Royal National Orthopaedic Hospital NHS
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BONE CEMENT IMPLANTATION SYNDROME: A CASE REPORT.

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INTRODUCTION

Bone cement implantation syndrome (BCIS) remains a poorly understood transient phenomenon, and yet is an important cause of intraoperative morbidity and mortality during orthopaedic surgery. It is characterised by hypoxia, hypotension and/or unexpected loss of consciousness occurring around the time of cementation, prosthesis insertion, reduction of the joint or limb tourniquet deflation.[1]

The risk has been particularly highlighted in frail patients undergoing cemented hip arthroplasty following hip fracture. It can however occur with any cemented bone surgery [2].

CASE REPORT

A 77 year old ASA II female presented for a revision of a left hip hemiarthroplasty.

Her past medical history consisted of morbid obesity (BMI 40), hypertension, previous deep venous thrombosis, osteoporosis and lumbar spondylosis. There were no known allergies.

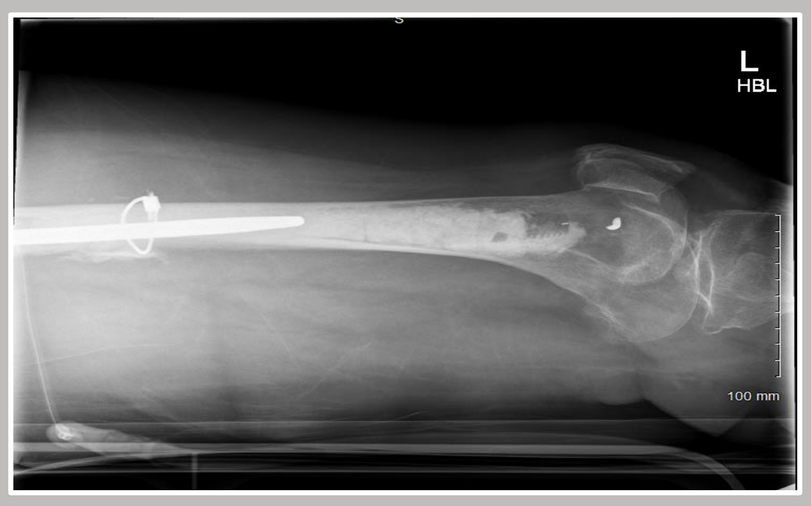
Her medication included: Bisoprolol, Ramipril, Risedronate, Adcal D3, Creon, Buscopan, Cocodamol, Bendroflumethiazide and Moxonidine.

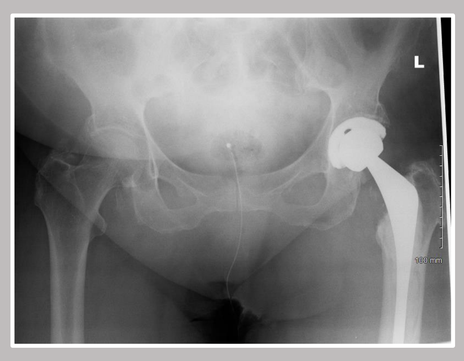
Preoperative investigations were within normal limits and echocardiogram showed mild left ventricular hypertrophy but good overall function.

Surgery proceeded under general anaesthesia and supplemental fascia iliaca block. Routine monitoring using electrocardiogram, non-invasive blood pressure, pulse oximetry and an oesophageal temperature probe was performed. Venous access consisted of 22- and 16-gauge cannulae. Induction of anaesthesia was unremarkable and the patient remained cardiovascularly stable prior to cementing. The blood gas at this stage showed a Hb of 11.2 g/dl.. 3000ml of intravenous crystalloid and 1000 ml of intravenous colloid had been administered to this point, and the estimated blood loss was 1000ml

The surgical procedure was complicated by difficulty removing the cement and the femur was perforated. A long-stemmed prosthesis was required and 120g of cement was used. Five minutes after cementing the non-invasive blood pressure (NIBP) dropped to 45/26 mmHg and was unresponsive to intravenous crystalloid, metaraminol and ephedrine. The patient rapidly developed bradycardia and pronounced ST depression. The end-tidal carbon dioxide fell to 1.8 kPa. The NIBP became un-recordable and the carotid pulse was lost. Cardio-pulmonary resuscitation as per the Advanced Life Support protocol was commenced, with return of spontaneous circulation occurring after two cycles of CPR and 2mg of epinephrine. The patient was transferred to the intensive care unit for supportive treatment for presumed bone cement implantation syndrome.

An echocardiogram performed on day 2 showed no left or right ventricular dysfunction and no evidence of pulmonary hypertension. A CT pulmonary angiogram was normal. The patient developed an acute kidney injury and initially had high inotropic and vasopressor requirements. However after four days the patient was extubated with complete neurological recovery and no lasting organ dysfunction.





Postoperative X-rays of the pelvis and left femur

DISCUSSION

The aetiology and pathophysiology of BCIS are uncertain.

Theories proposed include emboli formation, mediator release, complement activation, histamine release and endogenous cannabinoid-mediated vasodilatation.[1] The embolic model suggests fat, marrow, cement and bone particles, air, aggregates of platelets and fibrin – formed during cementing and prosthesis insertion - produce an increase of pulmonary vascular tone by both mechanical obstruction and mediator release.

A grading system of severity has been proposed:[1]

GRADE 1	moderate hypoxia (SpO2 < 94%) or >20% fall in systolic arterial pressure (SAP)
GRADE 2	severe hypoxia (SpO2 < 88%) or hypotension (fall in SAP > 40%)
GRADE 3	cardiovascular collapse requiring CPR

The incidence of less severe events are under reported. However, according to The National Reporting and Learning System between 2005 and 2012 one in every 2900 cemented hemiarthroplasties for fractured neck of femur suffered death or severe harm during or within a few minutes of cement insertion.[3]

Identification of high risk patients is essential.

RISKS FACTORS FOR DEVELOPING BCIS[4]	
PATIENT FACTORS	ASA III-IV
	Pre-existing pulmonary hypertension
	Significant cardiac disease
	Osteoporosis
SURGICAL FACTORS	Pathological fracture
	Intertrochanteric fracture
	Long-stem arthroplasty

The recognition and optimisation of patients at high risk combined with the selection of appropriate surgical technique and type of prosthesis can minimize complications.[4]

MEASURES TO REDUCE INCIDENCE OF BCIS[2]	
CONDUCT OF SURGERY	Good communication between surgeon and anaesthetist.
	Carefully prepare, wash and dry the femoral canal. Use of a pressurised lavage system.
	Use a distal suction catheter on top of an intramedullary plug.
	Insert cement from a gun retrogradely.
	Do not use excessive manual pressurisation devices in high risk patients.
CONDUCT OF ANAESTHESIA	Ensure that the patient is adequately hydrated.
	Close vigilance for possible cardiovascular events during instrumentation of the femoral canal.
	Maintain the SAP within 20% of pre-induction values.
	Be ready to give vasopressors in response to hypotension.

Once BCIS occurs the inspired oxygen concentration should be increased to 100% and aggressive resuscitation with IV fluids and alpha-agonist drugs started.[1] It is also essential to follow standard resuscitation protocols to reduce the morbidity and mortality.

In conclusion, BCIS is a life threatening but reversible phenomenon.[4] Identification of high risk patients is a priority. Early recognition and prompt management is essential to improve survival and reduce morbidity.

REFERENCES

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