Case Report

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Transient Mesenteric Ischemia: A Complication of Carbon Dioxide Angiography

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THE use of intraarterial carbon dioxide for diagnostic angiography was pioneered by Hawkins in the 1970s and 1980s (1). Recent improvements in digital subtraction angiography, postprocessing computer software, and CO₂ delivery systems have resulted in an increased interest in the use of CO₂ as an alternative intravascular contrast agent in patients with life-threatening reactions to iodinated contrast material or a history of renal insufficiency. CO₂ is appealing because it is a natural product of metabolism, is highly soluble in aqueous solution, has no known nephrotoxicity, and has been documented to be safe when used intravascularly (2–4). However, serious side effects related to the use of CO₂ have been described (5–7). We report a case in which a patient developed transient mesenteric ischemia after intraarterial use of CO₂.

CASE REPORT

A 62-year-old woman with a history of insulin-dependent diabetes, hypertension, chronic renal insufficiency, and a long history of cigarette use presented with a nonhealing ulcer involving the right lower extremity. Physical examination revealed a nonhealing ulcer over the anterior surface of the right lower leg. The right femoral and popliteal pulses were diminished and the right pedal pulses were only detectable with Doppler. Laboratory tests revealed a preprocedure blood urea nitrogen level of 66 mg/dL (23.6 mmol/L of urea) and a creatinine level of 6.5 mg/dL (575 μmol/L). Because of the marked renal insufficiency, a CO₂ angiogram was requested.

Consent was obtained from the patient regarding the use of CO₂ as an intravascular contrast agent. From a left common femoral artery approach, a CO₂ angiogram was obtained with use of a plastic bag delivery system (Angiodynamics, Glen Falls, NY) described by Hawkins (8) (Fig 1). The reservoir bag, tubing, and syringes were set up and carefully purged with CO₂ to eliminate any chance of contamination of the system with room air (8). Care was taken to flush the catheter with saline to prevent thrombus formation. There was no evidence of a leak in the CO₂ system during the course of the procedure.

The abdominal aortogram was obtained with use of a 5-F Sos Omni selective (2) catheter with side holes (Angiodynamics) positioned at the level of the renal arteries. Three separate manual injections of 40–60 mL of CO₂ were made into the abdominal aorta at 2–3-minute intervals to define the abdominal aorta, renal artery origins, aortic bifurcation, and iliac arteries. After the first injection of CO₂ into the abdominal aorta, the patient immediately reported abdominal pain and nausea. The patient was given antiemetics (4 mg of ondansetron hydrochloride; GlaxoWellcome, Research Triangle Park, NC) intravenously. Several minutes later, the study was resumed. In addition, during the course of the remainder of the examination, the patient was rotated from side to side to promote dissipation of the CO₂ from the visceral vessels. The patient report no further episodes of abdominal pain or nausea during the procedure.

The Sos Omni selective (2) catheter was advanced around the aortic bifurcation into the right common iliac artery, and a right lower extremity arteriogram was obtained with use of several 30-cm³ manual injections of CO₂. Approximately 2 minutes separated each CO₂ injection.

The study revealed extensive atherosclerotic disease of the abdominal
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Figure 1. CO₂ plastic bag delivery system consists of a reservoir bag (solid straight arrow), a 60-cm³ CO₂ delivery syringe (open arrow), and a 3-ml purge syringe (curved arrow).

The aorta. The celiac and superior mesenteric arteries were widely patent, but the inferior mesenteric artery was occluded (Fig 2). A high-grade (80%–90%) stenosis was present in the right external iliac artery.

After consultation with the vascular surgeon, balloon angioplasty of the high-grade right external iliac lesion was successfully performed. The entire diagnostic study and intervention were performed without the use of any iodinated contrast material. The patient received approximately 800–1,000 cm³ of CO₂ during the examination. At the conclusion of the procedure, the patient reported the urge to defecate, followed shortly by a single episode of diarrhea. This episode was not associated with any abdominal pain. The patient was closely monitored overnight, but reported no abdominal pain, nausea, or urge to defecate.

The next morning (postprocedure day 1), the patient reported the onset of vague abdominal pain accompanied by diarrhea. The patient had several loose bowel movements, which eventually became bloody. The abdominal examination revealed a soft abdomen. Blood urea nitrogen level had increased to 83 mg/dL (29.6 mmol/L) and the creatinine level had increased to 7.8 mg/dL (689.5 μmol/L). The white blood cell count had increased from 14.0 K/μL to 26.9 K/μL, and the hematocrit had increased from 34% to 52.6%. The serum glucose rose from 154 to 321 mg/dL (8.5–17.8 mmol/L). No ketones were present in the urine. The blood eosinophil count was 0.1%. Stool cultures were also obtained and were found to be negative. The patient was afebrile with a blood pressure of 112/80 mm Hg and a heart rate of 105. The patient reported no nausea and there was no vomiting. There were no skin changes to suggest cholesterol emboli.

Our assessment was that the patient had dehydration and hypovolemic. Intravenous hydration was initiated, and the general surgery and gastrointestinal medicine services were consulted for further evaluation of the bloody diarrhea. On the second postprocedure day, lower gastrointestinal endoscopy was performed and revealed pale colonic mucosa with scattered areas of hyperemia and punctate hemorrhages in the nondes- tinate portions of the colon, specifically the hepatic flexure, splenic flexure, and sigmoid colon. The mucosa appeared viable throughout the colon (Fig 3).

The patient was managed conservatively with hydration and bowel rest. The patient's renal function and hematocrit returned to baseline during the next 5 days with gentle hydration. The patient was discharged home on the 7th postprocedural day without diarrhea, abdominal pain, or nausea, and was able to tolerate a normal diet. The presumed diagnosis was transient mesenteric ischemia secondary to CO₂ administration.

DISCUSSION

Interest in CO₂ as an intravascular contrast agent for patients with renal insufficiency or a history of severe contrast material reaction is increasing because of improvements in digital imaging technology and CO₂ delivery system. Because CO₂ is a gas, it does not mix with blood, but rather “floats” above it. To accurately assess the luminal characteristics of a vessel, the blood within the vessel must be transiently replaced by CO₂. Therefore, enough CO₂ must be administered to fill the entire vessel to avoid underestimating the diameter of the vessel. An inadequate volume of CO₂ results in the gas floating to the anterior surface, incompletely delineating the posterior wall of the vessel. For larger vessels, such as the aorta, a rapid manual injection of 40–60 cm³ of CO₂ is typically used to deliver an adequate volume of CO₂. In addition, with the bag delivery system used in our patient, 3 cm³ of CO₂ is delivered via a separate 3-mL syringe to purge the catheter of blood or saline immediately prior to each digital acquisition. By eliminating the fluid from the catheter prior to the 40–60-cm³ bolus injection, the explosive delivery associated with compression of the CO₂ behind the fluid in the catheter is minimized (6). In our experience, use of less than 40 cm³ of CO₂ in the abdominal aorta results in underfilling of the abdominal aorta, and the potential for overestimation of disease
within the abdominal aorta and proximal renal arteries.

The bag delivery system (Angiodynamics) contains a series of one-way valves within glued three-way stopcocks and ports for attaching the reservoir, 50-mL delivery syringe, and 3-mL "purge" syringe. When assembled, the bag delivery system minimizes the chance for inadvertent delivery of room air into the patient. Room air could be delivered to the patient if the syringes attached to the bag delivery system are loose or the tubing from the CO₂ reservoir is loose or detached. Prior to each injection in our patient, these three potential sources of air contamination were checked to ensure that there was no leakage.

The supine patient, the anterior position of the celiac, superior mesenteric, and inferior mesenteric arteries leads to preferential filling of these vessels with CO₂ during CO₂ injections into the abdominal aorta. In addition, during selective injection of the iliac vessels, reflux of CO₂ may lead to filling of the inferior mesenteric artery. This phenomenon has been previously described (6).

Accumulation of CO₂ in these nondependent vessels can subsequently result in trapping of the CO₂, causing a "vapor lock" in the vessel. When a large interface is present between the blood and CO₂, the CO₂ normally dissolves very quickly and is eliminated ultimately by the lungs (6). Occasionally, a bolus of CO₂ can become "trapped" within a vessel, especially a nondependent vessel, and cause a vapor lock obstructing the normal flow of blood. In addition, the pressure of the gas may prevent collateral flow from entering the affected vascular distribution (9), thereby compromising tissue perfusion.

Cardi and Hawkins have described two patients with abdominal aortic aneurysms in whom abdominal pain, cramping, and diarrhea developed after CO₂ angiography (6). It was postulated that vapor lock contributed substantially to the symptoms. Clinically, these patients were believed to manifest transient mesenteric ischemia without infarction (6). Our patient initially developed abdominal pain and nausea. Because of concern about a vapor lock phenomenon, the amount of CO₂ injected was reduced, the time interval between CO₂ injections was increased, and the patient was intermittently rolled into different positions to make the visceral vessels more dependent and to facilitate blood flow and CO₂ dissipation. The subsequent clinical findings of bloody diarrhea, vague abdominal pain, abdominal distension, dehydration with increased fluid requirements, and the laboratory findings of leukocytosis and hemocoagulation, were suggestive of mesenteric ischemia. The endoscopic findings were also consistent with mesenteric ischemia.

It is unlikely that an embolus or in situ thrombosis of the superior mesenteric artery or its branches would be the cause for the clinical and endoscopic findings because the entire colon was affected in a segmental distribution (hepatic and splenic flexures and sigmoid area). Likewise, venous thrombosis would also be an unlikely cause of transient mesenteric ischemia in this patient. A low-flow state could cause transient mesenteric ischemia, but there was no episode of hypotension or cardiac instability during or immediately after the arteriogram. An air embolus due to contamination of our bag delivery system with room air could also cause transient mesenteric ischemia. However, this is unlikely because every effort was made to prevent accidental injection of room air and no problems with the plastic bag delivery system occurred. Infectious colitis could present with a similar clinical picture, however, negative stool cultures, negative stool smear for leukocytes, and typical endoscopic findings for infectious colitis make this etiology unlikely.

The diagnosis of cholesterol embolization syndrome was also considered in this patient. The lack of the typical skin changes associated with cholesterol emboli in cholesterol embolization syndrome, the rapid improvement in the renal function after hydration, the unusual endoscopic findings localized to the nondependent portions of the bowel, and the lack of
eosinophilia make it unlikely that cholesterol embolization syndrome was the cause of mesenteric ischemia in this patient (10–16).

Therefore, we postulate that CO₂ gas caused a vapor lock and resulted in decreased perfusion to the colon. Because of the buoyancy of CO₂, the ischemic changes were localized to the less dependent portions of the colon (in the supine position), such as the hepatic flexure, splenic flexure, and sigmoid colon. The bloody diarrhea and associated dehydration suggest that the small bowel may also have been transiently affected (6).

In addition, with use of a gelatin vascular model, Hawkins found that CO₂ injected at 200 mL/sec through a multiple side hole catheter was less traumatic than iodinated contrast material injected at 12 mL/sec.

Defining what is an excess dose of CO₂ is quite difficult because it is patient specific and probably dependent on the patient’s underlying anatomy. In our patient, the inferior mesenteric artery was occluded. CO₂ injections were limited to volumes of 80 cm³ or less, the time intervals between CO₂ injections were at least 2 minutes, CO₂ injections were made in a nonexplosive manner with use of the bag delivery system (specifically designed for CO₂), and positional maneuvers were performed to minimize gas air trapping.

Despite these precautions, CO₂ was apparently trapped within the mesenteric distribution and, most likely, resulted in this patient’s transient mesenteric ischemia. The administration of pain medications and antiemetics may also have obscured the symptoms in the immediate postprocedure period. Ultimately, the benign abdominal examination and the visible appearance of the colon at endoscopy allowed us to manage the patient conservatively.

Development of abdominal pain, nausea, vomiting, or diarrhea during CO₂ angiography may herald the onset of CO₂ trapping in the mesenteric distribution. A decrease in the volume of CO₂ injections and an increased in time intervals between CO₂ injections, and positional maneuvers do not guarantee that mesenteric ischemia will be obviated, but such precautions may minimize the degree of ischemia that may occur. In addition, several hours may elapse before symptoms and signs of mesenteric ischemia appear. Therefore, patients in whom abdominal pain, nausea, and the urge to defecate develop during CO₂ angiography should be followed closely with serial abdominal examinations during the next 24 hours and observed for signs and symptoms of mesenteric ischemia.

In conclusion, we present a case in which CO₂ was the most likely cause for the development of transient mesenteric ischemia in a patient undergoing CO₂ angiography. The harbinger of this complication was the development of abdominal pain, nausea, and the urge to defecate during the procedure.

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References