Scholars Journal of Medical Case Reports
Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

OPEN ACCESS

Anesthesiology

Postoperative Presentation of Bone Cement Implantation Syndrome: Case Report

Chikhi Brahim[1*], Hmadate Ilyass[1], Aarjouni Youssef[1], Fakri Ahmed[1], Benani Mohamed[1], Chakib Chouikh[1], Balkhi Hicham[2]

[1]Department of Anesthesiology and Intensive Care, Military Hospital Mohammed V, Faculty of Medicine and Pharmacy of Rabat, Mohammed V University, Rabat, Morocco
[2]Head of the Anesthesiology and ICU Department, of The Mohamed V Military Training Hospital, Faculty of Medicine and Pharmacy of Rabat, Mohammed V University, Rabat, Morocco

DOI: https://doi.org/10.36347/sjmcr.2025.v13i03.042 | **Received:** 19.02.2025 | **Accepted:** 22.03.2025 | **Published:** 27.03.2025

***Corresponding author:** Chikhi Brahim
Department of Anesthesiology and Intensive Care, Military Hospital Mohammed V, Faculty of Medicine and Pharmacy of Rabat, Mohammed V University, Rabat, Morocco

Abstract **Case Report**

***Background*:** Bone Cement Implantation Syndrome (BCIS) is a potentially fatal complication associated with cemented orthopedic procedures, primarily hip arthroplasty. It is characterized by acute respiratory and cardiovascular instability, often occurring intraoperatively or immediately after cementation. However, delayed-onset BCIS remains a rare and under-recognized entity. ***Case Presentation*:** We report the case of a 75-year-old male with a history of well-controlled chronic obstructive pulmonary disease (COPD) who underwent cemented hemiarthroplasty for a right femoral neck fracture. The preoperative assessment classified him as ASA II, with no significant cardiopulmonary compromise. Surgery was uneventful under spinal anesthesia, with stable intraoperative hemodynamics. However, one hour postoperatively, the patient developed respiratory distress, profound hypoxia (SpO_2 85%), and hemodynamic instability (BP 87/66 mmHg, HR 119 bpm). Despite initial resuscitative measures, he deteriorated further, necessitating intensive care unit (ICU) admission, invasive monitoring, mechanical ventilation, and vasopressor support. Transthoracic echocardiography revealed acute right ventricular (RV) failure, a hallmark of severe BCIS. Over the following days, intensive supportive management led to gradual clinical improvement, and the patient was successfully extubated on day six. He was discharged from the ICU on day eight and later recovered without further complications. ***Conclusion*:** This case underscores the importance of heightened awareness of BCIS, particularly its delayed presentation. A multidisciplinary approach, prompt diagnosis, and aggressive supportive management are crucial in improving survival in high-risk patients undergoing cemented arthroplasty.

Keywords: Bone Cement Implantation Syndrome (BCIS), Delayed-onset BCIS, Cemented Hemiarthroplasty, Right Ventricular Failure, Respiratory Distress.

INTRODUCTION

Bone Cement Implantation Syndrome (BCIS) is a serious perioperative complication that can result in severe cardiovascular and respiratory instability. It is most frequently associated with cemented hip arthroplasty and is thought to arise from embolization of marrow and cement particles into the pulmonary circulation. This leads to increased pulmonary vascular resistance (PVR), right ventricular (RV) strain, and systemic hypotension. While BCIS is typically recognized intraoperatively or immediately post-cementation, delayed-onset cases are rare and underreported. Here, we describe a case of BCIS presenting one hour postoperatively, underscoring the need for continued vigilance and timely management to improve patient outcomes.

CASE REPORT

A 75-year-old patient presented to our hospital with a right-sided femoral neck fracture following a fall in the bathroom. He was a former smoker with a history of chronic obstructive pulmonary disease (COPD), which was well controlled with inhaled therapy (salmeterol plus fluticasone, and salbutamol as needed).

Pre-Anesthetic Assessment revealed a patient breathing normally at 18 breaths per minute, with no signs of respiratory distress. Dyspnea was classified as

NYHA stage II, and oxygen saturation was 97% on room air. Pulmonary auscultation revealed no wheezing or crackles. Cardiovascular examination showed a blood pressure of 118/65 mmHg, and a heart rate of 76 beats per minute functional capacity exceeding 4 metabolic equivalents of task (METs) prior to the fracture, no exertional angina, no peripheral signs of heart failure. Neurological evaluation found the patient to be conscious, alert, and without sensory or motor deficits. The anesthesia-focused airway assessment revealed no predictors of difficult intubation or ventilation.

Preoperative laboratory tests were within normal limits. Hemoglobin was 12.6 g/dL, and blood type was O +. Coagulation parameters, serum electrolytes, and renal function tests were all normal. Chest radiography revealed emphysematous lungs, and electrocardiogram (ECG) was unremarkable. No additional investigations, such as echocardiography or arterial blood gas analysis, were performed, as they were deemed unnecessary. The patient's COPD was well controlled, his exercise tolerance exceeded 4 METs before the fracture, and there were no clinical indicators of significant cardiopulmonary impairment. The case was classified as ASA II, and the patient was scheduled for hemiarthroplasty (partial hip replacement).

The patient was admitted to the operating room, where standard monitoring was initiated**,** including non-invasive blood pressure (NIBP), electrocardiography (ECG), and pulse oximetry. He was found to be hemodynamically stable, with a blood pressure of 127/69 mmHg, a heart rate of 78 beats per minute, and an oxygen saturation (SpO_2) of 96% on room air. As part of preoperative optimization, he received two puffs of Ventolin (salbutamol). After establishing peripheral intravenous access with an 18-gauge cannula, a prophylactic dose of 2 grams of cefazolin was administered. Under strict aseptic precautions**,** spinal anesthesia was performed at the L3-L4 interspace using a Quincke 25G needle. A total of 3 mL of 0.5% bupivacaine**,** along with 25 µg of fentanyl and 100 µg of morphine, was injected intrathecally through a midline approach**.** Intravenous bolus 3mg of ephedrine were administered to maintain blood pressure following administration of spinal anesthesia. After 10 min, he was put in a left lateral position. The procedure then conducted with close monitoring of vital signs. During cement application, several preventive measures were applied. These included increasing the inspired oxygen fraction (FiO_2), optimizing intravascular volume to avoid hypovolemia and proactively increasing arterial blood pressure. A **r**ight-sided cemented hemiarthroplasty was successfully performed. The patient's vitals remained stable throughout the procedure. The surgery lasted 1 hour and 30 minutes, with an estimated blood loss of 200 ml. Intraoperative fluid management included the administration of 1.5 liters of crystalloid solution.

Following the procedure, the patient was transferred to the post-anesthesia care unit (PACU) for monitoring. Postoperative monitoring showed no signs of cardiorespiratory distress, bleeding, or neurological deficits. His pain was well controlled with a multimodal analgesic approach. After an uneventful recovery period in the PACU, he was transferred to the orthopedic service for further postoperative management.

After one hour, the patient developed signs of respiratory and hemodynamic distress. He became tachypneic, exhibiting respiratory effort with accessory muscle use and a progressive oxygen desaturation to 85%, despite the absence of abnormal lung sounds on auscultation. Hemodynamically, he experienced a drop in blood pressure to 87/66 mmHg and developed sinus tachycardia at 119 beats per minute.

Initial resuscitative measures were undertaken, including intravenous fluid administration with normal saline 0.9% and supplemental oxygen therapy at 5 L/min via a nasal oxygen cannula. However, there was no significant clinical improvement. Further investigations were conducted to determine the underlying cause. Electrocardiogram (ECG) revealed sinus tachycardia without ischemic changes or arrhythmias. A chest X-ray showed no abnormalities, ruling out pulmonary edema or pneumothorax. Arterial blood gas analysis demonstrated type I respiratory failure with a pH of 7.36, PaO_2 of 55 mmHg, $PaCO_2$ of 32 mmHg, and HCO_3^- of 19 mEq/L, indicating hypoxemia without hypercapnia. Given the clinical suspicion of pulmonary embolism, a thoracic CT angiography (CTA) was performed, which ruled out an embolic event.

Due to persistent respiratory compromise and hemodynamic instability despite initial supportive measures, the patient was transferred to the intensive care unit (ICU) for further management, close monitoring, and advanced supportive care.

On admission to the intensive care unit, the patient was found to be in severe shock, with a blood pressure of 77/45 mmHg, a heart rate of 125 beats per minute, and an oxygen saturation of 80% despite receiving 5 L/min of supplemental oxygen. He was tachypneic and confused, suggesting significant respiratory and circulatory failure. Given the worsening hemodynamic instability, respiratory distress, and neurological deterioration, urgent endotracheal intubation was performed to secure the airway and optimize oxygenation.

A central venous catheter and an arterial line were placed for continuous hemodynamic monitoring and vasopressor support. Noradrenalin was initiated at 1.8 µg/kg/min. A bedside transthoracic echocardiogram (TTE) revealed acute right heart failure, characterized by a dilated right ventricle (RV) with severely impaired systolic function, significant tricuspid

regurgitation, while left ventricular ejection fraction was preserved (LVEF). Management of acute right heart failure focused on reducing RV afterload, optimizing preload, and improving inotropism. Dobutamine was administered at 5 µg/kg/min to enhance right ventricular contractility. Fluid resuscitation was cautiously adjusted using transthoracic echocardiography to assess hemodynamic status and guide ongoing therapy to maintain adequate preload without exacerbating RV strain. Mechanical ventilation was tailored to minimize intrathoracic pressure and avoid worsening right ventricular dysfunction. Laboratory tests were within normal limits, except for a positive troponin result 0.095 ng/mL.

Over time, the patient's hemodynamic status gradually improved. Vasopressor and inotropic support were progressively weaned off, and careful ventilator management allowed for a successful extubation on the sixth day. However, during the ICU stay, the patient developed ventilator-associated pneumonia (VAP), confirmed by clinical and microbiological findings. Broad-spectrum antibiotic therapy with piperacillin-tazobactam and amikacin was initiated and adjusted based on culture results and clinical response.

Through multidisciplinary critical care support, the patient ultimately showed progressive recovery, with resolution of shock and respiratory distress. By day 8, the was discharged from ICU to the orthopedic service.

DISCUSSION

Bone Cement Implantation Syndrome (BCIS) is a well-recognized but under-reported complication associated with orthopedic procedures that involve cemented prostheses. It is characterized by a spectrum of cardiovascular and respiratory disturbances, ranging from transient hypoxia and hypotension to fulminant cardiovascular collapse [1]. BCIS is most commonly seen in hip arthroplasty but has also been reported in other orthopedic procedures such as femoral reaming, acetabular prosthesis insertion, knee replacement, vertebroplasty, and tourniquet deflation [2]. The incidence of BCIS varies widely, with reports suggesting that it occurs in 25–30% of patients undergoing cemented hemiarthroplasty for hip fractures [3]. The severity of BCIS has been classified into three grades [4]:

- **Grade I:** mild hypoxia (SpO_2 < 94%) or a drop in systolic blood pressure (SBP) > 20% of baseline.
- **Grade II:** moderate hypoxia (SpO_2 < 88%), SBP drop > 40% of baseline, and transient loss of consciousness.
- **Grade III:** cardiopulmonary arrest.

Our patient developed delayed-onset Grade III BCIS, manifesting with profound hypoxia, hypotension, tachycardia, and subsequent right ventricular (RV) failure requiring ICU admission, vasopressor support, and mechanical ventilation. This delayed presentation, occurring over one hour postoperatively, is unusual, as BCIS is typically reported intraoperatively or immediately after cementation [1].

The pathophysiology of BCIS remains incompletely understood, but the predominant mechanism is believed to be hemodynamic compromise due to embolization of bone marrow, fat, and cement particles into the pulmonary circulation [5]. The embolization results from pressurization of the bone cavity, which can exceed 300 mmHg during cementation and prosthesis insertion. These emboli induce the release of vasoactive mediators, leading to increased pulmonary vascular resistance (PVR), RV dysfunction, and reduced cardiac output [4-6]. In severe cases, RV dilation causes a septal shift, impairing left ventricular (LV) filling and leading to systemic hypotension and shock [7].

Pulmonary embolization can also contribute to ventilation-perfusion mismatches, worsening hypoxia. Furthermore, embolic particles may reach the systemic circulation through a patent foramen oval (PFO) or via pulmonary arteriovenous shunts, potentially causing cerebral microembolization and postoperative delirium in elderly patients [1-8]. Our patient presented with severe hypoxia and confusion, requiring urgent intervention. However, the confusion could not be definitively attributed to BCIS, as we did not perform an MRI to assess for cerebral embolization, nor could we rule out hypotension or hypoxia as a contributing factor.

Several patient and procedure related factors increase the risk of BCIS, including advanced age (particularly > 75 years), high ASA grade (III or IV), pre-existing cardiopulmonary disease (e.g., COPD, Congestive heart failure, pulmonary hypertension), use of diuretics or warfarin, cemented prosthesis particularly with high-pressure cementation, long-stem prostheses and revision surgeries [1-9]. Our patient had multiple risk factors, including advanced age, ASA II status, COPD, and a cemented hemiarthroplasty, making him susceptible to BCIS. Additionally, COPD is often associated with pulmonary hypertension and vascular remodeling, which may exacerbate the effects of BCIS-related embolization and increased PVR [9]. Given the patient's clinical presentation, multiple high-risk factors, cemented arthroplasty, and evidence of right ventricular failure, BCIS emerged as the most probable diagnosis.

Preoperative evaluation and continuous intraoperative vigilance are critical in the prevention and management of BCIS due to a lack of consensus in the literature regarding the best anesthetic technique for cemented bone surgery [10]. BCIS prevention begins with identifying high-risk patients during the preoperative assessment and communication between surgical, anesthetic, and medical providers regarding the selection of the type of prosthesis, surgical procedure, and techniques to minimize the risk of cardiovascular

complications for high-risk patients with multiple or severe risk factors or comorbidities [8-11]. Anemia is common in adults over the age of 65 (17%), and the incidence is even higher in the hip fracture population. Thus, preoperative and intraoperative blood and fluid restoration of the patient should be considered, and intravascular fluid volume should be kept as close to normal as possible [12].

BCIS is a reversible, time-limited phenomenon, according to several studies. Pulmonary artery pressure can return to normal within 24 hour, and non-diseased hearts can recover in minutes to hours [8]. This means that early recognition of BCIS combined with appropriate management are essential in preventing further deterioration, and can significantly impact patient outcomes. A sudden drop in end-tidal CO_2 ($EtCO_2$) intraoperatively is a key diagnostic clue and should alert the anesthetist. Oesophageal Doppler measurements may detect impending BCIS at an earlier stage than standard hemodynamic monitoring [13]. Transesophageal echocardiography may reveal emboli that were named "snow flurry" by Lafont *et al*., these emboli were seen with both cemented and non-cemented procedures [14]. Early signs of BCIS in the awake patient undergoing regional anesthesia include dyspnea and altered sensorium [15]. If BCIS is suspected, immediate resuscitation should be based on general principles. Initially, the anesthetist should secure the airways, and increase the inspired oxygen concentration to 100% [8]. It has been suggested that cardiovascular collapse in the context of BCIS should be treated as RV failure [16]. This includes the administration fluid therapy to maintain normovolemia and avoid hypotension, the use of pulmonary vasodilators (inhaled nitric oxide and prostaglandin E1) to decrease the pulmonary artery pressure if hypoxemia and right ventricular dysfunction worsens, and the use of inotropes (dobutamine and milrinone) to preserve right ventricular contractility [3-6]. Additionally, to maintain hemodynamic stability, direct-acting α-1 agonists (epinephrine, norepinephrine) may be administered [1-19]. Additionally, in cases of bradycardia and hypotension, ephedrine, an α- and β-adrenergic agonist, may be given; however, giving any sympathomimetics without addressing pulmonary vasoconstriction may result in rapidly increased right ventricular preload and acute right ventricular failure [20]. In our case, we adhered to these management principles, implementing early resuscitative measures including securing airways, invasive monitoring, fluid therapy. Pulmonary vasodilators were not available, limiting our ability to further optimize pulmonary hemodynamics. Instead, we managed the patient with vasopressors, inotropes, and ventilatory support leading to a favorable outcome despite the severity of BCIS.

CONCLUSION

Bone Cement Implantation Syndrome is a serious perioperative complication that typically arises during cementation but can also present in a delayed manner. Early recognition and aggressive resuscitation are crucial to improving patient outcomes and minimizing the risk of unexpected fatalities during cemented arthroplasty procedures.

REFERENCES

1. Donaldson, AJ · Thomson, HE · Harper, Bone cement implantation syndrome Br J Anaesth. 2009, 102:12-22
2. Byrick, RJ · Forbes, D · Waddell, JP A monitored cardiovascular collapse during cemented total knee replacement Anesthesiology. 1986, 65:213-216
3. Govil P., Kakar P.N., Arora D., Das S., Gupta N., Govil D., et. al.: Bone cement implantation syndrome: a report of four cases. Indian J Anaesth 2009, 53: pp. 214-218.
4. D. So, C. Yu, M.A. Doane Subscribe to ATOTW Tutorials by Visiting Bone Cement Implantation Syndrome (2017), pp. 1-6
5. Urban M.K., Sheppard R., Gordon M.A., Urquhart B.L. Right ventricular function during revision total hip arthroplasty. Anesth. Analg. 1996, 82:1225–1229.
6. Hines C.B. Understanding Bone Cement Implantation Syndrome. AANA J. 2018, 86:433–441.
7. Sastre J.A., Lopez T., Dalmau M.J., Cuello R.E.: Bone cement implantation syndrome during spinal surgery requiring cardiac surgery. A&A Case Rep 2013, 1: pp. 82-85.
8. Khanna G., Cemovsky J.: Bone cement and the implication for anaesthesia. Contin Educ Anaesth Crit Care Pain 2012, 12: pp. 213-216.
9. Olsen F., Kotyra M., Houltz E., Ricksten S.-E. Bone cement implantation syndrome in cemented hemiarthroplasty for femoral neck fracture: Incidence, risk factors, and effect on outcome. Br. J. Anaesth. 2014, 113:800–806.
10. Chwarzkopf E., Sachdev R., Flynn J., Boddapati V., Padilla R.E., Prince D.E. Occurrence, risk factors, and outcomes of bone cement implantation syndrome after hemi and total hip arthroplasty in cancer patients. J. Surg. Oncol. 2019, 120:1008–1015.
11. Griffiths R., White S.M., Moppett I.K., Parker M.J., Chesser T.J., Costa M.L., Johansen A., Wilson H., Timperley A.J. Safety guideline: Reducing the risk from cemented hemiarthroplasty for hip fracture 2015, Association of Anaesthetists of Great Britain and Ireland British Orthopaedic Association British Geriatric Society. Anaesthesia. 2015, 70:623–626.
12. Rocos B., Whitehouse M.R., Kelly M.B. Resuscitation in hip fractures: A systematic review. BMJ Open. 2017, 7:e015906.

13. Clark, DI · Ahmed, AB · Baxendale, Cardiac output during hemiarthroplasty of the hip. A prospective, controlled trial of cemented and uncemented prostheses *J Bone Joint Surg*. 2001, **83**:414-418
14. Kalra, A., Sharma, A., Palaniswamy, C., El-Oshar, S., Desai, P., Yazbeck, M., & Zanotti-Cavazzoni, S. L. (2013). Diagnosis and management of bone cement implantation syndrome: Case report and brief review. *American journal of therapeutics*, *20*(1), 121-125.
15. Edmonds, CR · Barbut, D · Hager, Intraoperative cerebral arterial embolisation during total hip arthroplasty,*Anesthesiology*. 2000, 93:315-318.
16. Pietak, S · Holmes, J · Matthews, R .Cardiovascular collapse after femoral prosthesis surgery for acute hip fracture, *Can J Anesth*. 1997, 44:198-201.
17. Park H.J., Kang H., Lee J.W., Baek S.M., Seo J.S. Comparison of hemodynamic changes between old and very old patients undergoing cemented bipolar hemiarthroplasty under spinal anesthesia. Korean J. Anesth. 2015, 68:37–42.
18. Agpal J.K., Velankar P.M., Choudhary S., Nutangi V. Bone cement implantation syndrome: A dreaded complication during arthroplasty. Med. J. Dr. DY Patil Univ. 2015, 8:569
19. Chatterjee K., Mittadodla P.S., Colaco C., Jagana R. A Rare Cause of Pulmonary Edema in the Postoperative Period. Indian J. Crit. Care Med. 2017, 21:108–109.
20. Torres B. 5-HT3 receptor antagonism following suspected bone cement implantation syndrome [case report] Int. Student J. Nurse Anesth. 2013, 12:18–22.