Prof. Ivo Šteiner, MD, PhD, Editor

CASE 1-2010: ACUTE OCCLUSION OF ABDOMINAL AORTA

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Introduction

Complete aortic occlusion is rare, but potentially catastrophic. It usually occurs in patients with advanced aortoiliac atherosclerosis, and can cause severe ischaemic manifestations (7). Acute aortic occlusion bears an early mortality of 31–52 % (1, 7) and is caused either by embolic occlusion or by acute thrombosis of the infrarenal abdominal aorta. Between 75–80 % of cases of thrombotic aortic occlusion occur in the setting of underlying severe aortoiliac atherosclerotic occlusive disease, often precipitated by a low-flow state. A hypercoagulable state may precipitate thrombosis of an abdominal aortic aneurysm and lead to aortic occlusion (4, 6, 7). Acute occlusion of abdominal aorta has a high early mortality and requires prompt intervention. It remains a serious vascular surgical emergency with significant morbidity and mortality, even when recognized promptly and treated appropriately (6). Symptoms and imaging methods (ultrasonography, CT, angiography, MRI) are crucial for the diagnosis.

Clinical data

A 69-year-old man was admitted to our department after cardiopulmonary resuscitation for ventricular fibrillation. He had history of arterial hypertension, hyperlipidemia and smoking. Physical examination showed no significant pathology on admission. He was hemodynamically stable (no catecholaminergic support). On admission, diffuse ST segments denivelations on ECG were found and urgent coronary angiography indicated. Three – vessel disease with severe diffuse atherosclerotic changes (with no coronary occlusion) and large subrenal abdominal aortic aneurysm (65 mm) were found during angiography. Conservative treatment strategy was indicated (hemodynamic stability, no coronary occlusion) – arteficial ventilation, hypothermia for 24 hours after cardiac arrest, anticoagulants, fluids and diuretics, intermittent catecholaminergic support. The second day of hospitalization was complicated by gastrointestinal tract bleeding from diffuse ischemic mucosal changes (with no need for endoscopic intervention). During the third hospitalization day the patient's condition suddenly deteriorated; a suspicion of acute abdominal aortic occlusion was expressed (absence of pulsations, color and temperature changes on lower limbs). Complete occlusion of subrenal abdominal aorta was found on computer tomography (Fig. 1, 2). The clinical status deteriorated rapidly and severe shock developed. Surgical intervention was contraindicated in this polysclerotic patient with shock symptoms (hemodynamic instability, disseminated intravascular

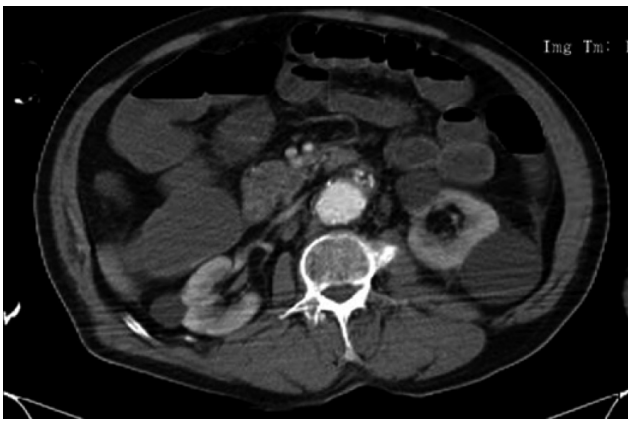

Fig. 1: Normal finding of contrast in abdominal aorta.

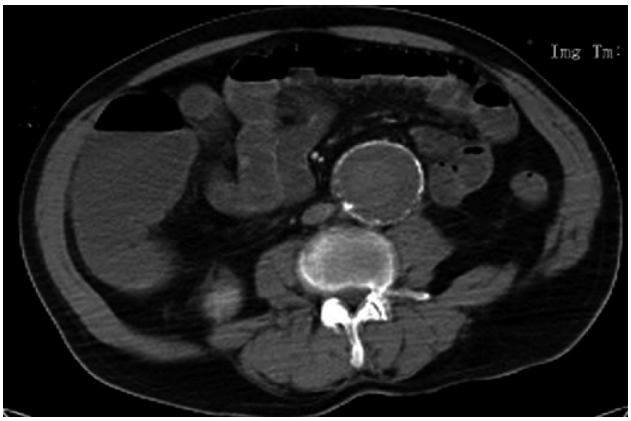

Fig. 2: Absence of contrast in abdominal aneurysm.

coagulopathy, lactic acidosis etc.). The patient died due to progressive shock.

Clinical diagnosis: Acute abdominal aortic occlusion.

Pathological findings

Grossly, the dominant finding was subrenal abdominal aortic atherosclerotic aneurysm 50 x 30 x 15 mm with its lumen completely obliterated by huge decolorated thrombus extending into both common iliac arteries. Pulmonary congestion, oedema (right lung 850 g, left lung 830 g) and a small infarction at the basis of the left lung were found. Severe atherosclerotic changes involved particularly the aorta with narrowing of orifices of the abdominal arteries by atherosclerotic plaques and mural thrombi and the coronary arteries with stenoses > 75 % in all major branches (without acute changes). There was marked left ventricular hypertrophy of the heart (wall thickness 20 mm, heart weight 620 g) and a fibrous scar in the posterior wall of the left ventricle. The intestinal mucosa from lower half of jejunum to rectosigma was violet coloured and oedematous. There were no other significant findings.

Microscopically, the gross findings were confirmed – pulmonary congestion with oedema and hemorrhagic infarction, fibrotic changes and residual cardiomyocyte hypertrophy in the posterior wall of left ventricle and marked congestion of the intestinal submucosa with early phlegmon (Fig. 3).

Pathological diagnosis: Thrombotic occlusion of abdominal aorta.

Discussion

Acute occlusion of the infrarenal abdominal aorta is a catastrophic event requiring early recognition and intervention. While traditional causes of occlusion (saddle embolus and thrombosis) are the most frequent, vasculitis and hypercoagulable states have recently also been suggested as etiologies. Acute occlusion in patiens with atherosclerosis is precipitated by low flow states (4). A thorough clinical and laboratory evaluation of such patients should be performed, since symptoms alone can be misleading, due to the development of extensive collateral vasculature, which may prevent manifestation of the acute ischaemic phenomena. Acute aortic occlusion should be considered when there is sudden onset of leg pain, pallor, paresethesia progressing to paralysis, associated with characteristic mottling of skin. Clinical examination of peripheral pulses in these cases is mandatory. Contrary to what might be expected, not all patients with sudden occlusion of abdominal aorta are admitted with symptoms of severe ischemia (14). Immediate intervention is obligatory when aortic occlusion is diagnosed. Without treatment, these patients have a poor prognosis. Although acute thrombosis is a recognized complication of aneurysms in general, it is rare in abdominal aortic aneurysm; its incidence is reported to be 0.6–1.8 % of AAA cases (8). Several mechanisms of complete occlusion of the aneurysm have been proposed. They include 1) acute low-flow state superimposed on a stenotic or occluded atherosclerotic distal vascular bed; 2) thrombemboli which are usually of cardiac origin; 3) fragments of mural

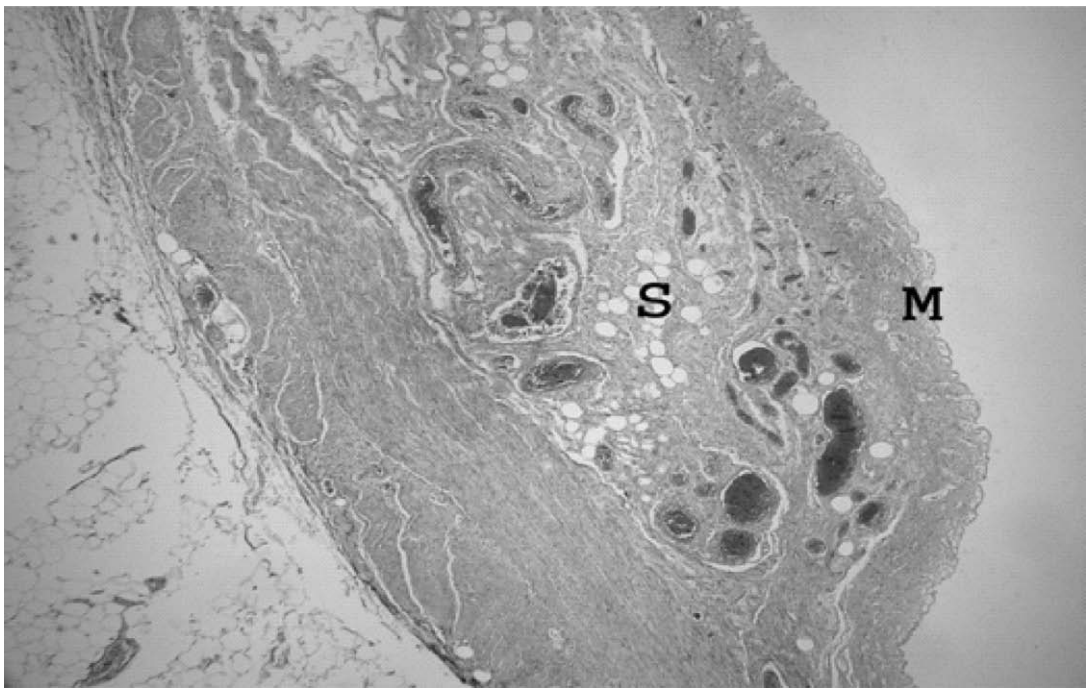

Fig. 3: Congestion and early phlegmon of the intestine. M – mucosa (autolysis); S – submucosa (congestion, early phlegmon).

thrombi from the aneurysm moving distally and leading to retrograde thrombosis, and 4) hypotension from hemorrhage or an acute cardiac problem possibly initiating the thrombosis (2, 3, 5, 8, 12). In our case, the potential etiology mechanisms were: low-flow state after cardiopulmonary resuscitation and cardiac failure superimposed on aortoiliac atherosclerosis and aortic wall injury with thrombosis induction in aortic aneurysm. There is no definite relationship between the aneurysm size and the likelihood of thrombosis, however, in some reported cases, the aneurysms tended to be smaller (8, 9). The key to successful management of abdominal aortic aneurysm thrombosis is prompt diagnosis and appropriate surgery (8, 9, 10, 11, 13).

Message from the Editor (Prof. Šteiner)

Abdominal aortic aneurysms are very common. The majority are atherosclerotic; it is estimated that at least 40 of every 1000 persons older that 50 years of age harbor such lesions. Often, they are asymptomatic until they get complicated, with catastrophic consequences and a low operative survival rate. Men are affected by atherosclerotic abdominal aneurysms more often than women, in a ratio of 3.8:1 to 6:1. Among risk factors are systemic hypertension and a long history of cigarette smoking.

The diameter of saccular abdominal aneurysms increases by a median rate of 0.21 cm/year. For those with a diameter greater than 5 cm, the risk of complications over 5 years is 25 %.

The most common complication is rupture of the aneurysm with massive retroperitoneal haemorrhage; it appears as the 13th most common cause of death in the USA. An unusual complication is infection of the aneurysm.

Almost all aneurysms contain laminated old thrombus which may become a source of peripheral embolization. In rare instances, as in the presented case, thrombus in an aneurysm completely seals off its lumen.

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