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Case Report

Diffuse Duodenal Nodular Lymphoid Hyperplasia in A Young Adult: A Case Report

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Abstract

Background: This is the case of a 25-year-old female patient with no known past medical history presenting for esophagogastroduodenoscopy (EGD) for intractable reflux symptoms. Endoscopy revealed a carpet-like distribution of innumerable sub-centimetric polyps protruding into the duodenal lumen between 2 to 5 mm in size. The mucosa of the duodenal bulb was devoid of any polypoid or nodular features. Similarly, the esophageal and gastric mucosa were normal upon direct endoscopic visualization.

Summary: A previously healthy 25-year-old female patient presented for EGD due to refractory reflux symptoms. Of note, Nodular lymphoid hyperplasia (NLH) was previously thought to be heralding an early manifestation of familial adenomatous polyposis (FAP). Upon endoscopic visualization, innumerable polyps were seen in the second part of the duodenum ranging in size between 2- and 5-mm. Biopsies were taken from the polyps and sent to pathology which revealed reactive lymphoid follicles corroborating a diagnosis of diffuse duodenal nodular lymphoid hyperplasia (DDNLH). Gastric biopsies were normal and negative for Helicobacter pylori.

Conclusion: DDNLH is a form of NLH characterized by a diffuse distribution of polyps in a carpet-like pattern throughout the duodenal mucosa and sparing the duodenal bulb. Biopsy reveals reactive lymphoid follicles. NLH is a common finding in children whereby it exhibits a spontaneous regression. Conversely, NLH is seldom seen in adults and it is usually considered a precursor of mucosal associated lymphoid tissue (MALT) lymphoma.

Keywords: Diffuse Duodenal Nodular Lymphoid Hyperplasia; MALT-type Lymphoma; Germinal Center Hyperplasia

Introduction

Nodular lymphoid hyperplasia (NLH) is usually attributed the term "follicular lymphoid hyperplasia". Previously, NLH had been coined the term "pseudo-lymphoma". However, this nomenclature is no longer endorsed in medical literature [1]. NLH embraces two forms: diffuse and focal [2]. NLH has a predilection for the terminal ileum, colon and rectum and is rarely seen in the duodenum [3]. Although a common finding in children, NLH is exceedingly rare in adults [4]. For instance, gastrointestinal (GI) tract in children is constantly sampling luminal contents and plays a key role in mitigating potential threats. This fact accounts for the hypertrophy of mucosal associated lymphoid tissue following exposure to a viral antigen. This underscores the adaptive immune system in pediatric population [5].

In adults, both immunodeficiency and immune hyperstimulation can trigger the development of NLH. On one hand, in immunodeficiency states, plasma cells fail to reach maturation and accumulate as functionally inadequate plasma cells. This maturational defect halts their immunological activity and results in lymphoid follicles hyperplasia and germinal center hypertrophy [6]. This is commonly seen in patients with common variable immunodeficiency (CVID) and selective IgA (sIgA) deficiency, respectively [7]. On the other hand, immune hyperstimulation of the lymphoid tissue can induce NLH. This stems for an infectious process whereby germinal centers hypertrophy as a result of constant antigenic stimulation, as exemplified in patients with giardiasis [6,7].

Familial adenomatous polyposis (FAP) and Gardner's syndrome can stimulate hypertrophy of the lymphoid tissue of the terminal ileum and thus developing NLH [1,2].

We herein describe a case of a 25-year-old female patient, previously healthy, presenting for EGD due to intractable reflux symptoms. She was found to have DDNLH upon endoscopic visualization. This fact highlights the rarity of the case because DDNLH is exceedingly rare in adults.

Case Presentation

A 25-year-old female patient, previously healthy, sought medical attention for intractable reflux symptoms. She was scheduled for upper endoscopy as part of her medical workup. Patient has no pertinent past medical history nor does she take any chronic medications. She denies any family history of GI malignancies. Her physical exam was unremarkable. Patient denies any weight loss, epigastric pain, and post-prandial nausea or vomiting. EGD revealed normal esophageal and gastric mucosa in the setting of a patulous gastro-esophageal (GE) junction with Hill's grade III. However, innumerable polyps were seen in the second portion of the duodenum distributed in a carpet-like pattern and protruding into the duodenal lumen (figures 1A and 1B). The duodenal bulb was spared. Biopsies were taken from duodenal polyps and sent to pathology for further characterization. Gastric biopsies were also taken. Duodenal biopsies revealed signs of chronic duodenitis along with the presence of reactive lymphoid follicles, corroborating a diagnosis of DDNLH. Gastric biopsies were normal and *H. pylori* was not detected. Patient was diagnosed with DDNLH and was subsequently prescribed a course of proton pump inhibitors (PPI). Her reflux symptoms were most likely attributed to her incompetent GE junction.

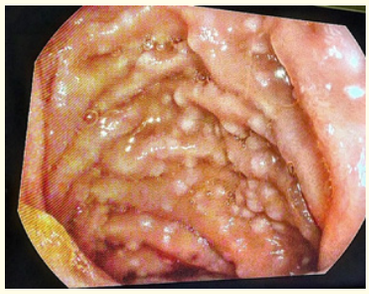

Figure 1A: Diffuse mucosal nodular lesions throughout the second portion of the duodenum. There is partial loss of Kerckring's folds.

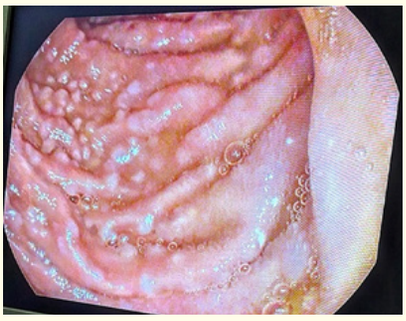

Figure 1B: Numerous sub-centimetric polypoid nodules measuring between 2 to 5 mm protruding from the lumen of the duodenum in a carpet-like pattern.

Discussion

DDNLH is characterized by the presence of innumerable subcentimetric polyps in a carpet-like distribution in the duodenum. The duodenal bulb is usually devoid of polyps or nodularity [1,3]. NLH has a predilection for the pediatric population and is commonly seen in the small intestine, colon and rectum. The terminal ileum is the most commonly affected part of the small intestine. Therefore, DDLNH is seldom seen in adult patients [4,7].

Histologically, NLH is characterized by germinal center hyperplasia and mitotically active lymphoid follicles whereby mantle cell lymphocytes densely populate the lamina propria [6].

NLH has an unclear symptomatology and it is incidentally encountered during endoscopic visualization. NLH can sometimes cause symptoms, such as chronic diarrhea, intestinal bleeding and non-specific abdominal pain [1,4,6]. Our patient solely had reflux-related symptoms most likely attributed to a patulous GE junction consistent with Hill's grade III. To date, there is no clear consensus regarding the etiology of DDNLH. Medical literatures argue that DDNLH can stem from both a state of immuno-deficiency and immune hyper-stimulation [5,7].

CVID and sIgA deficiency exemplify DDLNH caused by immunodeficiency. In light of this, maturation of plasma cells residing in germinal centers of lymphoid tissue in CVID and sIgA is hindered. Therefore, immature plasma cells accumulate in germinal centers and attempt to compensate for the inadequate immune competency [4,5]. Thus, this process results in germinal centers hypertrophy and lymphoid follicles hyperplasia.

Conversely, NLH can result from immune hyper-stimulation. For instance, it has been demonstrated that *H. pylori* infection can induce reactive hyperplasia, and thus NLH ensues [5]. It is worth mentioning that Khuroo et al. evaluated the association between *H. pylori* infection and NLH. They followed patients with NLH over the course of 5 years [5]. Every patient included in their study had *H. pylori*. Complete resolution of NLH was documented following *H. pylori* eradication therapy [5]. This process starkly establishes a causal relationship between *H. pylori* infection and DDNLH.

Celiac disease and giardiasis have been also linked to NLH manifestation [6,7]. In light of a negative *H. pylori* biopsy result, our patient needs to forgo further investigational workup to rule out CVID, sIgA, giardiasis and celiac disease, respectively.

It is worth mentioning that NLH is regarded as a pre-malignant entity for MALT-type lymphoma [7]. However, there are limited data concerning both the rate of transformation from NLH to lymphoma and surveillance strategies [4,7].

In general, NLH found solely in the absence of other abnormalities portends a normal finding [2].

Immuno-deficiency should be ruled-out when a large lesion is noted in the setting of flattened intestinal villi. Mucosal flattening and decreased intestinal surface area are conducive to malabsorptive manifestations [3]. For instance, massive NLH lesions have symptomatic implications, such as chronic diarrhea, intestinal obstruction and weight loss [2,3]. Oral glucocorticoids have proven to be effective in the treatment of symptomatic NLH. When patients exhibit NLH-induced intestinal obstruction, surgical intervention is warranted [3].

A causality between NLH and lymphoma remains a matter of debate.

Conclusion

DDNLH is displayed as innumerable polyps in a carpet-like pattern protruding into the duodenal lumen with polyps ranging in size between 2 to 5 mm. NLH is commonly encountered in pediatrics and usually ensues following an infectious process. Conversely, NLH is an exceedingly rare finding in adults during direct endoscopic visualization. NLH etiology in adults remains unclear and a causal relationship between MALT lymphoma and NLH remains debatable. Thus, this paper should trigger physicians to embark on further studies to elucidate protocolized surveillance strategies and to tailor therapeutic implications when they come across NLH during endoscopic visualization.

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