

Delayed presentation of bone cement implantation syndrome requiring extracorporeal membrane oxygenation support

Ekstrakorporeal membran oksijenizasyonu gerektiren geç seyirli kemik çimentosu implantasyon sendromu

Ulaş Kumbasar¹, Pramod Bonde²

Department of Cardiothoracic Surgery, Yale University School of Medicine, New Haven, USA

ABSTRACT

Bone cement implantation syndrome is a rare and potentially fatal complication which may occur following cemented bone surgery. Herein, we present a case of delayed and fatal presentation of bone cement implantation syndrome following cemented spinal surgery, despite mechanical support with extracorporeal mechanical oxygenation.

Keywords: Bone cement, extracorporeal mechanical oxygenation, implantation.

ÖZ

Kemik çimentosu implantasyon sendromu, çimento kullanılan kemik ameliyatları sonrası görülebilen nadir ve ölümcül olabilen bir komplikasyondur. Bu yazıda, çimento uygulanarak yapılan spinal cerrahi sonrası geç dönemde ortaya çıkan ve ekstrakorporeal membran oksijenizasyonu ile mekanik desteğe rağmen ölümcül seyreden kemik çimentosu implantasyon sendromu olgusu sunuldu.

Anahtar sözcükler: Kemik çimentosu, ekstrakorporeal membran oksijenizasyonu, implantasyon.

Bone cement implantation syndrome (BCIS) is a rare and potentially fatal complication characterized by hypoxia, hypotension, pulmonary hypertension, cardiac arrhythmias, neurological dysfunction, and cardiac arrest. It is an acute devastating event which may occur in patients undergoing cemented bone surgery.^[1] In this article, we present a case of delayed and fatal presentation of BCIS following cemented spinal surgery, despite mechanical support with extracorporeal mechanical oxygenation (ECMO).

CASE REPORT

A 76-year-old male patient with a past medical history of hyperlipidemia, mild aortic stenosis, recent lumbar spinal surgery, and recent postoperative mild subsegmental pulmonary embolism was admitted to an external institution for repeat spinal surgery. His preoperative investigations including

electrocardiogram (ECG) and transthoracic echocardiography (TTE) were within normal limits. He underwent revision of L2 and L4 screws with application of bone cement under general anesthesia. His intraoperative hemodynamic parameters were normal throughout the procedure. Postoperatively, he was transferred to post-anesthesia care unit (PACU). Low-dose Neo-Syneprine[®] (Baxter Healthcare Corporation, Deerfield, IL, USA) was started to maintain a mean arterial pressure (MAP) target of >65 mmHg. One unit of red blood cell was given to maintain a hemoglobin value of >9 g/dL. However, he started to become more hypotensive and was transferred to the surgical intensive care unit. He became increasingly hypotensive during evaluation, ultimately requiring three pressors, norepinephrine, vasopressin, and phenylephrine. He was intubated for airway protection. The ECG showed junctional

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Correspondence: Ulaş Kumbasar, MD. Department of Cardiothoracic Surgery, Yale University School of Medicine, 06525 New Haven, USA.
Tel: +1 (203) 506 7859 e-mail: ulaskumbasar@gmail.com

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bradycardia with ST changes in V1-V2. Due to the concern for right-sided infarction, right-sided ECG was performed which showed ST changes in right V3-V4. Urgent cardiac catheterization showed 60% eccentric stenosis of proximal left anterior descending (LAD) coronary artery; however, this was not considered the source of the acute shock episode. The TTE revealed newly reduced left ventricular ejection fraction (LVEF) of 37%, reduced right ventricular (RV) function, hypokinetic apex, apical anterior, apical lateral, apical septum, and mid-inferolateral segments. Computed tomographic (CT) pulmonary angiogram was done to rule out pulmonary embolism and was found to be negative. The creatinine, lactate and liver function test levels started to increase, while urinary output decreased. The patient was transferred to our institution for possible mechanical support. In our institution, he was urgently evaluated, and peripheral (right femoral artery and vein) venous arterial extracorporeal membrane oxygenation (VA-ECMO) was initiated for salvage therapy in the setting of profound shock and multisystem organ failure (Figure 1). His



Figure 1. Hip X-ray of right-sided hip showing the femoral artery and vein cannulas for initiation of venous arterial extracorporeal membrane oxygenation.

pressor requirements increased, and he remained increasingly acidotic, despite continuous veno-venous hemofiltration (CVVH) therapy. He continued to require high-dose pressors, volume boluses, and blood products. A cranial, thoracic and abdominal/pelvic non-contrast CT was unremarkable for acute process or reversible cause. Severe lactic acidosis persisted, despite all interventions. He started to have multiple episodes of ventricular fibrillation which he was shocked. The patient's condition was discussed with his family and ultimately made the decision to withdraw care. Autopsy report revealed four-chamber dilatation of the heart, infarction at the left lower lobe of the lung, and focal ischemic injury of the myocardium. Informed consent was taken from the patient's legal guardian.

DISCUSSION

Bone cement implantation syndrome is a rare and devastating postoperative complication which may occur in patients undergoing cemented bone surgery and may even lead to death in 0.6 to 1% of patients.^[2] It was first described by Powell^[3] in 1970. Since then, several case reports have been published with varying clinical presentations, including death.^[1-5] Its etiology and pathophysiology are still unclear. Proposed mechanisms include toxic effects of methyl methacrylate cement monomer, fat and bone marrow embolization, exothermic reaction, histamine release, complement activation, and endogenous cannabinoid mediated vasodilatation.^[4,5] Most recent studies have focus on embolism of medullary contents (i.e., fat, marrow, cement, air, and small bone fragments) formed during the cementation process. The cement undergoes an exothermic reaction and expands, trapping air and medullary contents under pressure, thereby, leading to embolism. Subsequently, RV failure occurs due to high pulmonary vascular resistance (PVR), resulting in a reduced cardiac output. Acute enlargement of the RV pushes the intraventricular septum to the left causing poor filling of the left ventricle, reduction in cardiac output, systemic hypotension, decrease in coronary perfusion pressure which may end up with cardiac arrest.^[1,5] Clinical decline usually occurs within minutes after cement implantation. However, delayed presentation, as in our patient, suspects the involvement of other mechanisms, as well.^[5]

Severity of BCIS has been classified into three grades: Grade I, moderate hypoxia (SpO₂ <94%) or hypotension (fall in systolic blood pressure [SBP] >20%); Grade II, severe hypoxia (SpO₂ <88%) or

hypotension (fall in SBP >40%) or unexpected loss of consciousness; and Grade III, cardiovascular collapse requiring cardiac resuscitation.

Management is basically supportive and includes administration of 100% oxygen, fluids, vasopressors (alpha-1 agonists), invasive monitoring, and intensive care. Preventive strategies, particularly in high-risk patients, include applying non-cemented prosthesis, use of low-viscosity cement, retrograde application of the cement, and avoidance of high-pressure during implantation.^[6,7] Although BCIS is an acute-onset and transitory process, and healthy patients improve even within minutes and high PVR returns to normal within 24 to 48 h, in patients with underlying risk factors and severe cardiopulmonary disease, as in our patient, it may lead to severe cardiopulmonary dysfunction with catastrophic outcomes.^[1,5,8] Early and aggressive resuscitation, as well as proper management of RV failure, are the mainstays of the treatment in high-grade patients. In patients in whom cardiopulmonary dysfunction persists despite all medical measures, bedside mechanical support should be considered as a salvage therapy, as utilized in patients with massive acute pulmonary embolism.^[9] Considering acute clinical presentation associated with underlying cardiopulmonary dysfunction, cementing surgery, and biventricular dysfunction without any other obvious reason, we concluded that BCIS was the most probable diagnosis in our patient. To the best of our knowledge, there is no such report of delayed postoperative presentation of BCIS managed with mechanical support to date.

In conclusion, bone cement implantation syndrome is a catastrophic complication that may also appear as a delayed presentation. It should be kept in mind in patients having cardiopulmonary compromise following cemented surgery and these patients should be treated aggressively. However, it is a potentially fatal phenomenon, despite all best treatment efforts.

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