Pulmonary edema during repair of mycotic aneurysm of descending thoracic aorta, in postpartum period

B. KUMAR (*), Y. K. BATRA (**), A. SINGH (***) and S. K. S. THINGNAM (****)

Abstract: Infective endocarditis is uncommon during pregnancy (1). The difficulty in diagnosing infective endocarditis during pregnancy, may increase the likelihood of development of complications like a mycotic aneurysm of the descending thoracic aorta. We describe a case of mycotic aneurysm in the postpartum period, as sequel to delayed diagnosis; who developed pulmonary edema during its repair.

Key words: Infective endocarditis; mycotic aneurysm; pulmonary edema.

INTRODUCTION

Infective endocarditis is rare and difficult to diagnose during pregnancy (1). The disease may progress rapidly and cause life threatening complications if the diagnosis is not made promptly or, antibiotic therapy is delayed. Although there have been several report of infective endocarditis during pregnancy (1, 2), there is few in literature regarding the development of mycotic aneurysm of the descending thoracic aorta and its management in the post partum period as a sequel of delayed diagnosis.

We here in describe a case of mycotic aneurysm of the descending thoracic aorta that resulted in pulmonary edema during its repair in the postpartum period. We discuss the reasons for delay in diagnosis of infective endocarditis during pregnancy and the pathophysiology of the development of pulmonary edema during repair of a mycotic aneurysm.

CASE REPORT

A 27 year old female weighing 35 kg and height of 155 cms with 8 month pregnancy came to our emergency with fever, chills and severe anemia. Her past history was not significant for congenital or rheumatic valvular heart disease and intravenous drug abuse. On admission, her hemoglobin was 3 gm/dl for which she received 6 units of packed red blood cell. On investigation, hemoglobin electrophoresis and G6PD level were normal. Ampicillin, Gentamycin and Metronidazole were started for her fever chills and rigors awaiting blood culture reports which were subsequently changed to Vancomycin for six weeks since the blood culture showed staphylococcus aureus. The patient had spontaneous labor and delivered a low birth weight child via assisted breech delivery during the 2nd week of admission. During the post partum period she developed delirium with fever one week later. Her physical examination revealed mild splenomegaly and a systolic murmur at the apex. Routine blood examination showed thrombocytopenia and anemia. Chest X-ray revealed mild cardiomegaly. Electrocardiogram (ECG) showed a 1st degree atrioventricular block. Computerized tomography (CT) examination of the head revealed hyperintensity in bilateral (left more than right) periventricular white matter, suggesting embolic infarction. Transthoracic echocardiography revealed two large vegetations, one each on the anterior and posterior mitral valve leaflet causing severe mitral regurgitation (MR), and ejection fraction of 60%. The transesophageal echocardiography finding correlated with the transthoracic echocardiogram. Her blood culture became sterile on the 3rd week. Further investigations with CT angio thorax revealed a $2.7 \times 2.6$ cm saccular aneurysm.
on the posterolateral aspect of descending thoracic aorta with perianeurysmal leak (Fig. 1). She was planned for repair of the aneurysm and mitral valve replacement in the 5th week of postpartum period.

She was premedicated with oral diazepam 0.15 mg/kg on the night before and 1 hour prior to surgery. In the operating room, after attaching routine monitors a 16G intravenous cannula was inserted to left hand vein and right radial artery was cannulated under local anesthesia. Anesthesia was induced with morphine 10 mg, midazolam 2 mg and thiopentone titrated to loss of consciousness. Vecuronium 0.1 mg/kg was used to facilitate endotracheal intubation with a 7 mm internal diameter single lumen endotracheal tube (ETT). After induction a central venous catheter was inserted into the left internal jugular vein and the femoral artery was cannulated to monitor distal perfusion pressure. Maintenance of anesthesia was done with oxygen-air mixture, isoflurane, morphine and intermittent boluses of vecuronium. Dexamethasone 8 mg and epsilon aminocaproic acid 150 mg/kg was administered after induction of anesthesia. The aneurysm was exposed via left thoracotomy approach and an aortic cross clamp (ACC) was applied just proximal and distal to aneurysm in supra celiac descending thoracic aorta. Mannitol 1 g/kg and heparin 1 mg/kg was administered intravenously 30 and 3 minutes prior to ACC respectively. Immediately after ACC, her systolic blood pressure increased from 100 mmHg to a maximum of 140 mmHg despite 1 µg/kg/min nitroglycerin (NTG) infusion, which subsequently settled after increasing NTG infusion and deepening of anesthesia. After few minutes of ACC patient started to desaturate and was able to maintain only 95% oxygen saturation on FiO2 of 1. Chest auscultation revealed equal air entry on both sides and inspiratory crepts on both lower axillary regions. There was a decrease in lung compliance and ETT suction showed blood stained secretions. Arterial blood gas (ABG) analysis on FiO2 1 revealed pH 7.2, PaO2 97 mmHg, PaCO2 42 mmHg, HCO3 19 and base deficit 7. The total ACC time was 17 minutes. Before removal of the ACC, NTG was stopped and the patient received 1 meq/kg of NaHCO3 together with 300 ml 0.9% normal saline. The systolic blood pressure dropped to 70 mmHg after removal of the ACC which subsequently normalised with infusion of dopamine 10 µg/kg/min. Desaturation and inspiratory crepts persisted even after full expansion of lungs. A total of 800 ml 0.9% normal saline was used during the intraoperative period. Furosemide 10 mg intravenously was administered once her hemodynamic condition became stable. Further ABG analysis showed improvement in PaO2. The mitral valve was then replaced via a median sternotomy with use of cardio pulmonary bypass (CPB) and cardioplegic arrest of the heart. The CPB time and ACC times were 80 and 45 minutes respectively. CPB was terminated with the use of dopamine 10 µg/kg/min and adrenaline 0.05 µg/kg/min. Her post operative chest X-ray showed bilateral congested lung fields but ABG after shifting to intensive care unit was unremarkable. She was extubated the next morning and her subsequent postoperative course was uneventful.

**DISCUSSION**

Infective endocarditis during pregnancy is rare with an incidence varying from 0.006 to 0.0125% (1) commonly predisposed by rheumatic and congenital heart disease. The diagnosis of this disease is difficult during pregnancy due to various reasons like unnoticed temperature, paucity of physical signs in early stage, common occurrence of innocent heart murmurs during pregnancy and frequent use of antibiotics for other infections such as urinary tract infection during pregnancy and frequent use of antibiotics for other infections such as urinary tract infection which may mask the disease and lead to negative blood cultures (2). This delay in diagnosis may lead to catastrophic outcomes for both mother and fetus. Although pregnancy itself does not seem to influence the natural history of infective endocarditis, reported maternal
PU L MO N A R Y  E D E M A  D UR IN G R EPA IR O F MY C O TIC A N EU R YSM

Mycotic aneurysm due to infective endocarditis is rare due to the widespread use of antibiotics. Common sites include the femoral artery, abdominal aorta, and peripheral arteries. Most frequent organisms are gram-positive bacteria such as Staphylococcus Aureus and Streptococcus species, however, Gram-negative organisms can also be implicated. The mechanisms of mycotic aneurysm, particularly in larger vessels, are multifactorial and include direct trauma with contamination, local extension of an infected focus, septic microemboli to the vasa vasorum, or hematogenous seeding from a remote focus. In our case, the delay in diagnosis and treatment is likely to have caused septic microemboli to the vasa vasorum resulting in the development of a mycotic aneurysm, as evidenced by positive blood cultures.

In mycotic aneurysm, it is desirable to delay surgical intervention until improvement of inflammation by antibiotics, but rupture or impending rupture necessitates emergency surgery. The type of reconstruction depends principally on the localization and the number of aneurysms and on the extent of destruction of the aortic wall. The gold standard technique consists of removal of all infected tissues combined with an extra-anatomic bypass. However low long-term permeability, “aortic stump blow-out syndrome”, and unsuitable anatomy are drawbacks associated with this technique. Alternatively, a Dacron patch angioplasty can be performed if the lesion is well circumscribed and the adjacent aorta is not dilated. We preferred Dacron patch angioplasty since it was a small necked well circumscribed aneurysm.

Our patient developed desaturation with decreased lung compliance, bilateral coarse crepts and blood stained secretion from ETT, suggestive of pulmonary edema within few minutes of ACC. This may be due to both cardiogenic and non cardiogenic causes. The cardiogenic pulmonary edema is more likely in patients with MR requiring ACC. With ACC there is a sudden increase in impedance to aortic flow causing increased afterload. There is also a significant increase in blood volume in the organs and tissue proximal to ACC. Preload increases substantially as manifested by the increase in filling pressure, end-diastolic and end-systolic ventricular volume. In addition, patients with MR preoperatively have the left atrium (LA) and left ventricle (LV) volume overload due to regurgitant volume into LA from LV in addition to normal pulmonary venous return. The LV afterload is reduced due to the alternate pathway of ejection of LV into LA. With ACC in such a patient further increase in regurgitant volume is expected due to both increased impedance to forward flow, and increase in preload causing increased LV end diastolic volume. This sudden increase in volume overload in an unprepared LA might increase LA and pulmonary venous pressure to the extent to cause pulmonary edema.

Another possible cause is reperfusion pulmonary edema. This is due to increase in microvascular permeability and rapid passage of protein rich fluid across the endothelial cell barrier following ischemia and reperfusion. The mechanism involved includes effects of various mediators, in addition to pulmonary hypervolemia.

In our patient a cardiogenic cause may have played a predominant role in causing pulmonary edema since the aortic cross clamp time was short and pulmonary edema developed before aortic cross clamp was removed i.e prior to reperfusion. Nonetheless the contribution of reperfusion injury in perpetuating pulmonary edema cannot be ruled out particularly in the setting of pre-existing sepsis.

CONCLUSION

Infective endocarditis is rare and difficult to diagnose during pregnancy. The possibility of exacerbation of infective endocarditis presenting as febrile episodes in a pregnant woman should be born in mind to prevent a delay in the diagnosis and late consequences such as mycotic aneurysm.

References