ISSN: 2349-9141

Available online at http://www.ijirr.com

International Journal of Information Research and Review
Vol. 2, Issue, 07, pp. 971-973, July, 2015

OPEN ACCESS JOURNAL

Full Length Research Paper

CARDIAC ARREST DURING CEMENTED FEMUR HEMIARTHROPLASTY- A CASE REPORT

[1,*]Dr. Kalpesh H Shah, [2]Dr. Neha H Mehta, [3]Dr. Tushar Agrawal and [4]Dr. Yash Shah

[1,2]Department of Anaesthesiology, Sanjeevani Hospital and Asatha Hospital, Mumbai
[3]Consultant Paediatric Orthopaedic Surgeon Bombay Hospital and CEO Asatha Hospital, Mumbai
[4]Assistant Professor Ortho Govt Medical College Miraj and CEO Matruseva Hosp Pune

***Corresponding Author**

Received 18th June 2015; Published 31st July 2015

Abstract

86 year old male with multiple comorbidities including a low EF was admitted to our hospital with fracture neck femur and was to undergo a cemented bipolar hemiarthroplasty. After optimization and taking a well-informed high risk consent, the patient was given PNS guided Lumbar plexus and Sciatic block. He remained hemodynamically stable till 5 min of cementing with high viscosity Polymethymethacrylate cement. There was sudden cardiovascular collapse which failed to show sustained return of spontaneous circulation inspite of all measures and lead to the death of the patient after 3 hours

Keywords: Cemented Bipolar Hemiarthroplasty, Co morbidities, Bone cement, Cardiac Arrest

To cite this paper: Dr. Kalpesh H Shah, Dr. Neha H Mehta, Dr. Tushar Agrawal and Dr. Yash Shah 2015 Cardiac arrest during cemented femur hemiarthroplasty- a case report, *International Journal of Information Research and Review.* Vol. 2, Issue, 07, pp. 971-973, July, 2015.

INTRODUCTION

Bone cement is the substance which is used in orthopaedic surgery for adhering implants to bone or to fill in bony defects. Bone cement related reactions have not been uncommon since its first use in THR in 1958 by John Charnley (Vaishya R *et al.,* 2013). Bone cement is not always 100% safe and may be associated with dangers of its own during any procedure. One such complication is Bone cement implantation syndrome which is quite severe in nature and can at times be fatal. However a recent retrospective study shows that regardless of its severity, bone cement implantation syndrome (BCIS) is a fairly common complication with an incidence of 25-30% and in more severe form confers a 16 fold increase in mortality (Olsen F *et al.,* 2014). We present one such case with severe BCIS with a discussion of pathogenesis, risk factors and management.

MATERIALS AND METHODS

Case History

A 86 year old male with sedentary lifestyle came with a 2 day old fracture neck femur. He was a well-controlled diabetic and hypertensive (on Glyburide and Carvedilol) with past history of MI 20 y ago treated medically with sequel of poor cardiac function. His hemoglobin was 7 g/dl with a corresponding low PCV. All other blood parameters were normal. ECG showed no fresh changes. A 2D Echo confirmed inferior wall akinesia with 30% EF, no valvular abnormality and pulmonary pressures normal. All vitals were within normal limits with no evidence of angina, failure or embolism. A right IJV 7 Fr catheter showed CVP of 2 cm. The patient was put on Enoxaparin 0.4mg SC, the last dose being 12 hours prior to surgery.3 units of packed cells were transfused bringing PCV upto 30. 2 more units were reserved for surgery.

A high risk well informed ASA 3 consent was taken explaining risk factors of age, DM, HT, IHD with low EF and the intra op consequences upto cardiac failure. On the day of surgery a 20g catheter was placed in non-dependent radial artery for invasive pressure monitoring.Spo2 was 100% on O2 2l/min, IBP 120/78,5 lead ECG showing HR 82, NSR. In lateral position with operative limb uppermost, PNS guided lumbar plexus and sciatic nerve block were given at appropriate end motor response. The 50 ml solution used comprised of 25 ml 0.75% Ropivacaine, 10ml 2% Lignocaine, 8mg Dexamethasone and 13 ml NS.A 16 G epidural catheter was sited. Maintenance crystalloid was given at 5ml/kg/hr. In 20 min with onset of adequate analgesia surgical preparation was started and incision was taken at 55 min. The patient remained haemodynamically stable. At 90 min after giving the block, high viscosity Gentamycin imbibed PMMA containing cement was introduced into a prepared femoral canal in a retrograde manner. Within 5 minutes while pressurizing the prosthetic femoral stem, the patient started talking irrelevantly and there was sudden hypotension to 90 systolic which failed to respond to 40ug boluses of phenylephrine.

1. Significant risk factors for developing BCIS [Donaldson AJ et al 2009]
Pre-existing disease
Pre-existing pulmonary hypertension
Significant cardiac disease
NYHA class 3 or 4
Surgical factors
Pathological fracture
Inter-trochanteric fracture
Long-stem arthroplasty

2. Anaesthesia risk reduction [Donaldson AJ et al 2009, Parvizi J et al 1999, Pietak S et al 1997 and Herrenbruck T et al 2002]
•High level of haemodynamic monitoring in high risk patients-CVP, cardiac output monitoring oesophageal Doppler.
•Avoid intravascular volume depletion
•Increase FiO2 in all patients at time of cementing
•Avoid N2O in high risk patients

3. Surgical risk reduction [Donaldson AJ et al 2009, Khanna G et al 2012 and Wheelwright et al 1993]
•Uncemented prosthesis- esp. long stem, pathological fracture
•Thorough medullary lavage
•Good haemostasis
•Venting hole in distal shaft
•Bone vacuum cementing technique
•Retrograde cement insertion(Cement Gun)
•Use of bone cement mixing set with vacuum

4. Management [Donaldson AJ et al 2009, Khanna G et al 2012]
Treat hemodynamic instability and cardiovascular collapse promptly and aggressively
Treat as Right heart failure-
Fluids
Alpha 1 agonists e.g. phenylephrine
Inotropes
Pulmonary vasodilators

The patient was given 100% O2 by mask. Nor adrenaline and dopamine infusions were started with minimal response. Next there was a steep drop in the heart rate which responded to two boluses of atropine. The patient became increasingly drowsy and thus was intubated in lateral position and mechanically ventilated with 100% O2.ECG at this point showed tachycardia with suspicious ST elevations. Packing the wound the patient was turned supine, Inj. Enoxaparin 0.4 IV was given and Inj. Nikorandil infusion was started after 2 mg bolus.

Suddenly there was severe bradycardia ending in arrest. CPCR was started as per ACLS and adrenaline 1mg injected and there was ROSC in one cycle. The ECG showed a broad complex tachycardia with ST elevations. A quick closure of the wound was done and the patient was shifted to the ICU with ongoing vasopressors and Nikorandil.

Arterial BP was 80/50.CBC and electrolytes were normal, ABG showed metabolic acidosis for which Sodium bicarbonate was given. Suddenly there was onset of P.VTach which was defibrillated with 120J biphasic charge followed by chest compressions which led to ill sustained ROSC. Defibrillation with 200 J and chest compressions were continued Inj. Amiodarone 300mg was given but to no response. The rhythm degenerated to VF and finally irreversible asystole.

DISCUSSION

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 (Donaldson *et al.,* 2009). The proposed severity classification (Donaldson AJ *et al.,* 2009) Grade 1: moderate hypoxia (SpO2<94%) or hypotension (fall in SBP>20%). Grade 2: severe hypoxia (SpO2< 88%) or hypotension (fall in SBP >40%) or unexpected loss of consciousness Grade 3: cardiovascular collapse requiring CPCR. In a study of 1016 cemented hip hemiarthroplasties the incidence of BCIS grade 1, 2, and 3 were 21%, 5.1%, and 1.7%, respectively. Early mortality in BCIS grade 2 (35%) and grade 3 (88%) were significantly higher when compared with grades 0 and 1 (Olsen *et al.,* 2014). It has been demonstrated that circulating methylmethacrylate monomers from the cement cause vasodilatation in vitro (Karlsson *et al.,* 1995) but it is not supported as a sole cause of BCIS in vivo. So, it has been suggested that the haemodynamic changes observed in BCIS are the result of an increase in intramedullary pressure at cementation leading to embolization (Orsini *et al.,* 1995). The cement undergoes an exothermic reaction and expands in the space between the prosthesis and bone, trapping air and medullary contents like includes fat, marrow, cement particles, air, bone particles and aggregates of platelets and

fibrin under pressure so that they are forced into the circulation (Orsini *et al.,* 1995, Michel R *et al.,* 1980, Parvizi *et al.,* 1999, Pietak *et al.,* 1997 and Hayakawa *et al.,* 2001). The physiological consequences of embolization are considered to be the result of both a mechanical effect (Byrick *et al.,* 1997, Orsini *et al.,* 1995) and mediator release, (Byrick *et al.,* 1997, Wheelwright *et al.,* 1993) which provokes increased pulmonary vascular tone (Lafont *et al.,* 1994) thus causing shunting of blood that is the most likely cause of the hypoxaemia. Some mediators cause a reduction in SVR (Wheelwright *et al.,* 1993). The effect of BCIS on haemodynamic variables will depend on the relative magnitude of the changes in PVR, SVR and myocardial contractility which will be altered by pre-existing co-morbidities. The anaesthetic team should be fully involved in the pre- operative assessment of patients scheduled for joint arthroplasty, allowing for full investigation of co-morbidity and preoperative optimization. In high risk cases discussion should be carried out between the surgeon and anaesthetist regarding the most appropriate anaesthetic and surgical technique, including the potential risk-benefit of uncemented compared with cemented arthroplasty. In patients with a history of previous severe BCIS, avoidance of a further cemented procedure may be prudent (Donaldson AJ *et al.,* 2009). Our patient was diagnosed to be at high risk for developing BCIS and the same was conveyed to the relatives giving option of uncemented procedure but they still chose to go ahead with the cemented procedure due to monetary constraints. The invasive monitors used helped us pick up the hemodynamic instability quickly; however as the patient had a very poor cardiac reserve it became impossible to reverse the cardiac arrest in spite of all efforts.

Conclusion

Patients due for cemented orthopaedic procedures should be carefully screened for all known risk factors and any case at high risk for developing BCIS should be discussed in depth by the surgeon and anaesthetist deciding judiciously the risk benefit ratio for use of cement. The patient and relatives should be made aware of cement related complications while taking informed consent. A high level of suspicion for symptoms of BCIS should be maintained including use of advanced hemodynamic monitoring. All surgical preventive measures should be employed and any hemodynamic instability should be promptly reversed. Cardiovascular collapse should be aggressively managed with principles of ACLS. Constant Vigilance seems to be the crux of prevention and management of BCIS.

LEGEND

BCIS – Bone Cement implantation Syndrome
ACLS – Advanced Cardiac Life Support
N2O – Nitrous Oxide
FiO2 – Fraction of Inspired Oxygen
PVR – Pulmonary vascular resistance
SVR – Systemic vascular resistance
CVP – Central venous Pressure
CPCR – Cardio-pulmonary Cerebral Resuscitation
SBP – Systolic Blood Pressure
ABG – Arterial Blood Gas
ROSC – Return of Spontaneous Circulation
NYHA – New York Heart Association
P.VTach – Pulse less Ventricular Tachycardia
VF – Ventricular Fibrillation
MI – Myocardial Infarction
PMMA- Poly methyl Meth Acrylate

****

REFERENCES

Byrick, R.J. 1997. Cement implantation syndrome: a time limited embolic phenomenon. *Can. J. Anaesth,* 44: 107–11

Donaldson, A.J., Thomson, H.E., Harper, N.J. and Kenny, N.W. 2009. Bone cement implantation syndrome. *Br. J. Anaesth,* 102:12-22.

Hayakawa, M., Fujioka, Y., Morimoto, Y., Okamura, A. and Kemmotsu, O. 2001. Pathological evaluation of venous emboli during total hip arthroplasty. Anaesthesia, 56: 571–5

Herrenbruck, T., Erickson, E.W., Damron, T.A., Heiner, J. 2002. Adverse clinical events during cemented long-stem femoral arthroplasty. *Clin. Orthop. Relat. Res*., 85: 154–63

Karlsson, J., Wendling, W. and Chen, D., et al. 1995. Methylmethacrylate monomer produces direct relaxation of vascular smooth muscle in vitro. Acta Anaesthesiol Scand, 39: 685–9

Khanna, G. and Cernovsky J. 2012. Bone cement and the implications for anaesthesia Continuing Education in Anaesthesia, Critical Care and Pain, 12 (4), pp. 213-216.

Lafont, N.D., Kostucki, W.M., Marchand, P.H., Michaux, M.N., Boogaerts, J.G. 1994. Embolism detected by transoesophageal echo- cardiography during hip arthroplasty. *Can, J. Anaesth,* 41: 850–3

Michel, R. 1980. Air embolism in hip surgery. Anaesthesia, 35: 858–62

Olsen, F., Kotyra, M., Houltz, E., Ricksten, S.E. July 16, 2014. Bone cement implantation syndrome in cemented Hemiarthroplasty for femoral neck fracture: incidence, risk factors, and effect on outcome. Br J Anaesth. Advance Access published on.

Orsini, E.C., Byrick, R.J., Mullen, J.B.M., Kay, J.C., Waddell, J.P. 1987. Cardiopulmonary function and pulmonary micro emboli during arthroplasty using cemented or non-cemented components the role of intramedullary pressure. *J Bone Joint Surg.,* 69: 822–32

Parvizi, J., Holliday, A.D., Ereth, M.H., Lewallen, D.G. 1999. Sudden death during primary hip arthroplasty. *Clin Orthop Relat Res.,* 369: 8–48

Pietak, S., Holmes, J., Matthews, R., Petrasek, A. and Porter, B. 1997. Cardiovascular collapse after femoral prosthesis surgery for acute hip fracture. *Can J. Anesth*, 44: 198–201

Vaishya, R., Chauhan, M., Vaish, A. Bone cement. Journal of Clinical Orthopaedics and Trauma 2013; 4(4):157-163. doi:10.1016/j.jcot.2013.11.005.

Wheelwright, E.F., Byrick, R.J., Wigglesworth, D.F. *et al*. 1993. Hypotension during cemented arthroplasty. Relationship to cardiac output and fat embolism. *J Bone Joint Surg.,* 75: 715–23