

A Review on Bone Cement Implantation Syndrome

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Incidence

- The true incidence of BCIS - unknown, & mortality data are not systematically collected or published.

Introduction

- BCIS –
 - Important cause of intraoperative mortality & morbidity in pts undergoing cemented joint arthroplasty.
 - Also seen in post op. period in a milder form causing hypoxia & confusion like CNS changes.
 - Bone cement is constituted by mixing a liquid component (containing methyl methacrylate & a activator like N,N-dimethyl-p-toluidine) with a powder component (containing PMMA, a initiator like benzoyl peroxide & antibiotics like gentamycin).
 - BCIS is ch/by hypoxia, hypotension or both and/or unexpected loss of consciousness occurring around time of cementation, prosthesis insertion, reduction of joint or, occasionally, limb tourniquet deflation in pt undergoing cemented bone surgery.
 - Other complications include pulmonary hypertension, pulmonary oedema, bronchoconstriction, cardiac dysarrhythmia, cardiac arrest, hypothermia & thrombocytopenia.
- BCIS severity-
- Grade 1: moderate hypoxia (Spo₂< 94%) or hypotension [20% fall in SBP].
 - Grade 2: severe hypoxia (Spo₂< 88%) or hypotension (40% fall in SBP) .or unexpected loss of consciousness.
 - Grade 3: cardiovascular collapse requiring CPR.

Clinical features

- Mainly develop nonfulminant BCIS ch/by a significant, transient reduction in arterial oxygen saturation ,& systemic blood pressure in peri-cementation period.
- Some develop fulminant BCIS resulting in profound intra-operative cardiovascular changes, which may proceed to arrhythmias, shock or cardiac arrest.
- The cardiovascular changes are more variable.
- MAP, stroke volume & cardiac output may be reduced.
- SVR may be reduced or increased.
- PVR and PAP may be increased.
- Rt-ventricular ejection fraction may be impaired.
- Increased PVR causes a reduced rt-ventricular ejection fraction, compliant rt ventricle (RV) distends & causes inter-ventricular septum to bulge into Lt ventricle (LV), further reducing LV filling, & therefore CO.
- Embolization of femoral canal contents to cerebral circulation either through a patent foramen ovale or after transit through pulmonary circulation may cause post-operative delirium.

Aetiology and pathophysiology

- Initial theories - release of MMA cement monomer into circulation during cementation.
- Recent research - role of emboli formed during cementing & prosthesis insertion.
- Several mechanisms e.g. histamine release, complement activation,& endogenous cannabinoid-mediated vasodilatation.

□ Monomer-mediated model

- Circulating MMA monomers cause vasodilatation in vitro.
- Not supported in vivo in a animal studies that have shown that the plasma MMA concentration after cemented hip arthroplasty is considerably lower than conc.. required to cause pulmonary or cardiovascular effects.

□ Embolic model

- ❖ Mechanism of emboli formation
- Embolization occurs as a result of high intramedullary pressures developing during cementation and prosthesis insertion. The cement undergoes an exothermic reaction & expands in the space b/w prosthesis & bone, trapping air & medullary contents under pressure so that they are forced into circulation.

- When cement is inserted into femur using a cement gun, pressures generated are almost double those seen when manual packing is used.
- Uncemented arthroplasty is associated with lower intramedullary pressure, fewer emboli, & less severe haemodynamic changes.

Haemodynamic effects of embolization

- The debris from medulla can embolize to lungs, Heart or paradoxically to cerebral & coronary circulations.
- Mediator release from emboli-
Simple mechanical obstruction of pulmonary circulation, leads to rt ventricular dysfunction than hypotension & hypoxia occurs.

First, mechanical stimulation or damage of endothelium may result in reflex vasoconstriction or release of endothelial mediators.

Second, it has been suggested that embolic material may release vasoactive or pro-inflammatory substances that directly increase PVR, such as thrombin & tissue thromboplastin.

- Therefore medullary lavage before insertion of cement significantly reduces release of mediators.
 - Mediator-induced vasoconstriction, in combination with mechanical obstruction from emboli, causes shunting of blood that is the most likely cause of hypoxaemia.
- Problems with embolic model :-
- Embolization - not always a/with haemodynamic changes, & degree of embolism correlates poorly with extent of hypotension or hypoxaemia. Studies using TOE show that embolic events are common & most patients tolerate them well.

Multimodal model

It is likely that a combination of the above processes is present in any individual patient who develops BCIS. The extent to which each of these models contributes to the clinical features may depend upon the individual's physiological response. For example, there is significant interpatient variability in the response to morphine-induced histamine release.^{58 71} Several of the phenomena may have opposing actions and the combined effects of BCIS on haemodynamic variables will depend on the relative magnitude of the changes in PVR, SVR, and myocardial contractility. It is reasonable to assume that the patient's pre-existing co-morbidities may alter the clinical features of BCIS. It would be expected that a patient with pre-existing impaired right-ventricular function would be more susceptible to the effects of a sudden increase in PVR .

- Anaphylaxis (Type 1 hypersensitivity)
- A significant increase in plasma histamine conc.. in hypotensive pts undergoing cementation has been demonstrated.
 - In a single study, blockade of histamine receptors with clemastin & cimetidine (H1 and H2 antagonists) appeared to a protective effect, but these findings have not been reproduced in more recent studies.
- Complement activation
- The anaphylatoxins C3a & C5a are potent mediators of vasoconstriction & bronchoconstriction. An increase in C3a and C5a levels, suggesting activation of complement pathway, has been demonstrated in cemented hemiarthroplasty but not in uncemented hemiarthroplasty.
 - Methylprednisolone appeared to prevent the release of anaphylatoxins & development of oxygen desaturation.

Risk factors

1. Old age,
2. Poor preexisting physical reserve,
3. Impaired cardiopulmonary function,
4. Pre-existing pulmonary hypertension
5. Osteoporosis,
6. Bony metastases,
7. Concomitant hip fractures, particularly pathological or inter-trochanteric fracture { These factors associated with increased or abnormal vascular channels through which marrow contents can migrate into circulation. Pts with a patent foramen ovale or ASD may be at increased risk of paradoxical emboli & neurological sequelae. }
8. Patients with un-instrumented femoral canal may be at higher risk of developing the syndrome than those undergoing revision surgery.
9. Two possible mechanisms, First, there is more potentially embolic material present in an un-instrumented femur. Second, once canal has been instrumented & cemented, inner surface of femur becomes smooth & sclerotic ; offers a less permeable surface.

10. Long-stem femoral component increases likelihood of developing BCIS.

☐ **Anaesthetic risk reduction**

- Anaesthetic team should be fully involved in preop. assessment of pts scheduled for joint arthroplasty, allowing for full investigation of co-morbidity & pre-optimization.
- Particular attention - to pts undergoing a cemented procedure with cardiac, respiratory or metastatic disease, pts with a femoral fracture & those having a long-stem prosthesis inserted.
- In high risk cases discussion should occur b/w surgeon & anaesthetist regarding most appropriate anaesthetic & surgical technique, including potential risk-benefit of uncemented compared with cemented arthroplasty.
- Although - no clear evidence regarding impact of anaesthetic technique on severity of BCIS, an animal study has suggested that volatile anaesthetic agents may be a/with a greater HD change for same embolic load.
- The avoidance of nitrous oxide should be considered in high risk pts to avoid exacerbating air embolism.
- Increasing inspired oxygen conc.. should be considered in all pts at time of cementation, especially in high risk pt of BCIS.
- Advanced haemodynamic monitoring should be considered in high risk patients.
- CVP monitoring help to volume optimization & inotrope administration but changes in CVP may correlate especially poorly with changes in PAP in BCIS.
- An oesophageal doppler, TEE or PA catheter has been suggested in high risk patients.

☐ **Surgical risk reduction**

- Medullary lavage,
- Good haemostasis before cement insertion,
- Minimizing length of prosthesis,
- Using non-cemented prosthesis (especially if using long-stem implant)
- Venting medulla.
- ❖ Venting the bone permits air to escape from end of cement plug & reduces risk of air embolus. {Unfortunately, drilling a hole in cortical bone to create a pressure-relieving vent can increase risk of femoral fracture.}
- Low viscosity cementation & retrograde insertion have been suggested to reducing incidence of BCIS in high risk group pts.
- Although cementing by guns - result in more even pressure distribution in medullary cavity, & less reduction in oxygen saturation as described in some studies.
- Paradoxically, it has been demonstrated that intramedullary pressures are higher when cementation is performed with a cement gun rather than finger packing ; so chances of embolisation syndrome are high with cement gun.
- Vacuum mixing of cement also help to reduce BCIS.
- Reduction of prosthetic femoral head is also a time of increased risk becoz previously occluded vessels are re-opened and accumulated debris may be allowed into circulation.
- Significant venous emboli released at time of tourniquet deflation during knee arthroplasty.
- Working the cement to remove volatile vasodilatory compounds has also been recommended.

• **Management**

- High risk consent
 - Communication b/w surgeon & anaesthesiologist prior to surgery.
 - Advance invasive monitoring for CVP & arterial BP.
 - A fall in EtCO₂ conc.. may be first indication of clinically significant BCIS in anaesthetized patient.
 - Early diagnosis - BCIS in awake pt undergoing regional anaesthesia include dyspnoea & altered sensorium.
 - Role of steroid – controversial..??
 - Prevention is better than cure.
 - Avoid hypovolemia, hypoxia, hypercarbia. If BCIS suspected, inspired oxygen conc.. should be increased to 100% & supplementary oxygen should be continued into post-op period.
 - Aggressive resuscitation with i.v. fluids has been recommended. Although CVP monitoring does not accurately reflect PAP, central venous catheter - indicated for inotropic drugs.
 - Sympathetic alpha-1 agonists should be first-line agent in context of right heart dysfunction & vasodilatation.
 - Advance CPR
 - Post-op intensive care and monitoring is essential for high risk pt.
- ☐ **Anaesthetic options for elderly and sick pt for cemented arthroplasty**
- CSE with very low dose spinal

- Graded epidural anaesthesia
- General anaesthesia combined with epidural anaesthesia.
- Single shot lumbar plexus block technique or
- Continuous catheter for lumbar plexus block.
- ❖ Invasive monitoring for BP, CVP, PAP is essential for elderly pt with poor cardiopulmonary reserve.
- ❖ TEE probe should be readily available in operation theatre when dealing of such patients.