Case reports

Pseudo Tamponade Soon After Cardiac Surgery: A Report of Three Cases

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ABSTRACT

Objective: *To describe a syndrome suggestive of tamponade but secondary to increased intra-thoracic pressure due to undetected dyssynchronous expiratory efforts in mechanically ventilated patients soon after cardiac surgery.*

Methods: *A retrospective chart review of three patients immediately after cardiac surgery.*

Results: *All patients developed the tetrad of decreased blood pressure, increased central venous pressure, decreased cardiac index and decreased urine output soon after cardiac surgery without other obvious reasons. In all patients, tamponade was suspected. In all cases, physical examination found no marked evidence of patient-ventilator dyssynchrony but evidence of abdominal muscle tensing and intermittent expiratory efforts not in synchrony with the ventilator cycle. In all patients, sedation and muscle relaxation led to a decrease in central venous pressure, improved blood pressure and improved cardiac index and urine output with full resolution of the syndrome. All patients were successfully extubated within 24 hours of surgery.*

Conclusions: *In some postoperative cardiac surgery patients expiratory efforts which are not in synchrony with mechanical ventilation and not immediately obvious to clinicians or intensive care unit nurses can simulate tamponade. Greater awareness of this syndrome may decrease the chance of diagnostic error.* (**Critical Care and Resuscitation 2004; 6: 193-196**)

Key words: Cardiac tamponade, mechanical ventilation dyssynchrony

Cardiac tamponade immediately after cardiac surgery is a potentially life-threatening complication. While it may sometimes be clinically obvious, it may also be difficult to diagnose, even with the assistance of transoesophageal echocardiography.[1-3] Such diagnostic difficulty arises from the fact that pericardial effusions are present in up to 64% of postoperative patients, even when there is no clinical suspicion of tamponade.[4] Furthermore, it is relatively common for tamponade to be due to the presence of a localised pericardial clot,[5] which may be difficult to determine.[6,7]

Because of these diagnostic difficulties and because of concerns about the adverse consequences of delayed diagnosis, patients often return to the operating theatre for exploration on the basis of clinical suspicion and clinical findings alone. It is therefore important for intensive care clinicians to make the correct diagnosis in detecting cardiac tamponade and not cause the patient to have an unnecessary second operation. Several conditions can mimic some, or most, of the clinical features of tamponade in the immediate post-operative period, including tension pnemothorax, relative hypovolaemia, right ventricular failure and global myocardial dysfunction.

In this case report, we describe three postoperative cardiac surgical patients who developed clinical findings consistent with tamponade secondary to undetected

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patient-ventilator dyssynchrony.

CASE REPORTS

Patient 1

A 66 year old man was admitted to the intensive care unit (ICU) following elective coronary artery bypass grafts surgery (CABGS), using bilateral internal thoracic artery grafts and a single radial artery graft. His risk factors included hypertension and smoking. He also had mild chronic obstructive pulmonary disease.

The operation was performed under a relaxant general anaesthetic (total intravenous anaesthesia with propofol target-controlled infusion and alfentanil infusion). Cardiopulmonary bypass time was 163 minutes. He did not require inotropic drugs after bypass surgery, although he required external atrial pacing. At the conclusion of the operation, he was admitted to the ICU, intubated and ventilated. Drainage from his chest drains was minimal.

Approximately 4.5 hours later, he began to wake up but remained disoriented. There was no obvious use of his accessory muscles of respiration and no clear evidence that he was "fighting the ventilator". His central venous pressure began to rise (7 to 14 mmHg) and both his mean arterial pressure (80 to 62 mmHg) and urine output (175 to 15 ml/hr) fell. His cardiac index was low (2.2 L/min/m^2) and pericardial tamponade was considered.

On general physical examination, his abdominal muscles were found to be tense during expiration, although the effort did not appear to be isolated to the expiratory cycle of the ventilator. The patient was then sedated with propofol and paralysed with vecuronium. He rapidly improved with his central venous pressure decreasing, his blood pressure remained stable and his cardiac index (2.7 L/min/m^2) improving. A propofol infusion was commenced and continued for 4 hours. The patient woke up after the propofol infusion was ceased and was extubated successfully. His haemodymamic problem did not recur.

Patient 2

A 60 year old man was admitted to the ICU following elective CABGS, using sequential left internal thoracic artery grafts and bilateral radial artery grafts. His medical history included an acute myocardial infarction in 1994 and a coronary angioplasty in 1995. His cardiac risk factors included hypertension, dyslipidaemia and smoking. He also had mild chronic renal impairment.

The operation was performed under a relaxant general anaesthetic with both volatile (sevoflurane) and intravenous anaesthetics (propofol target-controlled infusion and remifentanil infusions). Cardiopulmonary bypass time was 91 minutes. An intraoperative transoesophageal echocardiogram revealed a dilated left ventricle with segmental systolic dysfunction. After coronary artery bypass, there was improvement in systolic function but there was persistent posterolateral dyskinesis. A milrinone infusion was commenced to maintain cardiac contractility and for radial artery graft vasodilatation. At the conclusion of the operation, he was admitted to the ICU, intubated and ventilated. Drainage from his chest was < 100 mL/hr.

Approximately, 1.5 hours after surgery, the patient opened his eyes but was not able to obey commands. His central venous pressure rose markedly (12 to 20 mmHg) and his mean arterial pressure decreased from 80 to 60 mmHg. His cardiac output also decreased. Pericardial tamponade was considered. However, on further examination, his abdominal muscles were found to be contracting in what appeared to be expiratory efforts during both the expiratory and inspiratory ventilator cycle. He was sedated immediately with propofol and morphine. However, clinical improvement was slow and his abdominal muscles remained tense. He was then paralysed with rocuronium. Immediately, his central venous pressure decreased to 10 mmHg and his mean arterial pressure increased to 75 mmHg. The milrinone infusion was not increase and his cardiac output was preserved. He was extubated successfully 6 hours later with no further haemodynamic abnormality.

Patient 3

A 57 year old man was admitted to the ICU following elective coronary artery bypass grafts surgery, using the internal thoracic artery graft and two radial artery grafts for triple vessel disease. His echocardiogram had shown normal systolic function. He had a past history of hypertension and was a non-smoker.

The operation was performed under a relaxant general anaesthetic (total intravenous anaesthesia with propofol target-controlled infusion and fentanil). Cardiopulmonary bypass time was 150 minutes. At the conclusion of the operation, he was admitted to the ICU, intubated and ventilated. Drainage from his chest drains was approximately 30 mL/hr. He was receiving milrinone for radial artery vasodilatation and required noradrenaline at 5 μg/min to maintain a blood pressure of 85 mmHg.

Approximately 2 hours later. His central venous pressure began to increase (10 to 15 mmHg) and his noradrenaline dose was increased from 5 to 9 μg/min to maintain a blood pressure > 80 mmHg. His urine output fell from 220 to 40 mL/hr. His cardiac index decreased from 3.2 to 2 L/min/m^2 and a pericardial tamponade was considered.

However, on general physical examination, his abdominal muscles demonstrated active contraction and felt tense in what appeared to be expiratory efforts. These efforts were noted to be dyssynchronous with the ventilator cycle. He was sedated with propofol and paralysed with vecuronium. Following this he rapidly improved with his central venous pressure decreasing by 4 mmHg and his cardiac index increasing to 2.9 L/min/m^2. A propofol infusion was commenced and continued for 9 hours. The patient woke up and was co-operative following discontinuation of the propofol infusion. He was successfully extubated and his haemodynamic problem did not recur.

DISCUSSION

In this case series, we describe three patients with a syndrome characterised by a rising right sided venous pressure, hypotension, decreased cardiac output and oliguria in patients soon after cardiac surgery. In these patients, the clinical picture suggested the possibility of cardiac tamponade. No obvious clinical findings were present to suggest ventilatory dyscoordination. On further physical examination, evidence of active expiratory effort with contraction of the abdominal wall muscles, which was dyssynchronous with the ventilator cycle was revealed. Sedation and neuromuscular relaxation resulted in resolution of the syndrome suggesting the diagnosis of undetected patient-ventilator dyssynchrony. This syndrome of "pseudo-tamponade" did not recur in any of the patients.

Patient-ventilator dyssynchrony or asynchrony is a well described phenomenon.[8] Several possible triggers have been described and are summarised in Table 1. However, using electronic reference libraries and search terms such as "tamponade", "intrathoracic pressure", "valsalva", "cardiac surgery", "cardiac output", "cardiac index", "patient-ventilator interaction" "patient-ventilator asynchrony" and "patient-ventilator dyssynchrony" we were unable to find any articles reporting a syndrome similar to that described in our article. Furthermore, the typical description of patients who are diagnosed with patient-ventilator dyssynchrony emphasise the presence of anxiety or agitation,[9] tachypnea, diaphoresis, nasal flaring, use of accessory muscles of respiration and tachycardia. These features were not observed in our patients.

Previous publications also note the importance of an asynchronous or paradoxical motion of the rib cage and abdomen.[10,11] However, consideration of mechanical ventilatory dyssynchrony is often triggered by other signs of respiratory distress and may not occur if clinicians focus on haemodynamic changes and cardiovasular events alone. In our patients, abdominal muscle contraction and paradoxical expiratory effort, which did not cycle with the ventilator, was only partly evident on inspection but was clearly evident on palpation. Such findings, however, may be overlooked by inexperienced clinicians.

Table 1. Possible triggers for patient-ventilator dyssynchrony

Patient-related factors
Bronchospasm
Pneumothorax
Pulmonary edema
Lung collapse
Pulmonary emboli
Dynamic hyperinflation
Drug-induced
Ileus with abdominal distention
Psychomotor agitation
Gas trapping in the stomach
Secretions
Airway factors
Blocked or kinked endotracheal tube
Migration of the endotracheal tube:
into the right main bronchus
above the vocal cords
Cuff herniation or rupture
Ventilator factors
Undetected leak
Autocycling
Inadequate F_IO_2
Inadequate flow
Circuit malfunction
Inadequate ventilator support

We used sedation and neuromuscular blockade to assist with the diagnosis of patient-ventilator dyssynchrony and found that the abnormalities of central venous pressure, cardiac output and blood pressure resolved with their use. We note that, once sedation has been applied with neuromuscular blockade, intravascular volume expansion may become necessary if the central venous pressure is low (<10 mmHg) in patients with known pre-operative myocardial dysfunction.

In summary, we report three patients who developed clinical findings consistent with tamponade and without obvious clinical signs of patient-ventilator dyssynchrony soon after cardiac surgery. A careful examination of the abdominal wall muscles may suggest the latter diagnosis. This diagnosis was confirmed by the effect of neuromuscular blockade and subsequent improvement in the clinical course.

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