BONE CEMENT IMPLANTATION SYNDROME (BCIS)

Dr. Ekambarakrishnan, MD., DNB. Bone & Joint Foundation, Coimbatore

Bone Cement Implantation Syndrome is an important cause of intra operative 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. patient may have co-existing pathologies which can increase the likelihood of developing BCIS.

DEFINITION:

BCIS is characterized by hypoxia, hypotension or both and/or unexpected loss of consciousness occurring around the time of cementation, prosthesis insertion, reduction of the joint or, occasionally, limb tourniquet deflation in a patient undergoing cemented bone surgery.

SEVERITY CLASSIFICATION:

* Grade 1: moderate hypoxia (SpO2<94%) or hypotension [fall in systolic blood pressure (SBP) >20%].
* Grade 2: severe hypoxia (SpO2<88%) or hypotension (fall in SBP >40%) or unexpected loss of consciousness.
* Grade 3: cardiovascular collapse requiring CPR.

CLINICAL FEATURES:

Many patients undergoing cemented hip arthroplasty develop non-fulminant BCIS characterized by a significant, transient reduction in arterial oxygen saturation, and systemic blood pressure in the peri-cementation period. A smaller proportion of patients develop fulminant BCIS resulting in profound intraoperative cardiovascular changes, which may proceed to arrhythmias, shock or cardiac arrest.

It has been suggested that embolization of femoral canal contents to the cerebral circulation either through a patent foramen ovale or after transit through the pulmonary circulation may cause postoperative delirium. Transcranial Doppler ultrasonography demonstrated cerebral emboli in 40–60% of patients undergoing joint arthroplasty in two small studies but none of these patients developed delirium or a focal neurological deficit.

AETIOLOGY AND PATHOPHYSIOLOGY:

The aetiology and pathophysiology of BCIS are not fully understood.

* **Monomer-mediated model**

It has been demonstrated that circulating MMA monomers cause vasodilatation in vitro. This hypothesis is not supported in vivo in a number of animal studies

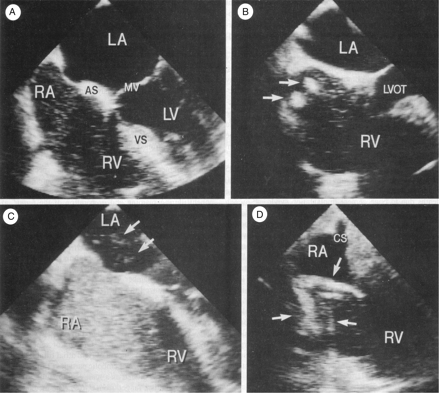
* **Embolic model**

Embolic showers have been detected using echocardiography in the right atrium, RV, and pulmonary artery during surgery. Post-mortem studies have demonstrated pulmonary embolization in animals and man. The physiological consequences of embolization are considered to be the result of both a mechanical effect and mediator release,  which provokes increased pulmonary vascular tone

Embolization occurs as a result of high intramedullary pressures developing during cementation and prosthesis insertion. The cement undergoes an exothermic reaction and expands in the space between the prosthesis and bone, trapping air and medullary contents under pressure so that they are forced into the circulation. The temperature of the cement can increase as high as 96°C 6 min after mixing the components.

Cementation is achieved either with a cement gun or by manually packing the femoral canal. Regardless of the method of cementation, prosthesis insertion into the cemented femur is associated with a considerably greater pressure than cementation alone. When cement is inserted into the femur using a cement gun, the pressures generated are almost double those seen when manual packing is used

Transoesophageal echocardiography:



TOE has demonstrated that the embolic load is greater in cemented compared with uncemented hip arthroplasty. ‘Snow flurries’ were visible on TOE intermittently from the start of reaming until the end of the surgical procedure in both cemented and uncemented arthroplasty. In the cemented group, these were most marked during reaming of both the femur and acetabulum, and during insertion of the femoral component and reduction of the hip joint. In the group undergoing uncemented arthroplasty, the incidence and duration of the flurries did not increase at any specific point during the procedure.

Post-mortem examinations performed after intraoperative deaths during cemented arthroplasty also confirm the presence of marrow, fat, bone emboli, and MMA microparticles in the lungs. Fat emboli have also been demonstrated post-mortem in the brain, kidneys, and myocardium of a patient who had a cardiac arrest during insertion of the femoral prosthesis.

Uncemented arthroplasty is associated with lower intramedullary pressure, fewer emboli, and less severe haemodynamic changes. The degree of embolization may be related to the peak pressure generated in the femoral canal.  high intramedullary pressure per se is an important factor in the genesis of BCIS.

In addition to simple mechanical obstruction of the pulmonary circulation, there are several possible mechanisms by which emboli may result in an increase in PVR. First, mechanical stimulation or damage of endothelium may result in reflex vasoconstriction or release of endothelial mediators. Second, it has been suggested that the embolic material may release vasoactive or pro-inflammatory substances that directly increase PVR, such as thrombin and tissue thromboplastin, or act indirectly by promoting release of further mediators which increase PVR. Histamine release, hypersensitivity, Complement activation and a combination of the above process may responsible for BCIS.

**PATIENT RISK FACTORS:**

Numerous patient-related risk factors have been implicated in the genesis of BCIS including old age, poor pre-existing physical reserve, impaired cardiopulmonary function, pre-existing pulmonary hypertension osteoporosis, bony metastases, and concomitant hip fractures, particularly pathological or intertrochanteric fractures.

MANAGEMENT:

Communication between the surgeon and the anaesthetist is important. In addition to the hazards of cement implantation and prosthesis insertion, reduction of the prosthetic femoral head is also a time of increased risk because previously occluded vessels are re-opened and accumulated debris may be allowed into the circulation. During knee arthroplasty, significant venous emboli are released at the time of tourniquet deflation and this may also be a high risk period.

A fall in end tidal carbon dioxide concentration may be the first indication of clinically significant BCIS in the anaesthetized patient and should alert the anaesthetist. Oesophageal Doppler measurements may detect impending BCIS at an earlier stage than standard haemodynamic monitoring. Early signs of BCIS in the awake patient undergoing regional anaesthesia include dyspnoea and altered sensorium.

If BCIS is suspected, the inspired oxygen concentration should be increased to 100% and supplementary oxygen should be continued into the postoperative period. It has been suggested that cardiovascular collapse in the context of BCIS be treated as RV failure. Aggressive resuscitation with iv fluids has been recommended. Although CVP monitoring does not accurately reflect PAP, a central venous catheter may be indicated for the administration of inotropic drugs. Opinion is divided as to the relative merits of a pure alpha adrenergic agonistor a mixed alpha and beta receptor agonist. The choice of vasopressor is facilitated by the presence  of non-invasive CO monitoring or a pulmonary artery flotation catheter.

Haemodynamic instability should be treated with the potential aetiology in mind. Sympathetic α1 agonists should be first-line agent in the context of right heart dysfunction and vasodilatation. Fluid resuscitation should then be commenced if there is insufficient pre-load.

SUMMARY:

Specific groups of patient who are particularly at risk of developing BCIS in order to minimise this risk the following suggestions are recommended :

liation with the surgeon regarding the type of prosthesis, generous intra-vascular fluid repletion, increased vigilance at the periods of highest risk, and invasive vital-signs monitoring. cemented prosthesis should be avoided in patients who are at high risk of BCIS unless there are overriding orthopaedic considerations.

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