Hepatic portal venous gas in blunt abdominal trauma: A case report

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1. Introduction

Historically, hepatic portal venous gas (HPVG) used to be diagnosed on plain X-rays in the late or terminal stages of ischaemic or obstructive diseases of the bowel. It gained therefore the reputation of a catastrophic prognosis sign. This bad name tends to persist even today in spite of the demonstration that echography and CT scanning also show HPVG in benign and reversible conditions. We report such a case where HPVG disappeared a few days after a blunt abdominal traumatism.

2. Case report

A 44-year-old man with a history of asthma, major depression and chronic alcoholism was admitted intoxicated with ethanol (2.27 g/L) 1 h after a brawl. He was conscious but did not remember the incident. Witnesses reported a fall, many hits to the abdomen, vomiting and seizures. He presented with hypotension (86/60 mm Hg) and tachycardia (130 min^-1) that responded well to crystalloid boluses but the patient remained agitated and complained of pain at the left upper abdominal quadrant on physical examination. The admission haemoglobin was 135 g/L. A CT-scan revealed (Fig. 1 and 2): 1—a major hemoperitoneum around the spleen and liver as well as in the pelvis and both paracolic gutters; 2—a spleen inferior pole transection (Grade IV injury); 3— intrahepatic (portal vein) and extrhepatic (inferior mesenteric vein) venous gas; 4—heterogeneity of the caecal wall and surrounding fat. As the haemoglobin fell to 90 g/L, a laparotomy was performed: 2.5 L of blood were suctioned and the actively bleeding spleen was removed. An ileo-colic resection with primary anastomosis was done because of a haematoma of the caecal wall (Fig. 3). The pathologist reports the presence of a transmural haematoma with rupture of muscularis layer.

The postoperative course was complicated by alcohol withdrawal symptoms, pneumonia, wound infection, thrombocytosis and an incisional hernia but the patient eventually recovered. A week after surgery, a CT-scan evidenced the complete clearance of the hepatic portal and mesenteric vein gas.

3. Discussion

HPVG is regarded nowadays as a non-specific sign that can be observed in many conditions, both in children and adults. These can range from benign to lethal and include endoscopy, enteritis, colitis, diverticulitis, abdominal abscess, cancer, cholangitis, caustic ingestion, intestinal obstruction, bowel ischaemia and – as in our case – blunt abdominal trauma and seizures. Clinically, pain, nausea, vomiting, diarrhoea, abdominal distension, signs of peritoneal irritation and acidosis suggest an ischaemic aetiology but are non-specific. Other CT findings such as a distended bowel, ascites, calcified mesenteric arteries, colonic wall thickening and mesenteric oedema contribute marginally to the diagnosis and the treatment. In contrast, pneumatoasis intestinalis (PI) is present in 73% of HPVG cases, is associated with ischaemic causes and is a recognized sign of severity. As reported by Peloponissios et al., PI is hardly ever seen in benign causes of HPVG and could therefore be used to discriminate patients needing emergency surgery from those that can be treated conservatively. In this context, the available case reports of HPVG in blunt abdominal trauma indicate that the mortality is low and that this sign is not per se an indication for surgery. Our patient showed no PI and was indeed operated for haemodynamic instability. The complete clearance of his HPVG within a week strengthens the view that it was a benign occurrence.

Several non mutually exclusive mechanisms may explain the pathophysiology of HPVG. Intestinal air may enter venous or capillaries altered by erosion, ulceration, haematoma or necrosis. Alternatively gas may be generated by anaerobic bacteria within the gut wall. Finally, air may be forced through the intestinal layers into the venous circulation by a sudden increase of the intraluminal pressure. Whereas the first 2 mechanisms are thought to initiate ischaemia related HPVG, it is likely that the 3rd one predominates in abdominal blunt trauma. In our patient, the seizures may have also contributed to an increased intra-abdominal pressure, as first reported by Chen et al.

If this and other case reports point to the benign nature of HPVG without PI in blunt trauma, it is however essential to keep a low threshold for surgical exploration in case of a haemodynamic
4. Conclusion

Hepatic Portal Venous Gas has a different clinical relevance pathophysiology and treatment algorithm according to the underlying cause. In the setting of abdominal blunt trauma, bowel necrosis/ischaemia must be ruled out and a complete abdominal and pelvic CT-scan examination is mandatory: if it shows HPVG without PI and no obvious bowel necrosis, observation may be envisaged. On the contrary, HPVG with PI and/or any suspicion of bowel injury requires surgical exploration.

References