Case Report

HENOCH-SCHONLEIN PURPURA ASSOCIATED WITH GASTRIC HELICOBACTER PYLORI INFECTION

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ABSTRACT: This is a case report of a 40 year old patient with Henoch-Schonlein Purpura, who presented with upper abdominal pain, retrosternal burning and palpable purpura over the lower limbs. His upper GI Endoscopy revealed erythematous lesions in the body of stomach and more severe antral gastritis, which was later on proved to be due to Helicobacter Pylori (H. Pylori). The patient was started on anti-H.Pylori treatment with omeprazole, clarithromycin and amoxicillin and responded very well, with remission of symptoms. H.Pylori infection was the most probable cause of Henoch-Schonlein purpura in this patient.

KEYWORDS: Henoch-Schonlein purpura, Antral Gastritis, Helicobacter Pylori

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INTRODUCTION

Helicobacter Pylori is a curved, gram –ve , microaerophillic, oxidase, catalase and urease-positive bacillus[1]. Its serpositivity rate among the general population varies in the different regions of the world and increases with age. The H. Pylori seropositivity rate is directly related to the age, and inversely proportional to the socio-economic class[2]. It is highly adapted to the gastric environment, so that humans and primates are the only species that acquire the infection naturally[3]. The discovery of H. Pylori as a gastrointestinal pathogen has had a profound effect on the current concept of peptic ulcer disease pathogenesis. These bacteria most likely spread from person to person through foecal-oral or oral-oral routes. Since the source of H. Pylori is not definitely known, recommendations for avoiding infection have not been made. An association between H. Pylori infection and various extraintestinal pathologies has also been revealed. In this case report, association between H. Pylori infection and Henoch-Schonlein Purpura is described.

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CASE REPORT

This 40 years old male was admitted with complaints of upper abdominal pain, retrosternal burning and later developing palpable purpura over the lower limbs.

The patient had been well two weeks prior to admission, when he developed localized, epigastric pain of moderate to severe intensity. The pain was burning in nature and episodic in character occurring four to five times a day. Other symptoms were nausea, belching and

retrosternal burning which were temporarily relieved by H2 receptor antagonists. He then started developing rashes on his lower limbs and buttocks which during his admission enlarged to become palpable purpura. During admission he was also diagnosed as hypertensive for the first time. He had no history of breathlessness, arthralgia and weight loss. He was non-smoker and non-alcoholic and married with three children. Family history was found to be unremarkable.

On examination, he was of average height and built, weighing 80 kg. His pulse was 100/bpm, blood pressure 140/100 mm Hg, afebrile at the time of admission. He was not anaemic. There was no lymphadenopathy, oedema or clubbing. On abdominal examination, there was no visceromegaly but tenderness in epigastrium was noted. Respiratory and Cardiovascular examinations were unremarkable.

Investigations revealed Hb. 14.2 Gm% with normocytic & normochromic picture. Total leucocyte count was 9800/cmm with normal differential and ESR was 40mm/1hr. His platelet count was within normal limit. Urine analysis was normal. LFTs showed total bilirubin 0.6 mg%, SGPT 35 U/L, Alkaline phosphatase 450 U/L. Serum urea, creatinine and blood sugar all were within normal limits. Electrolytes were normal. His fasting serum cholesterol was 180 mg%.

Ultrasound abdomen showed fatty infiltration in the liver. Chest X-ray and ECG were normal. The upper gastrointestinal endoscopy showed gastritis in body and more so in antrum. The antral biopsy showed presence of H. Pylori.

He was put on omeprazole, clarithromycin and amoxicillin. His main symptom of abdominal pain resolved and so was purpura.

DISCUSSION

Helicobacter Pylori is one of the most frequent causes of gastrointestinal infections worldwide. The microorganism is not only involved in the pathogenesis of peptic ulcer, it is also involved in the pathogenesis of non-atrophic and multifocal atrophic gastritis[4] and in the risk of developing gastric cancer and gastric lymphoma[5]. It has therefore been included by the World Health Organization among the Type-I carcinogens[6].

In the past, several studies have been carried out on the association between H. Pylori infection and a miscellany of extradigestive diseases, such as immunological, cardiovascular, and various other pathologies[7]. (Table-1)[8]. The mechanisms behind these clinical observations still remain unclear. However, if confirmed, these findings could revise the diagnostic and therapeutic approach to diseases previously considered as idiopathic[9] in nature.

Henoch-Schonlein Purpura is characterized by palpable purpura, colicky abdominal pain, gastrointestinal haemorrhage, arthralgia, and renal involvement, occuring fundamentally after streptococcal upper airway infections[10]. Aetiological investigations are required as a triggering factor is found in approximately half

Table-I: Extraintestinal diseases that have been associated to Helicobacter pylori infection[8]

Cardiovascular Diseases
Arteriosclerosis, myocardial ischaemia
Primary Raynaud's phenomenon
Vascular migraine
Autoimmune Diseases
Sjogren's syndrome
Henoch-Schonlein purpura
Autoimmune thyroiditis
Idiopathic arrhythmias
Parkinson's disease
Ischaemic non-arterial anterior optic neuropathy
Skin Diseases
Chronic idiopathic urticaria
Rosacea
Alopecia areata
Other Diseases
Iron – deficiency anaemia
Growth retardation
Late menarche
Extragastric MALT lymphoma
Hepatic encephalopathy

of the patients. As there were no other predisposing factors for development of HSP in this patient, like upper respiratory tract infection, H. Pylori infection was the most probable causative factor.

Reinauer et al.[11] were the first to report such an association between H.Pylori and HSP. They described a case of 21 year old female with clearing of purpuric manifestations after eradication therapy for H. Pylori. Further cases have been reported by Cecchi and Torelli[12], Machet et al.[13] and Mozrzymas et al.[14] They have reported one case each of clearing of symptoms after H.Pylori eradication therapy. In Pakistan, one such case has been reported[15].

An antigenic similarity between H.Pylori and host antigens could be responsible for auto immunity in some infected patients. The presence of more toxic strains of H. Pylori and the multiplicity of the toxic strains produced, directly or indirectly, in response to the bacterium, besides a genetic predisposition of the individual, are the factors which may enhance the ability of H. Pylori to generate both local and systemic damage[16].

As H. Pylori eradication often leads to the disappearance of or an improvement in some extradigestive pathologies, further well designed epidemiological and controlled intervention studies, with special refernce to Cag A status of infecting strains, are needed in order to identify whether and by which molecular mechanisms H. Pylori may cause extradigestive manifestations[7].

It is emphasized that it would be useful to look for H. Pylori infection in patients with Gastro-Intestinal manifestations of HSP such as epigastric pain.

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