CASE REPORT

A case of abdominal apoplexy because of the rupture of the short gastric vessel

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Abstract

Abdominal apoplexy or idiopathic spontaneous intraperitoneal haemorrhage is defined as the presence of free blood within the peritoneal cavity. Non-traumatic and non-iatrogenic causes may cause abdominal apoplexy. It has a variable clinical presentation, with abdominal pain being an early and non-specific symptom. We report a rare case of a 23-year-old male with abdominal apoplexy because of rupture of the short gastric artery. He presented to our department with abdominal pain. Later, he developed signs of shock, and was found to have haemoperitoneum on laparotomy. We ligated the short gastric artery, which was the bleeding source, and he had an uneventful postoperative course. We also review the literature on existing cases of short gastric vessel rupture.

INTRODUCTION

Idiopathic spontaneous intraperitoneal haemorrhage (ISIH) was first reported by Barber in 1909 [1]. He described the case of a pregnant woman who suffered intraperitoneal haemorrhage in the absence of trauma; the source of bleeding was not identified. Green and Powers [2] termed the condition abdominal apoplexy in 1931.

Abdominal apoplexy has a low reported incidence. In their report, Carmeci et al. [3] found only 110 cases between 1909 and 1998. It occurs more frequently in males than in females at a ratio of 3:2, and most cases occur in later middle age between 55 and 64 years [4]. We report the case of a young male presenting with abdominal apoplexy because of rupture of the short gastric arteries.

CASE REPORT

A 23-year-old male, of Polish origin, was brought in by the ambulance to the emergency department. He complained of abdominal pain which had started the night previously. The duration from pain onset to presentation was ~15 h. On that morning, he complained of feeling light headed and had experienced an episode of syncope. There was no history of trauma, nausea or vomiting.

On examination, he was tender predominantly in the epigastri c and right iliac fossa sections of the abdomen. No abdominal distension was noted. There were no signs of peritonism. The remainder of the physical examination was unremarkable.

In terms of laboratory tests, he had an Hb of 89 g/l, an MCV of 92 fl, a platelet count of 146 × 10⁹/l, a white cell count of 14.5 × 10⁹/l and a CRP of <1 mg/l.

The working diagnosis at this time was of acute appendicitis. He was placed nil by mouth and started on intravenous fluid resuscitation. However, while awaiting a chest X-ray, he experienced two further episodes of syncope. He was taken to theatre for an exploratory laparoscopy.

During the laparoscopy, the peritoneal cavity was full of blood, with clots in the upper abdomen. The operation was converted to open laparotomy for better visualization. The peritoneal cavity was full of blood. The short gastric vessels were identified and ligated. The patient made a full recovery and was discharged home on the 10th postoperative day.
because of a predisposing vascular lesion [5]. In older patients, in the literature (Table 1).

Key features of 13 cases of short gastric vessel rupture identified in order of decreasing frequency. We have summarized the predispositions in the medial coat of the visceral arteries are proposed to be responsible. In our case, we were unable to identify any predisposition towards abdominal vessel rupture.

Vomiting has been proposed as a common antecedent towards short gastric vessel rupture. Eight of the 13 cases, we identified occluded following bouts of vomiting (Table 1). Hayes et al. [12] proposed that retching may cause a partial, gastric volvulus. This may then pull on the gastroplenic ligament. This shearing force may result in a tear of the short gastric artery. Our case is unusual because there was no emesis. This suggests that other mechanisms may explain the occurrence short gastric vessel rupture.

The symptoms of haemoperitoneum may be divided into three phases [19]. There is an initial phase of mild to severe abdominal pain. It is important to note that blood acts only as a minor peri-
am open laparotomy as the source of bleeding could not be identified. This revealed a bleeding point from a short gastric vessel. Ligation with 2/0 prolene was used to achieve haemostasis. He was transfused two units of red blood cells and achieved an Hb of 10 g/l prior to discharge. The postoperative course was uneventful and discharge occurred 5 days later.

**DISCUSSION**

Rupture of the short gastric vessels is a rare case of abdominal apoplexy. In their review of 85 patients with abdominal apoplexy, Carter and Gosney [5] did not identify a single case of short gastric vessel rupture. The left gastric artery, superior mesenteric artery and splenic artery were listed as the most common sites of rupture in order of decreasing frequency. We have summarized the key features of 13 cases of short gastric vessel rupture identified in the literature (Table 1).

Most cases of abdominal apoplexy are believed to occur because of a predisposing vascular lesion [5]. In older patients, arteriosclerosis with or without hypertension is believed to be the most common cause. With younger patients, congenital defects in the mediolateral wall of the visceral arteries are proposed to be responsible. In our case, we were unable to identify any predisposition towards abdominal vessel rupture.

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The symptoms of haemoperitoneum may be divided into three phases [19]. There is an initial phase of mild to severe abdominal pain. It is important to note that blood acts only as a minor peritoneal irritant. The severity of the pain is related to the volume and rapidity of expansion of the blood volume. This is followed by a latent period, devoid of symptoms, lasting from hours to days. In the terminal phase, there is an increasing severity of symptoms and the manifestations of shock become evident. Our patient appears to have presented in the early terminal phase.

In terms of investigations, computed tomography of the abdomen has been reported as the most useful [4]. In haemodynamically unstable patients, FAST (focused assessment by sonography in trauma) examination may be useful to detect intra-abdominal haemorrhage. Our case of abdominal apoplexy was diagnosed by exploratory laparotomy. However, it was necessary to convert to laparotomy via an upper midline incision to identify the bleeding point.

Surgery remains the definitive treatment option for abdominal apoplexy [5]. This should be coupled with replacement of circulating volume to combat shock. Transarterial embolization has also been described as a treatment option [4]. The choice of intervention is determined by the age and condition of the patient, as well as the site of bleeding.

Prognosis for the patient is dependent on early diagnosis and intervention. In patients who do not undergo surgery, mortality has historically approached 100% [4]. However, prompt surgical intervention has been described to reduce mortality to 8.6%, especially where a definite bleeding point can be located and ligated [5].

Diagnosis of abdominal apoplexy is dependent on maintaining an awareness of the condition in one’s differential diagnoses. Unfortunately, lack of suspicion continues to delay diagnosis, worsening the prognosis for the patient [4]. One should suspect abdominal apoplexy in individual presenting with atypical abdominal pain and signs of shock. Vomiting, hypertension and arteriosclerosis should further raise suspicion of possible vessel rupture. In cases where abdominal apoplexy is suspected, one should progress quickly towards laparotomy and surgical ligation.

**CONFLICT OF INTEREST STATEMENT**

None declared.

**REFERENCES**