Negative results - Assisted circulation

Huge intracardiac thrombosis in a patient on veno-arterial extracorporeal membrane oxygenation support

Florian Weis a,*, Andres Beiras-Fernandez b, Dirk Bruegger c, Simone Kreth a, Ralf Sodian b, Felix Kur b, Marion Weis a, Konstantin Nikolaou c

a Department of Anesthesiology, University Hospital Grosshadern, Ludwig-Maximilians-University, 81377 Munich, Germany
b Department of Cardiac Surgery, University Hospital Grosshadern, Ludwig-Maximilians-University, 81377 Munich, Germany
c Department of Clinical Radiology, University Hospital Grosshadern, Ludwig-Maximilians-University, 81377 Munich, Germany

Received 23 September 2008; received in revised form 22 October 2008; accepted 23 October 2008

Abstract

A young woman suffering from congenital hypertrophic obstructive cardiomyopathy (HOCM) received a transcoryonary ablation of the septal hypertrophy (TASH) and an automated Cardioverter/Defibrillator (AICD) for the relief of progressive symptoms of heart failure. She developed an acute heart failure in the perioperative period and had to be put on veno-arterial extracorporeal membrane oxygenation. Following this the patient developed a nearly complete thrombosis of the left-sided cardiac chambers, despite successful laboratory anticoagulation.

Keywords: Extracorporeal membrane oxygenation; Intracardiac thrombosis; Anticoagulation; Heart transplantation

1. Case report

We present the case of a 20-year-old woman with congenital hypertrophic obstructive cardiomyopathy (HOCM). She had undergone resection of a subvalvular aortic stenosis at the age of four and was now considered suitable for transcoronary ablation of the septal hypertrophy (TASH procedure) [1] to relieve increasing symptoms of heart failure and for prophylactic implantation of an Automated Cardioverter/Defibrillator (AICD) [2]. A few days after the successful TASH procedure, the AICD was implanted. The shock-wire was tested. Despite a successful defibrillation the patient’s hemodynamic status worsened dramatically. She needed high doses of catecholamines and had to be resuscitated mechanically for an hour. As hemodynamics did not stabilize despite excessive pharmacologic support (epinephrine, milrinone, and vasopressin) and pulmonary function was severely impaired (arterial oxygen tension was below 40 mmHg at an inspired fraction of oxygen of 1.0 and a positive expiratory pressure of 15 mbar) a veno-arterial extracorporeal membrane oxygenation device (Biomedicus 540, Medtronic, Minneapolis, USA) was implanted via the left femoral vessels (Arterial cannula: Medtronic EOPA 20f, Medtronic, Minneapolis, USA; 2-Stage venous cannula: Medtronic Bio-medicus 20f, Medtronic, Minneapolis, USA). She developed a marked hypoperfusion of the left limb and therefore the arterial cannula was placed into the ascending aorta via median sternotomy.

The patient developed a severe adult respiratory distress syndrome (ARDS) with tidal volumes of less than 50 ml at a positive end-expiratory pressure of 25 mbar and an inspiratory peak pressure of 45 mbar. The extracorporeal membrane oxygenation (ECMO) flow was adjusted according to measurements of the mixed venous or central venous saturation of oxygen (target > 70%). To achieve this, a flow of 3.5 to 4.5 l/min was necessary. The anticoagulation was guided by the activated clotting time, which was kept strictly above 160 s throughout the whole period of treatment. This resulted in partial thromboplastin time (PTT) levels between 44 and 65 s. Additionally, the thrombocyte count was between 35 and 75 G/l throughout the whole period of treatment, which was probably caused by the ECMO and the continuous veno-venous hemofiltration. A heparin-induced thrombocytopenia could be ruled out by testing for platelet factor 4-heparin complexes (ELISA) [3].

The patient suffered from intermittent (more than five periods per day, lasting for up to an hour) ventricular tachycardia (VT) which could not be ameliorated despite high doses of amiodarone and magnesium. However, even during the periods of VT the patient was hemodynamically stable and a left ventricular ejection could be detected on the arterial pressure curve. The patient was listed for combined heart and lung transplantation on a high urgency level. After four days of ECMO therapy serum levels of lactatdehydrogenase (maximum level: 58860 U/l) and hydroxybutyrat-dehydrogenase (maximum level: 3710 U/l) rose progressively, indicating massive hemolysis. Thus, the oxygenators and the pump of the assist device were
changed. However, this did not result in an improvement of the laboratory figures. Echocardiographical studies were performed transthoracically during that period, as the patient suffered from intermittent spontaneous bleeding from the oro-pharynx (probably caused by thrombocytopenia). After 48 h of severe hemolysis the patient became hemodynamically unstable and needed high doses of catecholamines. Under these circumstances a transesophageal echocardiography was performed and detected a huge thrombus emanating from the pulmonary veins and filling out at least the hole left atrium and parts of the left ventricle. A CT-scan was performed to estimate the whole dimension of the thrombus and to find out if thrombotic material had already been torn off via the systemic circulation, potentially causing cerebral embolism. As there were no cerebral abnormalities found on the CT-scan, the patient underwent an operative closure of the proximal ascending aorta to eliminate the risk of cerebral embolism. Unfortunately, the patient developed a severe acute liver failure after the operation and died. The CT-scan showed a severely impaired hepatic perfusion, probably caused by multiple embolisms (Figs. 1 and 2).

2. Discussion

Left ventricular thrombus formation in patients with HOCM has been described, but only in patients with apical aneurysms [4]. We assume that this huge intracardiac thrombosis was the consequence of two facts:

1. Pulmonary hypertension (PH) is a characteristic feature of ARDS. The magnitude of PH has been shown to correlate with the severity of lung injury in patients with ARDS. Pulmonary vasoconstriction, thromboembolism and interstitial edema contribute to the development of PH even in the early histopathological evolution of ARDS [5]. Resulting in a high right ventricular afterload, these pathomechanisms may have led to a hypoperfusion of the lungs, and thus to a reduced blood supply to the left atrium and ventricle.

2. The small dimension of the left ventricular cavity in a patient with HOCM (preoperatively 32 mm) in combination with the severely reduced left ventricular function, especially in phases of ventricular tachycardia may have contributed to the impaired left ventricular filling.

The consequence of this case should be to keep in mind that in patients with anatomical cardiac abnormalities and cardiac failure, anticoagulation may have to be more intensive than in patients who do not fulfill these conditions. Veno-arterial ECMO in patients with respiratory- and cardiac failure is a rare clinical situation and requires sophisticated monitoring. If transesophageal echocardiography is not possible, as in our case, early CT diagnosis is warranted if intracardiac thrombosis is suspected.

References


