

# Portal vein Gas detected by Point of care ultrasound

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## Abstract

Portal venous gas can be detected by a bedside point of care ultrasound examination. We present the case of a 73-year-old female with advanced ischemic cardiomyopathy. She developed vague abdominal pain. Her findings were consistent with portal venous gas with point-of-care ultrasound. She had nonobstructive mesenteric ischemia.

Hepatic Portal vein Gas detected by Point of care ultrasound

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Portal vein Gas detected by Point of care ultrasound.

## Key Clinical Message

Portal vein gas detected by point-of-care ultrasound in critical care medicine, in situations of unidentified shock and hypoxemia indicates a high likelihood of underlying intestinal ischemia.

Abstract

Background:

Portal venous gas is a rare finding in adults and is typically associated with underlying intestinal ischemia. Portal venous gas can be detected by a bedside point of care ultrasound examination in adult patients in critical care units(CCU). Findings include echogenic bubbles flowing centrifugally throughout the portal venous system.

We present the case of a 73-year-old female with advanced ischemic cardiomyopathy and cardiorenal syndrome who was managed in the CCU. She developed vague abdominal pain and respiratory depression requiring intubation and dialysis during her course of treatment in the CCU. Her findings were consistent

with portal venous gas upon point-of-care ultrasound, prompting computed tomography of her abdomen and surgical consultation. She was ultimately found to have nonobstructive mesenteric ischemia.

## Introduction

Nonocclusive mesenteric ischemia (NOMI) is a rare complication in critically ill patients with multiple comorbidities and dialysis patients, with potentially fatal consequences if not recognized early (1). Usually, it presents with nonspecific symptoms in critically ill patients who might be intubated, leading to a delay in diagnosis (1). POCUS has an evolving role in establishing a diagnosis of unexplained shock and deterioration of critically ill patients at the bedside (2). We present a critically ill patient who initially improved and then deteriorated acutely and was found to have intestinal ischemia after detecting portal vein gas by POCUS.

## Case report

A 73-year-old female presents with a medical history significant for hypertension, diabetes mellitus type 2, diabetic nephropathy with chronic renal impairment, dual pacemaker due to sick sinus syndrome, and an ST elevation myocardial infarction 10 months earlier managed with three drug-eluting stents (one in the left main coronary artery, one in the proximal right coronary artery, and another in the proximal circumflex artery). She was fed by PEG 6 months ago because of refusal to eat and the possibility of true bulbar palsy and early signs of Alzheimer's disease. Her medications prior to admission were furosemide 80 mg twice daily, carvedilol 6.25 mg twice daily, spironolactone 50 mg once daily, and clopidogrel 75 mg once daily.

The patient was admitted with hypotension and pulmonary edema and received ventilator support after tracheal intubation. She was initially managed with inotropes and renal replacement therapy with improvement, and on hospital day 3, she was extubated. This event was followed by acute on chronic renal failure, requiring a restart of renal replacement therapy.

Echocardiogram showed severe global hypokinesia of the left ventricle with a low ejection fraction (EF) (< 20%), bilateral lung ultrasonic B lines, bilateral mild pleural effusions, and low cardiac output. There was generalized edema.

She improved initially and infusions of noradrenaline and dobutamine were discontinued.

On day 7 after extubation, the patient underwent hemodialysis 9 hours before her acute deterioration with an episode of hypotension (blood pressure, 85/42 mm Hg) during hemofiltration that resolved with temporary use of a noradrenaline infusion during the procedure. The patient developed tachypnea (33 breaths/min) and vague abdominal pain. Physical examination showed a blood pressure of 105/45 mm Hg and a heart rate of 93 beats/min. Her abdomen was slightly distended but nontender, with hypoactive bowel sounds throughout. Blood work showed elevations of alanine aminotransferase (360.1 U/L), aspartate aminotransferase (577.3 U/L), lactate dehydrogenase (381 U/L), white blood cell count ( $16.00 \times 10^3/\text{L}$  [ $12.45 \times 10^9/\text{L}$ ]), and C-reactive protein (83.4 mg/L) levels. Anion gap metabolic acidosis with lactate rising from 1.8 to 8.6 mmol/L (0.5 to 1.5 mmol/L), and pH 7.00 constituting lactic acidosis was also observed.

The above episode necessitated the restart of the noradrenaline and dobutamine infusions. Due to tachypnea and hypoxemia, she was reintubated. POCUS to the abdomen showed sluggish movement of the intestines. Initially, bedside echocardiography was performed and showed severely impaired systolic function. While assessing the inferior vena cava, hyperechoic dot artifacts were observed throughout the left upper lobe of the liver, which were consistent with air extending to the periphery of the liver and hilum (Videos 1 and 2, Figure 1). Furthermore, discrete echogenic bubbles flowed centrifugally throughout the portal vein with disruption of the normal Doppler pulse because of air in the hepatic portal vein. These findings were consistent with portal venous gas (PVG), raising the possibility for bowel ischemia. CT of the abdomen with intravenous and oral contrast showed portal venous gas (Figure 4) in its tributaries, as well as in the superior mesenteric vein, which is a feature highly suggestive of bowel ischemia. Foci of gas were observed in the walls of the stomach, right side of the colon and some segments of small bowel loops (pneumatosis intestinalis), which also raises the possibility of ischemic bowel. Marked atherosclerotic changes in the aorta and its great branches were noted, with an attenuated hepatic artery, superior mesenteric artery and celiac

trunk with nonvisualized inferior mesenteric artery. Minimal fluid was found in the pelvis. Despite extensive calcification of the superior mesenteric artery and its branches, neither occlusive thrombosis nor significant luminal narrowing was detected.

The surgeon evaluated her and diagnosed nonocclusive diffuse intestinal ischemia. The family was counselled, and no further interventions were recommended. She passed away 3 days later.

## Discussion

PVG is an ominous radiological sign and reflects an abdominal surgical emergency. Intestinal ischemia is the typical pathology and cause of PVG, occurring in up to 72% of cases (3). PVG has been reported in cases with underlying inflammatory bowel disease when there was mucosal damage and infection with gas-producing organisms (4). However, infection with gas-forming organisms, especially *Clostridium*, and anaerobic streptococci can lead to gas in the portal vein and intestinal walls (5). Complicated diverticular disease of the colon and infection with gas-forming organisms has also been demonstrated in a case study (6). Other causes of PVG include intestinal perforation, enterovenous fistula following surgical procedures, and malignant bowel obstruction complicated by ischemia (5).

Our case report showed both PVG and pneumatosis intestinalis. Both signs occurred together in 41.7% of the cases. PVG occurred separately in 33.3% of the cases, while PI occurred separately in 25% of the cases (7). Many studies have claimed that, when accompanied by PVG, PI is often associated with bowel ischemia (7,8). The combination of pneumatosis intestinalis, portal venous gas, and acidosis typically portends bowel ischemia and inevitable necrosis (9). Furthermore, in cases with bowel ischemia, the presence of PVG and pneumatosis intestinalis is highly suggestive of transmural bowel ischemia (91%) and is associated with higher mortality (72%), whereas each sign cannot independently differentiate between transmural and partial thickness bowel ischemia. It was associated with lower mortality rates, 44% and 56% for PI and PVG, respectively (10).

Acute mesenteric ischemia and hypoperfusion of the small bowel could be due to occlusive or nonocclusive obstruction of the arterial blood supply or obstruction of venous outflow. The superior mesenteric artery (SMA) is commonly occluded by emboli or thrombosis, while venous thrombosis commonly affects the superior and inferior mesenteric veins. NOMI accounts for 5% to 15% of patients with acute mesenteric ischemia (11). The mechanism behind hypoperfusion of the bowels in NOMI is related to the maintenance of cerebral and cardiac blood flow at the expense of the bowels (12). Neurohormonal mediators (vasopressin and angiotensin), along with other vasoactive and chronotropic drugs, can trigger spasms and decrease blood flow to the intestines(12).

Classically, NOMI occurs in critically ill patients with severe cardiovascular disease; has life-threatening complications (sepsis, myocardial infarction, and congestive heart failure), with chronic renal impairment/hemodialysis; and patients are often receiving multiple drugs, including vasopressors and inotropes (13).

Usually, the early symptoms of NOMI are mild and nonspecific, and abdominal pain is absent in 25% of patients. In addition, the presence of precipitating factors and comorbidities in the majority of cases, such as congestive heart failure and renal failure requiring hemodialysis that are usually complicated with hypotension, intubation and sedation, can hide the early signs of NOMI [14]. Necrosis and perforation can occur, and in many cases, the diagnosis can be confirmed. The patient typically develops hypotension and shock of underdetermined cause. POCUS plays an important role in detecting the sources of surgical emergencies and detecting serious surgical conditions.

With the increasing practice of and need for point-of-care ultrasound in critical care and emergency medicine, especially in situations of unidentified shock and hypoxemia, and the limited knowledge about portal venous gas in the point-of-care setting, intensive care physicians should be aware of findings of concern related to air in the portal system as a bedside test indicating a potential need for more advanced imaging or surgical consultation, given the high likelihood of underlying intestinal ischemia.

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## CONFLICT OF INTEREST

The authors declare no conflicts of interest.

## Contributions

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Mohamed Elsayed Elhabibi; literature search, data acquisition, and help in manuscript preparation

Ossama Sajeh Maadarani; data acquisition, and help in manuscript preparation

Ali Khalifa Albirami; data acquisition, and help in manuscript preparation

Mahmoud Mostafa Elzouairy; reviewing content in terms of writing principles and help in literature search.

Tamer Mohamed Zaalouk; literature search, data acquisition

## Consent

**Images are entirely unidentifiable, and consent for publication is obtained.**

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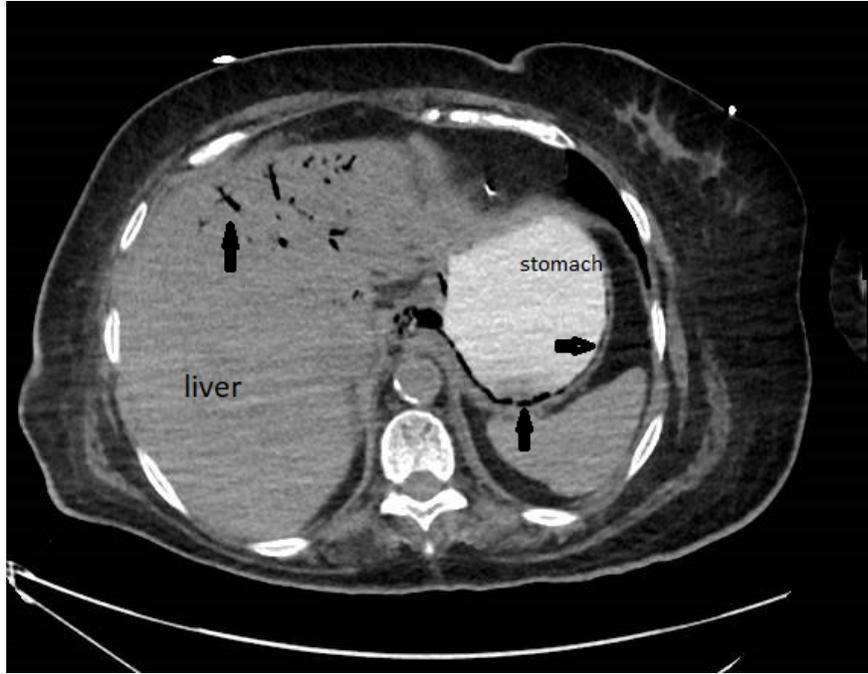
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Figure 1. Ultrasound image showing hyperechoic dots (arrow) within the liver parenchyma and portal vein representing gas.

Videos 1 and 2. Ultrasound videos. Arrows show moving hyperechoic dots, representing air within the portal vein and liver parenchyma. G: gallbladder, L: liver.

Figure 2. Contrast-enhanced phase multidetector CT axial image shows gas in the intrahepatic portal veins and wall of the stomach (black arrow) as multiple areas of low attenuation.





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