

Gas Embolic Stroke Secondary to Bowel Infarction

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A 69-year-old gentleman with metastatic esophageal adenocarcinoma presented with acute abdominal pain to the emergency medicine department and subsequently developed an acute left hemiplegia while in the resuscitation unit. An unenhanced computed tomography (CT) scan of the head showed right frontal cerebral gas emboli while an unenhanced CT scan of the abdomen and pelvis showed extensive portal venous gas and pneumatosis intestinalis, presumed secondary to bowel infarction. **Key Words:** Stroke—gas embolism—bowel infarction—ischemia—cerebral—Batson.

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Case Report

A 69-year-old gentleman with a background history of type II junctional adenocarcinoma of the esophagus presented to the emergency department with a history of acute onset generalized abdominal pain and vomiting. The patient had an esophagectomy 4 months before presentation and a loop ileostomy done 1 month earlier for large bowel obstruction. Widespread peritoneal metastatic disease and a left upper quadrant abdominal mass was found on the latter presentation. The patient was awaiting oncology follow-up to discuss second-line chemotherapy at the time of acute presentation.

The patient had presented to the emergency department with complaints of generalized lethargy and abdominal pain. Pain was more marked at the ileostomy

site with no change in stoma drainage. He was noted to be tachypnoeic with a respiratory rate of 24, had oxygen saturations of 96% on room air, and was tachycardic with a heart rate of 120. His Glasgow Coma Scale (GCS) was 15/15. On physical examination, the abdomen was firm with generalized tenderness and reduced bowel sounds. Initial venous blood gas was unremarkable except for a raised lactate of 6.7. Within 20 minutes of presentation, the patient's GCS dropped to 6/15 (E4V1M1) and the blood pressure to 80/40 mmHg. Pupils remained equal and reactive at 3 mm in diameter. With Trendelenburg's maneuver and fluid resuscitation, the patient's blood pressure came up to 120/80 mmHg within minutes, with his GCS improving 10 minutes later to 11/15 (E4V1M6). The patient was found to have a new dense left hemiplegia at this point with Medical Research Council (MRC) power of grade 0/5, diminished deep tendon reflexes on the left side, and extensor plantar reflex on the left.

An unenhanced head computed tomography (CT) (Fig 1) was subsequently performed which showed gas within the confines of multiple right frontal lobe sulci. A CT scan of the abdomen and pelvis performed at the same time (Fig 2) demonstrated extensive portal venous gas (Fig 3), spilling into systemic veins and also extensive bowel pneumatosis, presumed due to small-bowel ischemia.

After subsequent intensive care and surgical evaluation, it was deemed the patient's condition was

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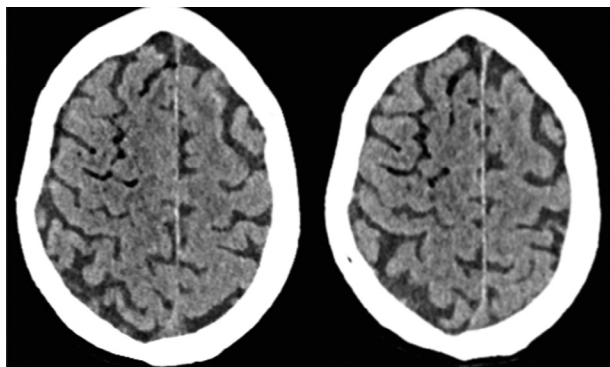


Figure 1. Axial CT head images. There is air-density gas in the sulcal spaces of the right frontal lobe confined within tubular spaces and following branching patterns, making this cerebral venous air. Abbreviation: CT, computed tomography.

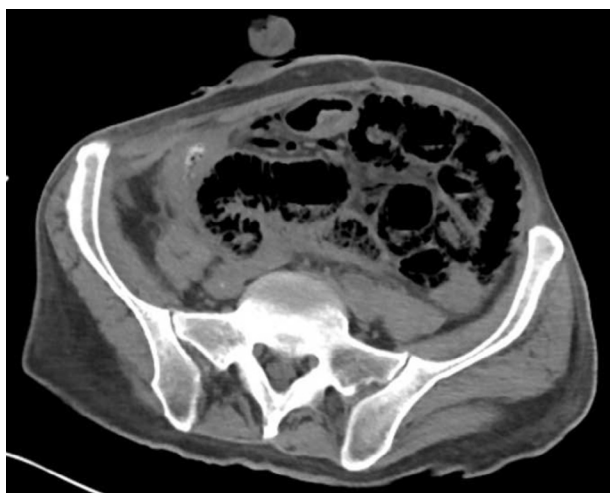


Figure 2. Axial non-contrast-enhanced CT scan of the abdomen shows extensive small-bowel gaseous distension, pneumatosis, and gas within mesenteric veins. Abbreviation: CT, computed tomography.

unsurvivable and the patient and family were appropriately informed. Routine monitoring was stopped, intravenous morphine administered for comfort, and the patient died 2 hours later with his family at his bedside.

Discussion

Gas emboli arise when the intravascular pressure is lower than that of the surrounding gas. Portal venous gas due to a disruption of bowel mucosa is well described and is a poor prognostic indicator in the context of ischemic bowel.¹ While gas in both the portal venous and systemic venous system is in itself an extremely rare finding, to the best of our knowledge, cerebral gas embolism secondary to bowel ischemia has never been described. Gas in the cerebral vasculature can cause ischemia through mechanical obstruction and vasospasm, causing endothelial injury resulting in an inflammatory reaction.² The gas emboli in our patient were seen in multiple sulci



Figure 3. Axial non-contrast-enhanced CT scan of the abdomen shows extensive portal venous gas. Abbreviation: CT, computed tomography.

following a serpiginous pattern, localized within the cortical veins. The distribution of the gas emboli in the right frontal lobe adjacent to the motor cortex (Fig 1) supports the clinical manifestation of a left hemiparesis that was observed.

The route of extension of the portal venous gas to cerebral circulation to cause venous embolic stroke is unclear. A number of mechanisms have been postulated for gas emboli to be shunted from the portal circulation to systemic venous circulation. Extensive intrahepatic portal venous gas may cause widespread sinusoidal obstruction resulting in portal hypertension.³ Subsequent gas emboli may thus be forced through anatomical portosystemic anastomoses such as those at the lower

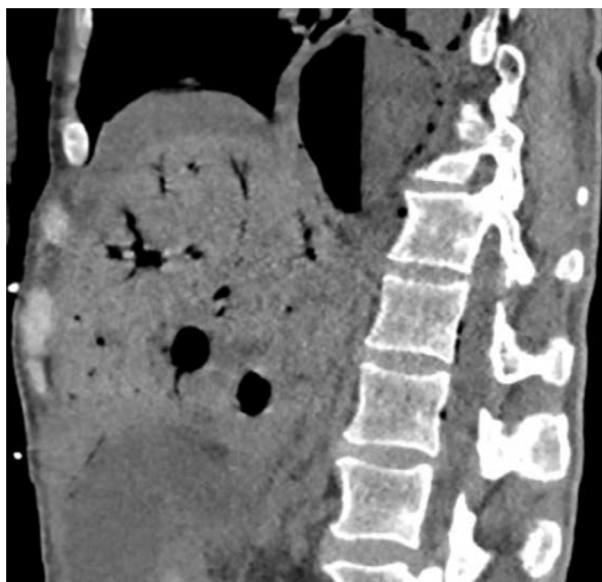


Figure 4. Reformatted sagittal image from non-contrast-enhanced CT scan of the abdomen shows air within the basivertebral vein and also air in the spinal canal, likely within the epidural venous plexus. Abbreviation: CT, computed tomography.

esophagus or upper anal canal, including rarely, through a patent ductus venosus. Instead of obstructing the hepatic sinusoids, 1 report suggested that gas emboli might pass directly from intrahepatic portal veins through distended sinusoids and into hepatic veins as has been observed in real-time sonography.⁴ Previous abdominal surgery, a history of which was present in our patient, may result in the development of systemic venous drainage of the bowel or the creation of portosystemic venous fistulas,⁴ and this may be why extensive gas is seen in both the portal and systemic venous systems. It has also been suggested, after a case of portosystemic venous air postlumbar puncture, that the vertebral venous plexus, or Batson's venous plexus, is a potential route of portosystemic shunting.⁵ The veins of the vertebral venous plexus are longitudinally orientated, contain no valves, and form complex connections with the femoral and iliac veins, inferior and superior vena cava, and pelvic venous structures.⁶ The CT scan in our patient showed vertebral venous air emboli in the basivertebral vein (Fig 4). Furthermore, it is recognized that there is continuity of the vertebral and cerebral venous systems⁶ and that the vertebral venous plexus therefore provides a potential route for the spread of infection, tumors,

and, importantly in this case, emboli.⁷ This, therefore, is a possible mechanism by which our patient developed an embolic stroke.

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