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International Journal of Current Research
Vol. 8, Issue, 12, pp.44274-44276, December, 2016

INTERNATIONAL JOURNAL OF CURRENT RESEARCH

ISSN: 0975-833X

RESEARCH ARTICLE

PRIMARY ACUTE EOSINOPHILIC APPENDICITIS: A CASE REPORT WITH LITERATURE REVIEW

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ARTICLE INFO

Article History:
Received 25th September, 2016
Received in revised form
22nd October, 2016
Accepted 18th November, 2016
Published online 30th December, 2016

Key words:

Acute appendicitis,
Eosinophilic appendicitis.

ABSTRACT

Acute appendicitis is a common surgical emergency. The histopathological diagnosis of a classic suppurative appendicitis is usually made by the presence of neutrophils in the muscularis propria. "Acute eosinophilic appendicitis" is a rare variant of acute appendicitis. It clinically mimics acute suppurative appendicitis and is characterised by a grossly inflammed edematous appendix, but with the absence of neutrophils in the muscle layer. The clinical significance of this less understood variant of acute appendicitis is that these patients seldom proceed on to suppuration prior to surgery. However, this entity can be diagnosed only on histopathology of the appendectomy specimen. The histological hallmark of this entity is eosinophilic infiltration of the muscularis layer with accompanying oedema separating the muscle fibres, typically called the "eosinophil- edema lesion". We herein, present the case of a 34 year old male presenting classically as acute appendicitis but was found to have acute eosinophilic appendicitis on histology.

Citation: Dr. Gayatri Deshpande , Dr. Murtaza Akhtar, Dr. Divish Saxena and Dr. Kanav Kumar, 2016. Primary acute eosinophilic appendicitis: a case report with literature review", *International Journal of Current Research*, 8, (12), 44274-44276.

INTRODUCTION

Acute appendicitis is a common surgical emergency and most often appendectomy is performed as the treatment. The histopathological diagnosis of a classic suppurative appendicitis is usually made by the presence of neutrophils in the muscularis propria. Acute eosinophilic appendicitis (AEA) is a rare variant of appendicitis which does not show neutrophils in the muscle layer, instead a marked eosinophilic infiltration is observed. We herein, present the case clinically presenting as classical acute appendicitis, which on histopathology was found to be acute eosinophilic appendicitis. This is a rare variant of acute appendicitis and less understood entity.

CASE REPORT

A 34 year old male was admitted with pain in the right lower quadrant of the abdomen for two days. He did not have nausea, fever or diarrhoea. His medical history was not significant. On physical examination, vitals were stable and there was rebound tenderness at the Mc Burney's point. In his laboratory tests, the total leucocyte count was 12,200 mm with normal eosinophil count. Abdominal ultrasound revealed gross thickening of the caecal wall with surrounding fat infiltration and sub centimetre lymphadenopathy with a blind ending compressible tubular structure in the right iliac fossa.

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Imaging features were suggestive of acute appendicitis. As the clinical and radiological findings were suggestive of Acute Appendicitis, he underwent emergent laparoscopic appendectomy. On laparoscopy, the appendix was extremely edematous (Figure 1). Exudation or suppuration was not observed on the surface of the appendix. Post operative period was uneventful.

HISTOPATHOLOGY

Histopathological examination of the appendectomy specimen revealed an intense eosinophilic infiltration and edema extending upto the serosa. Few congested and dilated blood vessels are seen in the serosa. Histopathological features were suggestive of Acute Eosinophilic appendicitis (Figure 2).

DISCUSSION

Although vermiform appendix is considered to be a vestigial organ, it has a propensity for acute inflammation leading to complications. Reginald Fitz first recognised acute appendicitis as a clinical entity. Later in the 1500s, Charles Mc Burney described the clinical manifestations of acute appendicitis. The peak incidence of acute appendicitis is seen in the second decade of life (O'Connell, 2010). Based on symptoms, signs and diagnostic tests in patients presenting with right iliac fossa pain, acute appendicitis can be diagnosed using a scoring system called the Alvarado scoring system. A score of seven or more is strongly predictive of acute appendicitis (Alvarado, 1986).

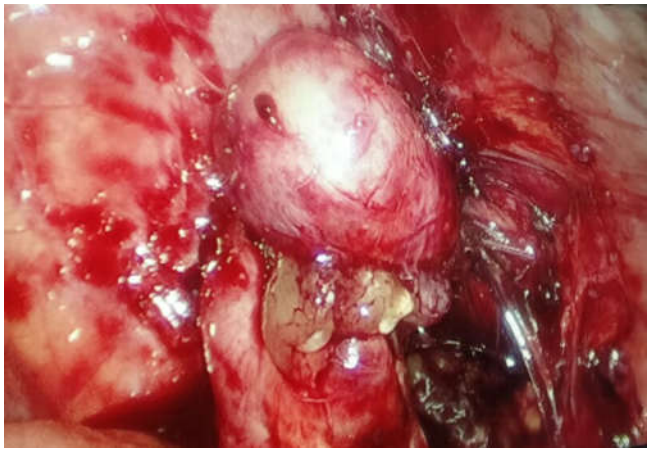

Figure 1. Intraoperative photograph showing edematous appendix

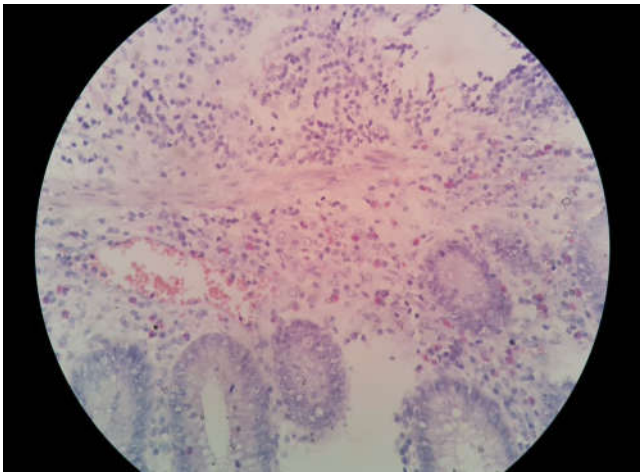

Figure 2. Microphotograph showing intense infiltration by eosinophils. (H and E ×100)

One of the important etiologies of acute appendicitis is luminal obstruction due to faecolith, fibrosis, or stricture. Luminal narrowing may also be due to lymphoid hyperplasia. Once luminal obstruction occurs, continued mucus secretion and inflammatory exudation leads to increased intraluminal pressure resulting in obstruction of lymphatic drainage. Thereby, leading to intense edema, appendiceal gangrene and finally perforation. In a study by Shreshtha *et al*, fecoliths were found in 40% cases of acute appendicitis leading to gangrenous appendicitis with or without rupture in a majority of cases (Shreshtha *et al*., 2012). Bickell *et al* have reported that the rate of appendicular perforation increases by 5% every 12 hour, after a period of 36 hours after the onset of symptoms (Bickell, 2006). This emphasises the need for expedient diagnosis and treatment of this condition (Bhangu, 2015).

The need for histopathological examination of all the appendectomy specimens has long been debated. However, in their study Sinha *et al* have highlighted that histologically unusual pathologies may coexist in appendectomy specimens (Sinha, 2016). Thus, justifying the need of histopathological examination of all appendectomy specimens. Duzgun *et al* found that histopathological examination of the appendectomy specimen may also disclose additional pathological findings that may not be evident on gross examination intra operatively but may affect subsequent clinical management of the patient (Duzgun *et al*., 2004).

Traditionally, suppuration or neutrophilic infiltration has been the pathgnomonic feature of acute appendicitis on histopathology (Liu *et al*., 2004). Eosinophilic gastroenteritis is a known entity, characterised by eosinophilic infiltration of one or more areas of the gastrointestinal tract extending from oesophagus to colon with presence of gastrointestinal symptoms. Although peripheral blood eosinophilia is not a universal finding in eosinophilic gastroenteritis, it occurs in 20-90% of cases (Blackshaw *et al*., 1986; Cello, 1979; Klein *et al*., 1971; Tally *et al*., 1990). Stephenson *et al* described the presence of eosinophilic infiltrate in the muscle layer in appendicitis. They called this entity as 'subacute appendicitis' (Stephenson, 1961).

Jona *et al* described a similar entity in children presenting as acute appendicitis (Jona *et al*., 1976). However, they included these cases in the spectrum of eosinophilic gastroenteritis rather than considering them a variant of acute appendicitis. Primary eosinophilic appendicitis is a condition which primarily affects the appendix characterised by transmural eosinophilic infiltration more than 25 eosinophils per 10 high power field with absence of neutrophils or any other pathology in the wall with no known cause for eosinophilia including drug reaction, parasitic infections and malignancy (Carr, 2002; Rothenberg, 2004). In our case, eosinophils were present in all the layers including muscularis propria of the appendix without eosinophilia in the peripheral smear. Thus, our case fulfilled the criteria for primary acute eosinophilic appendicitis.

The term "Acute eosinophilic appendicitis (AEA)" was first proposed by Arvindan *et al* for a rare variant of appendicitis (Aravindan, 1997). The histological hallmark of this entity is eosinophilic infiltration of the muscularis layer with accompanying oedema separating the muscle fibres, typically called the "eosinophil- edema lesion"(Aravindan *et al*., 2010). It has been observed that eosinophils mixed with lymphocytes without edema may also be seen in cases of resolving appendicitis (Ciani *et al*., 2000). However, the presence of the characteristic eosinophil- edema lesion helps to differentiate the cases with uninflammed appendices from primary AEA (Aravindan *et al*., 2010). The pathogenesis of primary AEA is largely unknown. The theory of allergic etiology for AEA has been suggested (Weiss, 2002).

Aravindan *et al* suggested that eosinophilic infiltrate seen in acute appendicitis is an early event linked possibly to type I hypersensitivity (Aravindan, 1997). One of the hypothesis is the association of parasitic infestation as a cause of acute appendicitis. The reported incidence of *Enterobius* infestation in patients with symptoms of appendicitis ranges from 0.2% to 41% (Aydin, 2007). However, Sterba *et al* have reported that appendicitis due to pinworm infestation is not associated with high eosinophil infiltration of the muscular layer (Stěrba and Vlcek, 1984). It has also been hypothesised that primary AEA may merely be those cases that do not proceed on to suppuration (Aravindan, 1997).

Conclusion

Primary acute eosinophilic appendicitis although rare, is an existing entity. This rare variant of acute appendicitis can be diagnosed only on histopathology of the appendectomy specimen. Hence, surgery is the mainstay of treatment of primary acute eosinophilic appendicitis.

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