Bone Cement Implantation Syndrome (BCIS)

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Abstract

Bone cement implantation syndrome (BCIS) is a poorly understood entity. It is an important cause of intraoperative mortality and morbidity in patients undergoing cemented hip arthroplasty and may also be seen in the postoperative period in a milder form causing hypoxia and confusion. Hip arthroplasty is being increasingly resorted to in the ageing population. We here report a severe hemodynamic derangement and transient hypoxemia observed during cemented arthro-plasty of hip in a 65 years old lady. Peripheral vasodilatory effects of the cement monomer, fat and marrow embolism and activation of the clotting cascade in the lungs, all contribute to bone cement implantation syndrome(BCIS). Early and aggressive resuscitation using vasopressors, establishment of invasive hemodynamic monitoring and surgical operative modifications are the key to prevention of this catastrophic syndrome.

Keywords- *Bone Cement, Complications, Hip arthroplasty*

Introduction

Joint replacement surgery, a common surgical procedure performed in the fast growing elderly population is primarily done to minimize disability, optimize function, and eliminate pain to improve the quality of life.

Bone cement implantation syndrome is a significant cause of morbidity and mortality in patients subjected to cemented hip arthroplasty. Total hip replacement (THR) and hemiarthroplasty are performed in patients with disabling arthritis or for femoral fracture either as a primary procedure or for failed internal fixation. Hip arthroplasties may be cemented, uncemented or hybrid. Cemented prosthesis are recommended for their better long term viability as well as due to the higher cost of noncemented prosthesis which have minimal significant potential benefits. Hazards of methyl methacrylate (MMA) like hypotension[2,4,12,13,14] hypoxia[1,5,11,12,13,14], arrhythmias[1,4,11], increased pulmonary vascular resistance (PVR) and cardiac arrest(1,4,5,11,14) have been recognised but the exact pathophysiology is not yet fully understood.

Intra operative cardio respiratory changes during total hip arthroplasties have been reported since cemented components were introduced in 1961[5,6].

BCIS has a varied presentation with a wide spectrum of severity. It is characterized by hypoxia, hypotension or both and/or unexpected loss of consciousness around the time of cementation/ prosthesis insertion/ reduction of joint or occasionally after tourniquet deflation.

We report a case of BCIS of grade 2 severity which was recognised early and managed empirically on basic physiological principles. Various risk factors and recent proposed theories of BCIS are discussed.

Case Report

Sixty five years old female was hospitalized with complaints of progressively worsening pain in the region of right hip and inability to walk since one month. She gave history of having sustained fracture of right femur due to an accidental fall for which she underwent an operation in a private hospital about a year back. Details

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of the procedure were not available. She was a known case of hypertension for which she used to take irregular medication for last ten years or so. She was used to regularly chewing tobacco for previous twenty five years.

She was posted for right hemiarthroplasty with cemented bipolar fixation. Preoperatively she was noted to be fully conscious, oriented with good cognition and had a pulse rate of 86/min and her blood pressure was 144/88 mmHg. Her systemic examination was within normal limits. Her haemoglobin was 12.84% and all her other routine investigations including blood sugar level, renal and liver function test, electrocardiography (EEG) and chest x-ray were essentially normal. X-ray spine showed severe osteoporotic changes. Patient preferred to undergo surgery under general anaesthesia.

On the day of surgery, she was taken for taken to operating room where all routine monitors including pulse oximeter, non-invasive blood pressure, skin thermometer probe, electrocardigraphy(ECG) and capnometer (for end tidal carbon dioxide) were connected and basal recording noted.

Preoxygenation with 100% oxygen, she was induced with inhalational agents, supplemented with 100mg injection Thiopentone sodium 2.5% intravenous, injection Fentanyl 80 mcg intravenous and injection diclofenac 75 mg intramuscular were given as preemptive analgesic agents before commencement of surgery and subsequently additional doses of injection fentanyl and infusion of paracetamol were administered. Maintainance anaesthesia was through cuffed oroendotracheal tube with oxygen, nitrous oxide and halothane by utilizing synchronized intermittent mandatory ventilation (SIMV) mode of ventilation.

During insertion of MMA cement into the bone marrow a sudden decrease in end-tidal carbon dioxide followed by fall in oxygen saturation and blood pressure was noticed. Sequential parameter recordings were

11:30 AM- HR 61/MIN, SPO2 -99%, ETCO2-31mm of Hg, BP-120/70mm of Hg

11:31 AM- HR 59/MIN, SPO2-94%, ETCO2-21mm of Hg, BP-104/64mm of Hg.

11:33 AM- HR 58/min, SPO2- 88%, ETCO2- 18mm of Hg, BP- 75/50mm of Hg.

Later the fall in ETCO2 & SPO2 was dramatic. Halothane delivery was 1.5 MAC at that time.

11:35 AM- HR-60/MIN, SPO2-80%, ETCO2 -12mm of Hg, BP 66/80mm of Hg

HALOTHANE -0.4 MAC (Decreased)

As the saturation dropped further she was ventilated with 100% oxygen with minimal halothane on Bain's circuit with Positive end expiratory pressure(PEEP). Flow rate of IV fluids was maximal.

11:36 AM- HR 75/MIN, SPO2-90%, ETCO2 - 20mm of Hg, BP- 114/72mm Hg

HALOTHANE -0.3 Minimum alveolar concentration(MAC)

Patient response was encouraging & ventilation with Bain's circuit continued. BP was maintained with Halothane – 0.6MAC.

11:45 AM- HR 86/MIN, BP 162/100 mm Hg, ETCO2 – 22 mm Hg, SPO2-99%

HALOTHANE -0.6MAC

After she stabilized mechanical ventilation by Synchronized intermittent mandatory ventilation(SIMV) mode was restarted. Her End- tidal carbon dioxide (30-35mm Hg) and Oxygen saturation (95-99%) stabilized after 20-30 min and remained so throughout the rest of surgery. **I/v fluids –** Ringer lactate (RL) 3 points and Haes-Steril 1 point was given intra-operatively. Extubation was done after full recovery of spontaneous breathing and stable vital parameters.

Patient was kept in recovery room for 2 hours with oxygen supplementation by mask and her vitals were monitored and were noted to be stable. Her subsequent postoperative course was uneventful.

Discussion

A fall in end tidal carbon dioxide concentration was first indication of BCIS in our patient alerting us to look for various different causes. This was followed by fall in SpO_2 and hypotension. Immediate empirical corrective measures based on basic physiological principles prevented further deterioration and catastrophic results.

Haemodynamic variations noted around the time of bone cementing in patients with BCIS were earlier thought to be due to the toxic effects of methyl methacrylate causing severe vasodilatation[6,7,8,9], but this line of thinking is not supported by different animal studies which have shown plasma methyl methacrylate(MMA) concentrations are much lower than concentrations required for causing pulmonary and cardiovascular system(CVS) effects.

Later studies looking for causation of bone cement implantation syndrome focused on possibility of

embolisation[2,11,14] of fat, marrow, air, cement/ bone particles and/or aggregates of platelets and fibrin due to increase in intramedullary pressure at the time of cementation. Exothermic reaction with increase in temperature as high as 96^0 C, six minutes after mixing of cement components is known to expand the space between prosthesis and bone, trapping air and forcing the medullary contents into the circulation[7,9]. Pressure generated by cement gun is almost double than manual packing of cement. During surgery methyl methacrylate(MMA) is intentionally forced into the interstices of bone for improved bonding by increasing contact surface area.

Table 3 : Mean and peak intramedullary pressures generated during cementation and prosthesis insertion in vented and unvented femurs.

	Peak pressure (mean, mmHg)	Finger packing Mean pressure (mean, mmHg)	Peak pressure (mean, mmHg)	Cement gun Mean pressure (mean, mmHg)
Unvented femur Cementation[15]	608	127	1177	322
Unvented femur (cadaveric studies) Cementation[15]	881	229	2051	374
Prosthesis insertion[15]	4931	3140	5003	3008
Vented femur Cementation [65] Unvented femurProsthes	>117			
is insertion[65]	>190			

Although the degree of embolic flurry is proportional to the intramedullary pressure, a ceiling effect due to the finite amount of debris present in the femoral canal has been recognised.

Pulmonary embolism of medullary contents results in characteristic hypoxia[1,5,11,12,13,14], hypotension[2,4,5,12,13,14] due to right ventricular(RV) dysfunction and emboli in cerebral circulation, through persistant foramen ovale(PFO) or transiting through pulmonary circulation may cause postoperative delirium or focal neurological deficit.

The physiological consequences of embolization[2,11,14] are considered to result from both mechanical effects and due to proinflammatory / vasoactive mediators (like thrombin, tissue thromboplastin, histamine, C3a, C5a complement activation) released, which provoke increase in pulmonary vascular tone.

It is rather difficult to pinpoint which of the above factors are seen in a individual patient as there is significant inter patient variability depending on pre-existing comorbidity and on the relative magnitude of changes in pulmonary vascular resistance(PVR), systemic vascular resistance(SVR) and myocardial contractility occurring after the embolic event.

Implicated patient risk factors for bone cement implantation syndrome include old age, impaired cardiopulmonary function, pulmonary hypertension[2,11,12,13,14], osteoporosis, bony metastases, concomitant hip fracture-pathological or intertrochanteric fracture. Patients with previously uninstrumented femoral canal are at a higher risk for bone cement implantation syndrome. Our patient was old, osteoporotic, hypertensive on irregular treatment and was operated earlier for fracture femur which are known risk factors[7,9].

Anaesthetic management of the bone implan-tation syndrome is supportive. Administration of 100% inspired oxygen with control of airway, invasive hemo-dynamic monitoring, aggressive volume therapy and use of vasopressors is required [15,16] .Surgical modifications to prevent excessive cement pressurization are para-mount in avoiding or minimizing bone cement implantation syndrome as complete elimi-nation of the embolic phenomenon is probably impos-sible [15,16,17]. Suggestion for prophylactic use of antihistaminics or ste-roids for treatment of cement embolism could not be found in the literature search.

To conclude bone cement implantation syndrome is a significant cause of mor-bidity and mortality in orthopaedic surgery. High index of suspicion and close monitoring is required at the time of cement insertion for early clinical diagnosis. Surgical procedural modifications, early recognition and aggressive resuscitation initially, based on physiological principles and later as per requirement with the use of vasopressors and invasive haemodynamic moni-toring are the key to the prevention of a catastrophic outcome.

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