Case Report

Journal of Orthopaedic Education
Volume 2 Number 2, July - December 2016
DOI: http://dx.doi.org/10.21088/joe.2454.7956.2216.5

Cement Implantation Syndrome During Cemented Hemiarthroplasty and Review of Literature

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Abstract

Bone Cement Implantation Syndrome (BCIS) is a highly under-reported rare fatal complication of cementation, characterized by hypoxia, hypotension, cardiac arrhythmias, and cardiac arrest. Such intra-operative deaths are usually not reported. We report such a rare adverse complication of cementation in a 65 years old female who had fracture neck of femer and sustained a fatal cardiac arrest while performing cemented hemiarthroplasty, with aim to alert surgeons about rare, but serious harm due to BCIS and review the available literature, with aim to educate the them regarding risk, clinical features, preventive measures and management.

Keywords: Bone Cement; Cement Implantation Syndrome; Hemiarthroplasty.

Introduction

Bone Cement Implantation Syndrome (BCIS) is a very rare, but fatal complication of cementation [1]. It is most commonly associated with hip arthroplasty, but not restricted to it [2]. It is characterized by number of clinical features including hypoxia, hypotension, cardiac arrhythmias, and increased pulmonary vascular resistant and cardiac arrest [1-3]. We report such a rare case of bone cement implantation syndrome in a 65 years old female who had fracture neck of femur and sustained a fatal cardiac arrest during cementation while performing cemented hemiarthroplasty.

Case Report

A 65 years old female was referred to our tertiary trauma center from a rural center, after she sustained trauma due to road traffic accident around two days ago. She complained of pain localized to left hip, along with difficulty in walking. On examination she had moderate tenderness in scarpa's triangle, along with external rotation deformity and mild shortening of left lower limb. Her medical history and personal history was non-significant. Her radiological examination revealed subcapital fracture neck of femur on left side.

Her haematological and biochemical profile was within normal limtis. Her ECG and chest X rays didnot reveal any abnormality. After medical clearance, pre-anaesthetic evaluation and written informed consent, patient was planned for cemented hemiarthroplasty by bipolar prosthesis. The episodic hypotension following the spinal anaesthesia was well treated and the patient's haemodynamic parameters were well maintained and stable. Following spinal anaesthesia she was taken in right lateral position and hip was approached by posterior approach. After head extraction and gradual rasping of the femur canal, the femoral cannal preparations were done. The canal was thoroughly lavaged with

high volume saline and dried to remove small debris and a cement restrictor was applied. The anaesthetic was informed regarding the starting of cementing process and at this point the patient was infused 8mg of dexamethasone and 100mg of hydrocortisone. The prepared cement after mixing the components was inserted into the femoral cannal in a retrograde manner with help of a cement gun. During this time a suction catheter was put in the femur canal to suck out trapped air, which was pulled back, as the cementing was done. The appropriate sized bipolar prosthesis was pressurized into the cemented canal.

Approximately 2 minutes following the insertion and pressurization of implant in the cemented canal, the patient became unresponsive along with severe fall in BP and bradycardia. The heart rate fell from 100 to 30 bpm and the BP, saturation and peripheral pulses were not recordable. ECG monitors showed arrhythmias with electromechanical dissociation. The surgical site was immediately sealed and the patient turned in supine position and intubated and ventilated with 100% oxygen. Cardiopulmonary resuscitation was started and adrenaline and atropine were given intravenously and repeated at regular interval. A subclavian central venous line inserted. Cardiac compressions continued.

After about 30 minutes of cardiac massage and CPR, crystalloid, vasopressors and supportive treatment, spontaneous cardiac rhythm and blood pressure was attained. Patient was revived and maintained oxygen with IPPV and at this time the heart rate, BP and SpO2 was 120/min, 150/90, 98% respectively. Following this as the patient was haemodynamically stable the surgery was resumed to reduction of hip and rapid closure of the wound. Patient was rapidly shifted to the ICU unit and connected to ventilator. But just after 15 min, the episode repeated again with fall in heart rate to 32/min and BP and SpO2 not recordable. Vasopressors, inotropics and cardiopulmonary resuscitation and three shocks of defribrillator at interval of 5min each with 150J, 200J and 200 J continued to follow the sequence, but the patient didn't responded and after prolonged efforts of resuscitation, patient could not be revived and she died. An autopsy was conducted which showed confirmed diagnosis of BCIS. Histopathological examination of the various organs, exhibited disseminated microembolization of medullary substances and amorphous material.

Discussion

Bone Cement Implantation syndrome (BCIS) is a rare fatal complications of cementation, which is highly underestimated and under-reported, mainly because details of patient who die intraoperatively, is less likely to be reported. Further lesser degrees of BCIS are not reported at all or not systematically collected or published [2,4]. Formal trials and case series are difficult on BCIS, hence very few reports containing free-text description of the incident are only described [1-3,5-7]. Further lack of peer-reviewed literature on the topic, accepted definition and management recommendations adds to the problem. The purpose of this case report is to alert about rare, but serious harm due to BCIS and review the available literature, with aim to educate the surgeons regarding risk, clinical features, preventive measures and management. Such case reports may be the only powerful tool to pick such rare cause of harm.

The aetio-pathophysiology of bone cement implantation syndrome (BCIS) is poorly understood. Several mechanisms have been proposed, like toxic effects of systemic methyl methacrylate, embolic episode, histamine release, complement activation, and endogenous cannabinoid-mediated vasodilatation, but none of them have been proven completely [8-12]. It is likely that a combination of the above processes, with dominance of embolic hypothesis develops BCIS [2]. The extent to which each of these models contributes depends upon the physiological response and risk factors present in patients.

Numerous patient-related risk factors implicated in the genesis of BCIS includes old age, male sex, osteoporosis, use of diuretics, poor preexisting physical reserve, impaired cardiopulmonary function, pre-existing pulmonary hypertension, patent foramen ovale, atrial-septal defect, bony metastases and concomitant hip fractures, particularly pathological or intertrochanteric fractures [2,13,14]. Patients with a previously un-instrumented femoral canal and long-stem hip arthroplasty appear to be associated with a higher incidence of BCIS [6,8]. Our patient was an elderly female with osteoporosis with normal cardiopulmonary functions.

Pathophysiologically, after prosthesis insertion, the exothermic sealed cement expands between the prosthesis and bone, generating high intramedullary pressure (often>300mmHg), which per se is important factor in the genesis of BCIS. It forces "snow flurry", which contain fat, marrow, cement particles, air, bone particles, and aggregates of platelets and fibrin into blood circulation. The embolic snow flurry within the circulation causes mechanical and mediator release changes via histamine, complement activation, endogenous

cannabinoids, vasoactive or pro-inflammatory substances, thrombin and tissue thromboplastin etc, which manifests typical cardiovascular and hemodynamic changes of BCIS [2,5,8,9,15,16]. Degree of cardiovascular compromise is not necessarily proportional to the degree of the embolic load, which supports mediator release model in combination with mechanical effect [2,3,9,16-18]. These embolic snow flurries are visible on transoesphageal echocardiography (TOE) during reaming until the end of the surgical procedure in both cemented and uncemented arthroplasty although lower and fewer in number in uncemented type [3,5,9,15,18]. Post-mortem examinations after intraoperative deaths during cemented arthroplasty also confirm the presence of snow flurries in lungs, brain, kidneys, and myocardium [3,14]. Such findings were seen in autopsy in our case as well.

Due to differences in patient's risk factors, susceptibility and response, BCIS vary widely in spectrum of severity, from milder transient hypoxia, hypotension or confusion to fulminant cardiovascular changes, which may proceed to arrhythmias, shock or cardiac arrest. Depending on SpO_2 and fall in systolic pressure Donaldson proposed a severity classification, grading BCIS into three grade [2]. Our patient belonged to the grade 3, having cardiovascular collapse which required CPR. Grade 1 is moderate hypoxia (SpO_2 <94%) or fall in Systolic blood pressure >20%, whereas Grade 2 is severe hypoxia (SpO_2 <88%) or fall in Systolic blood pressure >40%.

BCIS has no agreed definition. An adverse, sudden, cardiovascular event, characterized by hypoxia, hypotension, unexpected loss of consciousness, cardiac arrhythmias, and cardiac arrest or combination of these, occurring within minutes of cementation, prosthesis insertion, joint reduction or occasionally, tourniquet deflation while cemented bone surgery can be referred as 'bone cement implantation syndrome' (BCIS) [1,2]. Other complications include bronchoconstriction, pulmonary hypertension, pulmonary edema, right ventricular dysfunction, increased central venous pressure, decreased EtCO2, hypothermia and thrombocytopenia [1,2,8,14,19]. It is not only restricted to hip, and has been described during other cemented procedures like knee or shoulder arthroplasty and vertebroplasty [5,20-22]. Due to finite amount of debris present in the femoral canal, BCIS is a reversible time-limited process with ceiling effect, which can recover within minutes, even from large embolic loads, if hemodynamic stability is maintained by supportive therapy [23]. Contrary to this, our patient after successful revival from initial episode of cardiopulmonary instability, sustained another same episode around 30 min after the first one, following which she couldn't be revived.

A fall in end tidal CO_2 concentration with dyspnoea or altered sensorium may be the first indication of clinically significant BCIS [2]. Invasive hemodynamic monitoring like Oesophageal Doppler [24], intraoperative pulmonary artery catheter [6,14] or transoesophageal echocardiography [17,18] can detect impending BCIS at an earlier stage than standard haemodynamic monitoring, but they are not routinely used and where not used by us as well. We used CVP monitoring after the initial episode, although it does not accurately reflected PAP, but it helped us in drug administration and volume optimization [2,9].

Surgical measures undertaken to reduce the risk of BCIS include medullary lavage, good haemostasis before cement insertion, prevent excessive cement pressurization, using low toxicity monomeric cement, minimizing the length of the prosthesis, vacuum cement mixing, retrograde application with cement gun with a suction catheter, intramedullary plug and venting the medulla [2-6,8,14]. Except for vacuum mixing and venting a hole, we followed all the other modern cementing techniques to minimize BCIS.

In the absence of clinical trials, current management recommendations are empiric and only guided by case reports and basic physiological principles. If BCIS is suspected, the management is supportive which includes administration of 100% oxygen with control of airway and supplementary oxygen should be continued into the postoperative period. BCIS be treated as RV failure with aggressive fluid therapy resuscitation, inotropics and vasopressors. Avoiding intravascular volume depletion may reduce the extent of the haemodynamic changes in BCIS [2-6,8,14,25,26]. Prophylactic use of antihistaminics or steroids for treatment of cement embolism could not be found in the literature search [5]. We tried all the supportive measures but couldn't revive the patient.

National Patient Safety Agency (NPSA) issued multidisciplinary clinical guidance in 2009, about the use of bone cement during hip arthroplasty focusing on both anaesthetists and surgeons highlighting the need for joint decision-making, team-working and attention to detail during the peri-operative period, especially in high risk patients. All hip fracture surgery should be undertaken or directly supervised by appropriately experienced anaesthetists and surgeons and ideally on planned trauma lists. The anaesthetic team should be fully

involved in the preoperative assessment, allowing full investigation of co-morbidity and pre-optimization. All members of the theatre team should be aware of the problems and in the event of a severe reaction or cardiopulmonary arrest, everyone should be aware of their defined roles in resuscitating the patient [27].

Conclusion

BCIS is a rare preventable complication of bone cementation characterized by hypotension, hypoxemia, bradycardia, unconsciousness and even cardiac arrest, especially in high risk patients. Constant monitoring and early diagnosis during and after bone cementation is crucial in determining the outcome. Management include surgical modification with modern cementing techniques and supportive measures along BLS and ACLS guidelines. Further registry-based or multicentre studies are needed on the topic for patient safety.

Conflict of Interest

Nil

Acknowledgements

Nil

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