

Simultaneous Thromboembolism Of The Superior Mesenteric And Both Renal Arteries

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Citation

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Abstract

Thromboembolism of the peripheral circulation is relatively common but that of the visceral arteries is rare. We report a case of simultaneous atheroembolism of the superior mesenteric artery and both renal arteries from a left ventricular mural thrombus, six weeks following a myocardial infarction in a 79-year-old woman. Remarkably, the peripheral circulation was unaffected. This has not been reported before. The patient died from multi-organ dysfunction syndrome. Whilst, bilateral renal infarction was diagnosed on the CT scan, the infarction of the small bowel was revealed only at the post-mortem examination. This case illustrates that emboli from the heart can lodge in an unusual manner in the visceral circulation without affecting the peripheral circulation. A high index of suspicion is required to diagnose mesenteric ischaemia, especially when complicated by other intra-abdominal pathologies like renal infarction.

INTRODUCTION

Atheroembolism or thromboembolism of the peripheral arteries of the lower limbs from a left ventricular mural thrombus is relatively common, whilst embolism of the superior mesenteric artery (SMA) is rare and that of the renal artery is rarer still. We report a case of simultaneous atheroembolism of both renal arteries and the SMA from a left ventricular mural thrombus, six weeks following a myocardial infarction. Remarkably, the peripheral circulation was unaffected.

CASE REPORT

A 79-year-old female presented to the Accident and Emergency department with a two-day history of sudden onset, severe abdominal pain accompanied by vomiting and diarrhoea. Her past medical history included diverticulosis, hypertension, and anterior Q-wave myocardial infarction 6 weeks ago (which required stenting of the left anterior descending coronary artery). Her medication on admission was Aspirin 75 mg and Clopidogrel 75 mg, which she took daily.

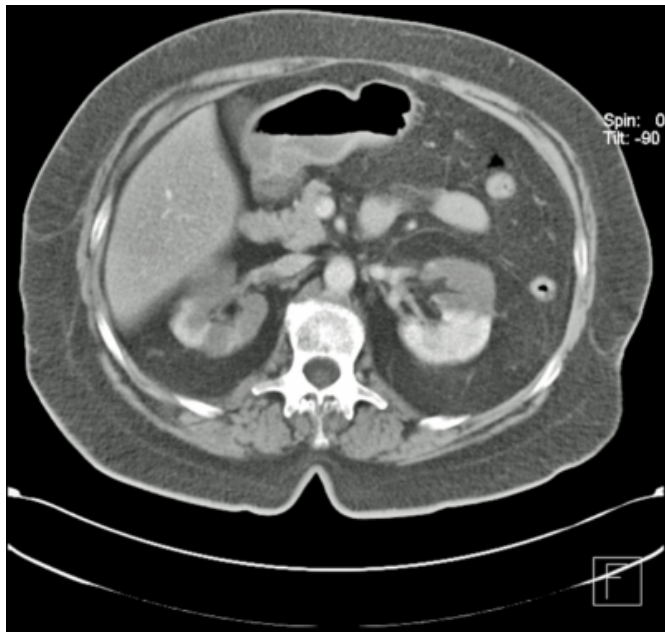
She was haemodynamically stable. Both the respiratory and cardiac examinations were unremarkable. There was a full complement of palpable peripheral pulses. The abdominal examination revealed moderate to severe tenderness, localized mainly over the left lower quadrant. Digital rectal examination was normal.

Blood tests on admission noted leukocytosis ($25.2 \times 10^9/L$) and an elevated serum urea and creatinine (11.0 mmol/L and 142 $\mu\text{mol/L}$ respectively). Arterial blood gases showed a base excess of minus 2.4, and the serum lactate was within normal limits. Chest and abdomen radiographs were both unremarkable, whilst an electrocardiogram noted sinus tachycardia with no acute ischaemic changes.

Based on these findings, a tentative diagnosis of acute diverticulitis was made. The deterioration in renal function was assumed to be pre-renal (due to dehydration). An abdominal CT scan (Fig 1) performed 6 hours later revealed patchy but well demarcated areas of cortical hypoperfusion in both the kidneys representing infarction. Diverticular disease was also noted in the sigmoid colon but there was no evidence of active inflammation.

Figure 1

Figure 1: Non-contrast CT scan of the abdomen showing patchy but well demarcated areas of cortical hypoperfusion in both the kidneys representing infarction



The patient was hereafter treated as a case of acute renal failure due to bilateral renal infarction (secondary to thromboembolism). The source of emboli was assumed to be the heart due to a recent history of myocardial infarction. Subsequent transthoracic echocardiogram, however, failed to detect any intra-cardiac thrombus.

The patient was managed on the ward. There was slight improvement in her gastrointestinal symptoms but her renal function continued to deteriorate. On day seven the serum creatinine rose to 526 $\mu\text{mol/L}$ and the urea to 29.6 mmol/L . Arterial blood gas noted a base excess of minus 9. She was transferred to the Intensive care unit for haemofiltration and responded to initial treatment. There was a marked improvement in her renal function and a fall in the blood white cell count, too. However, she continued to have mild to moderate abdominal pain and tenderness, which was attributed to her infarcted kidneys. On the tenth day following admission, there was a sudden deterioration in her condition. She became febrile and profoundly hypotensive. There was severe generalized abdominal tenderness. The white cell count was elevated to $33 \times 10^9/\text{L}$. She was ventilated and commenced on inotropes. Despite attempted resuscitation she had a cardiac arrest and died.

Post mortem examination revealed an old fibrotic infarction of the anterior wall of the left ventricle, measuring 35 x 7 mm, with central areas of haemorrhage. There was a

thrombus attached to the endocardial surface at the site of infarction. This measured 7 x 5 mm. The right renal artery had an occlusive red thrombus whilst the left renal and the superior mesenteric arteries had non-occlusive thrombi. The whole of the small intestine contained infarcted mucosa with relative preservation of the muscularis mucosa. The histology of the kidneys showed extensive coagulative necrosis.

DISCUSSION

Arterial atheroembolism has an incidence of about 10% in the visceral vessels, 20% in the cerebrovascular circulation and 70% in the peripheral circulation.⁽¹⁾ Visceral emboli preferentially lodge in the superior mesenteric artery (SMA) due to its oblique origin from the abdominal aorta.⁽²⁾ Thrombo-embolism of the renal arteries is rare whilst, simultaneous embolization of both renal arteries and the SMA has not been reported before. Assuming that a large embolus or multiple emboli inundated the circulation, we find it difficult to explain why the emboli selectively lodged in the SMA and the renal arteries and failed to reach the distal circulation.

The most important source of spontaneous arterial macroemboli is the heart. Two thirds to three fourths of these are due to dysarrhythmia and 20% are secondary to myocardial infarction.⁽³⁾ Left ventricular (LV) mural thrombus is a well-recognized complication of acute myocardial infarction. The pathophysiology involves a combination of dyskinesis and freshly infarcted inflamed myocardium containing abundant fibrin. This serves as a nidus for platelet aggravation and activation of the coagulation cascade.⁽⁴⁾ The resultant thrombus is composed of fibrin, red blood cells and platelets.⁽⁵⁾

The overall incidence of a mural thrombus at post-mortem in patients dying from myocardial infarction is reported to be 30-40%^(6, 7). In the patients who survive myocardial infarction, the incidence of LV mural thrombus depends on the size, site and the imaging modality used to detect it. Large anterior Q-wave infarction has higher incidence of mural thrombus as compared to inferior Q-wave and non-Q-wave infarctions⁽⁴⁾. The transthoracic 2-D echocardiography is the most common imaging modality used to detect a mural thrombus. It has a reported sensitivity of 95% and specificity of 86%.⁽⁵⁾ It must be noted that this is subject to limitations concerning patients' habitus and operator experience. Half of all mural thrombi are visible 48 hours after a myocardial infarction and 90% within the first week. Rarely, the

thrombus may appear one month after the infarction.⁽⁸⁾

Acute mesenteric ischemia results in bowel infarction that has a mortality rate of 60% to 80%.⁽⁹⁾ The infarcted bowel brings about severe metabolic changes which rapidly lead to multi organ dysfunction syndrome and death. Despite better understanding of the pathophysiology of acute mesenteric ischaemia and advances in imaging and treatment modalities, the mortality remains largely unchanged over the past 70 years. It is difficult to recognize the condition before infarction sets in.⁽¹⁰⁾ A dynamic contrast enhanced CT scan may improve the sensitivity to 64% and specificity to 92%⁽¹¹⁾. Mesenteric angiography using a multislice CT scanner can further improve the sensitivity.⁽¹²⁾

Use of papavarine infusion via angiography catheter can prevent arterial spasm and help in reperfusion of the intestine. If the mesenteric ischaemia has been diagnosed without angiogram, glucagon infusion may be used with similar effect.⁽¹³⁾ Embolectomy of the SMA and resection of necrosed bowel may sometimes be required.

In this particular case the initial signs and symptoms of mesenteric ischaemia were further masked by the presence of bilateral renal infarction and resulting acute renal failure. Furthermore, the emboli had not completely occluded the lumen of SMA, causing non-occlusive mesenteric ischaemia. This resulted in a more protracted course and widespread infarction of the mucosa of the small bowel with relative preservation of the muscularis and serosa.

CONCLUSION

This case illustrates that emboli from the heart can lodge in an unusual manner in visceral circulation without affecting the peripheral circulation. A high index of suspicion is required to diagnose mesenteric ischaemia especially when complicated by other intra-abdominal pathologies like renal infarction.

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