Clinical Report

Cardiovascular collapse after femoral prosthesis surgery for acute hip fracture

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Purpose: Prosthetic revision of hip fractures may result in embolization of tissue products leading to death. In this report, from cases reported to the Anaesthesia Advisory Committee to the Chief Coroner of Ontario, emphasis is placed on the immediate resuscitative procedures which may offset a fatal outcome.

Clinical features: Two elderly patients are reported in whom hip fractures necessitated primary prosthetic hip repair. The first patient, with a history of limited cardiac reserve and syncope, suffered a subcapital hip fracture. Under general anaesthesia, a Moore's prosthesis was inserted. The anaesthetic period remained relatively stable until surgical reaming of the femoral canal. Bradycardia, hypotension and cyanosis developed and quickly proceeded to a fatal cardiac arrest. Autopsy demonstrated diffuse pulmonary embolism of fat and thrombus. The second patient suffered a fracture around the stem of a previously inserted femoral prosthesis. Under general anaesthesia, a new cemented hip prosthesis was inserted, following which hypotension occurred. This was supported with small doses of ephedrine, ventilation was controlled with oxygen and the procedure was quickly terminated. Despite addition of a dopamine infusion, cardiac arrest and death followed. Autopsy disclosed massive fat and bone marrow embolization.

Conclusion: The combination of hip fracture, activated clotting factors and borderline cardiopulmonary function presents a risk of death from embolization of tissue products released during the placement of a cemented hip prosthesis. While the outcome of this catastrophe is generally poor, all practitioners should be prepared to immediately institute resuscitative procedures to manage the accompanying cardiovascular collapse.

Objectif : Les révisions de fractures de la hanche peuvent provoquer des embolies tissulaires fatales. Dans ce compte rendu, rédigé à partir de cas soumis au comité aviseur en anesthésie du coroner en chef de l'Ontario, on souligne l'importance de l'instauration immédiate de mesures de réanimation susceptibles de prévenir un dénouement fatal.

Éléments cliniques : Le cas de deux patients âgés chez qui des fractures de la hanche ont nécessité une réduction ouverte avec prothèse est rapporté. Le premier patient qui avait une histoire de réserve cardiaque diminuée et de syncope, avait subi une fracture sous-capitale du fémur. Sous anesthésie générale, une prothèse de Moore a été insérée. L'anesthésie est demeurée relativement stable jusqu'à l'alésage de la fracture. Une bradycardie, de l'hypotension et de la cyanose sont apparues et ont rapidement évolué vers l'arrêt cardiaque. L'autopsie a montré des embolies diffuses de graisse et des thrombi. Le second patient a subi une fracture sur une tige de prothèse préalablement implantée. Sous anesthésie générale, l'insertion d'une nouvelle prothèse cimentée de la hanche a provoqué une chute de pression. De petites doses d'éphédrine ont été administrées, la ventilation a été contrôlée, de l'oxygène administré et l'intervention terminée rapidement. Malgré l'ajout d'une perfusion de dopamine, un arrêt cardiaque et le décès ont suivi. L'autopsie a démontré une embolisation massive de graisse et de moelle osseuse.

Conclusion : L'association de fracture de la hanche, de facteurs de coagulation activés et d'une fonction cardiopulmonaire limite présente un risque de mortalité accru par embolisation de tissu libéré pendant le mise en place d'une prothèse cimentée de la hanche. Bien que le pronostic de cette catastrophe soit généralement mauvais, tous les intervenants devraient être prêts à appliquer les mesures usuelles de réanimation immédiates pour prendre en charge le collapsus cardiovasculaire concomitant.

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THE Anaesthesia Advisory Committee to the Chief Coroner of Ontario reviews anaesthetic related deaths referred by Regional Coroners. Over a two year period, four deaths were examined in elderly patients undergoing prosthetic revision of hip fractures.

In the presence of limited cardiopulmonary reserve, the management of catastrophic haemodynamic events following femoral canal reaming and cemented prosthesis insertion is frequently unsuccessful. Consequently, the literature emphasizes preventative measures.[1,2] The Committee, consisting of the above authors, is concerned that some anaesthetists may not recognize the event and patients at risk and do not have a resuscitation protocol to deal immediately with the problem, should it occur.

The purpose of this report is to emphasize that patients with pre-existing cardiopulmonary dysfunction having prosthetic revision of hip fractures, or other procedures involving pressurization of the femoral canal, are at high risk of cardiopulmonary instability. Two such cases, taken from the files of the Chief Coroner, are reported. As these cases represent summaries taken from patient charts, the details are limited.

Case #1

A 79 yr old woman with a previous history of coronary insufficiency, myocardial infarction, recurrent syncope and osteoporosis, fell and suffered a subcapital fracture of her right hip. Preoperative medications included isosorbide dinitrate, nitroglycerin, disopyramide phosphate and indomethacin, doses unknown.

Cardiopulmonary examination was unremarkable. Blood pressure was stable at 150/70 mmHg, pulse 78 bpm. Electrocardiogram showed non-diagnostic ST changes in anterior leads.

She was felt to have stable angina with her fall possibly related to syncope, which was not explored further. Clinical assessment revealed adequate hydration and an ASA rating of III was assigned, based on coronary insufficiency.

Premedication of 5 mg morphine *im*, one hour preoperatively, preceded general anaesthesia. Monitoring included non-invasive automatic blood pressure, lead II ECG, capnograph, pulse oximeter, inspired oxygen concentration, anaesthetic gas analyzer and temperature. Anaesthesia was induced with 1 mg midazolam, 0.625 mg droperidol, 75 μg fentanyl and 50 mg propofol. Tracheal intubation was facilitated with 80 mg succinylcholine. Anaesthesia was maintained with nitrous oxide, oxygen and isoflurane, dose unknown. Muscle relaxation was induced with 3 mg vecuronium. Ventilation was controlled using a semi-closed circle system.

During the procedure, blood pressure decreased from 140/70 to 90/60 mm Hg but stabilized after decreasing isoflurane concentration. Morphine was used to control blood pressure spikes. Heart rate remained stable at 70–80 bpm, SaO_2 97 % and $P_{ET}CO_2$ was 30–38 mmHg. Blood loss was minimal.

With reaming of the femoral canal before insertion of a Moore's prosthesis, sudden bradycardia, hypotension and cyanosis developed. Within minutes the patient developed cardiac arrest and failed to respond to CPR.

Autopsy demonstrated multiple fat emboli in both lungs, severe focal atherosclerosis of the dominant right coronary artery and peripheral organizing thromboembolus of the lung, without infarction. In addition, adenocarcinoma of the tail of the pancreas with metastatic disease in liver, lungs, spleen and femur was found.

Case #2

A 95 yr old woman fell and sustained a fracture around the stem of a previously inserted hip prosthesis. Her medical history included previous cerebrovascular accident and dementia but she was felt to be stable for revision of her Moore's prosthesis. Hydration was adequate and was maintained throughout the procedure. Preoperative haemoglobin concentration was 91 $g\text{-}dl^{-1}$ and blood replacement was started with commencement of the anaesthetic.

Anaesthetic monitors included non-invasive blood pressure, ECG, oesophageal stethoscope, pulse oximeter and capnograph (by mass spectrometry which failed part way through the procedure). Anaesthesia was induced with 1 mg alfentanil, 3 mg d-tubocurarine, 125 mg thiopentone, 80 mg succinylcholine and the trachea was intubated. Nitrous oxide, oxygen and enflurane 0.25% were used for maintenance. Muscle relaxation was obtained with vecuronium. Small doses of ephedrine were used intermittently to maintain blood pressure.

Approximately two minutes after insertion of a new cemented prosthesis, SaO_2 decreased from 98% to 72%, systolic blood pressure decreased from 120 to 80 mmHg and airway pressure increased from 24 to 36 cm H_20. The lungs were ventilated with 100% 0_2, and she was given ephedrine to support blood pressure, which remained low. The procedure was terminated with haste and the patient was transferred to the recovery room, where mechanical ventilation with 100% O_2 was continued. Cardiovascular support was provided with epinephrine and dopamine but bradycardia and cardiac arrest followed, with the decision to not pursue further resuscitation. Autopsy documented massive fat and bone marrow embolism.

Discussion

The two cases reported provide examples of the rapidity of haemodynamic deterioration and patient death that may follow femoral canal reaming and cemented prosthesis insertion. While the cases reported provide little detail in the sequence of the patient deterioration or the subsequent resuscitation, the intent is to demonstrate the immediate need for therapeutic modalities directed towards management of massive tissue product embolism with pulmonary hypertension and right heart failure. Regardless of therapy given, many of these patients will not survive and it is not the intent to presuppose success given different management of these patients.

Hypotension in cases following manipulation and pressurization of the femoral canal, with release of tissue products, is highly variable, reported between 5% and 50%. It is influenced by several variables, including surgical and anaesthetic techniques and type of prosthesis.[3,4] In some cases mortality may approach 10% following hypotensive episodes.[5]

Diffuse bone marrow fat embolism, while present in many fatal cases, does not necessarily produce the haemodynamic collapse described above. Poor correlation exists between the degree of stainable fat and the severity of cardiopulmonary dysfunction.[6,7] Release of vasoactive tissue thromboplastic products and prostanoids, perhaps generated by the fracture, has received more attention as causative factors.[2,6,7,8] Likewise, methyl methacrylate cement does not appear to be a major precipitating factor other than to seal the femoral shaft, with resulting intracavitary pressures of 600–900 mmHg during prosthesis insertion. It is this high shaft pressure that facilitates embolism of marrow and tissue products.

Prophylaxis against embolism classically relates to surgical technique, including preinsertion working of cement to remove volatile vasodilator compounds, profuse lavage of the femoral shaft to remove tissue products, and use of a femoral plug with venting to limit intracavitary pressure rises during cement and prosthesis insertion.[2,3] Where compromised cardiovascular status exists, the use of a non-cemented prosthesis may help prevent cardiovascular instability. Experimentally, bilateral hip replacement with cemented prostheses has been shown to be associated with greater cardiopulmonary derangement and is best avoided in the presence of cardiopulmonary dysfunction.[11]

Anaesthetic prophylaxis includes maintenance of normovolaemia, increased inspired oxygen concentration and decreased volatile agent concentration (where general anaesthesia is used), prior to prosthesis insertion.[2,3] Following serious embolization, these measures may prove to be inadequate since marked increases in pulmonary artery pressure and resistance can induce right ventricular failure that may persist for several hours.[11–13] Subsequent right ventricular dilatation may result in septal shift, increased intrapericardial tension, decreased left ventricular filling and cardiac output (CO).[13] Decreased CO with resultant decrease in mixed venous oxygen tension is responsible for the majority of the initial decrease in SaO_2.[11] High right ventricular end-diastolic pressure plus low systemic blood pressure results in ischaemia to the right ventricle (RV) that potentiates the vicious circle of RV depression, failure and death. It is towards this RV ischaemia and failure that initial therapy must be directed.

Fluid administration to augment RV preload may prove to be beneficial or harmful depending on the degree of septal shift already present.[13,14] After modest resuscitation volumes are administered, it is preferable to institute CVP monitoring and cease fluid loading when large increases in CVP begin.

To combat RV ischaemia and improve RV function, direct acting vasopressors such as phenylephrine or norepinephrine should be titrated until an adequate aortic perfusion pressure is restored.[15,16] Inotropes such as dobutamine can then be given to improve ventricular function, providing that the increased O_2 consumption induced by these agents is met by an adequate RV perfusion pressure.[14,17] For this reason, isoproterenol, which causes vasodilation, can result in further hypotension and deterioration, but in the face of adequate perfusion pressure can be beneficial.[15,17]

Conclusion

Recognition of patients at risk with hip fractures requiring prosthetic revision, is an important feature of the preanaesthetic assessment. Where severe cardiac or pulmonary derangement exist, invasive monitoring should be considered for the procedure. Patients with poor cardiopulmonary reserve should not be considered for bilateral prosthetic replacements but should undergo less hazardous unilateral surgical techniques such as a non-cemented prosthesis with extensive high pressure pulsatile lavage of the medullary cavity.

Where clinical evidence of embolization exists, namely hypotension, decreased SaO_2, decreased $P_{ET}CO_2$, tachycardia or bradycardia, therapy must be immediately instituted to restore RV function. This initially includes restoration of an adequate systemic perfusion pressure, augmentation of preload as RV filling pressures tolerate and then addition of inotropic agents to augment contractility.

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