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Recurrent Acute Pancreatitis Due to Eosinophilic Gastroenteritis

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aims: Eosinophilic gastroenteritis (EGE) is an unusual affection distinguished by punctual or abundant infiltration by eosinophilic cells in the gastrointestinal tracts, particularly the stomach or duodenum. Clinical aspects are inconstant.
Case Report: We record a case with EGE presenting as coexistent acute pancreatitis with ascites. Laboratory detections demonstrated elevated rate of lipase with elevated blood eosinophil cells numbers. A computed tomography of abdominal and pelvic region checkup appeared a normal pancreatic gland, minimal quantity of ascites in peritoneum and duodenal thickening. Duodenal Biopsies suggested eosinophilic cells infiltration in the lamina propria. This case was effectively treated using prednisolone.
Conclusion: in spite of its atypical circumstance, the discriminational opinion of inexplicable acute pancreatitis should include EGE, particularly if the patient has duodenal edema on imaging or peripheral eosinophilia.

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Keywords: Eosinophilic gastroenteritis; acute pancreatitis; ascites; eosinophilic infiltration.

1. INTRODUCTION

Eosinophilic gastroenteritis (EGE) is an unusual affection distinguished by eosinophilic cells infiltration in the gastrointestinal (GI) tract. Eosinophilic cells may concentrate in some section of the GI tract from upper digestive tract to the bottom. The most generally affected are the upper digestive tract specially stomach or duodenum [1,2]. Conferring to the Klein's classification, 3 distinct affection configurations of EGE may be differenced: mucosal, submucosal or serosal, even if further than 1 segment can be invested [1]. The aspects in clinical examinations depend on which bowel parts or which wall layer are generally invested.

GI Eosinophilic infiltration segments could be responsible for recurrent pancreatitis even though EGE presenting as pancreatitis is highly unusual. This article reports a patient with EGE causing intermittent acute pancreatitis associated with concomitant ascitis, with a debate about the clinical features and discriminational conclusion.

2. CASE REPORT

A 52- year-old woman was received in abdominal trouble, revulsion and vomiting. Without history of medicament allergy or allergic rhinitis. The patient suffered from diabetes under Metformin as well as sitagliptine for 10 years and hadn't been on any other drug lately. She had no history of alcohol consumption. 4 years ago, this patient was admitted to clinic with intense epigastric trouble and vomiting. Acute pancreatitis was retained in view of the clinical characters and the elevation of lipasemia rate in blood. Abdominal CT checkup demonstrated dilated small bowel circles with normal pancreas. On follow-up, the patient did not present intestinal obstacle, but there were edema in intestinal wall. Retrograde biliary catheterization proved edema and condensation of the second duodenum and presence of juxtapapillary diverticulum, but the biliary duts and the pancreatic ducts were normals.

Since this event, she had repeated episodes of abdominal pain and vomiting limiting feeding.

Presently, physical investigation was normal except for moderate epigastric tenderheartedness. Biological examinations suggested a numbers of white blood cells at 7800/ mm3 with high odds of eosinophils cells (19.6%). The biochemistry examination demonstrated a rate of ASAT at 48 IU/ L, a rate of ALAT at 30 IU/ L, a rate of lipase at 678.71 U/ L, and a rate of triglycerides at 69 mg/ dL. Serum immunoglobulin G4 (IgG4) and immunoglobulin E (IgE) ranks were normal. Auto antibodies were negative. Computed tomography of the abdominal and pelvic zones was executed for acute pancreatitis. It showed abundant duodenal wall thickening at the second and third portions, mild bowel edema and a soft quantity of ascites. Still, the pancreas was normal with no proof of peripancreatic fluid collection and no proof of gallstones. We retain the diagnostic of acute pancreatitis Balthazar grade A predicated on the high lipase rate with no modifications of the pancreas on imagery data. Esophagogastroduodenoscopy (EGD) was did to check the duodenal wall edema establish on imagery data. EGD discoveries showed slightly erythematous gastric mucosa and normal duodenal mucosa. Duodenal circles are also typical. Bowel rest and supporting management carrying acid- reducing agents with intravenous fluids were introduced. Lipase de-escalated on the succeeding day and her epigastric hurt gradationally dropped within a 7 days.

Still, moderate abdominal hurt perdured with later biological finding on the 8th medical center day established that eosinophil cells in blood augmented to 35, 9 % and its rate was 4.2 × 103/ µL (standard, 0.05 –0.6 × 103/ µL). Feces check for all infections was negative. Skin prickle test for allergy was negative. At that duration, we allowed of the eventuality of eosinophilic gastroenteritis because the persistence of her symptoms indeed taking treatment and the rate of eosinophilic cells was exacerbated with not any patent other etiology. EGD and colonoscopy were performed additionally specially to take a specimen from the GI tract for histological study. The second view endoscopy showed exacerbated antrum erythema and on the duodenum. Colonoscopy showed modifications of terminal ileum as erythematous mucosal. Biopsies were made from the regular mucosa and from the pathological lesions on multiples emplacement: the upper gastrointestinal tract and ileum. Histological investigation suggested confirmed inflammation with accumulated eosinophilic cells in the lamina propria from the pathological duodenal lesion (Fig. 1). The patient was administered 40 mg of prednisolone for four

weeks. Her symptoms ameliorated directly and the rate of eosinophilics cells regularized within two weeks. Treatment was decrescent over two months. After 12 months, we performed a second EGD, which revealed normal mucosa in antral and duodenal with the eosinophil numbers regularized.

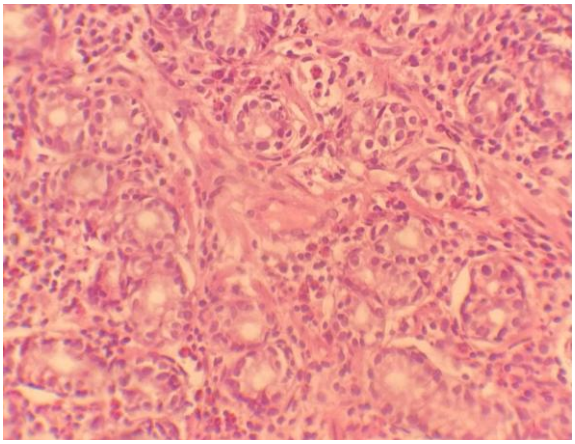

Fig. 1. Histopathology of duodenal mucosa showing dense eosinophilic infiltrate of the lamina propria. (Hematoxylin-eosin stain. Magnification x10)

3. DISCUSSION

EGE is an uncommon affection of unrecognized cause and is characterized as a GI disease of unresolved origin defined by infiltration of eosinophilic cells in the all GI tractus (upper or lower) [2]. Acute pancreatitis due to EGE was registered for the first time in 1973 [3]. Later, many articles described EGE combined with pancreatitis [4]. The conclusion of EGE could be delicate and need an elevated indicator of dubitation since this entity has an extensive range of aspects (clinical and biological). Our patient carried with epigastric hurt, vomiting, and high ranks of lipase. Initially, she also had blood eosinophilia. Still, the diagnosis of idiopathic acute pancreatitis was incorrectly made. We emphasize that EGE should be recall in every case with GI complaints conjoined with high peripheral eosinophilia because it's combined with blood anomaly in practically 30% to 80% of the cases [1]. The final conclusion of EGE is proved by detection of the infiltration by eosinophilic cells in laparotomic, laparoscopic or endoscopy biopsies. In case of EGE, the endoscopic aspects can be polymorphic: ranging from normal mucosa to erythema and ulcerations [5]. In this case, the diagnosis of EGE is based on the histological detections from the mucosal lesions of the upper gastrointestinal tract. Validating the diagnosis additionally requires rejection of other etiology of intestinal infiltrations by eosinophilic cells as food or drug allergy, medicine idiosyncrasy, parasitosis infection, autoimmune diseases, cancer, inflammatory bowel disease, and sprue, etc [2,6]. Since auto immune pancreatitis (AIP) can be combined with high rate of blood eosinophilic cell, with a frequency of 28% [7], it needs to be discerned from the present case. The diagnostic of autoimmune pancreatitis is based on the typical scannographic appearance of the pancreas as well as the blood level of immunoglubulins G4 [8]. Even so, our patient displayed a normal serum rate immunoglubulins G4 and normal pancreas on data computed tomography.

Conferring to the division by Klein, 3 separate affection configurations of eosinophilic gastroenteritis may be differenced mucosal, submucosal and serosal, even further than 1 level can be occupied. The less general figure of this entity is the infiltration of the subserosal layer, charactered by the appearance of infiltration of the gastro intestinal tract by eosinophilic cells and ascites rich in eosinophilic cells [1]. The clinical finding depends on which wall subcaste and which gastrointestinal parts are generally interested. Therefore, mucosal affection may influence in malabsorption, protein-losing enteropathy, and anemia, while the achievement of the muscle subcaste generally provoke intraluminal obstacle [1,2,9]. The infrequent figure is serosal affection with eosinophilic ascites [1,2]. Still, multiple of symptoms are not very specific, similar as vomiting, puking, epigastric hurt, and nausea, therefore doing a opinion may be delicate [1,2]. We believe our case could represent the subserosal type, even though we could not diagnose ascites rich in eosinophilic cells. Although it cannot be demonstrated that it is an ascites rich in eosinophils. The ascites was of low abundance and we could not do an exploratory puncture.

In our case, the important element that made us think of eosinophilic gastroenteritis is the duodenal thickening on imaging with blood hypereosinophilia. Eosinophilic infiltration of the upper duodenal digestive tract in particular can cause edema and deformation of the juxta ampullary region up to pancreatitis. [10], however, we don’t have confirmation of papillitis in this case. Radiological explorations have revealed, whether in our case or in those

reported in the literature, the presence of thickening and duodenal edema in cases of EGE with recurrent acute pancreatitis [10,11]. Thus, the diagnosis of eosinophilic gastroenteritis should be considered in the presence of any picture of recurrent acute pancreatitis or without obvious causes, especially in the event of the presence of duodenal thickening on imaging. In case of idiopathic pancreatitis, the duodenum must be examined by endoscopy, and multiple biopsies performed in the healthy area as in the diseased area to look for eosinophilic infiltration.

Steroids perdure the principal therapeutic in cases of EGE, even though no controlled tests are accessible. Lately, several options to steroids like as ketotifen, suplatast tosilate, sodium cromoglycate and montelukast have been employed [2]. Our department effectively treated this case using steroids firstly. The short and medium term evolution is acceptable. There are no further abdominal pain attacks and the blood eosinophilic rate is normal.

4. CONCLUSION

Eosinophilic gastroenteritis can be allowed as reasons of recurrent acute pancreatitis without flagrant causes establish. Edema and duodenal thickening caused by eosinophilic infiltration is the main pathogenesis of this disease. The treatment ground on steroids remains veritably effective in the medium and long term.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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