

A Case of Acute Type B Aortic Dissection: Limited Role of Laboratory Testing for the Diagnosis of Mesenteric Ischemia

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A 30-year-old man with severe back and abdominal pain was referred to our hospital because of a recurrence of acute type B aortic dissection. A computed tomography scan showed a 3-channel dissection and a severe narrowing of the true lumen of the descending aorta to the abdominal aorta because of the expansion of the newly formed second false lumen. Although laboratory testing, including creatine phosphokinase, lactate dehydrogenase, and lactate levels, indicated no visceral ischemia, abdominal pain requiring narcotics treatment had to be continued for more than 1 week. Based on the symptoms and computed tomography findings, the patient finally underwent aortic replacement, fenestration, and a reconstruction of the inferior mesenteric artery, after which the abdominal pain disappeared. Operative findings confirmed a pale shrunken intestine, indicative of mesenteric ischemia. The present case is a good demonstration revealing that mesenteric ischemia still remains a diagnostic challenge, and suggests that currently available laboratory markers are not sensitive enough to detect the presence of ischemia. A strong clinical suspicion for mesenteric ischemia may be the only key to preventing a catastrophic outcome in this condition. (Ann Thorac Cardiovasc Surg 2007; 13: 360–364)

Key words: acute aortic dissection, mesenteric ischemia, mesenteric necrosis, surgical repair

Introduction

Acute aortic dissection (AAD) is a lethal condition affecting the aorta. Without appropriate treatment, approximately 75% of patients with AAD die within 2 weeks of the disease onset.¹⁾ The principal cause of early death, particularly in patients with proximal dissection, is aortic rupture. However, dissection also involves the branches of the aorta and could obstruct the branch ostia. When malperfusion affects the central nervous system or abdominal viscera, the mortality rate increases dramatically.

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The reported mortality rates of patients presenting with mesenteric ischemia are 45%–87%.²⁻⁴⁾ According to the International Registry of Acute Aortic Dissection (IRAD) data, 15% of all the deaths of patients with type B dissection were related to mesenteric ischemia.⁵⁾ Although surgical therapy is required for patients with visceral ischemia, the repair is often delayed, and organ necrosis may develop. This is mainly because mesenteric ischemia is extremely difficult to diagnose before necrosis develops. Herein we describe the case of an AAD presenting with mesenteric ischemia in which repetitive laboratory testing failed to indicate it.

Case Report

A 30-year-old man was referred to our hospital for acute type B aortic dissection. He had no family history of aor-

Table 1. Clinical course

	Arrival ↓										Surgery ↓
	0	1	2	3	4	5	6	7	8	9	
Days after onset	0	1	2	3	4	5	6	7	8	9	10
Dose of pentazocine (mg)				15	15	15		30	30	30	
Computed tomography scan	∇	∇									∇
Intestinal gas on abdominal X-ray		-	-	-	-	-	-	-	-	-	
Occult blood in stool		+									
Blood tests:											
D-dimer (normal: <1.0 µg/mL)		16.0	12.5		10.8					6.2	
CRP (normal: <0.3 mg/dL)		1.3	1.7		13.9					14.9	
Lactate (normal: <17 mg/dL)		25	12		8		14			12	
Base excess (normal: -3.0 - +3.0 mmol/L)		-3.4	0.7	0.1	0.4	1.3	0.2	2.5	2.7	2.7	
CPK (normal: 62-287 IU/L)		84	73		74		72		43	40	

∇, performed computed tomography scan; +, present; -, absent. CRP, C-reactive protein; CPK, creatine phosphokinase.

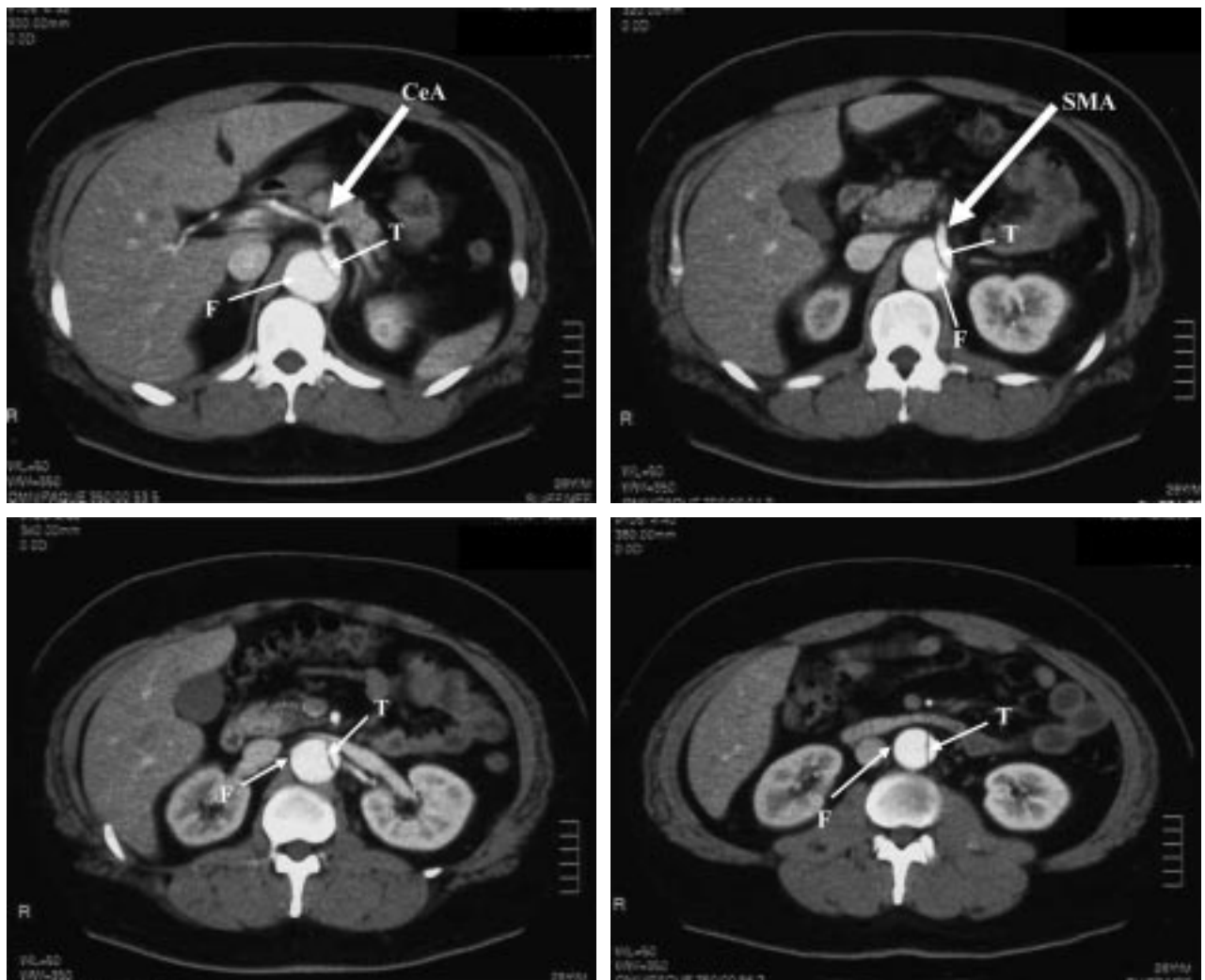


Fig. 1. A computed tomography (CT) scan obtained 12 months after the first occurrence of aortic dissection shows a true lumen (T) compressed by false lumen extending from the distal arch to the right common iliac artery. The celiac arteries (CeA), the superior mesenteric arteries (SMA), and the left renal arteries originate from the true lumen, whereas the right renal artery originates from the false lumen.

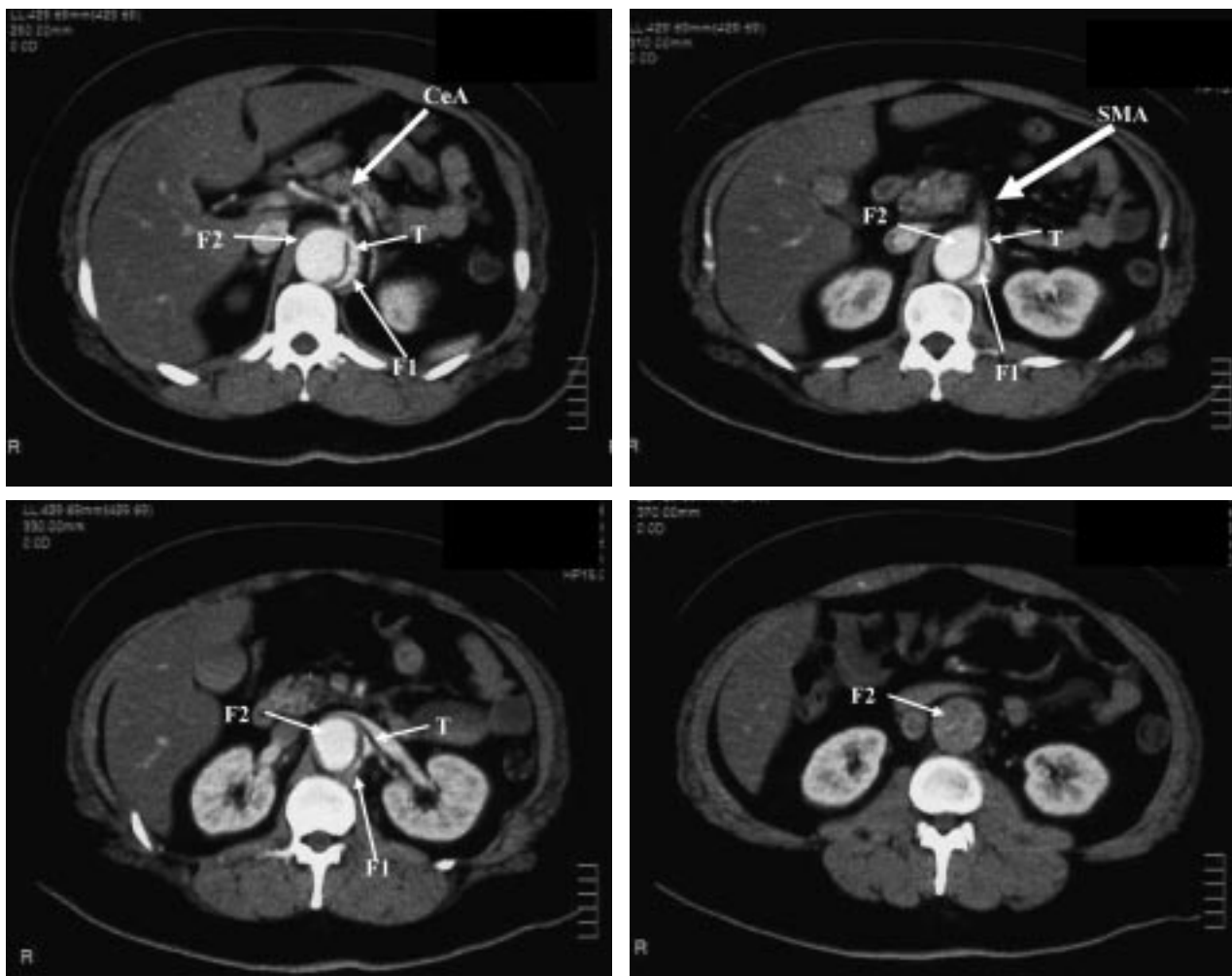


Fig. 2. A computed tomography (CT) scan obtained upon arrival (2 years after the first occurrence of aortic dissection) shows a 3-channel dissection. A newly formed false lumen (second false lumen: F2) is seen anterior to the true lumen (T). The first false lumen (F1), which developed at the time of the first occurrence of aortic dissection, is difficult to identify because of the compression by the second false lumen. The true lumen, from which the celiac and the superior mesenteric arteries arise, is also compressed by the second false lumen.

tic disease and did not meet the diagnostic criteria for Marfan syndrome. He had a history of hypertension and hyperlipidemia.

The patient first had acute type B aortic dissection 2 years earlier. A computed tomography (CT) scan at the time of the first occurrence of aortic dissection revealed an enlarged false aortic lumen, originating from the proximal descending thoracic aorta and extending to the right common iliac artery. The false aortic lumen had compressed the true lumen, from which the celiac, the superior mesenteric, and the left renal arteries originated. The right renal artery arose from the false lumen (Fig. 1).

At this second occurrence of aortic dissection, the patient suffered a severe tearing pain that migrated from his

chest to his back and then to his abdomen. The next day he was referred to our hospital. Physical examination upon arrival revealed blood pressure of 182/78 mmHg and abdominal tenderness around the navel. The pulsation in the lower limbs was decreased, which soon resolved on the day of admission.

A CT scan obtained upon arrival revealed a 3-channel dissection (Fig. 2). The newly formed false lumen (second false lumen, F2), originating from the level of tracheal bifurcation and extending down to the right common iliac artery, was completely thrombosed below the level of the renal arteries. Above this level, the expanded second false lumen severely compressed the true lumen (T), from which the celiac and superior mesenteric arter-

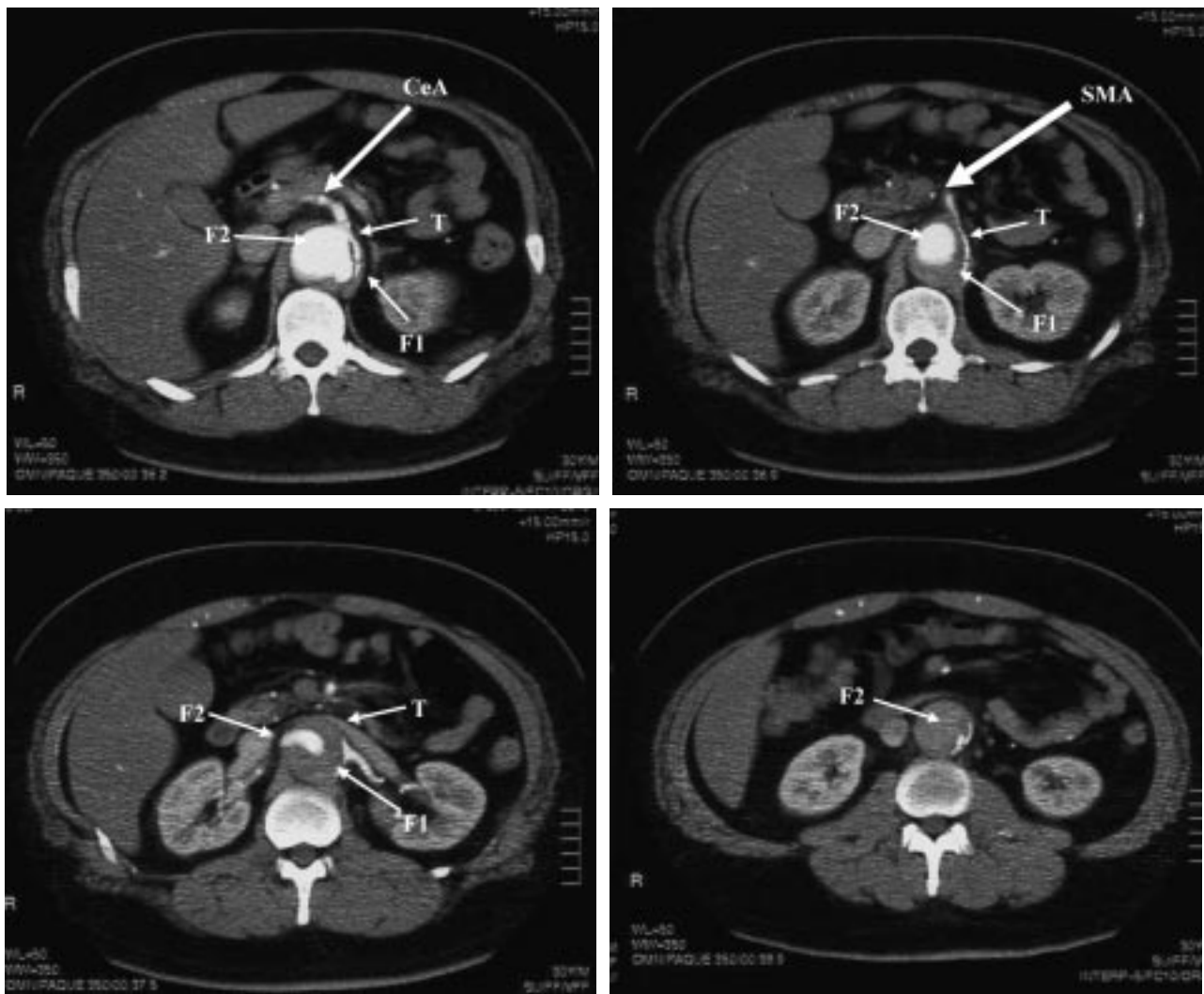


Fig. 3. A CT scan obtained 9 days after the onset of symptoms shows that the second false lumen (F2) is further expanded and that thrombosis has developed in it. The true lumen (T) is more severely compressed.

ies arose. The first false lumen (F1) that had developed with the first occurrence of aortic dissection was also severely compressed by the second false lumen and was difficult to identify. At the time of his arrival, despite these CT findings suggestive of severely disturbed abdominal flow, laboratory results, including creatine phosphokinase (CPK) and lactate dehydrogenase (LDH) levels, stayed within the normal limits. Slight acidemia (pH 7.38; base excess -3.2) and a slightly elevated lactate level (25 mg/dL; normal range: <17 mg/dL) that were observed upon arrival resolved soon after the admission (Table 1). With the administration of a β -blocking agent and a calcium channel blocker, the patient's systolic blood pressure decreased to 100 – 120 mmHg, and his pain was alleviated. However, severe abdominal pain recurred on the second

day and continued for more than 1 week. The patient required 15 – 30 mg of pentazocine per day for pain relief. Laboratory tests were performed repeatedly but showed no signs of mesenteric ischemia/necrosis. Abdominal X-ray examination was also performed repeatedly, but showed no intestinal gas or niveau formation, which would have been indicative of ischemic colitis. Although occult blood was detected once in his stool, another test was negative.

Nine days after the onset of symptoms, a follow-up CT scan was performed (Fig. 3). The second false lumen (F2) had expanded further, and as a result the true lumen (T) had become more severely compressed. Although during the admission, no definitive signs of mesenteric ischemia had been observed by laboratory testing, surgical repair was undertaken on the next day based on a strong clinical

suspicion.

A pale shrunken intestine was found during the surgery, indicative of mesenteric ischemia. The aorta was opened just below the renal arteries, and the thrombi inside the false lumens were removed. Two flaps were widely resected, and proximal anastomosis was performed with a knitted Dacron graft (GelsoftPlus, Vascutek, UK). Bilateral distal anastomoses were performed on the common iliac arteries, and the inferior mesenteric artery was attached to the left limb of the graft. The surgical repair alleviated the patient's abdominal pain. He started taking meals on postoperative day 12 and was discharged on postoperative day 27.

Discussion

Previous studies have indicated that medical therapy provides an excellent outcome for patients with uncomplicated distal dissection of the aorta. The 30-day survival rate for patients with distal dissection treated medically is as high as 92%.⁵⁾ Therefore medical therapy is preferred to surgical therapy for patients with uncomplicated distal dissection. However, the outcome of medical therapy may be poor when distal dissection is complicated by abdominal visceral ischemia, uncontrolled pain, and rapid expansion of the dissected aorta. Mesenteric ischemia and necrosis, for example, are potentially lethal complications of distal dissection seen in 3%–5% of cases.^{2,3,6,7)} The operative mortality rates for patients with mesenteric ischemia or necrosis are 45%–87%.²⁻⁴⁾ Despite successful visceral vascular reconstruction, death often results from the sequela of mesenteric necrosis.²⁾ In fact, mesenteric ischemia is also recognized as an independent risk factor for surgical death.⁸⁾ Thus it is strongly recommended that when patients with distal dissection are treated medically, they should be carefully monitored for any evidence of branch arterial malperfusion, particularly mesenteric ischemia. The clinical features of mesenteric ischemia, however, may be subtle and therefore can go unrecognized.

Laboratory testing, including the measurement of CPK⁹⁾ and lactate^{10,11)} levels, has been reported as a good diagnostic tool for mesenteric ischemia. When CPK and lactate levels are elevated, however, small intestine or colon necrosis may be in progress. By the time mesenteric ischemia is clinically obvious, irreversible organ damage may have already occurred. Thus it appears that these markers could be markers of necrosis rather than ischemia.

In this patient, lasting abdominal pain requiring analgesics and narrowing the true lumen on CT scan were clinical signs suggesting mesenteric ischemia. However, through the entire clinical course, repetitive laboratory testing showed no abnormalities. In this regard, the present case is a good demonstration that mesenteric ischemia still remains a diagnostic challenge and suggests that currently available laboratory markers are not sensitive enough to detect the presence of ischemia. Physicians cannot rely heavily on these markers; a strong clinical suspicion for mesenteric ischemia may be the only key to preventing a catastrophic outcome in this condition.

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