Surgical treatment of penetrating atherosclerotic ulcer of the descending aorta

Hirurško leženje penetrantnog aterosklerotskog ulkusa descendentne aorte

Pavle Kovačević*†, Lazar Velicki*†, Dušan Popović†, Vladimir Ivanović‡, Renata Mojaševi‡

*Faculty of Medicine, University of Novi Sad, Novi Sad, Serbia;
†Clinic of Cardiovascular Surgery, Institute of Cardiovascular Diseases of Vojvodina, Sremska Kamenica, Serbia

Abstract

Introduction. The term “penetrating atherosclerotic ulcer” (PAU) of the aorta describes the condition in which ulceration of an aortic atherosclerotic lesion penetrates the internal elastic lamina into media. PAU is a high-risk lesion due to its deleterious effects on the integrity of aortic wall, with potentially fatal outcome. Case report. A patient with intense, sharp chest pain irradiating to the back but with no signs of myocardial ischemia on an electrocardiogram was referred to our hospital. Transthoracic echocardiography showed no pathological changes of the ascending aorta. However, multislice computed tomography (CT) showed an aortic ulcer with varying degree of the subadventitial hemorrhage in the region of the thoracic aorta at the level of Th 8–9. Due to imminent rupture of the penetrating aortic ulcer, the patient was promptly prepared for surgery. A 15 cm long subadventitial hematoma was found intraoperatively in the right posterolateral aspect of the descending aorta, 5 cm above the diaphragm and 7 cm below the origin of the left subclavial artery. The affected segment of the aorta was resected, followed by an inlay aortic reconstruction with a Dacron tube graft of 24 mm. Control CT revealed satisfactory reconstruction of the descending aorta.

Conclusion. PAU is a rare, but potentially fatal disease. Open surgery in patients with PAU is an effective treatment strategy, although endovascular treatment options are emerging.

Key words: aorta, thoracic; aortic rupture; atherosclerosis; ulcer; diagnostic techniques and procedures; cardiovascular surgical procedures; transplants.

Apstrakt


Ključne reči: aorta, thoracic; aortic rupture; atherosclerosis; ulcer; diagnostic techniques and procedures; cardiovascular surgical procedures; transplants.
Introduction

The term “penetrating atherosclerotic ulcer” (PAU) of the aorta describes the condition in which ulceration of an aortic atherosclerotic lesion penetrates the internal elastic lamina into media. The progression of aneurysmal dilatation is usually slow. Aortic ulcers may break through into the adventitia to form a pseudoaneurysm. In this situation the hematoma is contained by the overlaying adventitia and some authors consider this type of aortic lesion a contained aortic rupture. With regard to the new aortic dissection classification, PAU is considered a form of aortic dissection. Mixed clinical presentation, varied patient population, and data limited to small numbers of retrospectively reviewed patients have made a data-driven algorithm for the surgical treatment of PAUs difficult to construct. Most authors believe that PAU is a high-risk lesion with potentially fatal outcome.

Case report

A 55-year-old man, with a history of hypertension and smoking, that suffered a sudden onset of severe chest pain was admitted to our hospital. Intensive, and sharp chest pain with irradiation to the back had appeared suddenly during the night. On admission, chest radiography was performed showing enlarged cardiac shadow mainly due to elongated and enlarged descending thoracic aorta. There was no evidence of myocardial ischemia on an electrocardiogram. Transthoracic echocardiography (TTE) registered no pathological changes of the descending aorta. Multislice computed tomography (CT) showed an aortic ulcer with varying degree of subadventitial hemorrhage in the region of the thoracic aorta at the level of Th 8–9. Magnetic resonance imaging (MRI) was performed subsequently in order to delineate the process extension, showing saccular aortic aneurysm (2 × 2 cm) with surrounding hematoma and left pleural hemorrhagic effusion, which in fact was PAU. Aortography revealed irregular edge of the aortic defect which corresponded to described aortic exulceration on CT (Figure 1). After the diagnosis of imminent PAU rupture was established, the patient was urgently transferred to the operating room for emergency surgery.

Cerebrospinal fluid (CSF) drainage was initiated at the L 2–3 level in order to obtain CSF pressure < 10 mmHg. A selective double-lumen endotracheal intubation was performed using a Carlen’s tube. Posterolateral thoracotomy was performed in the 5th left intercostal space. There was no need for phrenotomy neither extra- nor transperitoneal approach to the upper parts of the abdominal aorta. A 15 cm long subadventitial hematoma was found in the right posterolateral aspect of the descending aorta, 5 cm above the diaphragm and 7 cm below the origin of the left subclavial artery. Systemic heparinization was achieved with 5,000 IU of heparin. Operation was further conducted on partial extracorporeal circulation (ECC) – 2.5 L/min – with the left femoral artery and the inferior vena cava cannulation. In the upper parts of the body we obtained a controlled hypotension with maximal systolic arterial pressure between 80 and 100 mmHg. The patient was not cooled down in order to avoid fibrillation of the heart. Aortic clamping was performed above and under the level of the PAU. The affected segment of the aorta was resected, followed by an inlay aortic reconstruction with a Dacron tube graft 24 mm along with three intercostal arteries included in the distal anastomosis through a separate anastomosis (Figure 2). The patient was subsequently weaned from partial ECC. Partial ECC time was 71 minutes. Drainage of the left pleural space was achieved with two tube drains. The postoperative period was uneventful. Control CT revealed a satisfactory reconstruction of the de-
scending aorta. The patient was discharged in good postoperative condition with no postoperative complications, on the 20th postoperative day.

Discussion

The correct initial diagnosis and immediate appropriate management of PAU is essential. It is often very difficult for the attending physician to establish the diagnosis of PAU because the pathophysiology and diagnostic algorithm for PAU have not been fully understood, and the amount of comprehensive literature description is still lacking.

PAU can lead to the development of intramural hematoma, aortic dissection, aortic aneurysm or rupture. In a retrospective study of 198 patients in whom aortic dissection was initially diagnosed, 7.6% of patients were found to have PAU. In some cases, hematoma extension causes stretching of the weakened aortic wall, leading to the formation of a saccular aortic aneurysm. Yokoyama et al. suspected that some spontaneous aortic rupture due to atheromatous plaque as previously reported might have been due to the perforation of PAU. Therefore, we believe that PAU might be recognized as a cause of aortic rupture with increasing frequency in the future by sensitive imaging techniques such as CT, MRI and transesophageal echocardiography. In our case CT scan showed PAU with varying degree of surrounding hemorrhage located in the thoracic aorta with left pleural hemorrhagic effusion. In emergency situation, CT is the imaging modality of choice to identify and locate the PAU.

Haris et al. reported that the disease progression is slow, with a low prevalence of acute rupture or other life-threatening complications. Coady et al. reported that the risk of aortic rupture in patients with penetrating aortic ulcer is 40% compared to patients with Stanford type A or type B dissection where the risk is 7% and 3.6%, respectively.

Draining CSF from the lumbar region may reduce CSF pressure, improve blood flow to the spinal cord and reduce the risk of ischemic spinal cord injury. Partial ECC is also a very useful approach to avoid visceral and spinal ischemia during aorta cross clamping. In our patient no neurological deficit has been observed during postoperative period. Persistent or recurrent pain, hemodynamic instability and a rapidly expanding aortic diameter have been considered as indications for surgical treatment. The authors emphasized that most patients with PAU are at high risk for surgical intervention because of their advanced age and poor general health.

Murgo et al. emphasized that surgical repair of the descending thoracic aorta is frequently complicated by respiratory disease, renal insufficiency, or spinal ischemia and recommended the transluminal placement of endovascular stent-grafts for PAUs. In patients with PAUs initially managed with medical therapy, Cho et al. reported that one-third of these patients required surgical repair during follow-up for the progression of PAUs to aneurysms, dissections, or perforations. Despite improvements in surgical techniques and postoperative care, conventional operative repair of the descending thoracic aorta for PAUs is still associated with high morbidity and mortality rates. Endovascular repair seems to be a promising option showing lower morbidity and mortality rates with respect to open surgical repair. Specifically, there are no prospective randomized studies comparing open and endovascular treatment of PAU. Reports in the literature primarily include single-center experiences and non-randomized studies of open and endovascular stent graft procedures with limited follow-up.

Conclusion

PAU is a rare, but potentially fatal disease. It is typically seen in elderly individuals with hypertension and atherosclerosis and usually involves the descending thoracic aorta. With onset of acute chest pain in a patient with no evidence of myocardial ischemia, aortic dissection or rupture, PAU should be considered. In emergency situation, CT is the imaging modality of choice to identify PAU. Open surgery in patients with PAU is an effective treatment strategy, although endovascular treatment options are emerging.
REFERENCES


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