

Prevalence of Helicobacter Pylori in patients with Portal hypertensive gastropathy and determination of congestive index in portal vein and its collaterals.

Thesis

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Abstract

This study was conducted on twenty patients with liver cirrhosis and PHG, in addition to twenty controls with no evidence of liver cirrhosis or PHG were included in this study. Patients & Controls Subjected to full medical and clinical assessment, complete blood count, liver function tests, serum urea&creatinine, sodium,potassium,abdominal ultrasound&duplex ultrasonography of portal vein, upper endoscopy. H.pylori status were determined by two antral biopsies stained with haematoxylin, eosin,and giemsa stain .

Key Words :

Portal hypertensive gastropathy - portal vein and its collaterals .

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The candidate

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Introuduction & Aim of the work

Introduction

Helicobacter pylori is one of the most common bacterial pathogens in humans. *H. pylori* is now recognized as a world wide problem. It causes chronic gastritis, peptic ulcer disease, and lymphoproliferative disorders and is a major risk factor for gastric cancer (**Crew et al.,2006**).

Portal hypertensive gastropathy defines a pathological endoscopic picture characterized by the presence of alteration of the gastric mucosa found in patients with hepatopathy associated to an initial or evident portal hypertension. The role of *H. Pylori* in the development of these alterations, in terms of prevalence of infection in hepatopathic subjects is still controversial (**Urso et al., 2006**).

Portal hypertensive gastropathy is the most common gastric mucosal injury to patients with liver cirrhosis. The main histological change is that blood vessels in the mucosa and sub mucosa become dilated and twisted, and the vessel wall become thickened. In fact, PHG is the major factor in patients with liver cirrhosis who were accompanied with upper gastrointestinal haemorrhage (**Bayraktar, et al., 1996**).

Aim of the work

The aim of the present study was to ascertain the prevalence of *Helicobacter Pylori* in patients with portal hypertensive gastropathy and determine whether it contribute to the severity of the disease.

Review of Literature

History of Helicobacter pylori Detection

Pathologists have described spiral organisms in the stomach of man and animals since the last century (**Doenges, 1938**).

Steer and Colin, 1975) observed gram-negative curved bacilli in 80% of patients with gastric ulcer in the mucus layer of the stomach.

In Australia, **Warren and Marshall (1984)** described a spiral bacterium as a factor in the pathogenesis of gastritis and peptic ulcer disease. They named it *Campylobacter pyloridis*, as these spiral microaerophilic organisms resemble *Campylobacter* by light microscopy and in guanine plus cytosine content. However, several characters do not really fit the genus *campylobacter* as revealed by later studies depending on the ultra structure and fatty acid profile of this organism (**Goodwin et al., 1989**).

As well as the 16S-subunit ribosomal RNA sequences of these organisms (**Lau et al., 1987**), later on it has been renamed *Helicobacter pylori* because of its helix shape (**Thompson et al, 1988**).

Morphology of H. pylori

H.pylori is a microaerophilic, motile, gram negative curved or spiral bacteria, measuring 3µm in length and about 0.5 µm in diameter, the external wall is smooth, wavy, double layered with 4-6 flagellae arising from one pole of the cell, these flagellae are sheathed with a terminal bulb (**Marshall, 1991**).

The morphology of H.pylori observed by both light and electron microscopy is homogenous (**Mergraud, 1988**).

Epidemiology of Helicobacter pylori Infection

Helicobacter pylori infection is typically acquired in early childhood in both low and high income regions of the world and once established commonly persists lifelong unless treated (**Das et al., 2007**).

Prevalence of Infection

The increase in H.pylori prevalence with age is largely due to a birth cohort effect rather than a late acquisition of infection. In Sao Paulo, Brazil, **Zaterka et al., (2007)**, tested 993 blood donors with no dyspeptic symptoms for H.pylori infection and found a prevalence of 66.5% in men and 63.2% in women.

The prevalence increased with age and was higher in non-white population. The common risk factors, ie crowding, type of drinking water, lack of toilet facilities during childhood, low family income, low educational level and previous gastrointestinal endoscopy were observed. There is considerable difference in H.pylori prevalence between high and low income countries, and concerning children the prevalence ranges from less than 10% to more than 80% - respectively. In low income countries the rate of seroconversion tend to be higher and annual re infection rates after H.pylori eradication in some low income communities have been reported to be as high as 13 -24 %, thus comparable to the incidence in childhood(**kivi et al., 2006**) .

AS noted by **Muhsen et al., (2006)**, socioeconomic and living conditions are major risk factors for H.pylori infection and intrafamilial transmission in early childhood plays an important role.

Celinski et al., (2006), addressed this question by conducting a seroprevalence study in the Lublin region of Poland. The global prevalence was 78.5%. Eighty seven percent of those born in rural areas were infected compared to 78.4 % of those born in small towns and 64% for those born in big towns. A high prevalence was concerning personal hygiene. The percentage of H. pylori positive Subjects neglecting basic hygienic rule sometimes exceeded 90 %.

Transmission Routes

Human are the only known host for H. pylori. Its transmission route is not yet clearly understood. The human stomach is considered as the reservoir of this pathogen, and accepted routes of transmission are the fecal oral route in developing countries and the gastro oral route in developed countries, in which water could be a vehicle (**Das et al., 2007**).

Queralt and Araujo, (2007) studied the survival of H. pylori in a water model using culture, morphology and molecular methods. They showed that H. pylori survives in water but rapidly