Bone cement implantation syndrome

By the perioperativeCPD team

Introduction
Bone cement implantation syndrome (BCIS) is a potentially fatal complication of orthopaedic surgery. It is a cause of intraoperative mortality and morbidity and is most commonly associated with cemented hip arthroplasty. An aging population means that joint replacements are becoming more common. These older patients may have co-existing pathologies which can increase the likelihood of developing bone cement implantation syndrome (BCIS).

The ability to predict, recognise, and manage BCIS is important for everyone working in the perioperative environment.
**History**

Bone cements have been used successfully to anchor artificial joints (hip joints, knee joints, shoulder and elbow joints) for more than half a century.

Bone cement chemically is nothing more than Plexiglas (i.e. polymethyl methacrylate or PMMA) which was invented in the 1920’s and has been used in a huge number of applications, including transparent glass substitutes in windows, semiconductor research, and for the bodies of electric guitars.

The medical orthopaedic use of PMMA is universally credited to Sir John Charnley, who was inspired by his dentist to use dental acrylic for prosthetic fixation in total hip replacement (THR) procedures in the 1950’s. This was possible due to the excellent tissue compatibility of PMMA bone cement when in the body. In 1965, Charnley began to use bone cement that was developed specifically for THRs rather than dental acrylic.

**Why use bone cement?**

Artificial hip joints are commonly anchored with bone cement. This is necessary because the human hip is acted on by approximately 10-12 times the body weight and therefore the bone cement must absorb the forces acting on the hips to ensure that the artificial implant remain in place over the long term. The term “cement,” however, is misleading since bone cement acts more like a grout, filling in space in order to create a tight space to hold the implant against bone.

Millions of procedures of this type are conducted every year all over the world and more than half of them routinely use bone cements - and this proportion is increasing. Bone cement is considered a reliable anchorage material with its ease of use in clinical practice and particularly because of its proven long survival rate with cemented-in prostheses. Hip and knee registers for artificial joint replacements such as those in Sweden and Norway clearly demonstrate the advantages of cemented-in prosthesis. The NICE Guidelines for hip fracture management in adults suggests that cemented implants are preferable to uncemented implants in patients undergoing arthroplasty, as it results in increased postoperative pain-free mobility and reduces the risk of re-operation.
What is bone cement?

Polymethyl methacrylate (PMMA) is a sterile acrylic polymer. PMMA bone cements are usually supplied as two-component systems made up of a fine powder and a liquid. These two components are mixed to start a chemical reaction called polymerisation, which forms the polymethyl methacrylate (PMMA) cement. Polymerisation is a chemical reaction in which two or more small molecules combine to form larger molecules that contain repeating structural units of the original molecules.

PMMA bone cement typically consists of a powder which contains:

- pre-polymerised PMMA co-polymer beads and/or powder,
- radiodensity or radiopacity (x-ray) contrast compound,
- initiator or activator which starts the polymerisation reaction at room temperature when mixed with the accelerator,
- Antibiotics, if used.

and a liquid made of:

- MMA monomer (methyl methacrylate) liquid,
- Stabiliser or inhibitor to prevent polymerisation during storage,
- Accelerator

The two components are mixed and the bone cement viscosity changes over time from a runny liquid into a dough like state (2-3 minutes) that can be safely applied (5-8 minutes) and then finally hardens into solid hardened material. Hardening is influenced by the cement temperature, the operating theatre temperature, and the body temperature of the patient. Vigorous over mixing may also accelerate the setting of the cement. Total setting time is the time is about 8-10 minutes.

During the mixing process, the cement heats up. This polymerisation heat reaches temperatures of around 82-86 °C. This temperature is not all transferred to the body due to the relatively thin cement coating, which should not exceed 5 mm, and the temperature dissipation via the large prosthesis surface and the flow of blood.

Antibiotics (gentamicin, cefuroxime, vancomycin, clindamycin, etc.) can be incorporated within the cement in the form of a soluble powder that is subsequently released into the joint cavity. Antibiotic-loaded bone cement is a well-accepted adjunct for the treatment of an established infection. However, its role in the prevention of infection remains controversial.

Bone cement implantation syndrome (BCIS)

Bone cement implantation syndrome (BCIS) is a poorly understood phenomenon. It is an important cause of intraoperative mortality and morbidity and is most commonly, but not restricted to, being associated with cemented hip arthroplasty.

The clinical features of BCIS most often occur at the time of:

- cementation,
- prosthesis insertion,
- reduction in the joint,
- deflation of a limb tourniquet.
The clinical features of BCIS

Physiologically, BCIS results in reduced arterial oxygenation, characterised by a combination of clinical features:

- Hypoxia
- Sudden loss of arterial pressure
- Pulmonary hypertension
- Arrhythmias
- Loss of consciousness
- Cardiac arrest

BCIS has a wide spectrum of severity. A majority of affected patients develop mild BCIS (see below). Mild BCIS is characterised by a significant, yet transient, reduction in arterial oxygen saturation and systolic blood pressure (SBP) in the peri-cementation period.

A small proportion of patients will develop severe BCIS, with profound intraoperative cardiovascular changes, progressing to; arrhythmias, shock, or cardiac arrest.

The causes of BCIS

There has not been a proven cause of BCIS but several theories exist. The most commonly accepted cause is the embolic model.

Embolic model

During surgical cementation and prosthesis insertion, the cement is intentionally pressurised to force it into the small gaps/spaces of the bone, improving bonding between the cement and bone. The cement then expands in the space between the bone and the prosthesis, further pressurising air and the bone contents and potentially forcing them into the circulation. It has been demonstrated that these embolic contents include fat, marrow, cement, air, bone particles, and aggregates of platelets and fibrin.

This debris may reach the lungs, heart, and/or coronary circulation. It is hypothesised that showers of pulmonary emboli are the cause of the characteristic hypoxia and right ventricular dysfunction of BCIS, leading to hypotension. These physiological changes from embolisation can be attributed to both the mechanical effect of the emboli and a mediator release which provokes increased pulmonary vascular tone.

Other suspected causes include:

- The direct effect of the exothermic reaction of cement temperature
- Air or gas embolism caused by polymerisation of methyl methacrylate monomer
- Hypersensitivity/anaphylactic reaction to the monomer

Proposed definition of bone cement implantation syndrome

The true incidence of BCIS in cemented hemiarthroplasty for hip fractures is, however, not known mainly because this syndrome has, until recently, not had an agreed standard definition. Donaldson and colleagues* have proposed a standard definition of BCIS:

“BCIS is characterised 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.”

Proposed severity classification of bone cement implantation syndrome

As well as the proposed definition of BCIS Donaldson and colleagues proposed a severity classification.

<table>
<thead>
<tr>
<th>Grade 1:</th>
<th>Moderate hypoxia SpO2&lt;94%</th>
<th>Hypotension i.e. a fall in systolic blood pressure &gt;20%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade 2:</td>
<td>Severe hypoxia SpO2&lt;88%</td>
<td>Hypotension i.e. a fall in systolic blood pressure &gt;40% or unexpected loss of consciousness.</td>
</tr>
<tr>
<td>Grade 3:</td>
<td>Cardiovascular collapse requiring CPR</td>
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</tbody>
</table>

How common is BCIS?

Although exact figures are hard to come by, partly due to the lack of a formal definition, one commonly quoted study by Olsen and colleagues in 2014 reports the incidence of BCIS as:

<table>
<thead>
<tr>
<th>BCIS Incidence</th>
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<tbody>
<tr>
<td>BCIS Grade 1</td>
<td>21%</td>
</tr>
<tr>
<td>BCIS Grade 2</td>
<td>5.1%</td>
</tr>
<tr>
<td>BCIS Grade 3</td>
<td>1.7%</td>
</tr>
</tbody>
</table>

Risk factors for the development of BCIS:

Patient factors
- ASA III–IV
- Pre-existing pulmonary hypertension
- Significant cardiac disease
- Osteoporosis
- Male sex
- Increasing age

Surgical factors
- Pathological fracture
- Intertrochanteric fracture
- Long-stem arthroplasty

The effect of anaesthetic technique on the severity of BCIS

There is no clear evidence with regards to the effect of anaesthetic technique on the severity of BCIS. The general principles of management include the maintenance of normovolaemia to avoid the cardiovascular consequences of cementing and the maintenance of high inspired concentrations of oxygen. The use of high anaesthetic vapour concentrations should be avoided as it is associated with greater haemodynamic compromise.

The use of intraoperative cardiac output (CO) monitoring has been recommended in patients with one or more risk factors for BCIS but this is not always available or practical. It can be in the form of semi-invasive transoesophageal Doppler monitor or invasive CO monitor (pulmonary artery flotation catheter).
Role of surgery in prevention of bone cement-associated complications

Surgical measures that can reduce the incidence of complications associated with bone cement, especially BCIS, include thorough lavage of the canal of the femoral shaft in order to remove debris, good haemostasis, the use of non-cemented prostheses, and drilling a venting hole in the distal femoral shaft. The latter technique reduces air trapping during the cementing process and acts as a pressure-relieving opening during the reaming of the femoral canal. However, drilling a venting hole increases the risk of fracture and prosthetic instability.

The vacuum bone cementing technique has been shown to reduce the embolic load during total hip arthroplasty. The retrograde insertion of cement (from distal to proximal) by a cement gun also helps as it causes the compartmentalisation of the bone marrow contents. This can lead to a more uniform increase in pressure and less physiological disturbance.

Furthermore, mixing bone cement in a dedicated cement-mixing set, which is in a vacuum, reduces the load of volatile vasoactive compounds. This, and the use of low-viscosity cement, can reduce the incidence of BCIS.

Consider using non-cemented procedures in high-risk patients.

Cement Curfew

Effective communication between anaesthetists, surgeons, and theatre staff is essential to increase awareness of the potential for BCIS and optimise responses should BCIS occur. It has been suggested that the step of cementation be discussed as part of the World Healthcare Organisation Safer Surgical checklist in the relevant operative procedures.

A ‘Cement Curfew’ protocol has been adopted at some institutions, which involves all members of the operating theatre team assuming specific roles and focus around the time of prosthesis insertion. Music is turned off, important steps during the process are formally verbalised by the lead surgeon including the start and end of the curfew.
The management of BCIS

BCIS is a reversible time-limited phenomenon. The recovery time ranges from a few seconds to approximately 24 hours. The patient’s chance of survival is increased if the situation is immediately recognised and supportive measures are rapidly initiated. This means that immediate aggressive resuscitation and supportive treatment is essential to reduce the morbidity and mortality of this potentially life-threatening situation.

A fall in end tidal carbon dioxide concentration may be the first indication of clinically significant BCIS in the anaesthetised patient and should alert the anaesthetist. Early signs of BCIS in an awake patient undergoing regional anaesthesia include dyspnoea (breathlessness) and altered state of consciousness.

BCIS has a wide spectrum of severity with the majority of affected patients developing grade 1 BCIS. Grade 1 BCIS is characterised by a significant, yet temporary, reduction in arterial oxygen saturation and systolic blood pressure (SBP) in the peri-cementation period. This can be treated with increased increasing the oxygen to 100%, fluids and vasopressors if needed.

A small proportion of patients will develop grade 3 BCIS, with profound intraoperative cardiovascular changes, progressing to arrhythmias, shock, or cardiac arrest.

In cases of severe BCIS (when the patient has arrested, or in a peri-arrest condition), standard advanced cardiopulmonary life support (ACLS) algorithms and procedures should be followed. Fluid resuscitation to maintain right ventricle preload, and inotropes to support ventricular contractility are recommended. Vasopressors (such as phenylephrine and noradrenaline) can be used as they primarily cause peripheral vasoconstriction, increase aortic blood pressure, which in turn supports coronary artery blood flow, and thus improve myocardial perfusion and contractility. Invasive haemodynamic monitoring (if not already in place), should be established.

Use of vasopressors and inotropes should be continued into the postoperative period as necessary, under the management of the intensive care unit (ICU).

Conclusion

Although uncommon, severe BCIS is a risk in all cemented joint replacements, especially hip arthroplasty. It is most likely caused by pulmonary embolism resulting from debris from the bone canal. Rapid hypoxia, hypotension and loss of consciousness are the key early signs. Good intraoperative management of BCIS includes intraoperative vigilance, good team communication, and prompt resuscitation. Non-cemented prosthesis should be considered in high-risk patients.
References


