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Acute eosinophilic appendicitis: a radiologic-pathologic correlation

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ABSTRACT

Inflammation of the appendix is one of the most common conditions requiring emergent surgical intervention. Computed tomography commonly demonstrates a dilated appendix with adjacent inflammation. Traditionally, luminal obstruction of the appendix has been thought to be the primary etiology of appendicitis. However, current evidence suggests that etiology of appendicitis is multifactorial and can involve a number of different pathogenic pathways. Here we present a case of acute eosinophilic appendicitis with radiologic-pathologic correlation from a hypersensitivity reaction pathway. Acute eosinophilic appendicitis may represent an early precursor to conventional acute suppurative (phlegmonous) appendicitis, or a variant form of acute appendicitis.

1. Introduction

Inflammation of the vermiform appendix is one of the most common conditions requiring emergent surgical intervention in the world with a lifetime risk estimated to be 7–8% [1]. Computed tomography (CT) has become commonplace in the management of the acute abdomen, with a sensitivity and specificity for the detection of appendicitis ranging from 88%–100% and 91%–99%, respectively [2]. Acute appendicitis has its peak incidence in the 2nd or 3rd decade of life and is less commonly seen at the extremes of age [1]. The most specific sign for appendicitis on CT has been visualization of a dilated fluid-filled appendix over 6 mm [3]. Other signs on CT include increased enhancement of the appendix after intravenous contrast administration, appendiceal wall thickening greater than or equal to 3 mm, mural stratification of the wall, appendicolith, intramural gas and periappendiceal inflammation/fat stranding (the radiology correlate to gross pathology findings of edema and/or purulent exudate) [2, 3]. However, a subset of patients presents with clinical symptomology for acute appendicitis with non-specific CT findings. Here we present the first radiologic-pathologic correlation for a case of acute eosinophilic appendicitis.

2. Case report

A 40-year-old Latino male presented to our institution with two days of diffuse abdominal pain. He had an uncomplicated, elective hemorrhoidopexy and polypectomy one month prior to admission. His past medical history is significant for stage 2 chronic kidney disease, coronary artery disease with prior bypass grafting, deep vein thrombosis on treatment with factor-Xa inhibitors and allergies to levofloxacin and medical tape. Abdominal examination on admission was benign. He was afebrile and had low-normal blood pressure.

His initial laboratory workup demonstrated a white blood cell (WBC) count of 20.66×10^3 cells/mcL (normal ranges 4×10^3 cells/mcL–10.9×10^3 cells/mcL) with manual differential showing elevation of his neutrophils of 17.6×10^3 cells/mcL (normal 1.9×10^3 cells/mcL–8.6×10^3 cells/mcL). The manual eosinophil count was 190 cells/mcL (normal 0–870 cell/mcL). Stable elevation of his creatinine of 1.19 mg/dL (normal 0.67 mg/dL–1.17 mg/dL) was also noted. Initial workup including influenza swab, urinalysis, electrocardiogram, troponin levels and transthoracic echocardiogram was unremarkable.

A contrast-enhanced CT scan of the abdomen and pelvis was then performed to work up his abdominal pain and evaluate for any post-operative complication related to his recent hemorrhoidopexy and polypectomy. There were no organized fluid collections in the region of recent operative procedures. Of note, the retrocecal appendix was thickened (Figs. 1–3), measuring up to 1.3 cm in maximal diameter (outer to outer diameter) with subtle wall hyperemia as well as thickening of the appendiceal wall up to 5 mm. There were no significant inflammatory changes of the mesoappendix. Given the elevation in white blood cell count and abdominal pain, appendicitis was mentioned by the radiologist as a potential source of his pain.

The patient was treated with intravenous ceftriaxone and metronidazole with improvement in his white blood cell count overnight to 7.66×10^3 cells/mcL (normal ranges 4×10^3 cells/mcL–10.9×10^3 cells/mcL). Given the patient's comorbidities and improving clinical status, the general surgery team elected to treat his

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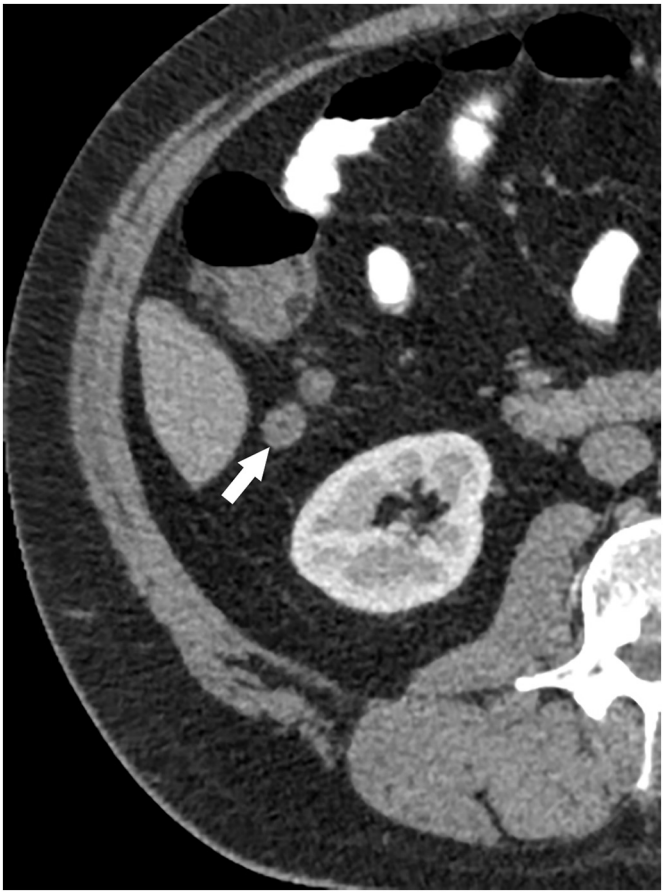

Fig. 1. Transverse contrast-enhanced CT image demonstrates a dilated retrocecal appendix (white arrow) with mural thickening, but without signs of periappendiceal inflammation.

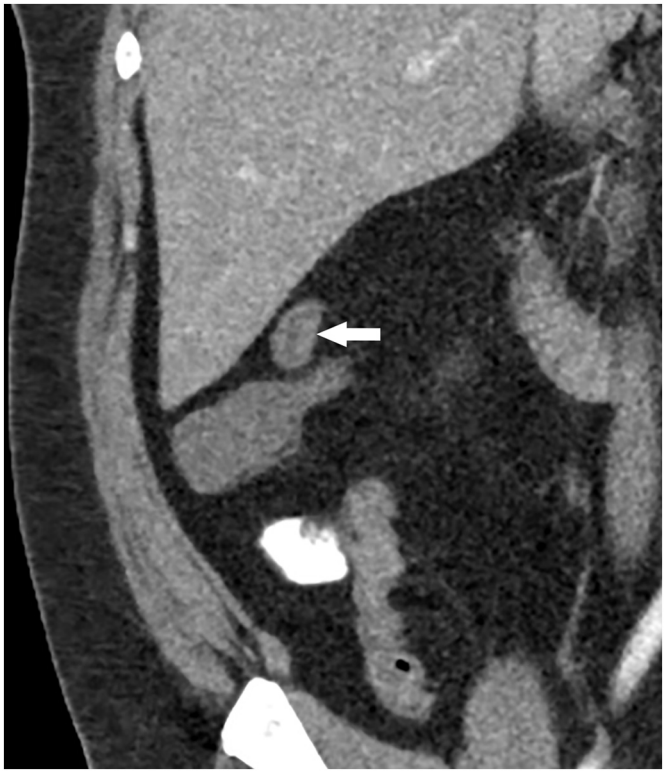

Fig. 2. Coronal contrast-enhanced CT image demonstrates a dilated retrocecal appendix with mural thickening (white arrow).

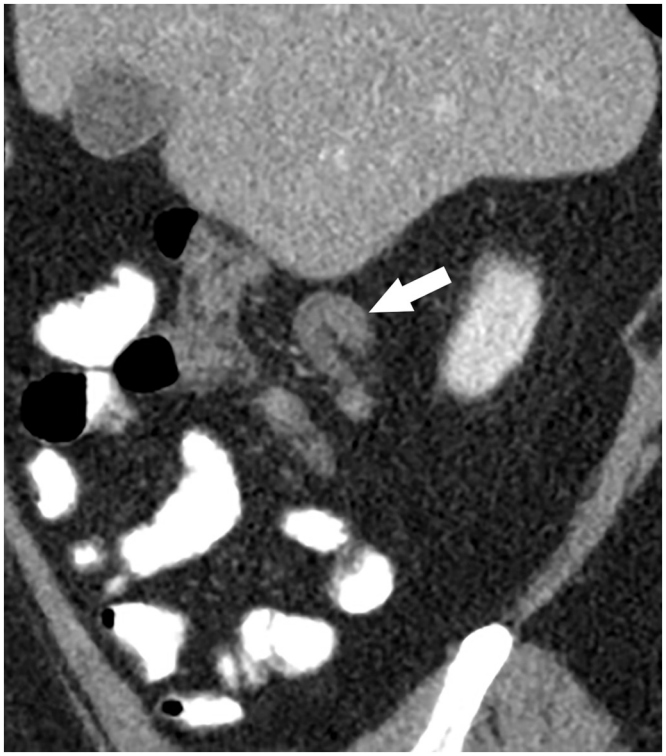

Fig. 3. Sagittal contrast-enhanced CT image demonstrates a dilated retrocecal appendix (white arrow) with mural thickening.

appendicitis non-operatively, and the patient was discharged on amoxicillin. Two days later, the patient had severe worsening right lower quadrant pain and again presented to our institution. A follow-up contrast-enhanced CT did not demonstrate any interval changes. A laparoscopic appendectomy was then performed due to failed non-operative management of the patient's uncomplicated appendicitis.

Gross pathologic analysis showed an appendix 1 cm in maximal diameter with scattered regions of punctuate hemorrhage. A pinpoint lumen was found with no luminal obstruction or appendicolith. No gross perforation was seen. The lumen contained some hemorrhagic content and was occluded in the distal aspect. The appendiceal wall measured 3 mm. Microscopic examination showed infiltration of eosinophils throughout the mucosa, submucosa and muscularis propria. No neutrophils were identified in the muscularis propria. Clusters of eosinophilic microabscesses were identified (Fig. 4). There was no evidence of parasites, ova or granulomas in the appendix. The findings were consistent with acute eosinophilic appendicitis.

Following surgery, there were no complications, and the patient had complete resolution of his abdominal pain. He was discharged in stable condition.

3. Discussion

Traditionally, luminal obstruction of the vermiform appendix by lymphoid hyperplasia, appendicolith, stool, or less commonly neoplasm has been thought to be the primary etiology of appendicitis. Obstruction of the lumen causes distention and increased pressure within the lumen, resulting in ischemic changes in the appendiceal mucosa and subsequent invasion by bacteria [4]. However, luminal obstruction theory appears to be the exception rather than the rule [1]. Current evidence suggests the etiology of appendicitis is multifactorial and can follow a number of different pathogenic pathways [1, 4]. Etiologies could include environmental factors, geographical