Abdominal apoplexy: A rare case of spontaneous rupture of the superior mesenteric artery in a hypertensive patient

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ABSTRACT

INTRODUCTION: Spontaneous rupture of an intra-abdominal visceral artery is an exceptionally rare and potentially fatal cause of abdominal apoplexy.

PRESENTATION OF CASE: We present a case of a 54-year-old hypertensive male who developed hypovolemic shock in our Emergency Department after presenting with abrupt onset of abdominal pain and diarrhea. Intra-operative findings revealed rupture of the superior mesenteric artery with massive hemoperitoneum. The bleeding vessel was ligated and the patient made a full recovery after 3 weeks in the Intensive Care Unit.

DISCUSSION: High index of suspicion is necessary for early preoperative diagnosis and must be considered in any patient with a history of hypertension presenting with abrupt abdominal pain, signs of peritoneal irritation and unexplained hypovolemic shock. Immediate resuscitation and prompt surgical control of bleeding is paramount in patient prognosis.

CONCLUSION: The seemingly unpredictable nature of abdominal apoplexy must be noted, a precipitating cause in most cases is untraceable and early diagnosis relies solely on awareness of the condition.

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1. Introduction

Intra-peritoneal hemorrhage due to any cause is potentially life threatening, it is especially daunting when presented with no discernible source, leading to a delay in diagnosis. Intra-abdominal apoplexy, more recently termed idiopathic spontaneous intra-peritoneal hemorrhage refers to the rupture of an intra-abdominal visceral vessel in the absence of trauma or underlying pathology, leading to massive hemoperitoneum. Although the condition is extremely rare and the real incidence is unknown abdominal apoplexy should be considered a differential diagnosis in a patient with a history of hypertension presenting with signs of acute abdomen and hemodynamic instability. Early diagnosis, immediate resuscitation and prompt surgical intervention are decisive factors in patient outcome.

2. Presentation of Case

A 54-year-old Pakistani male presented to the emergency department with abdominal pain and diarrhea for 15 h. He was driving home during the initial onset of pain, with no history of trauma or other precipitating factors. The pain was continuous, localized to the epigastrium, colicky with no radiation, and initially moderate in severity. He also passed 9–10 loose watery stools, with no associated relief in pain. There was no history of fever, vomiting, nausea, or anorexia. Past medical history was significant for hypertension for over 22 years, non-compliant to antihypertensive medication, untreated dyslipidemia for 6 years and gastroesophageal reflux disease with Barrett’s esophagus for 5 years. He was a non-smoker and did not consume alcohol.

During initial triaging at 11:18 his blood pressure (BP) was 229/126 mmHg, heart rate (HR) 130/min, respiratory rate 20/min and temperature 37.7 °C. On examination two hours later by the emergency physician, he was conscious, oriented, with a BP of 199/138 mmHg and HR 110/min. He appeared dehydrated and was started on 1 L of Ringers’ Lactate and captopril, with a working diagnosis of hypertensive urgency with acute gastroenteritis. Abdominal examination revealed only mild tenderness in the right upper quadrant. Laboratory data showed a white cell count of $10.5 \times 10^3/\mu$L, hemoglobin 16.9 g/dL, hematocrit 50%, platelet count $231 \times 10^3/\mu$L. An abdominal plain X-ray, serum lipase and amylase, n-dimers, cardiac enzymes, urea/creatinine/ electrolytes, urinalysis and liver function tests were all within normal limits.

At 15:45 the patient became diaphoretic and presyncopal, with BP 76/49 mmHg. He was given a crystalloid bolus and BP picked up to 127/65 mmHg. He began complaining of severe epigastric pain and on re-examination a mass was palpable in the right upper quadrant. He then became drowsy, diaphoretic with cold, clammy skin and BP dropped to 80/64 mmHg with Glasgow Coma Scale 14/15. FAST (Focused Assessment with Sonography for Trauma) at this time was equivocal. Fluid resuscitation with crystalloids was followed by 1 unit of packed red blood cells, after

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Fig. 1. Preoperative CT (coronal view) showing fluid collection at the level of the Superior Mesenteric Artery, around liver and extending to the right paracolic gutter and pelvis.

which his BP stabilized to 117/82 mmHg. He was sent for urgent abdominal computed tomography (CT) with intravenous contrast enhancement (Fig. 1), which showed: localized fluid collection in the intra and retroperitoneal region of the distal part of the stomach and proximal duodenum with stranding of mesenteric fat; a free pelvic collection was also noted with a focal cystic lesion in the posterior segment of the right lobe of the liver showing nodular peripheral enhancement, suspected hemangioma (Figs. 2 and 3).

Shortly after the CT the patient became confused, irritable and dyspeptic. He was intubated and received another unit of packed red cells. On examination by the surgeon on-call at 19:00 he was in profound shock, intubated and on inotropic support; BP was 78/40 mmHg, HR 48/min and his abdomen was hugely distended. Abdominoceustesis was positive for blood and he was immediately shifted to the operating theatre.

Emergency exploratory laparotomy revealed 4000 ml hemoperitoneum with fresh blood and clots and a large hematoma at the root of the mesentery. Further exploration revealed active arterial spurting from three small approximately 2 mm tears of the superior mesenteric artery, the vessel appeared grossly normal with no signs of aneurysmal changes, suture ligation was done with successful hemostasis. The liver was mobilized completely to locate the hemangioma seen on CT but appeared normal with no signs of bleeding. Spleen and pancreas were also normal and bowel vascularity intact. The abdomen was explored without further findings, one intra-abdominal drain was inserted and primary closure of laparotomy was done.

Intra-operatively the patient was hemodynamically unstable and received several units of packed red cells, platelets and plasma. Following surgery he was shifted to the Surgical Intensive Care Unit and managed for multiple organ dysfunction syndrome over the subsequent three weeks, following which he made an uneventful recovery and discharged on the 36th post-operative day.

CT angiogram (Figs. 4 and 5) of the abdomen done 4 days post-operatively revealed normal vasculature and perfusion.

Fig. 2. Preoperative CT (axial view) demonstrating fluid collection around liver and spleen (arrows), distended stomach with air fluid level and liver lesion suggestive of a hemangioma (asterisks).

Fig. 3. Preoperative CT (sagittal view) with fluid collection seen at the level of origin of the Superior Mesenteric Artery.
3. Discussion

‘Apoplexy’ originates from the Greek word apopléxia meaning to strike down and incapacitate, which until the first half of the twentieth century was used by physicians in reference to ‘cerebral apoplexy’, now universally termed cerebrovascular stroke. The term ‘Intra-Abdominal Apoplexy’ was originally coined by Green and Powers in 1931 to describe an event equally unpredictable and catastrophic involving the spontaneous rupture of an intra-abdominal vessel resulting in massive intraperitoneal or retroperitoneal hemorrhage.

Traditionally abdominal apoplexy (also known as idiopathic spontaneous intraperitoneal hemorrhage) refers to the spontaneous rupture of an abdominal splanchnic vessel after excluding hemorrhage from gross aneurysms, gynecological lesions, visceral malignancies, trauma and any inflammatory or obvious pathological states. The most common sources of non-traumatic visceral artery rupture are from aneurysms or pseudoaneurysms, with approximately 30% of cases showing no identifiable source.

Maurice Barber first reported a case of abdominal apoplexy in 1909 in a 32-year-old female two days post-partum, the next case was reported soon after by John Churchman in 1911, in an otherwise healthy 48-year-old male. Since then medical literature has sporadically recounted cases of abdominal apoplexy with Carmeci et al. counting only 110 cases in a review of literature spanning almost a century (1909 –1998) and in 1940 Lafferty and Pearson discovered only one case of abdominal apoplexy in 9560 autopsies performed over ten years at the State Charity Hospital of Louisiana.

In 1970, Kleinsasser in an extensive review of literature described 83 cases of abdominal apoplexy. Cases ranged from 2 to 84 years of age, with the highest incidence in the 50- to 59-year-old population, and a higher incidence seen in men. Over the years hypertension and arteriosclerosis have become well established predisposing factors for abdominal apoplexy in older patients. Carter and Gosney’s study in 1966 revealed elevated blood pressure in 41% of cases, in 1945 Marks and Freedlander reported 56% and in 1948 Brewer and Marcus reported 50% of patients had either hypertension or arteriosclerosis. In addition to this most of the reported cases were already in hypovolemic shock when first assessed, thus making the true incidence of hypertension much higher.

Clinical presentation of abdominal apoplexy is variable, with no identifiable pathognomonic sign or symptom. The universal symptom however is the sudden onset of abdominal pain, which may subside as the bleeding ceases or is minimal, but often rapidly progresses to profound shock with sudden, excruciating pain accompanied with a drop in hemoglobin and hematocrit (if enough time has elapsed since initial pain to collapse), with or without leukocytosis.

Over the last century since abdominal apoplexy was first described only a handful of cases have been accurately diagnosed pre-operatively, even today reaching a diagnosis in any patient is heavily dependent on imaging techniques. The recommended radiological approach if spontaneous abdominal hemorrhage is suspected is a Monophasic CT Angio with arterial phase only (without oral contrast media); it is an effective method for detecting hemoperitoneum and active arterial extravasation. In an unstable patient a FAST would be the alternative. Diagnostic Peritoneal Lavage (DPL) is the most sensitive (and most invasive) test to identify spontaneous intra-abdominal hemorrhage; however a negative result does not eliminate the diagnosis of abdominal
apoplexy. Above all else, awareness of the condition and a high index of suspicion are imperative in reaching a pre-operative diagnosis.

If active extravasation of blood or arterial bleeding is detected on CT, percutaneous transcatheter embolization therapy\textsuperscript{12,14} can be considered as an alternative option to surgery in the stable patient and if the patient is not a surgical candidate. It may also be used as an adjunct to surgery to overcome a life-threatening bleeding prior to definitive surgical treatment. Hemodynamically unstable patients require an immediate exploratory laparotomy.\textsuperscript{12}

As with any other cause of hypovolemic shock, fluid resuscitation and restoration of circulating volume must be rigorously maintained, including during and after surgical intervention. The surgical management is simple, based on numerous reports over the last century, locating and ligating the bleeding point is the recommended procedure. The bleeding vessel is usually located at the point of maximal hematoma, and maintaining adequate resuscitation and normal blood pressure aids in locating the defect.\textsuperscript{14}

Kleinsasser’s study revealed that if a bleeding point was located and ligated the mortality was 8.6%, in contrast to mortality as high as 56% in which a bleeding point could not be detected\textsuperscript{9} (non-therapeutic exploration). Therefore in the event of a non-therapeutic laparotomy a visceral arteriography and embolization for any arterial pathology must be carried out.\textsuperscript{15}

Of the 83 patients in Kleinsasser’s study a bleeding point was identified in 60; the most common sources in descending frequency were the middle colic, left gastric, splenic and superior mesenteric artery.\textsuperscript{5} Overall mortality was 44.6% as reported by Kleinsasser with 100% mortality in the patients that did not have any type of surgical intervention.\textsuperscript{9} Since abdominal apoplexy was first reported there have been no cases of recurrence after ligation.\textsuperscript{13}

4. Conclusions

Emergency physicians and surgeons must exercise a high index of suspicion in hypertensive patients, between the ages of 50–60 years presenting with a history of abrupt onset of abdominal pain and consequent shock. Awareness of abdominal apoplexy due to non-traumatic rupture of abdominal splanchnic vessels must be raised by its inclusion in more surgical text books and online references.

Conflict of interest statement

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Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Author contributions

LA and MA are interns who were involved in the acquisition of patient information, literature research and drafting of the manuscript. KP was the surgeon treating the patient and with FB was involved in case management and the reviewing and editing of the manuscript critically for intellectual content. All authors read and approved the final manuscript.

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