Mesenteric ischaemia in a case of acute anterior myocardial infarction: overlap of ischaemic types

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Acute mesenteric ischaemia is divided into different clinical entities which are usually considered separately. Here we report a case of acute mesenteric ischaemia complicated with acute anterior myocardial infarction. The clinical picture suggested that non-occlusive mesenteric ischaemia and acute mesenteric arterial thrombosis were both present in this case. Thus, non-occlusive and occlusive ischaemia may coexist in a coordinated and perceptible pattern.

KEYWORDS: acute myocardial infarction, non-occlusive mesenteric ischaemia, intestinal obstruction, perforation, stent placement

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Introduction

Acute mesenteric ischaemia (AMI) comprises a number of different primary clinical entities: non-occlusive mesenteric ischaemia (NOMI) and occlusive mesenteric arterial ischaemia (OMAI); OMAI may be further subdivided into acute mesenteric arterial embolism (AMAE) and acute mesenteric arterial thrombosis (AMAT). AMI as venous disease takes the form of mesenteric venous thrombosis (MVT).1

Each type has somewhat different predisposing factors, clinical features and prognoses. AMAE often has a cardiac aetiology, for example mural thrombi after myocardial infarction (MI), atrial thrombi associated with mitral stenosis and atrial fibrillation. Patients with AMAT have atherosclerotic disease. Low cardiac output resulting from MI or congestive heart failure (CHF) may cause AMI in a patient with visceral atherosclerosis. NOMI is precipitated by a severe reduction in mesenteric perfusion, with secondary arterial spasm from such causes as cardiac failure, septic shock, hypovolemia or the use of potent vasopressors.2

When mesenteric ischaemia occurs in a patient with myocardial infarction, both the occlusive and non-occlusive types may contribute to the pathophysiologic process. We report such a case, where AMI is complicated with bowel obstruction and delayed perforation in a patient with acute anterior myocardial infarction.

Case report

A 59-year-old man with a body mass index of 26 kg/m² presented to the chest pain centre with chest pain and tightness that had lasted for 1 hour. On arrival, systolic blood pressure was 90 mmHg, heart rate 89 bpm and respiratory rate 19 breaths/min. He had a medical history of atrial fibrillation and hypertension of 10 years’ duration, and had had a myocardial infarction 4 years ago with stent placement in the left anterior descending (LAD) artery.

The first ECG on arrival indicated atrial fibrillation and ST-T elevation in leads of V1-V3 (Fig 1). A blood examination on admission revealed elevated high-sensitive troponin I (52.0 ng/mL), N-terminal pro-brain natriuretic peptide (3,193 pg/mL), D-dimer (152 ng/mL) and creatinine kinase myocardial band (196.5 U/L). ECG and blood tests were repeated 1 hour after arrival. The dynamic change of troponin and wave of leads V1-5 indicated acute anterior intensive myocardial infarction. Emergency coronary angiography showed that the distal end of the stent placed 4 years ago in the LAD artery was 90% narrowed and no significant left main coronary artery stenosis. Two pieces of white embolism were successfully removed by aspiration from the ostium of the existing stent and a new stent was placed in the LAD next to the old one.

The day after his admission to the intensive care unit, the patient complained of aggressive abdominal distension with temporary nausea and vomiting. Intensive small bowel distension with pneumatisms and effusion was showed on uncontrasted abdominal CAT scan (Fig 2). Bladder pressure was 15 mmH₂O. NOMI and adynamic intestinal obstruction were diagnosed according to physical examination and radiologic features. Nasogastric suction and best supportive treatment had little effect on his abdominal symptoms in the following 3 days. In the assessment of the superior mesenteric artery (SMA) on computed tomographic angiography (CTA), moderate to heavy stenosis was found at the origin of SMA (Fig 3). The distal vessel supply was normal in angiography. Multidisciplinary team consultation concluded that NOMI and AMAT were both causes of bowel dysfunction, which should have recovered within a few days of the acute myocardial infarction, and the patient decided to continue with conservative treatment. The cardiovascular system recovered in process. On the eighth day of admission, the patient suffered from abdominal pain with signs of peritonitis and an abdominal CT scan indicated bowel perforation. Several lesions of necrosis and perforation were found in the terminal ileum in urgent laparotomy (Fig 4). Most segments of small bowel showed normal blood supply with adynamic and dilated appearance. The
Inflammatory change was verified by pathologic examination. On the eleventh day of admission, the patient underwent a stent placement in the superior mesenteric artery.

Characteristics of small bowel necrosis and perforation indicated an inflammatory result instead of main mesenteric artery thromboembolism.

**Fig 1.** ECG after admission, showing elevated lead V1-5.

**Fig 2.** Small bowel obstruction on computed tomography.
Fig 3. Stenosis at the origin of SMA (marked with arrow).

Fig 4. Perforation of the terminal ileum, with normal blood supply of adjacent segment.
fourteenth day of admission, symptoms of abdominal distension relieved after the recovery of bowel function. The patient was transferred to the common ward and discharged at the end of third week of admission. Follow-up in the surgery department was unremarkable. The patient had tested negative for COVID-19 throughout this time.

Discussion

The prevalence of each type of AMI has changed over recent decades. The prevalence of mesenteric arterial embolism has decreased to 25% of cases, and is no longer the first cause of AMI, while mesenteric arterial thrombosis has increased to 40% and is now the most common cause. NOMI accounts for 25% of cases, which is also an increase on the historical cohort. The border line between AMAT and NOMI might be unclear, particularly with cases of visceral atherosclerosis is rising.

The diagnosis of NOMI in this case was supported by three points. The first was absence of acute peritonitis. The second was the clinical scenario of myocardial infarction, which is regarded as one of most common causes of NOMI. The third was successful resection and anastomosis of the small bowel. Normal terminal blood perfusion was verified in the operation.

The diagnosis of AMAT was supported by two points. The first was heavy stenosis at the origin of the SMA observed in CTA. The second was that small bowel obstruction was released only after the placement of a stent in the SMA.

As we noted above, the trends in the likelihood of each type of AMI are changing and the border line between NOMI and AMAT can be unclear when a complicated case emerges in emergency room. In this case, non-occlusive and occlusive ischaemia coexisted in a coordinated and perceptible pattern. Overlapping of AMI types has not been widely discussed or reported before. The increasing number of illness and intensive care triggered more NOMI in recent years and the mechanism underlining was unclear.

The occlusive mesenteric ischaemia also played a role because the obstruction of the small bowel was relieved only after stent placement at the origin of arteries. This intervention was delayed by our initial diagnosis of NOMI, as well as by the medical burden caused by COVID-19 at the end of 2022 in China.

The analysis of clinical process indicates that an overlapping type of mesenteric ischaemia existed in this case. Resection and anastomosis of terminal ileum without any complication was strong testification of NOMI that played key role in early ischaemia. Recovered bowel function after stent placement was obvious evidence of AMAT. NOMI overlapped AMAT in this case. Such an overlap could be the aetiology of an ‘acute on chronic’ presentation.

References


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