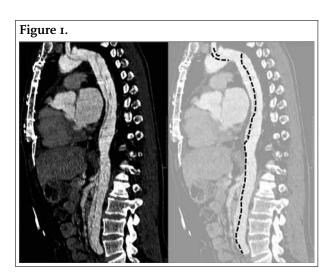
# Haematemesis, abdominal pain and a diastolic murmur in a cocaine user

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### CASE REPORT

A 39-year-old man was admitted to our hospital with haematemesis. After the use of cocaine earlier that same evening he experienced a tingling sensation, followed by haematemesis and upper abdominal pain. His medical history revealed cocaine and heroin abuse. He was not on any medication.



On physical examination the patient appeared to be alert and in no acute distress. His blood pressure was 115/35 mmHg, the pulse rate was 76 beats/min. Except for mild epigastric tenderness, no abnormalities were found. The electrocardiogram (ECG) showed sinus rhythm with nonspecific repolarisation changes. Routine laboratory tests and chest X-ray were normal.

The patient was admitted to medium care for observation of haematemesis in suspected upper gastrointestinal bleeding.

During the night the patient experienced increasing abdominal pain. Additionally he started to complain of back pain. Therefore the patient was reassessed. Blood pressure was 120/25 mmHg, in both arms. Auscultation now revealed a diastolic murmur in the left second intercostal space. His abdomen was more painful, with rebound tenderness. An emergency computed tomography angiography (CT-a) was performed (*figure 1*).

## WHAT IS YOUR DIAGNOSIS?

See page 426 for the answer to this photo quiz

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#### ANSWER TO PHOTO QUIZ (PAGE 422)

## HAEMATEMESIS, ABDOMINAL PAIN AND A DIASTOLIC MURMUR IN A COCAINE USER

## DIAGNOSIS

The presence of haematemesis and abdominal pain suggested acute abdominal disease. However, the combination of severe pain and signs of aortic insufficiency, namely diastolic murmur and high pulse pressure, made us think of aortic dissection. CT-a indeed showed a Stanford type-A aortic dissection.

The patient was immediately transferred to a cardiac surgery centre and a supracoronary aorta ascendens replacement was performed. The haematemesis resolved spontaneously and did not reoccur. No explanation was found as the CT scan showed neither perforation of the aorta into the oesophagus, nor signs of intestinal ischaemia. The patient recovered.

The clinical presentation of acute aortic dissection (AAD) can be atypical and often mimics other diseases.<sup>1,2</sup> This leads to a diagnostic delay in 39 to 85% of cases, thereby increasing mortality and morbidity.<sup>1,2</sup> Initially, 32% of AADs are mistaken for acute coronary syndrome.<sup>2</sup>

Without treatment, mortality of AAD is about 40 to 50% in the first 48 hours.<sup>1,3</sup> Therefore, it is vital to recognise risk factors and symptoms (*table 1*). The most important risk factors are hypertension and male gender.<sup>3</sup> Cocaine abuse, as in our patient, is increasingly reported as risk factor.<sup>3-5</sup>

The presenting symptoms are the result of local complications caused by the dissection.<sup>1,3</sup> The dissection flap can cause occlusion of a branch artery resulting in ischaemia of heart, brain, kidney, spinal cord, intestines and/or extremities.<sup>1,3,5</sup> If the tear communicates with the pericardium, pericardial tamponade results.<sup>1</sup>

Pain, usually very severe, is the most common presenting symptom (90%).<sup>1,3</sup> Importantly, in 10% of the cases AAD is painless.<sup>3</sup> Pulse deficit is present in less than half of the patients.<sup>1,3</sup> Diastolic murmur, a sign of aortic insufficiency, is usually present in type A dissection. Haematemesis can occur as a result of intestinal ischaemia or perforation to the oesophagus. Most patients have abnormal chest radiography and ECG.<sup>1,3</sup>

AAD should always be considered in patients presenting with unexplained chest, back or abdominal pain, neurological deficit, syncope, pulse deficit, diastolic murmur, high pulse pressure, kidney failure and/or ischaemia of limbs or intestines.

## A C K N O W L E D G E M E N T S

We thank R.H. Kruyt, radiologist, for providing us with the CT-angiography image.

 Table 1. Acute aortic dissection: associated factors,

 presenting features and findings in routine tests

	Type A (n=617)	Type B (n=384)
Associated factors		
Mean age (years)	61	65
Male sex	67%	71%
Hypertension	67%	80%
Atherosclerosis	28%	38%
Previous cardiovascular surgery*	16%	17%
Aortic aneurysm	7%	18%
Marfan's syndrome	6%	3%
Related to coronary angiography	6%	2%
Bicuspid aortic valve	4%	2%
Previous aortic dissection	3%	9%
Pregnancy	<1%	<1%
Cocaine abuse	<1%	1%
Symptoms and signs		
Pain		
Chest or back pain	85%	86%
• Abdominal pain	22%	43%
Severe or worst-ever pain	90%	90%
<ul> <li>Abrupt onset of pain</li> </ul>	91%	89%
Migrating pain	15%	25%
Hypotension, shock or tamponade	27%	3%
Hypertension	36%	69%
Any pulse deficit	31%	21%
Aortic regurgitation	44%	12%
Focal neurological deficit	17%	5%
Chest x-ray		
Normal	11%	21%
Widened mediastinum	63%	56%
Abnormal aortic contour	47%	49%
ECG		
Normal	30%	31%
Left ventricular hypertrophy	23%	32%
Myocardial ischaemia or infarction	24%	10%
The Classical triad <sup>#</sup>		
Aortic pain + pulse deficit + widened med	liastinum	
Triad present in 27% of patients <sup>1</sup>		
Presence of triad has a positive likelihood	l ratio of 66.01	

Data as published by the International Registry of Acute Aortic Dissection (IRAD)<sup>6</sup>; \*Vascular surgery includes coronary artery bypass surgery, aortic valve replacement, aortic aneurysm or dissection repair, mitralvalve replacement of repair, or other aortic surgery; \*Klompas' identified significant negative and positive indicators for AAD. The presence of pulse deficit or focal neurological deficit increases the probability of AAD. The classical triad of aortic pain, pulse deficit and mediastinal widening is a strong indicator of AAD. Negative indicators are absence of acute pain, normal chest X-ray, absence of symptoms from the triad.<sup>1</sup>

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