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Case Report

Abdominal apoplexy: a rare complication following Roux-en-Y gastric bypass

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ABSTRACT

Abdominal apoplexy, defined as spontaneous intra-abdominal haemorrhage, is an uncommon complication in patients with a history of bariatric surgery. This report highlights the diagnostic challenges and management approach of this rare entity. We describe the clinical presentation, diagnostic investigations and management approach for a patient who developed abdominal apoplexy five years following Roux-en-Y gastric bypass. A 48-years-old female presented with acute-onset severe abdominal pain. Her medical history included Roux-en-Y gastric bypass, laparoscopic appendectomy and hysterectomy. Examination revealed diffuse tenderness, predominantly in the upper quadrants. Laboratory results demonstrated anaemia. Imaging studies (Ultrasound and CT) revealed extensive intraperitoneal haemorrhage with no identifiable source. Diagnostic laparoscopy confirmed haemoperitoneum without active arterial bleeding, consistent with abdominal apoplexy. Surgeons and emergency clinicians should maintain a high suspicion of abdominal apoplexy in post-bariatric surgery patients presenting with unexplained acute abdominal pain. Early diagnostic laparoscopy is crucial for diagnosis and management.

Keywords: Abdominal apoplexy, Bariatric surgery, Haemoperitoneum, Roux-en-Y gastric bypass

INTRODUCTION

Abdominal apoplexy, also known as idiopathic spontaneous intraperitoneal haemorrhage, refers to a sudden, non-traumatic intra-abdominal haemorrhage. It is an exceedingly rare but life-threatening cause of acute abdomen.[1,2] With a growing obesity epidemic, Roux-en-Y Gastric bypass (RYGB) is Fastly becoming a more commonplace surgery. While postoperative haemorrhage is an uncommon complication (approximately 1-4% incidence), most bleeding is intraluminal in these instances.[3,4] True abdominal apoplexy following RYGB) is rare, with only a handful of cases reported. Historically, spontaneous intra-abdominal haemorrhage is very rare. A review by Carmeci et al, found only 110 cases of abdominal apoplexy reported between 1909 and 1998.[5] The majority of cases occurred in middle-aged to older patients (peak 55–64 years) with a slight male predominance (3:2).[5] post-bypass abdominal apoplexy can occur in the early postoperative period or several years later. Early bleeds usually manifest within the first few days after surgery. In one 21-year series of 2,639 RYGB patients, 2.7% had postoperative bleeding. Approximately 28% of these were extraluminal (intra-abdominal) haemorrhages.[6] Late spontaneous hemoperitoneum can occur months to years after RYGB due to delayed complications such as arterial pseudoaneurysm or ulcer erosion. Reported late cases include patients 5–14 years post-RYGB presenting with massive bleeding.[7,8] Notably, RYGB patients are often middle-aged and female which is reflective of the bariatric population, however, male patients may have higher risk of severe bleeds early on.[6] A case study of a patient who presented with spontaneous abdominal bleeding post-RYGB was conducted. Clinical records, radiological images and operative findings were reviewed and analysed.

CASE REPORT

A 48-years-old female presented with a three-day history of progressively worsening central abdominal pain. The pain was initially colicky and mild but escalated to severe by the morning of presentation, prompting the patient's presentation to hospital. She denied nausea, vomiting, fevers, urinary symptoms, or vaginal discharge but reported bloating and decreased passage of flatus. Bowel movements which had occurred the morning prior, were slightly loose and contained no blood. She had no previous history of similar symptoms.

Her past medical history included hypertension and diet-controlled diabetes, both of which had resolved following RYGB in 2016. She had significant weight loss from her pre-operative weight of 118 kg down to 76 kg. Additional surgical history included a hysterectomy 22 years earlier and a laparoscopic appendectomy. She had no allergies, took no regular medications, was a non-smoker and occasionally consumed alcohol. She lived with her husband and son and had recently been moving house, involving lifting heavy boxes, just before symptom onset.

On examination, her vital signs were within normal limits: blood pressure 130/85 mmHg, heart rate 60 bpm, respiratory rate 17 breaths per minute, temperature 36.9°C and oxygen saturation 99% on room air. Her abdomen was soft but diffusely tender, most pronounced in the right upper quadrant (RUQ) and left upper quadrant (LUQ), without evidence of peritoneal signs, palpable masses or organomegaly.

Initial laboratory investigations revealed normocytic anaemia (Hb 96 g/l, MCV 85.1 fl), a normal white cell count (5.9 × 10^9/l) and normal platelet count (181×10^9/l). Electrolytes and renal function were within normal ranges (Na 140 mmol/l, K 3.9 mmol/l, creatinine 64 µmol/l, eGFR >90). Liver function tests were unremarkable (bilirubin 7 µmol/l, AST 22 U/l, ALT 10 U/l, GGT 13 U/l, ALP 78 U/l).

Imaging investigations included an initial abdominal ultrasound, which demonstrated extensive particulate free fluid within the abdomen and pelvis, consistent with hemoperitoneum. The ovaries were not clearly identified, potentially atrophic and no adnexal masses or definitive bleeding source was seen. A follow-up contrast-enhanced CT scan of the abdomen and pelvis confirmed extensive high-density free fluid in keeping with haemoperitoneum, predominantly in the left upper quadrant surrounding the spleen and at the right upper omentum.

The liver, pancreas, gallbladder, adrenal glands, kidneys and bladder appeared unremarkable. No free intra-abdominal gas was detected. The bowel loops appeared normal within the limits of CT and there was no arterial phase contrast blush to suggest active haemorrhage. A linear hypoattenuation through the medial spleen was noted but interpreted as a capsular cleft rather than a laceration, given the absence of adjacent pleural or subcutaneous soft tissue abnormalities.

Given the persistent and unexplained haemoperitoneum, a diagnostic laparoscopy was performed. Intraoperatively, extensive haemoperitoneum was confirmed, but no clear source of active bleeding was identified.

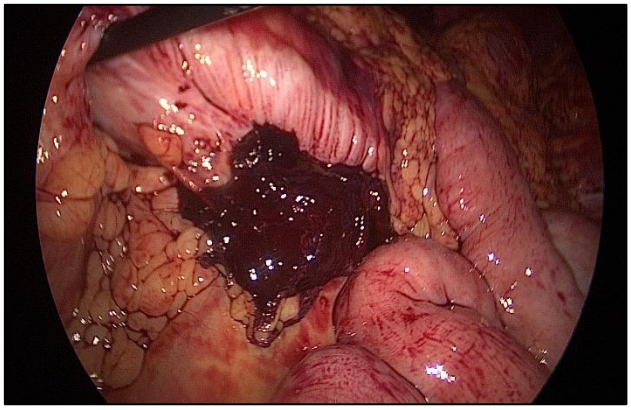

Figure 1: Intraoperative laparoscopic image demonstrating coagulated blood within the abdominal cavity, with no visible active arterial bleeding.

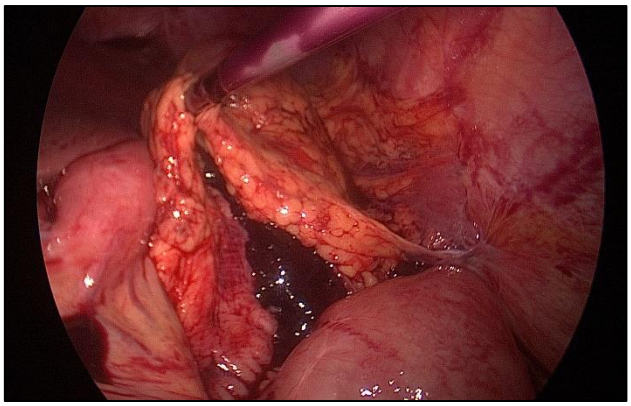

Figure 2: Laparoscopic view of the upper abdominal cavity, highlighting dense clotted blood beneath the omentum. Despite systematic exploration, no clear arterial bleeding site was identified.

The findings were consistent with abdominal apoplexy, a rare occurrence of spontaneous intra-abdominal haemorrhage without an identifiable bleeding vessel. The patient was managed conservatively with close monitoring, supportive care and serial hematologic assessments.

DISCUSSION

Abdominal apoplexy typically presents as acute abdominal pain and signs of haemorrhagic shock.[5] Patients often require aggressive resuscitation.[6] In hemodynamically stable patients, CT can confirm haemoperitoneum and sometimes identify the bleeding

source (e.g., active contrast extravasation or a vascular aneurysm/pseudoaneurysm).[8,9] In the case of suspected intraluminal bleeding It's worth noting that standard endoscopy often cannot reach the bypassed stomach and duodenum, so if GI bleeding is suspected in a RYGB patient, specialized techniques (e.g. enteroscopy or percutaneous remnant endoscopy) or imaging are needed.[2,4]

While conventional endoscopy has limited reach in RYGB anatomy, it still plays a role for bleeding that is partially intraluminal.[3,4] In scenarios where the bleeding site is identified on imaging, angiographic embolization can be a valuable, less invasive approach.[2] If the patient is unstable or if imaging is unavailable or inconclusive, urgent exploratory laparoscopy or laparotomy is indicated.[5] If the patient is in extremis, a damage-control approach may be needed to control bleeding and allow for subsequent resuscitation the patient before definitive repair at a later stage.[10]

Abdominal apoplexy carries a high mortality if untreated historically approaching 100% without surgical intervention.[5] If the patient recovers from the acute event, long-term outcomes are generally good. In cases caused by marginal ulcers, aggressive anti-ulcer therapy including proton pump inhibitors, *Helicobacter pylori* eradication and risk factor modification are needed to prevent recurrence.[4]

Although abdominal apoplexy is rare, it should be recognized as a possible cause of acute collapse in post-bypass patients. Presentations can resemble more common issues, so a high index of suspicion is required to avoid delays.[5] Prompt imaging and/or exploratory surgery are key to diagnosis. Management often involves aggressive resuscitation and urgent intervention (surgery or embolization) to stop the haemorrhage. Published cases demonstrate that outcomes are favourable if intervention is timely resulting in many patients recovering fully.[1,5] Conversely, delayed diagnosis can be fatal. Surgeons should be aware that in RYGB patients, bleeding can arise from unusual sites (mesenteric vessels, splenic artery, remnant ulcers) and both early and very late complications are possible.[2,3]

CONCLUSION

This case along with prior case reports, emphasizes early recognition and a team-based approach to successfully manage this dramatic complication of RYGB.

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