

THE ORIGIN OF YOUR GASTRITIS DISEASE IN CHILDREN

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Annotation: Gastritis, gastropathy, and peptic ulcer disease (PUD), collectively known as acid peptic disease, are often described as a spectrum of the same disease. Although these conditions are more common in adults, their incidence in the pediatric population is clinically significant. Left untreated, gastritis can progress to PUD, which can result in serious complications such as perforation, bleeding, bowel strictures, and obstruction. This article was written from internet sources and as a result of research conducted by scientists, and quotes from them without copyright infringement.

Key words: Gastritis, helicobacter pylori, chronic dyspeptic symptoms.

Studies describing the role of *Helicobacter pylori*, nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and other causes have helped to further expand our understanding of gastritis and PUD in children. Direct visualization and the ability to biopsy with endoscopy have revolutionized the diagnosis and treatment of these diseases.

Chronic dyspeptic symptoms such as abdominal pain, nausea, vomiting, or bloating are often suggestive for the diagnosis of gastritis and gastropathies among pediatric patients. A prompt diagnosis of these entities might prevent life-threatening complications such as peptic ulcer disease or gastric cancer, which develops after a long-time span of chronic gastric inflammation.

Helicobacter pylori (H pylori), one of the most frequent bacterial infection among humans, also represents the main etiologic factors of pediatric gastritis. Several studies reported an association between this pathogen and hematologic disorders, including immune thrombocytopenia. Recent literature data support the screening for **H pylori** infection in patients with idiopathic thrombocytopenic purpura, independently of the presence of gastrointestinal symptoms. An improvement of the number of platelets has been underlined after successful eradication of this bacteria. Still, this positive therapeutic response seems to be related to the pathogenicity of **H pylori** strains, especially the expression of CagA proteins, and therefore to the geographic variability of the infection severity. Hence, Eastern Asian populations diagnosed with thrombocytopenia were more likely proved to experience a benefic response after the eradication of **H pylori** infection.

In patients with **H pylori** positive gastritis, a useful tool for predicting a possible thrombocyte destruction is the assessment of platelet indexes. Thus, mean platelet volume (MPV) values were shown to be higher in patients diagnosed with this type of gastritis, as compared to patients without histopathological evidence of **H pylori**. This phenomenon might be explained by an ongoing process of platelet destruction, initially compensated by a continuous release of platelet precursor cells with an increased cellular volume into the blood flow.

Red blood cell parameters have also been evaluated in correlation with digestive disorders. Therefore, increased red blood cell volume distribution width (RDW) seems to be a predictive marker of different gastropathies, including gastric cancer, gastric ulcer, and chronic gastritis. **H pylori** has been shown to produce the same effect upon RDW, whereas lower values of the hematocrit (Htc), hemoglobin (Hb), and erythrocyte count have been associated with its presence

in adults. However, these changes seem to be reversible after **H pylori** eradication, due to an improvement in serum iron and vitamin B12 levels. A special attention has been given to iron refractory iron-deficiency anemia (IRIDA) in the past years, as **H pylori** infection eradication has been proved to improve the efficacy of iron supplementation in various pediatric studies evolving around this condition. Still, a recent review that summarized the findings of multiple studies and randomized control trials on this matter, underlined the need for higher quality, larger cohort researches to elucidate a possible association between iron deficiency anemia and **H pylori** infection in children.

The intestinal mucosa is composed of 3 layers: epithelium, lamina propria, and muscularis mucosa. Gastritis is described as the presence of inflammatory cells. Gastropathy occurs when there is gastric mucosal damage with no inflammatory cells. (2) Peptic ulcers occur when gastric or duodenal inflammation leads to defects of the muscularis mucosa. The acidic gastric contents, which normally aid in digestion, become corrosive when there is an increase in acid production or a disruption of protective factors. Parietal cells of the stomach produce gastric acid via proton pumps (H⁺/K⁺ ATPase) in response to acetylcholine from vagal efferents, histamine from enterochromaffin cells, and gastrin from G cells. There are several mechanisms to protect the gastric mucosa, including a mucus layer, a pH-neutral buffer zone, an epithelial layer, and a rich gastric blood supply. The mucus layer is composed of mucin secreted by surface foveolar cells. This mucus layer acts as a diffusion barrier and overlies a pH-neutral buffer zone composed of bicarbonate secreted by epithelial cells. Prostaglandin release mediates mucin secretion from surface foveolar cells and bicarbonate release from epithelial cells. Epithelial cells have tight junctions that act as an additional barrier of protection. A rich gastric blood supply redistributes excess protons that reach the lamina propria. Ultimately, peptic ulcers are formed when the damaging factors overcome the protective mechanisms.

This prospective study was conducted on a sample of 151 children aged between 1 and 17 years, who were admitted in a Pediatric Tertiary Hospital from Romania. Patients with chronic dyspeptic symptoms, such as abdominal pain, bloating, nausea, vomiting, or pyrosis were **included** in the study, between February 2018 and October 2019. **The exclusion criteria** were: patients with known chronic illnesses (including previously known hematologic disorders: immune thrombocytopenic purpura, acute lymphoblastic or myeloblastic leukemia, lymphoma, autoimmune hemolytic anemia, minor or major thalassemia), chronic medication which might alter the hematologic parameters, symptoms of other infectious diseases, parasitic infections or weight under 12kg (due to the characteristics of the video endoscope). The subjects were divided into three groups, depending on the histopathological findings of the gastric biopsies:

1. group 1—**H pylori** gastritis,
2. group 2—non-**H pylori** gastritis, and
3. group 3—control group, consisting of patients without any pathological changes.

Laboratory tests included a complete blood count performed by an automated hematology analyzer (Cobas Integra 400 plus automated analyzer, Roche Diagnostics GmbH, Mannheim, Germany), which revealed absolute values of erythrocytes, platelets, leukocytes, and the entire leukocyte formula, as well as platelet and erythrocyte indices, including MPV, Hb, Htc, MCV,

and RDW. The PLR and NLR values were calculate by dividing platelet/neutrophil count to lymphocyte count.

An abdominal ultrasound was performed for each patient, but no abnormalities were found. Moreover, in order to elucidate the etiology of the symptoms, a parasitology stool examination was recommended in every patients with complaints suggesting a parasitic infestation, such as abdominal pain, distension, flatulence, diarrhea, lack of appetite, or perianal pruritus. As functional abdominal pain and dyspepsia could have represented the cause of dyspeptic symptoms in certain cases, only patients with a family history of digestive disorders or with alarming symptoms were included in the study. Therefore, the decision of performing an upper digestive endoscopy was based upon negative abdominal ultrasound and parasitology stool examination (where applicable), positive family history of inflammatory bowel disease, celiac disease, peptic ulcer disease or **H pylori** infection and the presence of alarming symptoms, in accordance to the latest Rome IV criteria: dysphagia, odynophagia, persistent vomiting, hematemesis, melena, involuntary weight loss, delayed puberty, lack of appetite, symptoms persisting for more than 6 months, severe symptoms affecting daily activities, including sleep. Each patient underwent an upper digestive endoscopy with gastric biopsies (at least two samples taken from the antrum and at least two from the corpus) and a complete blood count. The upper digestive endoscopy was performed after a fastening period of at least 10h, with each patient benefiting from a mild sedation with Diazepam, approximately 20min prior to the procedure. All upper digestive endoscopies were performed by a single person using an Olympus gastroscope GIF P30. A microscopic examination was conducted in each case, with the help of Giemsa staining, used for identifying **H pylori**. The severity of gastritis was assessed in concordance with the modified, updated Sydney classification system, depending on the inflammatory modifications, activity, presence of atrophy, intestinal metaplasia and **H pylori** colonization, as described microscopically.

The initial step of examination includes evaluation of vital signs, including weight and height. It is important to determine appropriate growth trajectory. The Z-scores, standard deviations from the mean, for weight-for-height and/or BMI should be calculated. If the patient has a poor growth trajectory or Z-scores less than -1, this might indicate the presence of chronic disease or poor nutrition. There are no focal findings specific for acid peptic disease. Examination of the oropharynx should include evaluation of dentition for caries and eroded enamel that may indicate frequent vomiting or reflux. Pale conjunctiva, tachycardia, or flow murmur may indicate anemia associated with chronic disease or blood loss. Halitosis with regurgitation or dysphagia may indicate achalasia. Examination of the lungs may reveal wheezing, which can be seen from bronchospasm-associated chronic reflux. Abdominal examination should focus on determining areas of tenderness. Hepatomegaly and splenomegaly should also be ruled out. Rectal examination should determine the presence of fissures or rectal skin tags, which may be seen in Crohn disease. Fecal occult blood may also be useful for determining the presence of a gastrointestinal bleed. Potential extraintestinal manifestations of IBD should be noted on physical examination, including joint swelling, skin rashes, oral lesions, and eye abnormalities.

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