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Case Report

Gastric Xanthoma in a Pediatric Patient: A Case Report

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Abstract

Gastric xanthomas are cholesterol depositions commonly associated with dyslipidemia. These lesions are generally asymptomatic and are most typically incidental findings on upper endoscopy usually. Gastric xanthomas are infrequently described in children and the true association between incidental xanthoma findings and underlying systemic disease is unknown. Here we present the case of a 10-year-old male with gastric xanthomastosis discovered during workup for nausea, vomiting, upper gastrointestinal pain, and progressive dysphagia.

Keywords: Gastric Xanthoma; Gastric Xanthelasma; Pediatric; Ulcers; Foamy Histiocytes; Nausea; Upper GI Endoscopy

Introduction

Gastric xanthomas are the result of underlying pathology of lipid regulation among adults. However, because pediatric gastric xanthomas are infrequently described in literature, the true extent of the association between incidental xanthoma findings and underlying systemic disease is unknown and additional reporting is needed. This is of great importance with increasing pediatric obesity rates and well described assertions between lipid dysregulation and cardiovascular health risks among adults. We report the finding of a gastric xanthoma in a 10-year-old male with a symptomatic history of upper abdominal pain, nausea and vomiting, and progressive dysphagia first to solids then liquids.

Case Report

A 10-year-old male presented with complaints of nausea, vomiting, vague upper abdominal pain, and progressive dysphagia to solids then liquids. A previous trial of Omeprazole 40 mg once daily failed to alleviate his symptoms. Past medical history was notable for prematurity at 32 weeks gestation. In addition, the child's past medical history is significant for anxiety, attention deficit hyperactivity disorder (ADHD), Tourette's, and seizure disorder. Surgical history was notable for an appendectomy. His weight plotted at the 85th percentile and his height at the 25th percentile for age. The patient and his family denied any history of altered bowel patterns, hematemesis, hematochezia, or melena. The patient lives at home with his mother, brother, uncle, and maternal grandmother. The family deny a history of lipid disorders. The remainder of his physical examination was normal. A barium swallow was normal.

The patient then underwent an upper endoscopy, which revealed a grossly normal esophagus, mild gastritis, and a small polypoid lesion near the antrum. The lesion was pale tan and measured 3 mm in diameter with notable mucosal change at the tip of the lesion (see figure 1). The remainder of the upper endoscopy was normal. Esophageal, gastric, and duodenal histology revealed normal duodenal mucosa and chronic, non-specific, mild gastritis and esophagitis (Figure 3). Lesion histology revealed chronic inflammatory infiltrate with lymphoplasmacytic predominance, infiltration or destruction of isolated glands, and patchy areas of pale-staining foamy histiocyte expansion within the lamina propria, leading to diagnosis of gastric xanthoma (see figures 2). The PAS, mucicarmine, and iron stains are negative. Stains for fungi,

acid fast bacilli, and Whipple's Disease were negative. Additionally, following CD68 IHC stain, histiocytes were diffusely positive with CD68, but negative for CD1a and S100. Rapid urease testing for *H. pylori* was negative, as were fungus and acid-fast bacilli stains. After undergoing endoscopy, the patient was lost to follow-up, and did not return to our gastroenterology clinic for follow-up of biopsy results or undergo lipid testing. The patient presented to a clinic affiliated with our institution several months later with new onset of seizure disorder, but again was lost to follow-up before returning to gastroenterology clinic for evaluation.

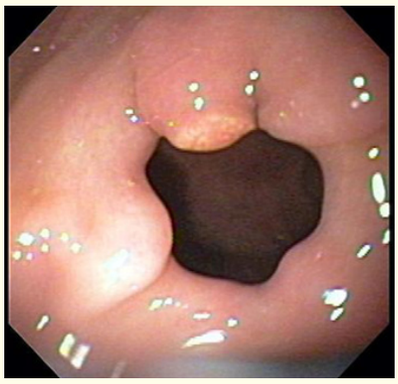

Figure 1: Polypoid lesion near the gastric antrum as visible on upper endoscopy (Image courtesy of Michael Wilsey MD).

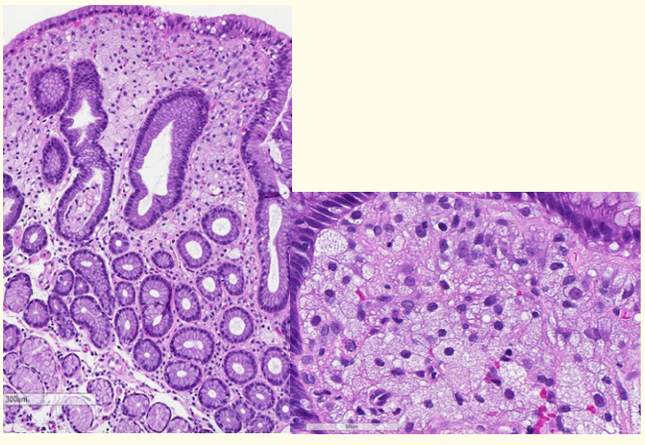

Figure 2 and 3: Histologic examination demonstrating foamy macrophages within the superficial lamina propria, H&E stain, 300 µm (left) and 60 µM (right). (Images courtesy of Wil Chamizo MD).

Discussion

There are few cases of gastric xanthoma reported in literature, particularly among patients below 18 years of age [1-3]. Gastric xanthomas are generally asymptomatic and found incidentally on upper endoscopy usually performed as part of a workup for nausea and vague abdominal pain [1-3]. They are primarily solitary lesions that appear most commonly in the antrum of the stomach, but may also be found within the lesser curvature, body, or fundus and vary in size from less than 5 mm in diameter up to 10 mm in diameter [4-6]. Xanthomas also have a polypoid, plaque-like appearance [7]. Diagnosis is determined by microscopy revealing lipid-laden histiocytes in the lamina propria [4].

The presence of pediatric xanthomatosis heralds underlying pathology and requires thorough evaluation, including genetic evaluation for heritable conditions [8]. Genetic alterations in the endogenous lipid pathway may contribute to xanthoma formation in primary hyperlipoproteinemia. This is commonly seen with syndromes such as familial lipoprotein lipase deficiency although children presenting with this syndrome more typically present with skin eruptions or symptoms of pancreatitis. Both of these were absent in our patient, whose endoscopy and physical exam were negative for further gastrointestinal, cutaneous, or periocular xanthomastosis.

Additional familial syndromes include familial defective apoprotein B-100, familial dysbetalipoproteinemia, and familial hyper-triglyceridemia among others. However, family history for our patient was negative for xanthomas or known lipid dysregulation disorders, making these diagnoses less likely. Because of our patient's past medical history of learning disorders, Tourette's syndrome, and ADHD, we would like to have performed a lipid profile and to have recommended genetic evaluation to investigate the presence of potential lysosomal storage disorders, given the existence of approximately 50 such disorders with varying presentation and inheritance [9]. However, the patient was lost to follow up before biopsy results could be discussed and before a lipid profile or genetic evaluation could take place. Additionally, diagnoses such as diabetes, hypothyroidism, or nephrotic syndrome may be considered as part of the differential for the formation of xanthomas. However, these comorbidities were not present in this case.

Conclusion

Although gastric xanthomas are benign lesions, they suggest the likely existence of potentially serious underlying disorders. This is particularly important among children, where a lifetime of lipid dysregulation has the potential for cumulative damage to the patient's cardiovascular system. Several studies have demonstrated that cardiovascular disease frequently starts in childhood and that early cardiovascular disease has detrimental long-term effects on mortality [10,11]. Due to the relative rarity of pediatric gastric xanthomas, the long-term impact of such findings is not yet under-

stood, and further research is needed on the topic. In this case, our patient presented with an isolated gastric xanthoma despite a family history and past medical history negative for associated disease. It is likely that an underlying genetic disorder affected our patient. However, he was unfortunately lost to follow up before this could be conclusively confirmed. The patient's development of seizure disorder was again concerning for a potential undiagnosed disorder.

This case of a pediatric gastric xanthoma highlights the complexities of appropriate diagnosis and management for this group of patients. When a clinician encounters this patient, it would be reasonable to consider further lipid metabolism and genetic work up for other underlying disorders.

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