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CASE REPORT

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Progressive abdominal pain in a 63-year-old man

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Abstract

50%–60% of patients with chronic mesenteric ischemia suffer from concomitant cardiovascular disease. We therefore suggest an extensive diagnostic screening to detect coronary artery and peripheral arterial disease in these patients.

KEYWORDS

atherosclerosis, mesenteric ischemia, renal artery obstruction

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We report on a patient with known metabolic syndrome and a newly diagnosed total occlusion of the superior mesenteric artery. The importance of evaluation of the overall atherosclerotic burden in these patients as well as therapy strategies is discussed.

A 63-year-old man presented in the emergency department with abdominal pain, postprandial nausea, and weight loss of 14 kg over the last 4 months. Signs of infection, fever, or night sweats were not present. A recent upper endoscopy and colonoscopy revealed an erosive antrum gastritis and duodenitis as well as a benign polyp in duodenum, which was successfully removed. The patient had a history of asymptomatic primary biliary cholangitis (PBC) confirmed by positive antimitochondrial antibodies and elevated alkaline phosphatase (AP) 3 years ago. A moderate to severe liver fibrosis (Metavir F2-F3) was also diagnosed. Liver cirrhosis was not present at the time of the diagnosis. A treatment with ursodeoxycholic acid was initiated leading to normalization of elevated transaminases. However, AP (initially, 355 U/L) and gamma-glutamyltransferase (GGT; 968 U/L), though decreasing continuously, remained slightly elevated after 3 years of treatment. Thus, therapy was intensified by adding a bezafibrate. Supporting the choice of the therapy was a newly diagnosed hypercholesterolemia (low-density cholesterol of 166 mg/dl) and elevated triglycerides (210 mg/dl). Other cardiovascular risk factors included arterial hypertension and a history of smoking (approximately 20 pack years with cessation 30 years ago). There was no history of diabetes mellitus or familial predisposition for cardiovascular diseases. The patient also suffered from chronic kidney disease of suspected vascular origin.

Initial abdominal ultrasound showed no signs of free intraperitoneal fluid, bowel obstruction, or suspicious masses. Lipid panel (low- and high-density cholesterol, triglycerides,

lipoprotein [a], transaminases, bilirubin, and AP) was normal at admission; there was a slight elevation of GGT present. A subsequent abdominal computed tomography (CT) revealed a severe atherosclerosis of the abdominal aorta with heavily calcified long chronic total occlusion (CTO) of the celiac axis and the superior mesenteric artery (SMA; Figure 1). In addition, the right renal artery was totally occluded and a severe stenosis of the left renal artery was present resulting in a single functional kidney.

The symptomatic SMA occlusion was treated via a transradial approach. After successful crossing of the occlusion the lesion was pre-dilated using balloon angioplasty (POBA) and subsequent stent implantation with a self-expandable stent (7/40 mm). A residual focal stenosis within the self-expandable stent was treated with a focal balloon-expandable stent. Final angiography revealed a significantly improved perfusion, despite a remaining slight stenosis. A total occlusion of the celiac axis was not intervened due to adequate collateral circulation. Dual antiplatelet therapy (DAPT) with acetylsalicylic acid and clopidogrel for 1 month as well as a statin therapy were initiated. Abdominal pain ceased completely shortly after the intervention and the patient reported a gain of 11 kg in the following 5 weeks.

To further determine the atherosclerotic burden, duplex ultrasonography of the extracranial and lower extremity arteries revealed minimal atherosclerotic changes and no relevant stenoses. Coronary angiography was performed and showed a three-vessel disease with normal systolic left ventricular function without significant stenoses.

Chronic mesenteric ischemia (CMI) is commonly caused by atherosclerotic narrowing of the celiac or mesenteric arteries. Other causes include median arcuate ligament syndrome (Dunbar syndrome), fibromuscular dysplasia, vasculitis etc.[1] Chronic mesenteric ischemia is a slowly progressing disease which remains asymptomatic over years in around 60% of the cases, because of compensating mesenteric collateral circulation.[2] Otherwise postprandial abdominal pain, weight loss, and diarrhea are typical symptoms of CMI.[3] The first-line therapy in patients with a symptomatic multi-vessel mesenteric artery stenosis is a percutaneous mesenteric artery stenting,[4] which represented the strategy in our case in addition to secondary prevention of atherosclerosis.

Even though no further signs or symptoms raising suspicion of coronary artery disease or peripheral arterial disease were present, we chose to perform an extensive diagnostic, partly invasive screening of coronary, carotid and lower extremity arteries, based on supporting data, that 50%–60% of patients with CMI suffer from concomitant cardiovascular disease.[5] Moreover, 42.7% of patients with coronary artery disease (CAD) had a concomitant mesenteric artery stenosis at the time of coronary angiography, in 20.4% CMI was clinically relevant.[6] A significant renal artery stenosis is a common concomitant finding with prevalence of 15% in patients undergoing a routine coronary angiography and 30%–56% in patients with peripheral arterial disease [7] and is significantly associated with mesenteric artery stenosis.[8] In the current case, no relevant arterial disease of coronary, carotid, and lower extremity arteries was found. Which vascular bed is affected by atherosclerotic changes, is determined by the patient's genetic background, gender, immune status, and oxidative stress, influencing the reaction of different vascular sites to flow disturbances, systemic risk factors.[9]

Primary biliary cholangitis is also associated with hyperlipidemia, which is reported in more than 75% patients with PBC.[10] There exist several mechanisms of hypercholesterolemia in PBC, including reduced hepatic clearance of cholesterol due to cholestasis and presence of lipoprotein-X, an abnormal lipoprotein rich in free cholesterol and phospholipids.[11] In the current case, however, the metabolic syndrome with multiple cardiovascular risk factors was rather the cause of CMI, as the cholestasis was well managed under the treatment with fibrates.

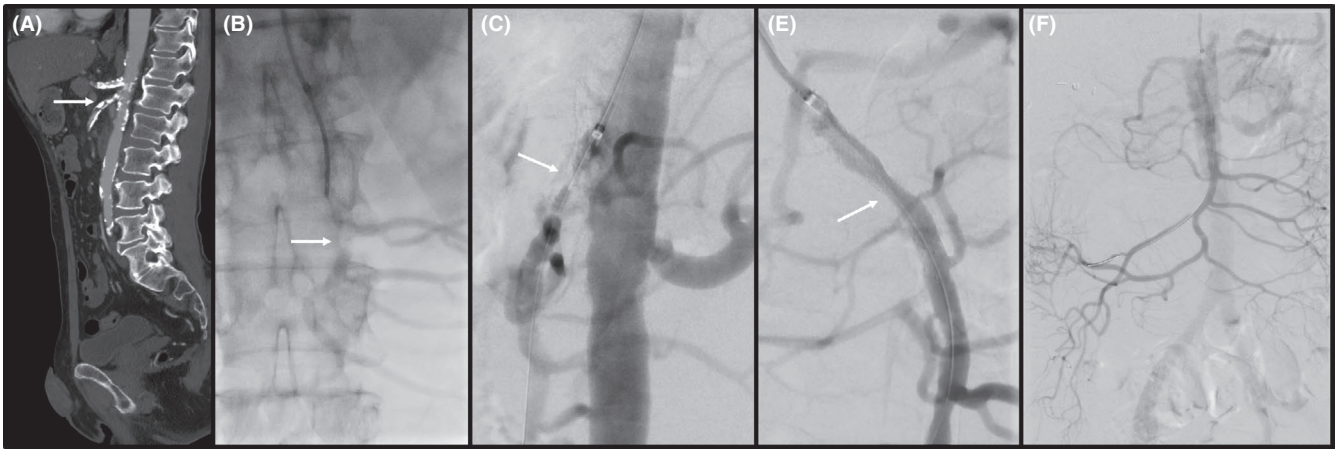

FIGURE 1 A, Chronic total occlusion of the celiac axis and superior mesenteric artery (SMA) in abdominal computed tomography; B, Sagittal view of the occluded SMA and its branches; C, SMA stenosis directly after balloon angioplasty; D, Remaining 30% SMA stenosis after stent implantation; E, Adequate mesenteric collateral circulation

Finally, worsening renal function as a result of ischemic nephropathy may have contributed to further vascular damage due to accelerated atherosclerosis, inflammation, and endothelial dysfunction.[12] In the current case, however, we opted for a conservative approach considering ischemic nephropathy, as revascularization was not found to significantly improve control of systolic blood pressure compared to medical therapy alone.[13] The effect of the percutaneous revascularization on renal function is still debatable, as only 25%–30% of the patients show improved renal function after renal artery revascularization.[14]

In conclusion, in patients with CMI, an early and extensive diagnostic screening should be considered due to high prevalence of concomitant peripheral arterial disease.

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CONFLICT OF INTEREST

None of the authors has any conflict of interest.

AUTHOR CONTRIBUTIONS

MZ: involved in conception, drafting, and critical revision of the manuscript. KW: involved in drafting and critical revision of the manuscript. DR: involved in drafting and critical revision of the manuscript. GG: involved in drafting and critical revision of the manuscript. K-PK: involved in critical revision of the manuscript. RH: involved in critical revision of the manuscript. MG: involved in critical revision of the manuscript.

ETHICAL APPROVAL

I hereby consciously assure that the manuscript is the authors' own original work, which has not been previously published elsewhere and is not currently being considered for publication elsewhere. All authors have been personally and actively involved in substantial work leading to the paper and will take public responsibility for its content.

CONSENT STATEMENT

Published with written consent of the patient.

DATA AVAILABILITY STATEMENT

Data sharing not applicable to this article as no datasets were generated or analysed during the current study.

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