Acute Primary Haemorrhagic Omental Torsion Mimicking Perforated Appendicitis: An Unorthodox Surgical Paradox

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ABSTRACT

Acute primary haemorrhagic omental torsion is an atypical and deceptive cause of acute abdomen that could closely mimic a myriad of intra-abdominal catastrophes, especially perforated appendicitis. The author reports a 30 years man who had presented with gradually worsening right-sided abdominal pain of 2 days duration. Laboratory work-up and abdominal radiographs were inconclusive. Abdominal sonography detected presence of free fluid in the pelvic cul-de-sac. Based on clinical and sonographic findings, presumptive diagnosis of perforated appendicitis was made and the patient was explored through extended Rockey-Davis incision. About 500 - 700 ml of dark-coloured blood (haemoperitoneum) was present in the peritoneal cavity and the pelvis secondary to acute haemorrhagic omental torsion. The appendix was grossly normal. Omentectomy and prophylactic appendicectomy resulted in uneventful recovery of the patient. Acute primary omental torsion is an uncommon pathology that must be kept in mind during differential diagnosis of acute abdomen, especially acute or perforated appendicitis.


INTRODUCTION

First described by Eitel,1,2 acute primary haemorrhagic omental torsion (PHOT) is an unusual cause of acute abdomen. It can mimic a number of abdominal emergencies, especially perforated appendicitis and represents < 0.1% of all the laparotomies performed for acute abdomen.1-3 Despite frequent use of sonography and computed tomography, an irrefutable diagnosis of PHOT is invariably achieved intraoperatively.2,3 More often than not, the underlying aetiology of omental torsion remains unsettled even after meticulous abdominal exploration (primary omental torsion) except in a handful of the cases where it is secondary to other intraabdominal pathologies (secondary omental torsion). Its rarity, suddenness in onset, non-specific symptomatology and the indecipherable mimicry of other acute abdominal conditions can pose perplexing diagnostic and therapeutic dilemmas even to the astute clinicians.

The prime rationale of reporting this case scenario is to apprise the healthcare professionals with aetiopathogenesis, clinical features, diagnostic work-up, and therapeutic strategies of PHOT.

CASE REPORT

A 30 years man presented with abdominal pain, nausea and vomiting of 2 days duration. Pain originated in the epigastrium and subsequently became localized in right lower quadrant (RLQ) of the abdomen. To begin with, it was mild to moderate in intensity but reached the crescendo within a couple of days. It was non-colicky in nature and had no relation with food intake. There were no aggravating or relieving factors. Pain was so excruciating that even strong narcotic analgesics failed to mitigate its intensity and intractability. The patient denied history of previous abdominal surgery.

On general physical examination, the patient exhibited haemodynamic stability and normothermia. Systemic examination was unremarkable. Abdominal examination revealed marked tenderness in RLQ of the abdomen while the rest of the abdomen showed mild non-specific tenderness. Bowel sounds were normal. Digital rectal examination showed no anorectal abnormality. Laboratory work-up revealed leukocytosis (15000/mm3) and raised C-reactive protein (125 mg/dl). Abdominal radiographs were indecisive. Based on clinical suspicion and laboratory data, a tentative diagnosis of perforated appendicitis was made and the patient was explored through extended Rockey-Davis incision. About 500-700 ml of dark-coloured blood (haemoperitoneum) was present in the peritoneal cavity and the pelvis secondary to acute haemorrhagic omental torsion. The appendix was apparently lily-white.
Prophylactic appendicectomy was done to avoid future diagnostic ambiguities. Postoperative course of the patient was uneventful. Histopathology of the resected omentum was consistent with acute omentitis (epiploitis) with haemorrhagic necrosis.

**DISCUSSION**

Acute primary haemorrhagic omental torsion is a deceitful clinical rarity that can notoriously mimic a multitude of acute abdominal conditions, particularly acute or perforated appendicitis. Although no age is exempt; PHOT predominantly afflicts the middle-aged population in their 4th and 5th decades of life with slight male preponderance.1-3

Pathologically, omental torsion (OT) is categorized as primary and secondary. Primary omental torsion occurs without any identifiable underlying aetiology (idiopathic). However, a number of omental anomalies like bifid omentum, accessory omentum, redundant omentum, omental adiposity and narrow omental pedicular attachment can predispose to OT.2,3 Nevertheless, a legionary of precipitating factors have been identified that could trigger the onset of OT. These factors include sudden rise in the intra-abdominal pressure due to forceful sneezing, violent coughing, straining at urination or defecation, heavy weight lifting, strong sexual activity, vigorous physical exercise, bicycle racing, forced reduction of obstructed hernia (omentocele), abdominal trauma, obesity or hyperperistalsis due to over-eating or over-purgation.2 In this patient, no precipitating or predisposing factor was discernible. PHOT is more frequent on the right side (90%) like in this case; probably due to the longer and bigger size of the omentum on right side.3 Conversely, secondary omental torsion occur as an aftermath of postsurgical adhesions, internal and external herniations, malignancies, cysts, and a sundry of inflammatory conditions. Pathogenically, OT is a sort of omental volvulus along its longitudinal axis in a clockwise direction. It is distal mobile part of the omentum that undergoes contortions around its pedicle. Depending upon severity and number of pedicular twists, initially venous return followed by arterial supply is jeopardized. Resultantly, the distal omentum becomes edematous, swollen and haemorrhagic with exudation of serosanguineous fluid into the peritoneal cavity. As the process of torsion advances, it may eventuate into omental infarction and gangrene with or without autoamputation.2-4

Clinically, PHOT present with abdominal pain, anorexia, nausea and vomiting. As progression of torsion is gradual, symptoms are less intense in the beginning causing significant delay on behalf of the patients before seeking medical advice and treatment. Characteristically, the pain of PHOT is out of proportion to the objective clinical signs. Abdominal palpation may reveal vague tender mass on right side of the abdomen with or without peritonism. Although vast majority of the patients suffers from single episode, recurrent attacks of OT with spontaneous derotation are not infrequent. Outwardly, the patients of OT look less systemically unwell as compared to those suffering from other abdominal catastrophes. An infallible pre-operative diagnosis of PHOT is well-nigh impossible due to its nebulous clinical manifestations. Common surgical catastrophes that must not be forgotten in differential diagnosis of PHOT include acute appendicitis, acute cholecystitis, caecal diverticulitis, Meckel's diverticulitis, epiploic appendagitis and adnexitis.5,6 Laboratory data apart from showing leucocytosis remains non-contributory. Likewise, abdominal skiagraphs are of no diagnostic value. Nonetheless, advent of modernistic imaging modalities, especially computed tomography has displayed reasonable success in pre-operative detection of PHOT. Abdominal sonography is operator-dependent imaging modality and has a limited sensitivity in this regard. However, sonography may detect free fluid in the peritoneal cavity. Contrarily, CT scan has emerged as the imaging modality of choice for elaboration of aetiology of acute abdomen. CT scan may show omental torsion as a well-demarcated, heterogeneous, hyperattenuating, cake-shaped inflammatory mass with interspersed hypoattenuated areas of omental necrosis and water-dense spiral streaks (whirling sign) in the periumbilical area. Notwithstanding, a precise diagnosis of PHOT is invariably established intraoperatively during laparotomy, laparoscopy or appendectomy.2,5-7
The mainstay of management of PHOT is surgical resection of the infarcted portion of the greater omentum (omentectomy) by open or laparoscopic technique. Now-a-days, cases of PHOT are being managed conservatively if accurate pre-operative diagnosis is established. Expectant management based on intravenous fluids, analgesics and prophylactic antibiotics (if necessary) usually results in amelioration of the symptoms within a couple of weeks. The cases of OT being managed expectantly must be kept under close clinical surveillance as OT is not always an innocuous and self-limiting disease. Surgical intervention becomes inevitable in case of diagnostic uncertainty, intractable symptomatology, persistent or worsening peritoneal findings, or onset of dreaded complications like intraperitoneal adhesions, intestinal obstruction, omental gangrene, abdominal abscess, peritonitis, septicaemia or septic shock. As PHOT is usually discovered intraoperatively, many surgeons outrightly advocate surgery as therapeutic modality of first choice instead of lingering on with the patients on conservative management with everlasting threats of development of lethal complications. Laparoscopic omentectomy has proven unprecedented supremacy over its conventional (open) counterpart; not only in being minimally-invasive technique but also in providing a golden opportunity for global visualization of the whole coelomic cavity for scrupulous exclusion and management of any synchronously encountered cause of acute abdomen during the indexed session.  

REFERENCES


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