Tropical Medicine and Gastroenterology, Assiut Faculty of Medicine. His planning, constant guidance and encouragement, together with valuable directions made the accomplishment of this work possible.

I am very grateful to Dr. Yossef Mohammed Swifee, Assistant Professor of Tropical Medicine and Gastroenterology Assiut Faculty of Medicine, for his sincere guidance, continuous help and great aid.

My great thanks to Dr. Osman Abdel-Hameed Osman Assistant Professor of Tropical Medicine and Gastroenterology Assiut Faculty of Medicine, for his support, encouragement and help.

Khairy Hammam Morsy Ahmed.

2001

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Introduction and Aim of the Work

- Bleeding oesophageal varices is one of the most important complications of liver cirrhosis.
- Compared with other causes of upper gastrointestinal haemorrhage it is usually the most serious and often life threatening.
- The risk of first gastrointestinal bleeding in cirrhotics with varices is approximately 30%, while the risk of rebleeding increases to be 70% or more (Burrough et al., 1989 &1992).
- Prophylaxis of haemorrhage from oesophageal varices in patients with liver cirrhosis aim at improving life expectancy by lowering the risk of this life-threatening event.
- In primary prophylaxis of bleeding from oesophageal varices, treatment with beta-blockers is neither an addendum nor an alternative to sclerotherapy, since sclerotherapy is not generally recommended in these patients (Van Ruiwyk and Byrd; 1992).
- Primary prophylaxis using beta-blockers was found in many studies to be effective in reducing the incidence of oesophageal variceal bleeding.
- Endoscopic sclerotherapy is widely used for treatment of haemorrhage from rupture oesophageal varices , both for haemostasis and for prevention of rebleeding (Hashizume et al., 1992).
- Both sclerotherapy and treatment with beta-blockers are effective in secondary prophylaxis of oesophageal varices bleeding.

- ***The aim of the work:***

1- To detect the role of propranolol in prevention of initial bleeding from oesophageal varices (primary prophylaxis).

2- To compare the effectiveness of sclerotherapy and administration of propranolol in preventing oesophageal varices rebleeding (Secondary prophylaxis).

3- To compare the effectiveness of sclerotherapy-propranolol combination and sclerotherapy alone or propranolol alone in secondary prophylaxis of oesophageal varices bleeding.

Portal Hypertension

Portal Hypertension and magnitude of the problem:

Portal hypertension is a frequent clinical syndrome caused by a pathological increase in hydrostatic pressure in the portal venous system. This makes the pressure gradient between the vena porta and the inferior vena cava (portal perfusion pressure of the liver or portal pressure gradient) rise above the normal values (1-5 mmHg). When the portal pressure gradient rises to 12 mmHg or above, complications of portal hypertension can arise. This value is considered the threshold for defining portal hypertension as clinically significant (Bosch et al., 1994).

The importance of this syndrome is defined by the frequency and seriousness of its complications: Digestive haemorrhage through rupture of oesophageal-gastric varices and through hypertensive gastropathy, ascites, hepatic encephalopathy, arterial hypoxemia, disorders in metabolism from drugs or endogenous substances that are normally eliminated by the liver, bacteremia and hypersplenism (Bosch et al., 1994& Burroughs et al., 1991).

Any pathological process that interferes with blood flow at any level within the portal venous system can provoke portal hypertension. According to the anatomical location of these processes we can distinguish portal hypertension of prehepatic, intrahepatic and posthepatic origin. The most frequent causes of portal hypertension is cirrhosis, either alcoholic or related to HVB or HVC infections (which causes more than 90% of portal hypertension cases in Europe), followed by schistosomiasis, which has a high prevalence in Northern Africa and a large part in Latin America (Bosch et al., 1994& Burroughs et al., 1991).

Portal hypertension is the most frequent clinical manifestation of cirrhosis, it is present in more than 80% of cirrhotic patients. At the time of diagnosis of cirrhosis, 50% of patients already have oesophageal varices upon endoscopy (D'Amico et al., 1995). The risk of bleeding increased if the varices have red spots (red whale marks, cherry-red spots, diffuse redness) and if the patients has any sign of clinical decompensation (Child-Pugh score B or C). Those who do not have varices are exposed to a considerable risk of developing them over time, so that 90% of patients who survive more than 10 years will have oesophageal varices (D'Amico et al., 1995). Bleeding from varices is a very serious complication with a mean mortality rate of around 30%. Intensive care treatment becomes necessary for these patients. Among the survivors, the risk of rebleeding is very high, approximately 70% in 2 years. Mortality from variceal rebleeding also average 30%. These figures probably underestimate the real seriousness of variceal bleeding since they do not include those patients whom died of massive haemorrhage before being admitted to the hospital. Bosch et al., (1994) have estimated that in Spain about 6,000 hospital admissions per years occur and 1,800 deaths from oesophageal bleeding Cirrhosis is the fifth leading cause of death in the USA in individuals under the age of 65. The fact that the average age of these patients is 50 years emphasizes the great socioeconomic repercussion of this syndrome.

*Anatomy of the portal venous system:

The portal venous system is formed from veins that carry blood from the abdominal part of alimentary tract, the spleen, pancreas and gall bladder. It is interposed between the capillaries of the splanchnic bed and the capillary network (Sinusoids) of the liver (Romanes, 1972 and Bott, 1985). The main portal vein is formed by the union of the superior mesenteric vein and the splenic vein, just anterior to the head of the pancreas at the level of second

lumber vertebra. Then it extends to the right of the midline for 5.5-8 cm to enter the liver at the porta hepatis . In front of the portal vein the bile duct lies to the right and hepatic artery to the left . It is a valveless vein, about 5.5-8cm long and 1.2 cm in diameter . At the porta hepatis the portal vein divided into right and left branches passing to the right and left lobes of the liver (Bott, 1985, Mahran,1987,Sherlock, 1993). The superior mesenteric vein is formed by tributaries from the small intestine, colon and head of pancreas and irregularly from the right gastroepiploic vein from the stomach. The splenic veins (5-15 branches), originate at the hilum of the spleen and join near the tail of pancreas with the short gastric veins to form the main splenic vein , it receives tributaries from the head of the pancreas and the left gastroepiploic. The inferior mesenteric vein bringing blood from the left part of the colon and rectum usually enters its medial third or the junction between superior mesenteric vein and splenic vein . It receives inferior (sigmoid) and superior left colic veins (Westaby and Wiliams, 1985). The left branch of the portal vein receives the paraumbilical veins running with ligamentum teres through the falciform ligament and connects the portal vein with the veins of anterior abdominal wall, it is also connected with the ligamentum venosum (Romanes 1972).