CASE REPORT

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Hepatic Portal Venous Gas After Blunt Abdominal Trauma In A Child

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ABSTRACT

Hepatic portal venous gas is defined as tubular areas with decreased enfeeblement around the periphery of the liver. It is a rare finding and associated with various underlying abdominal pathologies, from benign causes to potentially life threatening conditions. Rate of diagnosis of hepatic portal venous gas have been increased due to widespread use of advanced imaging techniques. Existence of gas in the portal veins after blunt abdominal trauma is a temporary incidental finding that could be resolved spontaneously. Here we report an extremely rare condition that describes a child who was suffering from air emboly of the portal veins due to blunt abdominal trauma. The condition was clinically under control and resolved spontaneously.

Keywords: Portal Vein Gas, Blunt Injury, Children

Introduction

Hepatic portal venous gas (HPVG) is defined as tubular areas with decreased enfeeblement around the periphery of the liver. It was first described in 1955 by Evens and Wolfe in a newborn baby with necrotizing enterocolitis (NEC) (1,2). It is a rare finding and associated with various underlying abdominal pathologies, from benign causes to potentially life threatening conditions (1). Rate of diagnosis of hepatic portal venous gas have been increased due to widespread use of advanced imaging techniques. Intestinal ischaemia is the most common cause of this condition. Blunt trauma has also been reported occasionally, in association with HPVG (1). Existence of gas in the portal veins after blunt abdominal trauma is a temporary incidental finding that could be resolved spontaneously. Here we report an extremely rare condition that describes a child who was suffering from air emboly of the portal veins due to blunt abdominal trauma. The condition was clinically under control and resolved spontaneously. Management of this case was discussed with the guidance of literature

Case Report

A 12 years old male patient was referred to our hospital after a motor vehicle accident. On initial assessment the patient was unconcious and intubated. He was initially hemodynamically stable but has bleeding from superficial scalp lesion. He had multiple abrasions and contusions on examination. His Glasgow Coma Scale (GCS) Score on admission was 7. His abdominal examination was normal. Shortly after arrival, he became hypotensive so normal saline and erythrocyte suspension transfused. The initial laboratory data results were as follows: White blood cells=11000 mm^3, Hemoglobin=10,8 g/dL, aspartate aminotransferase=435 IU/L, alanine aminotransferase=168 IU/L and lactat dehidrogenase= 3030 IU/L.

Radiological workup included plain film radiographs; and non-contrast computed tomograhy (CT) scan of the head, thorax and pelvis. Cranial CT was normal except with subcutanoeus hematoma. There was pulmonary contusion and minimally hemothorax on chest graphy. Contrast–enhanced abdominal CT scan revealed retroperitoneal fluid, edema in parahiler area and portal venous gas in hepatic portal system (Figure 1). Diagnostic paracentesis was normal. The patient was subsequently admitted to our intensive care unit, where he was managed with mechanical ventilation, broad-spectrum antibiotic coverage and repeated clinical evaluations and leukocyte counts. After two days patient was stable, extubated and a control CT and

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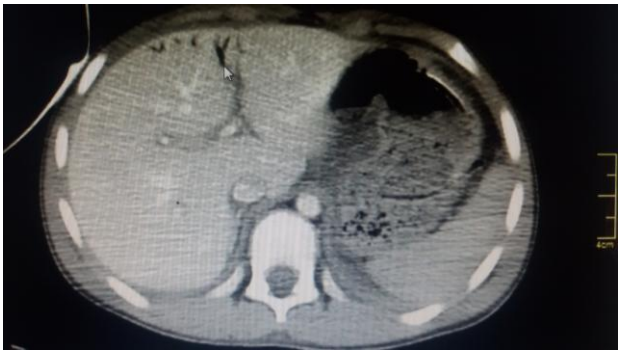

Fig.1. Ct Scan Shows Portal Venosus Gass

ultrasonography revealed no hepatic venous gas and a decreased abdominal fluid. This patient remained stable and was admitted to orthopedics clinic for fractures of right iliac crest for bed rest. His total hospital stay was eleven days, and was discharged with no neurological sequel.

Discussion

Diagnosis and management of the underlying surgical cause of hepatic portal venous gas is a challenge for physicians. It is not a illness itself; it is a ominous radiologic sign in patients who may be experience an intraabdominal severe pathology (1,3). Intestinal ischaemia is the most common cause of this condition and mesenteric vascular pathology in almost 60% of cases. This is occured after gastrointestinal tract inflammation (e.g. necrotizing enterocolitis, diverticulitis, inflammatory bowel disease), obstruction and dilatation (e.g. gastric dilatation, ileus), sepsis (e.g. septic thrombophlebitis), emphysematous cholecystitis and ascending cholangitis iatrogenic injury and malignancies (1,2,3,4,5). Majority of the reported patients are elderly with mesenteric vascular disease (6). The underlying causes of HPVG in childhood did not mainly differs from in adults. Necrotizing enterocolitis is leading eitology and HPVG has been described as a less prevalent but pathognomonic radiologic sign of NEC (7,8). Sharma et al. reported that HPVG was identified in 64 (33%) of the 194 infants with NEC (7). Furthermore, it has been described in association with congenital pyloric stenosis, child abuse and blunt abdominal trauma in childhood (9,10,11).

HPVG association with blunt abdominal trauma was first reported in 1988 by Matthews and Vauthey and only fourteen cases with HPVG due to blunt abdominal trauma have been reported in English-language medical literature, according to Medline (12,13). We identified four pediatric case in such association in literature (10,11, 14,15). In addition four cases with pneumatosis intestinalis after blunt abdominal trauma in childhood have also been reported with or without hepatic portal involvement (16,17).

The pathophysiology of HPVG has not been well understood. Some authors propose that mucosal damage allows gas to enter the mesenteric veins because of the mechanical distension due to increased intraluminal pressure. Whether microbiological factors include the following: (1) escape of gas produced by gas-producing organisms in the intestinal lumen or in an abscess which head towards to the liver or (2) the presence of gas-forming organisms in the portal venous system with passage of gas into the circulation (1,2,18). Similarly, the bacterias which fermentate substrates in milk are claimed to produce gas in newborns with NEC (7). On the other hand, blunt abdominal trauma and the other conditions (e.g. gastric dilatation, ileus) prove that the increased intraluminal pressure causes micro ruptures in mucosa of intestinal wall, which leads gas invasion to submucosal veins. And then gas may flow to the hepatic portal vein without bowel ischemia and gas-producing organism proliferation (1,12).

The diagnosis of HPVG is usually put by erect abdominal plain x-ray, computered tomograpy or abdominal ultrasonography (19). abdominal plain x-ray fail to show portal gas in 80% of the cases (19). Computered tomograpy is the gold standard for diagnosis of HPVG and its etiology (3).

Subcapsular, parenchymal, and intravascular gas can be seen with and without visceral injury and the spectrum of causes is various (5). But it is actually unusual finding that retrospective reviews of CT scans identified 17 cases in 14 000 in a study and 11 in 19 000 in another (2). After the detection of air in the liver, the initial differentiation that must be made is between pneumobilia and portal venous gas (20). While portal gas is generally accumulated in periphery of liver tissue, usually within 2 cm of capsule of Glisson (18), pneumobilia is located in central (20).

However, mortality could reach 75% in HPVG specially with ischemic conditions in elderly, most of these conditions which are reported in the literature were suitable for conservative treatment (3). Only half of cases with HPVG after blunt trauma that reported past years have resulted in surgery (13). But all of pediatric cases reported in literature have been underwent laparotomy except present case. Our patient was managed conservatively without any surgical aproach and

made a successful recovery. but a serious blunt abdominal trauma may lead to necrosis of the intestines and peritonitis due to mechanical perforation. When HPVG seen after abdominal trauma with free abdominal gas, pneumatosis intestinalis and peritonitis, surgical approach is always necessary (1). But without peritonitis signs and intestinal necrosis may allow an observation period (16). As in present case management without surgical intervention can be adequate and safe. The presence of gas in the hepatic portal system may indicate either a benign or potentially life-threatening condition. We can suggest that when clinical presentation and radiological modalities prove the absence of intestinal necrosis in a clinical stable patient, conservative treatment with nasogastric decompression, hemodynamic support, antibiotherapy and clinical observation is suitable in management of HPVG. However, the amount of reported cases of HPVG after blunt abdominal trauma is not enough to make a consensus of opinion.

In conclusion, we report a 12-year-old child who presents HPVG after an abdominal trauma that managed conservatively. To our knowledge, this is the first report to describe such association in children that managed conservatively.

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