EJCRIM
European Journal
of Case Reports
in Internal Medicine

REVERSIBLE 'UNSTABLE' ABDOMINAL ANGINA CAUSED BY RUPTURED PLAQUE OF THE SUPERIOR MESENTERIC ARTERY: CLINICAL AND RADIOLOGICAL CORRELATIONS

Shaul Yaari[1], Nurith Hiller[2], Yacov Samet[3], Samuel N. Heyman[1]

[1] Department of Medicine, Hadassah Hebrew University Hospital, Mount Scopus, Jerusalem, Israel
[2] Department of Radiology, Hadassah Hebrew University Hospital, Mount Scopus, Jerusalem, Israel
[3] Department of Vascular Surgery, Shaare Zedek Medical Center, Jerusalem, Israel

Corresponding author: Samuel N. Heyman **e-mail:** Heyman@cc.huji.ac.il

Received: 16/01/2023 **Accepted:** 22/02/2023 **Published:** 30/03/2023

Conflicts of Interests: The Authors declare that there are no competing interests.

How to cite this article: Yaari S, Hiller N, Samet Y, Heyman SN. Reversible 'unstable' abdominal angina caused by ruptured plaque of the superior mesenteric artery: clinical and radiological correlations. *EJCRIM* 2023;**10**:doi:10.12890/2023_003766.

ABSTRACT

Unstable angina, characteristic of coronary artery disease, is caused by in-situ clot formation complicating ruptured atheromatous plaque. Abdominal angina, however, usually reflects chronic mesenteric ischaemia, caused by multi-vessel stable plaques involving mesenteric arteries. Herein, we describe a patient with new-onset abdominal pain caused by a ruptured atheromatous plaque at the superior mesenteric root. The diagnosis was based on an evident reversible epigastric bruit and high-degree eccentric stenosis caused by a non-calcified atheroma. Symptoms and bruit resolved within 3 weeks on aspirin and statins with regression of the stenotic lesion. Although the condition is likely common, this is the first clear-cut report compatible with 'unstable' abdominal angina, resolved by conservative treatment.

KEYWORDS

Mesenteric ischemia, abdominal angina, atherosclerosis, unstable angina, superior mesenteric artery, atheromatous plaque

LEARNING POINTS

- Resembling unstable angina pectoris, ruptured atheromatous plaque in mesenteric vessels can develop, clinically manifested by new-onset abdominal angina.
- This condition may be reversible under treatment with antiplatelet medications and statins.
- Searching for abdominal bruit is invaluable in the assessment of unexplained abdominal pain.

BACKGROUND

Mesenteric ischaemia is classified as either chronic or acute (CMI and AMI, respectively). Atherosclerosis has been implicated in the aetiology of the overwhelming majority of cases of CMI, characterized by chronic abdominal visceral pain aggravated by meals and associated with anorexia and weight loss[1]. In most cases, CMI reflects multi-vessel chronic stable mesenteric disease, with substantial mesenteric ischaemia beyond the mitigating capacity of hypoxia adaptation[2,3] and the formation of collaterals[4,5]. By contrast, AMI reflects an acute arterial occlusion, manifested by an abrupt and catastrophic presentation. The superior mesenteric artery

EFIM
European Federation of Internal Medicine

Publisher
smc media srl

(SMA) is involved in 67% of patients with AMI, its occlusion most often caused by an arterial embolus, facilitated by its branching at a narrow angle from the aorta. Nonetheless, plaque rupture and in-situ thrombosis is the cause of acute mesenteric arterial occlusion in some 42% of patients[6].

Thus, the pathogenesis of CMI and atherosclerosis-related AMI parallel that of stable angina pectoris and acute myocardial infarction, respectively. While CMI reflects gut ischaemia during enhanced oxygen requirement due to chronically restricted blood supply, cases of atherosclerosis-related AMI are considered to represent total occlusion of arterial flow caused by in-situ thrombus formation.

We report a patient with an unusual reversible sub-acute presentation of mesenteric ischaemia, likely caused by a ruptured plaque, resembling pathophysiologically the cardiac clinical disorder termed 'unstable angina'. Although the condition is likely clinically prevalent, surprisingly, it has not previously been reported with imaging.

CASE DESCRIPTION

A 55-year-old woman was evaluated in our outpatient clinic for epigastric abdominal pain lasting 3 weeks. The pain started abruptly, became continuous, and was associated with loss of appetite. The pain was dull, non-colic, non-radiating and unaffected by posture. There was some intensification of pain an hour or so following meals. Weight remained stable. Her medical history was remarkable for untreated hypertension and dyslipidaemia and recent cessation of smoking following a 30 pack-year history.

The patient had been evaluated repeatedly during the preceding 3 weeks in different Emergency Medicine departments, where epigastric tenderness was noted as the sole clinical finding. She underwent broad laboratory evaluation, abdominal ultrasound and contrast-enhanced computerized tomography (CT), all reported as unremarkable. She was treated with omeprazole 40 mg daily without amelioration of symptoms. Subsequent

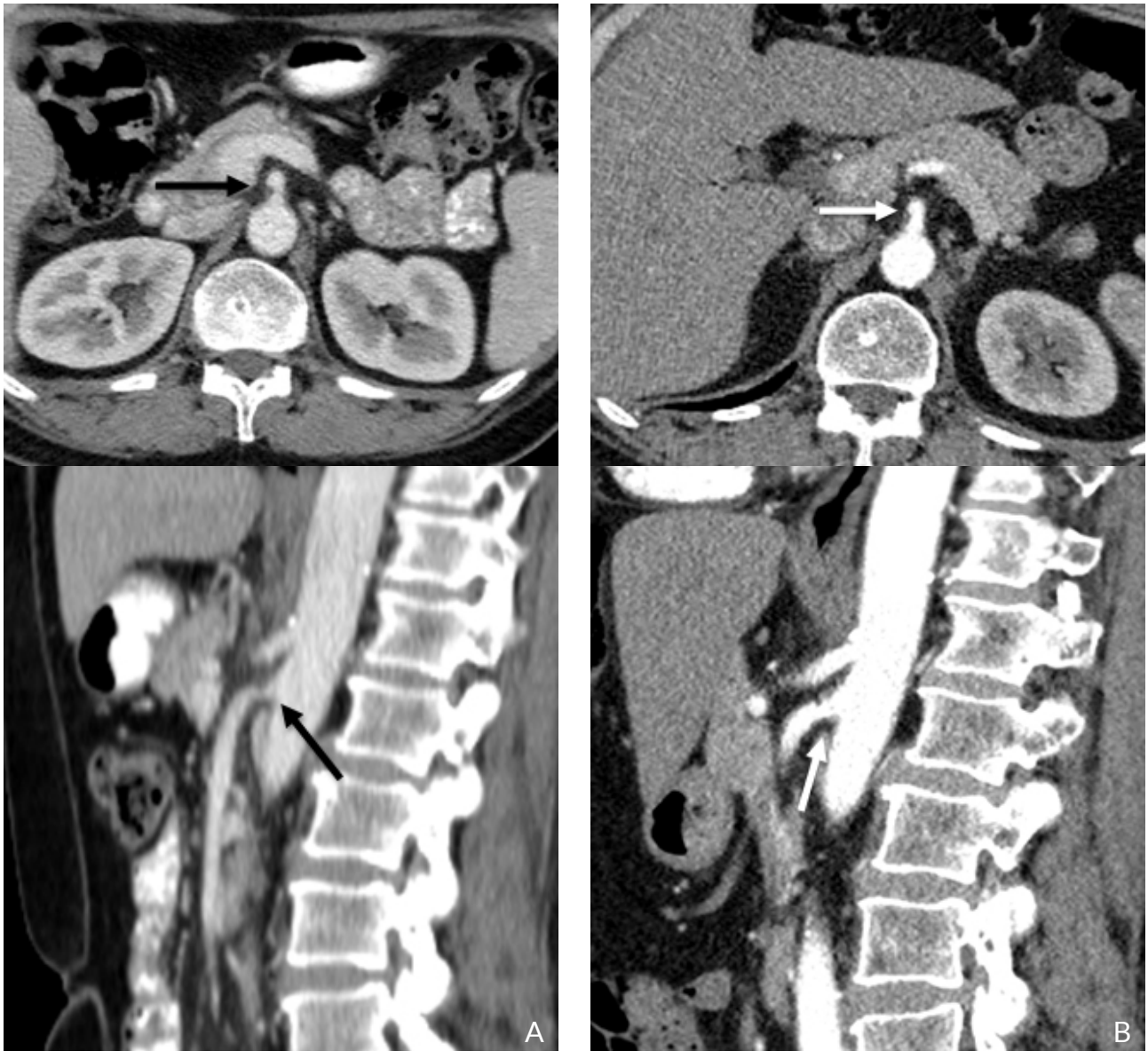

Figure 1. Imaging of changes in superior mesenteric artery (SMA) stenosis. (A) Contrast-enhanced CT of the abdomen revealing eccentric plaque at the origin of the SMA causing 60–80% stenosis (black arrows). (B) CT angiography, performed 4 weeks later, shows regression of the high-degree stenosis to about 20% of the cross-sectional area (white arrows), with disappearance of the flap caused by the ruptured plaque

evaluation in a gastroenterology clinic, complemented by oesophagogastroduodenoscopy and colonoscopy, was unremarkable, and an endoscopic retrograde cholangiopancreatography (ERCP) was scheduled.

On examination in our outpatient clinic, epigastric tenderness and a distinct regional bruit were noted, prompting revision of the CT. Indeed, an overlooked high-grade non-calcified eccentric stenosis of approximately 60–80% was noted at the origin of the SMA *(Fig. 1A)*. Calcified atheromas were detected in the abdominal aorta and coronary arteries; however, the remaining abdominal arteries, including the celiac and inferior mesenteric arteries, were intact.

The patient was given aspirin and atorvastatin. Pain declined and resolved within 3 weeks and the abdominal tenderness and bruit disappeared. Repeat CT revealed regression of the stenosis at the SMA origin to approximately 20% *(Fig. 1B)*. Blood pressure was controlled with ramipril and the patient has remained well for the past 6 years.

DISCUSSION

Our patient presented with reversible mesenteric ischaemia, likely caused by a ruptured atheromatous plaque at the origin of the SMA. Our assumption is based on the concomitant association of symptoms with the presence of bruit and documented high-degree stenosis, all regressing in parallel, and with the absence of alternative diagnoses.

This report describes an uncommon and possibly overlooked presentation of gut ischaemia related to atherosclerosis. Following reported cases of acute episodes of abdominal pain predating a fatal acute mesenteric event, Berman and Russo coined the term 'abdominal angina', making an analogy between intra-abdominal vascular disease and the vessels of the heart[7]. In the 1980s and early 1990s, a series of studies elucidated the pathological changes underlying the various clinical entities comprising acute coronary syndromes[8,9]. These studies demonstrated that unstable angina, a term adopted by Wood in 1961[10], is caused by ruptured plaque of the coronary artery and incomplete thrombotic occlusion, causing chest pain without elevation of cardiac enzymes indicating muscle necrosis. Clot forms on top of the ruptured atheromatous plaque but its growth is countered by activated local fibrinolysis. With anti-platelet agents, anticoagulants and statins, thrombolysis prevails over thrombogenesis, resulting in 'stabilization' of the ruptured plaques. However, left untreated, unstable angina usually intensifies, with an advancing limited functional capacity, reflecting thrombus expansion and progressing luminal occlusion. Acute myocardial infarction finally develops, with acute thrombosis completely obliterating the diseased vascular segment.

With advanced imaging modalities and physical examination, we were able to document an acute and protracted episode of abdominal pain associated with non-occlusive high-degree atherosclerotic stenosis of the SMA that resolved with anti-platelet treatment and statins. Thus, we would like to take Berman and Russo's analogy[7] a little step forward and name this clinical presentation as a case of 'unstable' abdominal angina. A Medline search was conducted using the terms 'unstable angina', 'abdominal arteries' and 'mesenteric arteries', but failed to identify any other cases of unstable abdominal angina in the literature. It is likely that this presentation is not rare, although under-detected, and reflects abrupt partial occlusion of a single mesenteric artery, that may resolve or even remain asymptomatic with the generation of collateral circulation or with resolution of the clot adhering to the unstable plaque.

CONCLUSIONS

As opposed to an acute mesenteric event that progresses to gut necrosis, abdominal angina is a reversible pain caused by transient sub-lethal mesenteric ischaemia. It is aggravated by meals, reflecting enhanced post-prandial mesenteric oxygen demand, with mesenteric stenosis most often due to a stable atheromatous plaque, limiting adequate enhancement of regional blood flow. Gut viability is often maintained by compensatory collateral circulation. Herein, we provide for the first time radiological evidence for a comparable pattern of symptoms that developed acutely, caused by an unstable atheromatous plaque. We term this phenomenon 'unstable' abdominal angina, as it pathophysiologically parallels unstable angina due to ruptured coronary atheromatous plaque that often precedes acute myocardial infarction. Likely, 'unstable' abdominal angina eventually resolves spontaneously even if the mesenteric vessel becomes occluded, as collateral circulation develops, occasionally leading to 'stable' abdominal angina.

This case report highlights the importance of auscultation, looking for abdominal bruit, in the evaluation of patients with unexplained abdominal pain.

REFERENCES

1. Derrick JR, Pollard HS, Moore RM. The pattern of arteriosclerotic narrowing of the celiac and superior mesenteric arteries. *Ann Surg* 1959;**149**:684–689.
2. Heyman SN, Leibowitz D, Mor-Yosef Levi I, Liberman A, Eisenkraft A, Alcalai R, et al. Hypoxia-adaptive response and remote ischemia preconditioning: a new hypoxia inducible factors era in clinical medicine. *Acta Physiol (Oxf)* 2016;**216**:395–406.
3. Wong YL, Lautenschläger I, Hummitzsch L, Zitta K, Cossais F, Wedel T, et al. Effects of different ischemic preconditioning strategies on physiological and cellular mechanisms of intestinal ischemia/reperfusion injury: implication from an isolated perfused rat small intestine model. *PLoS One* 2021;**16**:e0256957.
4. Sardar P, White CJ. Chronic mesenteric ischemia: diagnosis and management. *Prog Cardiovasc Dis* 2021;**65**:71–75.
5. van Petersen AS, Kolkman JJ, Meerwaldt R, Huisman AB, van der Palen J, Zeebregts CJ, et al. Mesenteric stenosis, collaterals, and compensatory blood flow. *J Vasc Surg* 2014;**60**:111–119.
6. Acosta S. Epidemiology of mesenteric vascular disease: clinical implications. *Semin Vasc Surg* 2010;**23**:4–8.
7. Berman LG, Russo FR. Abdominal angina. *N Engl J Med* 1950;**242**:611–613.
8. Fuster V, Badimon L, Badimon JJ, Chesebro JH. The pathogenesis of coronary artery disease and the acute coronary syndromes. *N Engl J Med* 1992;**326**:310–318.
9. Sherman CT, Litvack F, Grundfest W, Lee M, Hickey A, Chaux A, et al. Coronary angioscopy in patients with unstable angina pectoris. *N Engl J Med* 1986;**315**:913–919.
10. Wood P. Acute and subacute coronary insufficiency. *Br Med J* 1961;**1**(5242):1779–1782.