Letter to Editor

Acute eosinophilic appendicitis in a case of chronic abdominal pain

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cute appendicitis is one of the most common surgical emergencies worldwide [1]. Although the etiology is multifactorial, direct luminal obstruction mainly by a fecolith is reported to be the primary and principal cause. Acute eosinophilic appendicitis (AEA) was first described in 1997 by Aravindan *et al.* described as a rare variant of acute appendicitis [2]. With more research, they proposed that a type I hypersensitivity reaction may be the underlying cause. Before histopathologic analysis, it may be difficult to distinguish AEA from conventional acute appendicitis because these two conditions are often similar in their clinical presentation, laboratory results, and radiographic features.

A 40-year-old male sought evaluation at the surgical outpatient department due to a 6-month history of diffuse, intermittent abdominal pain. There was no fever, anorexia, or nausea along with the pain. The clinical examination was unremarkable. Hematologic and biochemical tests, including eosinophil count, were all within normal limits. Due to the prolonged duration of the intermittent pain, an abdominal ultrasound was performed, which revealed no abnormalities. Subsequently, an upper gastrointestinal endoscopy and a colonoscopy were performed, both of which yielded normal results.

To further investigate the condition, a contrast-enhanced computed tomography scan of the abdomen was performed, which revealed a concentric, thickened, and slightly enhancing appendix wall, with minimal periappendiceal fat stranding (Fig. 1). The appendix appeared non-opacified and coiled on itself. These radiological findings suggested subacute chronic appendicitis. Based on these findings, the initial diagnosis was subacute chronic appendicitis, and an open appendectomy was carried out. The surgical specimen displayed edematous, congested, and dilated characteristics, with no signs of suppuration (Fig. 2). Histopathological examination identified transmural infiltration of acute inflammatory cells, predominantly eosinophils. Furthermore, eosinophilic infiltration and edema between muscle fibers were observed in the muscularis propria (Fig. 3). No parasites were detected. The histopathological diagnosis

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Figure 1: Contrast-enhanced computed tomography of the abdomen shows concentric mild enhancing wall thickening of the appendix with minimal periappendiceal fat stranding

was conclusively reported as AEA. Subsequently, stool tests for parasites were conducted, all of which returned negative results. The patient was empirically treated with anti-helminthic drugs in accordance with previous reports in the literature. The patient remained asymptomatic and underwent regular follow-up.

Acute appendicitis predominantly affects a younger population with the highest incidence occurring in the second decade of life [3]. Luminal obstruction by a fecolith is one of the primary factors leading to acute appendicitis. Once luminal obstruction occurs, continued mucus secretion and the exudation of inflammatory substances elevate intraluminal pressure, resulting in lymphatic drainage obstruction. Histopathological examination shows inflammatory exudation, characterized by neutrophilic infiltration within the muscularis propria layer [4]. The pathogenesis of AEA remains incompletely understood, with the most widely accepted theory suggesting a type I hypersensitivity reaction or parasitic bowel infection [4,5]. The literature supports the occurrence of infections caused by Enterobius vermicularis or Taenia saginata [6,7], and it is worth noting that the absolute eosinophil count tends to be elevated in most affected patients. An alternative theory proposed by Aravindan et al. suggested that this might represent an early

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Figure 2: Intraoperative picture showing inflamed and dilated appendix



Figure 3: Hematoxylin and eosin with ×40 magnification showing infiltration of muscularis propria with eosinophils

stage in the development of acute phlegmonous appendicitis, potentially representing cases that do not progress to suppuration. Some cases of these lesions have been found to serve as foci for lower gastrointestinal bleeding, as observed by Shrestha *et al.* [8]. In contrast to previously reported cases, our patient is a man in his fourth decade of life who presented without acute symptoms and did not show an increase in total leukocyte count. Only crosssectional imaging could confirm the presence of subacute or chronic appendicitis, which was subsequently confirmed as AEA by histopathological examination.

In conclusion, primary AEA is a rare entity. This unique variant, which occurs in association with chronic abdominal

pain, is extremely rare and can only be definitively diagnosed by histopathological examination of the appendectomy specimen. Therefore, cross-sectional imaging combined with histopathology serves as the cornerstone for the diagnosis of this uncommon clinical condition. Surgery remains the primary treatment modality for primary AEA. Clinicians should consider AEA as a differential diagnosis in cases of abdominal pain in the right lower quadrant, whether acute or chronic.

Consent for Publication

Written informed consent was obtained from the patient for publication of this case report and all accompanying images.

AUTHORS' CONTRIBUTIONS

All authors contributed to the completion of this work. The final manuscript was read and approved by all authors.

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