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Case report

Benign portal venous gas after blunt abdominal trauma

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1. Introduction

Air in the portal venous system is considered an ominous sign of life-threatening intra-abdominal pathology.[2] We present a patient with portal venous gas following blunt abdominal trauma who recovered uneventfully and describe the occurrence and importance of intravascular gas in this setting.

2. Case report

A 44 year old male patient presented to the Level 1 Trauma Unit at Inkosi Albert Luthuli Central Hospital (IALCH), Durban, South Africa, having sustained blunt injuries following a road accident. The patient was a cyclist involved in a collision with a passenger motor vehicle. He arrived at the Trauma Unit 24 h following the injury, having been referred from an outlying rural hospital for mechanical ventilation due to his chest injury. He was not distressed, was noted to be haemodynamically stable with a pulse rate of 101, blood pressure 132/42, respiration rate 24 and a Glasgow Coma Score of 15/15.

External injuries included a superficial laceration of the forehead and minimal cervical spine tenderness. A thoracic cage deformity was evident with a depression of the left antero-superior chest (stove-in-chest) and decreased air entry over that region. Air entry was also decreased in the lower zones bilaterally. The abdomen was tender, but with no evidence of peritonitis or guarding. A contaminated degloving wound over the right elbow with involvement of the joint was also present.

IALCH utilises a 128-slice Multiple Detector Computerised Tomography scanner (Somatom Definition AS, Siemens, Germany). The patient underwent a full-body CT-Angiogram and portal-venous phase scan to delineate the extent of the chest injury and exclude significant abdominal injury. There were stable fractures of the C5 + T1 vertebral body, C7 left pedicle, lamina + facet joints; a sternal fracture with a retro-sternal haematoma; a right neck of scapula fracture and multiple left and right rib fractures with underlying lung contusions and associated haemothoraces. The CT scan of the abdomen and pelvis demonstrated air in the portal system of the left lobe of the liver as well as an ectopic right kidney, widened right sacro-iliac joint and right superior and inferior pubic rami fractures (Figs. 1 and 2).

Mechanical ventilation was not required. Following debridement of the contaminated elbow wound, the patient was observed in the Trauma ICU for signs suggestive of hollow abdominal visceral injury. The abdominal tenderness subsided and he was discharged back to the referral hospital after 7 days.

3. Discussion

The presumed pathophysiology of pneumatosis intestinalis (PI) and portal venous gas (PVG) in the trauma setting is mechanical injection by a concussive force.[2] The presence is suggestive of mucosal disruption, but does not necessarily imply intestinal necrosis or perforation. Intravascular gas may occur as a transient incidental finding after blunt abdominal trauma.[10]

The diagnosis PVG can be made by plain abdominal radiography, sonography, color Doppler flow imaging, or computed tomography (CT) scan. The characteristic finding on plain abdominal radiography is a branching radiolucency extending to within 2 cm beneath the liver capsule. This is because of the centrifugal flow of portal venous blood, which carries portal venous gas peripherally, in contrast to biliary gas, which tends to collect centrally as a result of the centripetal movement of bile. Although PVG may be diagnosed by conventional radiography, detection is difficult and it is easily overlooked.[9]

Sonography, color Doppler flow imaging, and CT scan have been reported to be superior to abdominal radiographs in identifying PVG. Sonography coupled to Doppler is very sensitive for PVG detection and its follow-up, and can be useful as an initial screening examination.[4,12] However, use of ultrasound is limited because of its high inter-operator variability and lack of full-time access. The typical ultrasonographic features of PVG are (1) highly echogenic particles flowing within the portal vein or (2) poorly defined, highly echogenic patches within the hepatic parenchyma which are most apparent in the non-dependent part.[15] With the regular use of screening ultrasonography (US) in the setting of

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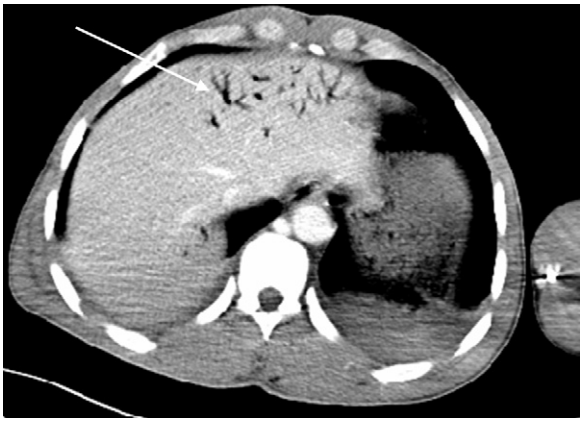

Fig. 1. Axial transhepatic cut (T10 level) of the portal-venous phase of the Trauma CT-Angiogram demonstrating the peripheral portal venous gas pattern (white arrow).

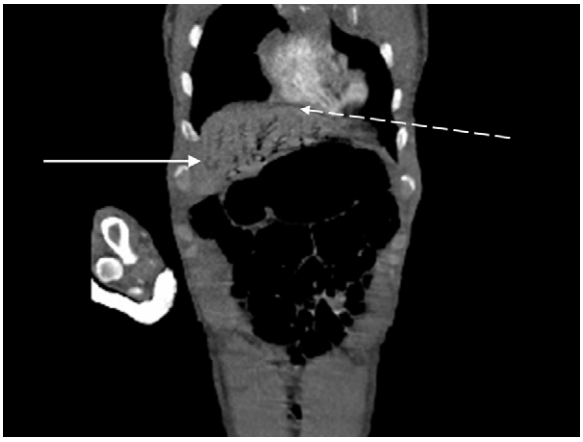

Fig. 2. Coronal reconstruction of the Trauma CT-Angiogram phase demonstrating portal venous gas (solid white arrow) and absence of sub-phrenic free air (dashed white arrow).

blunt abdominal trauma, identification of intravascular gas as a transient finding has been observed, without clinical consequence, in upper abdominal veins.[13] Ultrasound was not used in the index patient.

Color Doppler flow imaging shows hyperechogenic foci moving within the lumen of the portal vein, producing sharp bidirectional spikes superimposed on the normal monophasic portal vein wave pattern. CT scanning has a high sensitivity for detection of HPVG and can detect the underlying pathology.[14,3] PVG is characteristically associated with peripheral gas lucencies, which branch out and are noted even within the last 2 cm beneath the liver capsule. This peripheral gas distribution is related to the direction of blood flow into the liver. It is crucial to differentiate it from pneumobilia, which is centrally located because of the biliary anatomy and the direction of bile flow.[17,5] CT scan may also reveal gas in the bowel wall (pneumatosis intestinalis) and in the extrahepatic portal vein or splanchnic vasculature.

The underlying pathology inducing PVG is an important determinant of prognosis and historically has been associated with serious underlying disease and a high mortality rate.[2] PVG is often associated with intestinal pneumatosis or bowel necrosis and the reported causes are listed in Table 1.[2] Early reports of portal venous gas described mortality rates near 90% in contrast with recent studies where rates as low as 29% have been documented.[8,7] The decline in mortality is due not to improved therapy but rather

Table 1
Causes of portal venous gas (derived from intravascular gas as an incidental finding at US after blunt abdominal trauma[2] and hepatic portal venous gas: physiopathology, aetiology, prognosis and treatment[17]).

Mesenteric infarction
Intestinal obstruction
Abdominal abscess
Neonatal necrotising enterocolitis
Crohn's disease and ulcerative colitis
Acute diverticulitis
Perforated gastric carcinoma or ulcer
Suppurative cholangitis
Caustic corrosive ingestion
Acute pancreatitis
Obstructive pyelonephritis after extracorporeal shock wave lithotripsy
Acute appendicitis
Cholangitis
Gastro-jejunal anastomotic leak after laparoscopic gastric bypass
Uterine gangrene
Percutaneous endoscopic gastrostomy tube placement
In the presence of a jejuna feeding tube
Following esophageal variceal band ligation
Gastrointestinal perforation with amyloidosis
Severe hyperglycemic shock
Superior mesenteric artery syndrome
Chemotherapy
Liver transplantation

to the recognition of an increasing number of clinically unimportant causes of portal venous gas. This recognition has been facilitated by the more widespread availability and application of sensitive imaging modalities to a wide range of diseases.[2]Portal venous gas due to blunt trauma was initially reported to herald vascular injury and bowel necrosis, as concluded in reports as recently as in 1995. However, a few case reports of benign portal venous gas visualised on CT scans in patients with blunt abdominal trauma have been described.[6,16,11]

The most likely mechanism in cases of blunt abdominal trauma is that of sudden increase in intra-abdominal pressure caused by the impact, with consequent increased intra-luminal pressure causing mucosal tears. Intra-luminal gas is forced into the bowel wall, and enters the submucosal veins where it is absorbed into the portal circulation.[1] Once in the portal system, the gas may pass into the hepatic veins via the sinusoids. Gas in the inferior vena cava may arise from sites of porto-systemic communication.

In the recent literature the recommendation is for selective non-operative management and in the absence of other indications for surgical exploration, observation and monitoring in a high care setting is recommended. The presence of peritonism however, suggests intestinal necrosis and mandates operative intervention. In the patient described in this report selective non-operative expectant management was successful.

4. Conclusion

A case of transient PVG following blunt abdominal trauma which was successfully managed non-operatively has been presented. Benign forms of PVG are being increasingly reported on imaging and must be considered in the context of the patient's clinical picture in order to avoid unnecessary invasive procedures. In the absence of clinical indications for surgery, selective non-operative management is safe.

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