Case report

Spontaneous resolution late after aortic dissection

C.J.A.M. Zeebregts a,*, M.A.A.M. Schepens b, F.E.E. Vermeulen b

a Department of Surgery, University Hospital, P.O. Box 30001, 9700 RB Groningen, The Netherlands
b Department of Cardiovascular and Cardio-Pulmonary Surgery, St. Antonius Hospital, Nieuwegein, The Netherlands

Received 3 March 1997; received in revised form 26 May 1997; accepted 3 June 1997

Abstract

A 50-year-old man was operated on for acute type I (DeBakey classification) aortic dissection. The supracoronary ascending aorta was replaced with an interposition graft. Postoperative computed tomography and angiography clearly revealed a double-barrelled aortic arch, left common carotid artery and descending thoracoabdominal aorta with contrast filling of both true and false lumen starting from the distal anastomosis. The same finding was noted at 1 year follow-up with severe compression of the true lumen by the false lumen. At this time, anticoagulation therapy was stopped. One year later, computed tomography showed spontaneous resolution of the dissection in the aortic arch, left common carotid artery and descending aorta over its full length. This was confirmed by angiography. This case report illustrates that spontaneous resolution of a dissected descending aorta can occur late after surgery from type I dissection, but it remains very rare. © 1997 Elsevier Science B.V.

Keywords: Aortic dissection; Natural history; Spontaneous resolution

1. Introduction

Acute aortic dissection is a life-threatening disease and requires prompt diagnosis and treatment. Patients with acute DeBakey type I and II dissection require emergency surgery, whereas type III can usually be managed medically [1,2].

Spontaneous resolution of aortic dissection is very rare. To our knowledge, only three in vivo cases have previously been reported in the literature [3-5]. We present such a case in which a spontaneous regression was observed at 2-year follow-up.

2. Case report

A 50-year-old man with no medical history presented in October 1994 with acute dyspnoea and squeezing chest pain. Blood pressure was 80/40 mmHg. Physical examination revealed a grade III/VI systolic murmur. Electrocardiography showed a sinus rhythm at 70 beats/min with S-T segment elevation in leads V₃-V₆, I, II, aVL and aVF. Chest X-ray showed signs of pulmonary oedema. A contrast computed tomographic (CT) scan revealed ascending aortic dissection. This was confirmed by transoesophageal echocardiography, which also demonstrated aortic valve regurgitation.

An emergency operation was carried out. Intra-operatively, it appeared that the primary entry of the dissection was located just above the left coronary ostium. Dissection sites were also noted at the commissure between the non-coronary and left coronary cusp, and between the left and right coronary cusp. Both coronary ostia were themselves partially dissected. Proximally, the dissection extended over a circumference of 110°, progressing distally to almost 360°. Resuspension of the aortic valve and reconstruction of both coronary ostia was performed. Biological glue® (GRF, Saint Just
Malmont, France) was used proximally, as well as around the coronary ostia and also distally, to reattach the aortic layers. Before the glue was applied distally, a continuous mattress suture was placed approximately 10 mm distally of the transected wall. Interposition with a Gelweave® 24-mm prosthetic graft (Vascutek, Inchinnan, Renfrewshire, UK) was performed with an aortic clamp placed. The proximal anastomosis was located just above the sinotubular junction. Total perfusion time was 97 min with an aortic crossclamp time of 62 min. Histological examination showed atherosclerotic aortic tissue without signs of medial degeneration.

Postoperative intravenous angiography, confirmed by catheter angiography (Fig. 1a + b), revealed filling of contrast at the distal anastomotic suture line into the true and false lumen, showing dissection in the left common carotid artery (LCC) and the thoracoabdominal aorta. Five weeks after surgery the patient was discharged home in good condition with furosemide, enalapril and metoprolol tartrate as chronic medical treatment.

One year after discharge, CT scan showed dissection in the aortic arch, LCC, and thoracoabdominal aorta, extending distally into the left iliac artery with a false lumen which was far larger (75%) than the true lumen (Fig. 2a + b). At this time, anticoagulation was stopped. However, 6 weeks thereafter, the patient was re-admitted suffering severe chest pain followed by epileptic insults due to ischaemic infarction located in the medial cerebral artery area. Acetyl salicylic acid and anti-epileptic medication was started.

In October 1996, at 2-year follow-up, the patient was in good health and repeated CT scan was performed routinely. Diameters were as follows: ascending aorta prosthesis 25 mm, distal ascending aorta 30 mm and descending aorta (both proximally and distally) 25 mm. Remarkably, unlike the small true lumen seen on CT scan performed in October 1995, the true lumen had completely normalised (Fig. 2c + d). The partially dissected common carotid arteries showed the same pattern of spontaneous regression. To verify this change, angiography was performed which also showed the resolution, when compared to the angiogram made 2 years before (Fig. 1c + d).

3. Discussion

Natural history of aortic dissection includes either endothelialization of the false lumen forming a double-barrelled aorta with patent blood flow through both true and false lumina, or thrombosis of the sac leading to fibrosis and scarring. Major risks for conservatively treated aortic dissections are aneurysmal dilatation of the wall of the false lumen until it ruptures and/or extension of the dissection into coronary, carotid, renal, mesenteric and/or iliac arteries, causing sometimes critical vascular obstruction and malperfusion syndromes.
Also, patients with surgically repaired aortic dissection remain at considerable risk for secondary dilatation, aneurysmal transformation and malperfusion syndromes. To minimize these risks, medical treatment is instituted as soon as possible after admission, involving antihypertensive treatment with a vasodilator and the administration of a $\beta$-blocking agent to depress left ventricular contractility [1]. Furthermore, it is advisable to conduct contrast-enhanced CT scans and/or magnetic resonance imaging for repeated follow-up at regular times, also in asymptomatic patients [1].

Complete healing of dissections have been reported in coronary arteries [6], carotid arteries [7] and renal arteries [8], but in the aorta such healing is rare, with to our knowledge up to now only three reported in vivo cases [3–5]. All previous cases were non-operated type B aortic dissections without the use of anticoagulant therapy. This phenomenon seems to differ from characteristic thrombosis of the false channel. Therefore, it is remarkable that in our patient a typical dissection pattern, with such a severe narrowing of the true lumen through compression by the false lumen more than a year after admission, appeared to be almost completely reversible over its full length another year later. Possible explanation for this event may be the withdrawal of anticoagulant therapy (acenocoumarol) from October 1995 initiating thrombotic obliteration of the false lumen, combined with the maintenance of normal and stable blood pressure, the latter also being regarded as a favourable factor in the case described by Hoshino and colleagues [4]. The time period between the acute dissection and the complete resolution varied from 6 months to almost 1 year in the previous cases. In our patient no healing was present on CT scan at 1 year follow-up, but total healing was diagnosed 2 years after the acute incident.

Although the mechanism of spontaneous resolution of dissection of the thoracoabdominal aorta remains unknown, it underscores the thesis that without severe complication, conservative treatment of distal residual dissection may be an acceptable alternative.

References