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Relationship Between Liver Stiffness, Platelet Count, Spleen Size and Presence of Clinically Significant Portal Hypertension

Thesis

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رسالة

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DEDICATIONS

I dedicate this work to my beloved family especially my parents who always show so much care, aid, support and patience.

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سورة البقرة الآية: ٣٢

Introduction

Cirrhosis is the end stage of every chronic liver diseases resulting in formation of fibrous tissue, disorganization of liver architecture,and nodule formation which interferes with liver function and results in portal hypertension which is associated with development of hyperdynamic circulation and complications such as oesophago-gastric varices, ascites, hepatic encephalopathy. Patients with cirrhosis and gastro-oesophageal varices have a hepatic venous pressure gradient during haemodynamic catheterization of at least 0-12mmHg(1).

Oesophagal varices are present at diagnosis in approximately 50% of cirrhotic patients being more common in Child-Pugh class C patients compared to class A patients (85% versus 40%)(1, 2). Denovo formation of varices occurs at a rate of 5% per year, with a high incidence in patients who consume alcohol or with worsening liver function (2).

Once varices form they enlarge from small to large at a rate of 5-12% per year(2) and bleed at a rate of 5-15% per year the greatest bleeding risk is seen in large varices classified as being >5mm diameter and is also influenced by severity as assessed by Child-Pugh score, and presence of red wale markings on varices at endoscopy.

Therefore these factors should also be taken into consideration to classify high-risk varices (3).

Bleeding from oesophageal varices remains of significant clinical important and early diagnosis of varices before the first bleed is essential as studies of primary prophylaxis clearly show that the risk of variceal haemorrhge can be reduced by 50%to about15% for large varices.(4) Current guidelines therefore recommended that all cirrhotic patients should be screened for varices at diagnosis with follow up every 2-3 years for patients without varices and 1-2 years for patients with small varices to assess for enlargement of varices and need for prophylactic treatment.(5)

Upper GIT endoscopy remains the gold standard for screening diagnosis and grading of oesophageal varices also trials to predict oesophageal varices by non invasive methods as:

Physical signs and variables related to liver function:

Child-Pugh classification, serum albumin and prothrombin time, a low albumin and low platelet count were shown to be independent risk factors for prescence varices in a study by Garcia-tsao et al.(6), In a further study by Berzigotti et al, spider naevi, ALT,and albumin were found to predict oesophageal varices.(7)

Variables releated to portal hypertension and hypersplenism:

Thrombocytopenia may occur in portal hypertension-induced splenomegaly, A longitudinal study by Qamar et al demonstrated that the median platelet count at the time of occurrence of varices was 91,000, however no platelet count could be identified that accurately predicted the presence of oesophageal varices and they therefore concluded that platelet count is an inadequant for prediction of presence of oesophageal varices.(8)

To improve the predictive value of platelet count it has been combined with other variables such as spleen size ratio calculated by dividing platelet number by the maximum spleen bipolar diameter in mm as estimated by abdominal ultrasound, There have been a number of studies assessing this first by Giannini et al.in 2003 reported that ratio between platelet count and spleen diameter to be the only independent variable associated with presence of oesophageal varices (9).

Liver stiffness:

Transient elastography is noninvasive technique developed to assess hepatic fibrosis in patients with chronic liver diseases, fibrosis causes an increase in liver stiffness and measurement of this forms the basis of transient elastography which is painless, rapid and easy to perform a wide range of

liver stiffness values have been reported ranging from 2.5 to 75 kpa, being influenced by gender, body mass index, disease aetiology,and presence of necroinflammatory changes (10). As a rough guide normal Transient elastography value are to be 3.8-8 kpa in men and 3.3-7.8kpa in women significant fibrosis 7.8 kpa and cirrhosis 13-17kpa (11).

Aim of the Work

To find a relationship between liver stiffness [LS], LSPS [LS × spleen size/platelet count] and presence of CSPH [clinically significant portal hypertension], and oesophagel varices (detected by upper GIT endoscopy), in compensated cirrhotic patients, and try to correlate it with the grade of oesophageal varices.

Liver Cirrhosis

Definition:

Necrosis of liver cells followed by fibrosis and nodule formation. The liver architecture is diffusely abnormal and this interferes with liver blood flow and function. This derangement produces the clinical features of portal hypertension and impaired liver cell function.(*12*)

Aetiology:

a) Chronic viral hepatitis (B or C)

b) Alcohol

c) Non-alcoholic fatty liver disease

d) Immune:
   - Primary sclerosing cholangitis
   - Autoimmune liver disease.

e) Biliary:
   - Primary biliary cirrhosis
   - Cystic fibrosis

f) Genetic:
   - Haemochromatosis
   - α_1-antitrypsin deficiency

- Wilson's disease
- Galactosaemia
- Glycogen storage disease

g) Vascular:

- Cardiac cirrhosis following right sided failure
- Budd-Chiari syndrome
- Veno-occlusive disease

h) Cryptogenic (unknown).

World-wide, the most common causes of cirrhosis are viral hepatitis and prolonged excessive alcohol consumption. Prolonged biliary damage or obstruction, as in primary biliary cirrhosis, sclerosing cholangitis and post-surgical biliary strictures will also result in cirrhosis. Persistent blockage of the venous return from the liver e.g. veno-occlusive disease and Budd-Chiari syndrome will eventually result in liver cirrhosis.(13), (14)

Epidemiology:

Overall incidence of cirrhosis in the United States is estimated at 360 per 100,000 population or approximately