Reperfusion Injury—Postendovascular Recanalization in Chronic Mesenteric Ischemia: A Rare Clinical Case Scenario

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Abstract

Regardless of the number of vessels involved endovascular recanalization of mesenteric vessels is the treatment of choice for chronic mesenteric ischemia. Reperfusion injury post-endovascular recanalization in chronic mesenteric ischemia is a rare clinical scenario as it is mostly encountered in cases of acute mesenteric ischemia. Here in, we describe a case with characteristic clinical and imaging findings of reperfusion syndrome, post-endovascular recanalization of chronically occluded superior mesenteric artery and severely stenosed celiac trunk in a patient with chronic mesenteric ischemia.

Keywords

- chronic mesenteric ischemia
- reperfusion bowel injury
- endovascular recanalization

Introduction

Patients with chronic mesenteric ischemia (CMI) typically present with insidious onset of postprandial abdominal pain associated with significant weight loss. The association of pain with food intake often leads to food aversion. It is commonly seen in elderly patients with risk factors for atherosclerosis. Smoking, diabetes mellitus, and hypertension are most common risk factor.[1] Because of the extensive collateral network, usually at least two of the three visceral vessels are affected before patients develop symptoms.[2] However, given the spectrum of gastrointestinal disturbances associated with abdominal pain and weight loss, the diagnosis is often made late.[3]

Most patients present with occlusion or stenosis of superior mesenteric artery (SMA) and at least one other mesenteric vessel, most commonly the celiac trunk. Endovascular therapy is the first line of treatment.[4] Procedure-related complications such as hematoma at the access site, dissection, thrombosis, distal embolism, and contrast-induced nephropathy are the most commonly described complications after endovascular therapy.[5] Intestinal ischemic reperfusion injury (RI) is a life-threatening condition with high morbidity and mortality and is almost always seen in the setting of acute mesenteric ischemia. Contrary to the expectation, restoration of blood flow to the ischemic bowel further exaggerates cell injury. We present a case in which recanalization of SMA and celiac artery in single sitting for CMI was accompanied by the typical manifestation of reperfusion bowel injury.

Case Presentation

The patient is a 50-year-old male who had insidious onset severe postprandial abdominal pain associated with weight loss of 6 to 7 kg in 2 to 3 months. He had aversion to solid food and was on liquid diet. The patient was a chronic smoker and had smoked about 2 packs of cigarettes daily for 25 years. There was no history of diabetes or hypertension. On computed tomography (CT) angiography, there was complete atherosclerotic occlusion of SMA (15 mm length) and inferior mesenteric artery (IMA) for a length of 60 mm. There was also 90% stenosis involving the celiac trunk for a length of 4 mm (►**Fig. 1A**).

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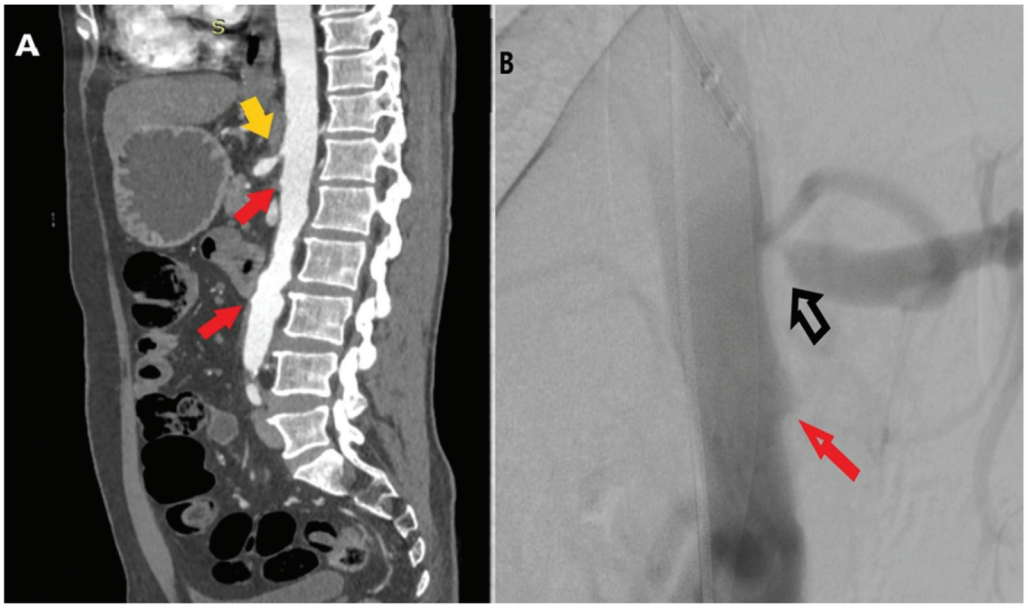

Fig. 1 (**A**) Computed tomography sagittal section showing 90% stenosis of celiac trunk (yellow arrow) with complete occlusion of superior mesenteric artery (SMA) and inferior mesenteric artery (red arrow). (**B**) Digital subtraction angiography lateral view showing 90% stenosis of celiac trunk (black arrow) with complete occlusion of SMA (red arrow).

The patient was referred to interventional radiology department for endovascular recanalization from gastroenterology department.

Through right brachial approach, aortography performed in lateral view confirmed complete atherosclerotic occlusion of the SMA and IMA with a critical stenosis of 90% involving the celiac trunk (►**Fig. 1B**). An intravenous heparin bolus (5000 IU) was administered. A 6F 90cm guiding sheath (Destination; Terumo, Tokyo, Japan) was placed in the abdominal aorta and SMA ostium was engaged with a 6F multipurpose angiographic guide catheter. The lesion in SMA was crossed with a 0.014-inch microwire (Command ES; Abbott vascular, Illinois, United States) and angioplasty was performed with 5×40 mm balloon (►**Fig. 2A**). The SMA was stented with an 8×27 mm balloon expandable stent (EXPRESS LD, Boston Scientific, Massachusetts, United States; ►**Fig. 2B**). Similarly, the celiac trunk was cannulated, the lesion was crossed with a 0.014-inch microwire, and balloon angioplasty was performed with a 5×40-mm balloon (►**Fig. 2C**). The celiac trunk was stented with an expandable 8×29 balloon stent (OMNILINK ELITE; Abbott vascular, Illinois, United States). Complete recanalization of SMA and celiac trunk was achieved (►**Fig. 2D**).

Post-procedure next day, postprandial abdominal pain was replaced by mild generalized abdominal discomfort. Ultrasonography of abdomen showed patent stents and mild diffuse bowel wall edema and hyperperistaltic bowel loops. After 48 hours, abdominal discomfort increased and was associated with bloating, abdominal distention and two episodes of vomiting. He was afebrile. There was no guarding or tenderness in abdomen. There was no signs of infection and hemoglobin was stable. Ultrasonography and plain CT of abdomen revealed edematous bowel loop and mild-to-moderate free fluid in the peritoneal cavity (►**Fig. 3**). The condition was consistent with reperfusion bowel injury secondary to hyperperfusion following mesenteric artery revascularization. Bowel rest was given, and patient was kept nil per oral for 48 hours followed by only sips of water for next 24 hours. He was on parenteral fluid therapy and gradually liquid diet followed by soft diet was started for him. Ultrasonography on 6th post-procedure day showed near complete resolution of bowel edema and ascites. Patient was discharged on seventh post-procedure day tolerating soft diet.

Discussion

RI is caused following the restoration of blood flow to an ischemic organ resulting in additional cellular damage. The underlying mechanism of RI is not specific to the intestine and is a consequence of oxidative stress following reperfusion, leading to an increase in reactive oxygen species (ROS), and local inflammation leading to cell death. This complex and multifactorial pathophysiological process is not yet fully understood and has been studied in preclinical models in various organs, particularly the heart, brain, liver, and intestine.[3]

The intestine is one of the most sensitive organs for RI. In RI injury, the barrier function of the mucosa is destroyed, and vascular permeability is increased. The increased vascular permeability allows inflammatory cells to activate and adhere. These inflammatory cells release ROS, proinflammatory chemokines, and protein kinases. In addition, disruption of the intestinal barrier allows bacterial translocation that further stimulates inflammatory response.[6]

In the setting of RI, CT scan shows bowel wall thickening (>5mm) in association with mucosal hyperenhancement, submucosal edema, mesenteric stranding, or free fluid. It may also be associated with venous dilatation in the adjacent mesentery.[5]

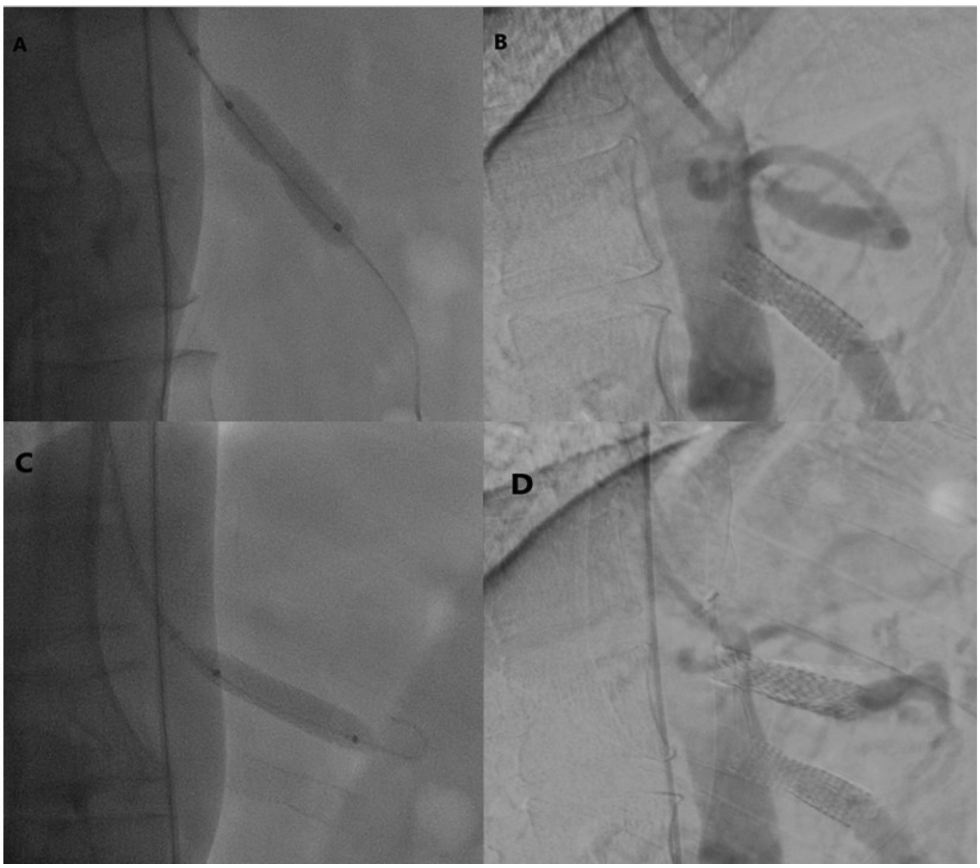

Fig. 2 (**A**) Digital subtraction angiography (DSA) lateral view showing representative image of superior mesenteric artery (SMA) balloon angioplasty. (**B**) DSA lateral view showing angiographic run post-SMA stenting. (**C**) DSA lateral view showing representative image of celiac trunk balloon angioplasty. (**D**) DSA lateral view showing angiographic run post-SMA and celiac trunk stenting.

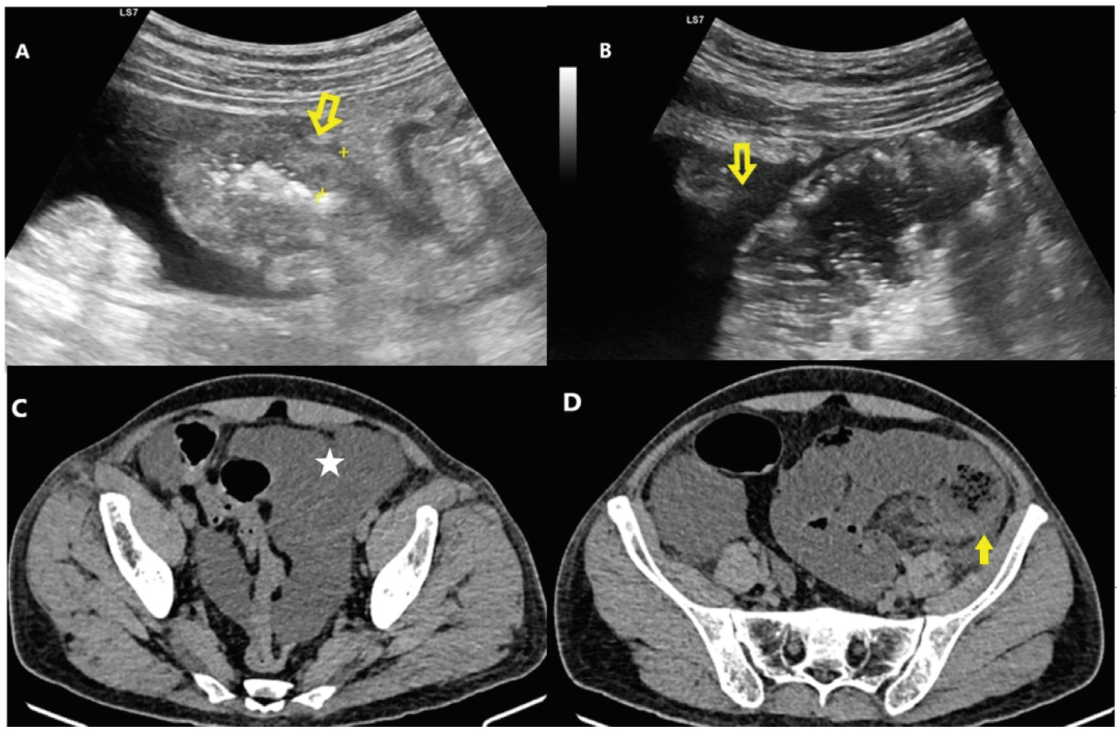

Fig. 3 (**A**) Ultrasound image showing thickened edematous bowel wall (yellow arrow). (**B**) Ultrasound image showing peritoneal free fluid (yellow arrow). (**C**) Computed tomography (CT) image showing pelvic free fluid (asterisk). (**D**) CT image showing edematous bowel wall thickening (yellow arrow).

The severity of RI varies with severity of ischemia (partial vs. complete), onset of ischemia (acute vs. chronic), duration of time ischemia has been present, and the bowel segment involved. This phenomenon primarily occurs when there is abrupt interruption of blood flow in circumstances like embolic phenomenon, acute mesenteric thrombosis, hypovolemic shock, aortic dissection, and aortic aneurysm surgery.[4]

In CMI due to gradual nature of occlusion and secondary collateralization, there is some degree of ischemia conditioning of the bowel. Therefore, RI is uncommon in CMI. Moreover, the degree of severity of RI in this scenario even if it develops is relatively mild considering the chronic nature of disease where bowel is already primed to ischemia. We could find only a single report of reperfusion bowel injury post-endovascular revascularization in CMI. Here also both celiac trunk and SMA were revascularized in a single session.[3] Clinical and imaging findings resolved completely within a week. If this is a complication unique to simultaneous revascularization of celiac artery and SMA, we do not have an answer.

It is generally recommended to avoid simultaneous revascularization of both carotid arteries in single sitting to avoid reperfusion brain injury. Deriving a corollary from carotid circulation and considering the common pathophysiology of RI irrespective of the organ involved, it seems reasonable to avoid simultaneous revascularization of celiac artery and SMA.

Patient Consent

Informed consent was obtained from the patient for publication of the case report and accompanying images.

Funding

None.

Conflict of Interest

None declared.

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