Case Report

Pressure bandage over venous conduit harvesting site causing compartment syndrome in a patient with intra-aortic balloon pump: An unusual cause

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ABSTRACT

Intra Aortic Balloon Pump (IABP) is conventionally used to support coronary perfusion and weaning from cardiopulmonary bypass. IABP in situ has its own share of complications. We present a case where a patient on IABP support who had reduced peripheral pulsations of the ipsilateral limb and was initially misdiagnosed as IABP catheter associated thromboembolism. A negative embolectomy ruled out the same. Further looking for the cause of reduction of ipsilateral pulses it was found that the tight compressive bandage at saphenous vein conduit harvesting site had led to development of compartment syndrome (CS).

Key words: Compartment syndrome; Thromboembolism; Venous conduit

INTRODUCTION

Use of intra-aortic balloon pump (IABP) to support coronary perfusion and weaning from cardiopulmonary bypass (CPB) is not uncommon. Use of IABP mandates vigilant postoperative surveillance as it is associated with a multitude of complications. We encountered a case where a patient on IABP support was initially misdiagnosed as suffering from IABP catheter-associated thromboembolism. Later on, it was found that the tight compressive bandage at saphenous vein conduit harvesting site was the offending agent which had led to the development of compartment syndrome (CS). After taking written and informed consent from the patient, we wish to report this case.

CASE REPORT

A 44-year-old male patient, diagnosed with coronary artery disease and mitral valve regurgitation was posted for coronary artery bypass grafting (CABG) along with mitral valve replacement.

Preoperative coronary angiogram revealed triple vessel disease with echocardiogram showing a dilated left ventricle with an ejection fraction of 45%, hypokinesia of posterior wall, and interventricular septum with severe mitral regurgitation.

General anesthesia was administered. Intraoperative monitoring included electrocardiography, pulse oximetry, invasive blood pressure (left radial and right femoral), temperature, central venous pressure, urine output, and transesophageal echocardiography (TEE). After primary median sternotomy, CPB was established. Activated clotting time (ACT) was maintained more than 480 s throughout the bypass period. Grafts performed were left internal mammary artery to left anterior descending, right internal mammary artery to...
posterior descending and reverse right great saphenous vein graft to the obtuse marginal artery. Further heart was arrested, and the mitral valve was replaced with mitral valve prosthesis.

Post-surgery, weaning from CPB failed initially due to severe left ventricular dysfunction seen on TEE. An IABP (8.0 Fr) via right femoral artery had to be inserted (1:1 mode) and the patient was shifted to Intensive Care Unit (ICU) with vasoactive support of adrenaline (0.1 mcg/kg/min), dobutamine (5 mcg/kg/min), and noradrenaline (0.1 mcg/kg/min). ACT was kept more than 200 s with heparin infusion as IABP was in-situ. For sedation midazolam and fentanyl infusions were used.

In ICU, following the improvement of hemodynamic status, weaning from inotropic and IABP support was initiated. IABP’s mode was de-escalated to 1:3 and the patient maintained on noradrenaline (0.05 mcg/kg/min). Palpation of peripheral pulses and color Doppler was done every 2 h. After 12 h, it was observed that there was a reduction in Doppler flow signals associated with reduced right sided dorsalis pedis and tibialis anterior pulses. This reduction of Doppler flow was also associated with increasing trends of serial lactate levels. Contemplating IABP associated thromboembolism, the patient was shifted to the operating room for embolectomy.

General anesthesia was administered with the same monitoring as mentioned beforehand. IABP site was explored, and it was removed after taking proximal and distal control. Embolectomy was attempted by Fogarty’s catheter (3 Fr and 5 Fr both proximally and distally) but no thrombus/clot could be retrieved. There was excellent antegrade and retrograde flow at IABP arteriotomy site. However, neither the Doppler flow nor the pulses showed any improvement. In the absence of any clot on embolectomy, our focus shifted to other causes of reduced right lower limb pulsations. To rule out the cause, the dressing of the right lower limb from where saphenous vein graft conduit was harvested was removed, and dressing was found to be more tight than usual. The limb’s skin was found to be cold, tense, and shiny and had developed blisters [Figure 1]. Diagnosis of CS was made and fasciotomy [Figure 2] was done to relieve intra-compartmental tension.

Doppler flow, pulsations, and serial lactate levels gradually improved over 4 h after fasciotomy. Next day, the patient was extubated and subsequently shifted to the ward after 3 days of ICU stay. Patient was referred to plastic surgery for fasciotomy wound.

**DISCUSSION**

There is abundant medical literature with case reports, reviews, and studies highlighting complications of IABP. CS is a relatively rare albeit dangerous, limb-threatening complication of CABG, and intra-aortic balloon catheter insertion.

Initially, we had suspected thromboembolic complication of IABP which is a known complication but a negative embolectomy ruled out the same. Furthermore, removal of IABP failed to normalize the pulses. However, a trivial maneuver of removal of the tight occlusive dressing over venous conduit harvesting site resulted in improvement of flow on color Doppler along with improvement in pulses.

Sensory and pain changes in a lower limb with an intra-aortic balloon catheter in place should necessitate evaluation for acute CS even in the presence of normal pulses. Diagnosis of lower limb CS in an unconscious,
ventilated, hemodynamically compromised patient is difficult, if not observed very keenly. Postoperative cardiac patients are usually kept sedated to reduce myocardial demand in early postoperative hours which may delay the diagnosis of CS.[7]

The maintenance of ACT above 200 s also increased the chances of development of CS.[8] As CS was clinically evident, immediate fasciotomy was done to decrease tissue pressure, restore blood flow, and minimize tissue damage. Clinical observation of the limb and serial use of Doppler color flow aids in early diagnosis of CS. Any alteration in vascular flow is it due to thromboembolism (commonly) or CS as in our case can be ascertained.

Tight bandage can increase intra-compartmental pressure and close thin-walled venules resulting in hypertension at the venous end of the capillary beds. Subsequently, arteriolar compression may lead to muscle and nerve ischemia. The primary treatment here should be loosening the occlusive dressing and urgent surgical decompression (fasciotomy).

Similar cases where a tight crepe bandage lead to CS (although in different surgeries) have been reported by Danner et al. who described four patients developing CSs, due to constrictive bandages applied after varicose veins stripping.[9]

Thus, tight pressure bandage over venous conduit harvesting site in a post-CABG patient must be kept in deliberation as a basis of CS in a patient with IABP. Serial clinical assessment of both limbs and pressure bandages over venous conduit harvesting site must be done at 2 hourly intervals to minimize vascular complications.

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