Thrombotic formations within the aortic arch as source of embolization in patients with coagulopathy

M. Roth*, M. Schönburg, W.P. Kloekerkorn, E.P. Bauer

Department of Cardiovascular and Thoracic Surgery, Kerckhoff Clinic Foundation, Bad Nauheim, Germany

Received 30 November 2000; received in revised form 17 February 2001; accepted 20 February 2001

Abstract

Thrombotic formations on atherosclerotic lesions of the thoracic aorta are potential sources of cerebral and systemic embolization. Especially younger patients without calcifications of atherosclerotic plaques or coagulation disorders have a higher risk for embolization. Magnetic resonance imaging and transesophageal echocardiography are the diagnostic methods of choice. As an alternative to anticoagulation surgical therapy is indicated to prevent severe brain damage or multiorgan failure in patients with mobile thrombotic formations. Herein we describe two patients in whom successful surgical treatment was performed in deep hypothermic circulatory arrest by excision of the aortic arch atheroma.

Keywords: Thrombosis; Aortic arch

1. Introduction

Prospective case controlled anatomical studies of patients with atherosclerotic aortic arch lesions showed, that there is a substantial risk of cerebral or peripheral embolic complications up to 33% [1,2]. Transesophageal echocardiography (TEE) and magnetic resonance imaging (MRI) are the methods of choice to visualize such lesions [2,3]. Several factors are known to increase embolic complications such as elevated plasma fibrinogen, presence of circulating anticoagulants, elevated plasma homocysteine or polycythemia vera [4–6]. There is still controversy how to treat patients with thrombotic lesions of the aortic arch. If cerebral infarction occurs antplatelet therapy has to be initiated. However, as soon as thrombus formation on atherosclerotic plaques is diagnosed full anticoagulation with warfarin is necessary. When there is a highly mobile thrombus or when embolic complications occurs despite anticoagulation thrombolysis or surgical therapy [3,7,8] is indicated.

2. Case report

2.1. Case 1

A 64 year old female patient was admitted in March 1999 due to sudden-onset of embolization into both iliac and femoral arteries. Long-term arterial hypertension and hyperlipidemia were identified as cardiovascular risk factors. Urgent surgical bilateral embolectomy was performed. MRI and angiography of the aortic arch and the descending aorta showed a 13 × 19 mm thrombus formation at the medial side of the aortic arch (Fig. 1). TEE showed atherosclerotic plaque with highly mobile thrombotic components on the concavity of the posterior segment of the aortic arch. Aortic arch and descending aorta were exposed via left lateral thoracotomy. The patient was put on femoro-femoral cardiopulmonary bypass (CPB) and cooled down to a core temperature of 17.1°C. The left ventricle was vented via the left atrial appendix. Thereafter total circulatory arrest was initiated. The aortic arch was opened in-between left subclavian artery and left carotid artery. Thrombus formation (15 × 20 mm in diameter) was found opposite left carotid artery (Fig. 1). After thrombectomy the entire atheroma was excised and the arteriotomy was closed with polypropylene suture. CPB was restarted after 10 min. Patient was rewarmed and weaned from CPB after 75 min. The postoperative course was complicated by a small pulmonary embolus. Histologic examination of the specimen confirmed...
an atherosclerotic process and adherent fibrin thrombus. Signs of arteritis were not found. Analysis of coagulation system showed a decreased protein S activity of 58.7% (normal 71%) and high level of D-Dimer 1000 ng/ml (normal, 200 ng/ml). Full anticoagulation with warfarin was initiated. MRI twelve month after surgery showed no recurrent thrombus. Twenty months after operation the patient is well without further embolic events.

2.2. Case 2

A 46 year old male patient had embolization into the right popliteal artery. After embolectomy he was referred to our department. TEE showed severe atherosclerotic plaques in the aortic arch with mobile thrombotic formations (15 £ 25 mm). MRI showed a structure in the ascending aorta and the aortic arch (16 £ 6 mm) (Fig. 2). CPB was initiated as described in case 1. Circulatory arrest was initiated at 19°C during 32 min. After aortic incision in the ascending aorta and thrombectomy of two thrombus formations (Fig. 2) the atherosclerotic segment of the distal ascending aorta was excised and the aortic wall was reconstructed with a Dacron patch. Histologic examination of the aortic wall confirmed atherosclerotic lesions with ulcerative plaques and a 40 £ 30 £ 15 mm thrombus formation. Coagulation parameters were normal, except heparin-associated thrombocytopenia with heparin-PF4-ELISA 113.5 Units (normal <30 units) and positive functional testing against heparin and orgaran (HIPA test positive) was diagnosed and treated with hirudine. After normalisation of platelet count anticoagulation was started. The patient had uneventful recovery. MRI control 8 months after surgery showed some smaller atherosclerotic plaques in the aortic arch but no recurrent mobile thrombotic formations. There was no further embolic event after 15 months.

3. Discussion

Atherosclerotic plaques of the aortic arch have been considered to be a rare cause of thromboembolic events [1]. But Tunick [1] found a prevalence of approximately 27% in patients with previous embolic events with a 12% risk of recurrent stroke within 1 year. The prevalence of insertion of thrombi on the wall opposite the ostia of the aortic arch, is striking. Laperche [7] identified only 23 out of 27 855 patients with arterial embolic events which 61% had a protruding thrombus in the posterior segment of the aortic arch, 17% in the horizontal aorta and 22% near the ostium of the left subclavian artery. If recurrent peripheral embolization occurs, we recommend routine TEE and/or MRI of thoracic aorta and aortic arch, respectively.

Optimal treatment strategies for atherosclerotic aortic plaques remains unclear. In some cases, anticoagulation with warfarin may prevent thrombotic formations and cerebral embolism [9]. However, recurrent embolic events may occur despite anticoagulant therapy in patients who do not undergo surgical treatment of atheromas [7]. Mobile thrombotic formations should undergo surgical resection to prevent embolization. Surgical removal of symptomatic atherosclerotic lesions of the aortic arch are described rarely in the literature. Swanson [8] performed an endarterectomy in deep hypothermic circulatory arrest in a patient with focal atheromas in the aortic arch. Bojar [3] recommended surgical removal of atherosclerotic plaques with thrombotic components to prevent further embolization. Belden [10] reported a successful aortectomy with graft replacement of the aortic arch in a patient with a mobile atheroma in the transverse aortic arch. Local endarterectomy should be performed when aortic atheroma is strictly localised, which was the case in patient 1. If multiple atherosclerotic plaques are present, aortic wall must be
excised and repaired by a patch (patient 2). If mobile thrombi are situated in the aortic arch or proximal descending aorta surgery had to be performed in circulatory arrest. Likewise in the ascending aorta crossclamping of the diseased aortic wall should be avoided.

In patients with mobile thrombotic formations in the aortic arch the risk of fatal embolization is high. If recurrent embolization of thrombotic formations persist after full anticoagulation resection of atheroma in deep hypothermic circulatory arrest is a safe procedure.

References