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A Case of Portal Venous Gas Secondary to Acute Appendicitis Detected on Gray Scale Sonography but Not Computed Tomography

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The presence of portal venous gas (PVG) has been reported most often in association with bowel ischemia or infarction but may result from nonischemic conditions such as bowel trauma or surgery, inflammatory or tumoral ulcerative disease, or infectious or inflammatory bowel disease. In a setting of mesenteric infarction, the presence of PVG has been reported to be an ominous finding; however, when PVG is related to nonischemic conditions, clinical outcome seems to be determined by the underlying disease and not by the presence of PVG. Therefore, diagnosis of PVG is of clinical importance because quick medical or surgical treatment of the underlying condition may be warranted. Both computed tomography (CT) and gray scale or Doppler sonography contribute to the detection of PVG, with CT being more sensitive in identifying the underlying cause of PVG. Recent reports suggest that sonography may be more sensitive in detecting PVG than CT.

We report a case of PVG detected on gray scale sonography that went undetected on CT. The cause of PVG in this case was acute appendicitis, which to our knowledge has never been reported in the literature.

Case Report

A 38-year-old man had acute abdominal pain associated with high fever, nausea, and vomiting for 24 hours. On admission the patient had a 38°C fever and signs of peritonitis predominating in the right lower quadrant. Laboratory workup revealed a white blood cell count of 10,500, a 3% shift to the left, a C-reactive protein level of 8 mg/L, and no acidosis or high lactate blood levels.

Abdominal sonographic examination performed on admission showed PVG detected as myriads of hyperechoic foci flowing within the intrahaepatic and extrahepatic portions of the portal vein and disseminated in the center and at the periphery of the liver parenchyma (Figure 1). Aside from showing a small quantity of free fluid in the pelvis, sonographic examination was nonconclusive; in particular, the appendix could not be visualized.

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Figure 1. A and B. Gray scale sonograms of the liver. Note diffuse hyperechoic foci (arrows) within the liver parenchyma and the portal vein compatible with PVG.

Figure 2. Computed tomographic images with typical acute appendicitis findings obtained 95 minutes after sonography. A and B. Postcontrast CT, serial axial images at the level of the cecum. The origin of the appendix (white arrow) is surrounded by periappendiceal fat infiltration. A small appendicolith (white arrowhead) is shown. C. Non–contrast-enhanced CT showing the middle portion of the appendix containing a second appendicolith (arrow). D. The cross-sectional diameter of the appendix is increased to 13.8 mm. Note the absence of air bubbles within the appendiceal wall or in the surrounding tissues.
Non–contrast- and contrast-enhanced CT was performed 95 minutes after sonographic examination. The CT findings showed an enlarged appendix with a cross-sectional diameter of 13.8 mm, associated with periappendiceal fat infiltration, and 2 appendicoliths, compatible with acute appendicitis (Figure 2). Non–contrast- and contrast-enhanced CT failed to show the presence of PVG (Figure 3) or air bubbles within the appendiceal wall or in the appendiceal mesentery (Figure 2). Sonographic examination was repeated immediately after the CT. It confirmed the presence of PVG, although visualization of the inflamed appendix remained impossible.

The patient underwent emergency laparoscopic appendectomy, which confirmed preperforated phlegmonous appendicitis. Results of the postoperative sonographic examination of the liver were normal. Surgical follow-up was uneventful after 3 days of prophylactic intravenous imipenem; in particular, repeated liver sonographic findings and liver transaminase levels were consistently normal. Blood culture results were negative. The pathologic examination of the appendix was compatible with acute, ulcerohemorrhagic appendicitis.

Discussion

This case corroborates the finding that PVG can be missed on CT but detected on sonography. Only 7 similar cases were found in the literature, suggesting that this is a rare finding. Although Doppler studies, which have been advocated as being more sensitive than gray scale sonography in detecting PVG, were not performed in our case, the gray scale sonographic findings were characteristic of PVG. Thus, other differential diagnoses to echogenic foci moving in the portal vein, such as erythrocyte “rouleaux formations,” were not retained. This case provides further evidence that sonography, even when only gray scale imaging is performed, is more sensitive in the detection of PVG than CT.

There may be several reasons for this. Intermittent or transitory discharge of gas into the portal venous system could result in the disappearance of PVG by the time CT is performed. This is unlikely in our case, however, because a second sonographic examination was performed several minutes after the negative CT results, confirming the presence of PVG. Lafortune et al suggested that gases within the intrahepatic portal venous system may become rapidly soluble, fail to accumulate inside the terminal portal venous branches, and thus escape detection on CT. This also seems unlikely in our case because static gas was detected in the periphery of the intrahepatic portal system as nonmoving, reverberating, hyperechoic foci and theoretically should have been detected on CT. Although the volume of air bubbles passing through the portal vein seemed large to the eye during real-time sonographic observation, in fact it could have been overestimated by the high sensitivity of sonography in showing up to a few moving air bubbles at a time. Therefore, the volume of PVG observed in our case could have been very small and could have fallen under the spatial resolution of CT, even when differences in density were accentuated after contrast product administration.

Infectious and inflammatory bowel disease has been reported as a cause of PVG, and Tsai et al reported a case of PVG and small-bowel...
obstruction appearing after appendectomy; however, we have found no mention in the literature of PVG in association with acute appendicitis. In the case discussed here, we considered PVG to be secondary to acute appendicitis, given the absence of other radiologically or surgically identifiable large- or small-bowel disease and because PVG was no longer sonographically detectable after appendectomy. Although inflammatory appendices do not generally contain gas within their lumen, endoluminal gas formation secondary to bacterial proliferation or infectious damage to the appendicular wall could explain the passage of air into the portal system. Both of these mechanisms are probable in this case because the appendix was found to be ulcerohemorrhagic on pathologic examination.

The recognition of PVG modified the clinical strategy because the patient was considered to have a higher risk for developing secondary liver abscesses.18,19 This prompted rapid surgical treatment and large-spectrum intravenous antibiotic therapy during the 3 days after surgery. Postoperative follow-up was uneventful.

References