

Case Studies

Isolated Internal Carotid Artery Thrombus and Cerebral Infarction in a Patient with Necrotizing Pancreatitis: Case Report

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Isolated internal carotid artery (ICA) thrombus in the absence of underlying atherosclerotic disease is a rare entity. We report a case of a patient presenting with right arm weakness, slurred speech, and altered mental status in the setting of acute on chronic pancreatitis. The patient was found to have scattered left cerebral hemisphere cortical infarctions, and catheter angiography confirmed the presence of intraluminal left ICA thrombus, with no evidence of atherosclerotic disease in the cervical or intracranial vasculature. Further workup also demonstrated the presence of anemia of chronic disease. The patient was initiated on anticoagulation, and follow-up imaging demonstrated a complete resolution of the left ICA thrombus. In the reported case, coagulopathy in the setting of acute on chronic pancreatitis was presumably the primary etiology. Anemia of chronic disease, related to a proinflammatory state, may also play a contributory role. **Key Words:** Stroke—carotid thrombus—coagulopathy—pancreatitis—anemia.

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Introduction

Thrombus within the carotid artery is uncommon and is generally associated with an underlying atherosclerotic plaque. However, multiple causal factors have been identified in carotid arterial thrombus formation in the

absence of underlying atherosclerosis, including a hypercoagulable state,¹⁻⁴ iron deficiency anemia with thrombocytosis,^{5,6} and the use of stimulant drugs.⁷ Carotid thrombus formation has also been reported in proinflammatory states such as Crohn disease and active pulmonary tuberculosis.^{8,9} We report a case of isolated thrombus in an internal carotid artery (ICA) with an associated middle cerebral artery (MCA) infarction occurring in a patient with necrotizing pancreatitis and anemia of chronic disease.

Case Report

A 37-year-old man with a past medical history of hypertension, gastroesophageal reflux disease, pancreatitis, and alcohol abuse was in his usual state of health until being admitted to the hospital for acute necrotizing

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Received June 6, 2017; accepted August 12, 2017.

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1052-3057/\$ - see front matter

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<http://dx.doi.org/10.1016/j.jstrokecerebrovasdis.2017.08.012>

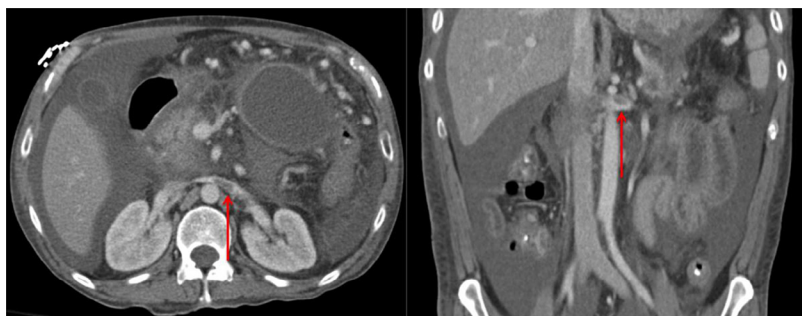


Figure 1. Nonocclusive renal vein thrombus. Contrast-enhanced axial and coronal computed tomography images of the abdomen demonstrate nonocclusive thrombus in the left renal vein (arrow). In addition, there is evidence of acute pancreatitis (pancreatic swelling and extensive peripancreatic fat standing), a rim enhancing intra-abdominal collection, and moderate volume reactive ascites.

pancreatitis, presumed to be secondary to alcohol abuse. A computed tomography (CT) scan of the abdomen and the pelvis at that time demonstrated acute pancreatitis with associated secondary findings, including splenic vein thrombosis; however, anticoagulation was not initiated. The patient was discharged after 1 week and experienced persistent diarrhea and weight loss until he experienced an acute neurologic event that brought him back to the hospital.

One week after the patient was discharged from the hospital, he presented to the emergency department with right arm weakness, slurred speech, and altered mental status. The patient was tachycardic (heart rate 140 beats per minute), hypotensive (blood pressure 87/72 mm of Hg), and febrile (38.9°). Laboratory evaluation demonstrated leukocytosis (17.2 K/mm³), anemia (hemoglobin 10.3 g/dL, mean corpuscular volume 87 fL), with platelets of 365 K/mm³, and elevated lipase (117 units/L). A noncontrast CT of the head showed no evidence of acute infarction or intracranial hemorrhage. Because the patient was outside the time window for thrombolytic therapy, a CT angiogram was not performed to evaluate for large-vessel occlusion. Cerebrospinal fluid evaluation did not demonstrate any abnormal findings. CT evaluation of the abdomen and pelvis showed findings of acute pancreatitis superimposed upon chronic pancreatitis, with necrosis of the pancreatic tail, multiple rim enhancing intra-abdominal fluid collections, reactive enteritis, moderate volume ascites, and splenic vein thrombosis. The patient was also found to have a new

nonocclusive thrombus in the left renal vein (Fig 1). He was admitted to the intensive care unit and started on broad-spectrum antibiotics.

Brain magnetic resonance imaging 3 days after admission showed scattered left cerebral hemisphere cortical and subcortical acute infarcts with hemorrhagic transformation (Fig 2). Concurrent magnetic resonance angiography suggested the presence of a filling defect within the proximal left ICA. Catheter angiography performed the following day confirmed the presence of intraluminal left ICA thrombus, with no evidence of atherosclerotic disease in the cervical or intracranial vasculature (Figs 3, 4). Echocardiography revealed no intracardiac source of thrombus, and there was no evidence of a patent foramen ovale.

The patient was started on parenteral heparin. As the patient's fever remitted and leukocytosis improved, antibiotics were changed from broad spectrum to narrow spectrum. The patient's intra-abdominal collections were managed conservatively. Iron (Fe) studies showed an Fe of 54 mcg/dL (normal range: 50-150 mcg/dL), a total iron-binding capacity of 210 mcg/dL (normal range: 250-400 mcg/dL), a transferrin saturation of 26% (normal range: 20%-50%), and ferritin of 836 ng/mL (normal range: 30-400 ng/mL), suggestive of anemia of chronic disease. The follow-up CT angiogram 1 week later showed an evolving cerebral infarction and a slight interval decrease in the size of the internal carotid arterial thrombus (Fig 5). The patient was transitioned to warfarin with an enoxaparin bridge and was started on a statin before discharge.

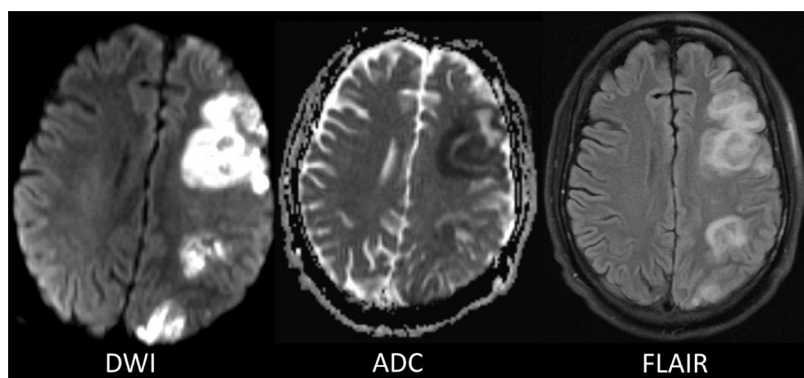


Figure 2. Left middle cerebral artery territory infarction. Magnetic resonance images of the brain demonstrate confluent and punctate areas of diffusion restriction within the territory of the left middle cerebral artery with associated FLAIR signal hyperintensity, consistent with acute infarction. Abbreviations: ADC, apparent diffusion coefficient; DWI, diffusion-weighted imaging; FLAIR, fluid-attenuated inversion recovery.

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