Clinical Report

Monitoring complication due to a pulsatile femoral vein from tricuspid regurgitation

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This clinical report illustrates a monitoring complication due to a pulsatile femoral vein from tricuspid regurgitation. In this case, the femoral vein of a patient was inadvertently cannulated and exhibited a pulsatile tracing which was mistaken for an arterial pressure tracing. This led to the initial inappropriate use of vasopressors which actually augmented the pulsatility and delayed detection of the error. Although this may be a rare occurrence, the clinician should be aware of its possibility when using invasive monitoring in a patient with tricuspid regurgitation.

Cette observation illustre une complication de monitoring causée par la pulsativité de la veine fémorale secondaire à une régurgitation mitrale. La veine fémorale est canulée par mégarde et son tracé pulsatile est faussement interprété comme le tracé de l'artère fémorale. Cette erreur mène à l'utilisation inopportune de vasopresseurs qui augmentent la pulsativité et retardent la découverte de l'erreur. Bien qu'il s'agisse là d'un événement rare, le clinicien devrait être au courant de cette possibilité lorsqu'il installe un monitoring invasif chez un patient porteur de régurgitation tricuspédienne.

We present a case of a patient in which the femoral vein was inadvertently cannulated and exhibited a pulsatile tracing which was mistaken for an arterial pressure tracing. This led to the initial inappropriate use of vasopres-

sors which actually augmented the pulsatility and delayed detection of this error. There have been previous reports of pulsatile veins in patients with tricuspid regurgitation¹⁻⁷ in other clinical settings.

Case report

A 72-yr-old man with known ischaemic heart disease and chronic congestive heart failure was admitted to hospital following a transient ischaemic attack. He was volume-overloaded on admission and thus aggressively diuresed. He then became oliguric and this was believed to be due to overdiuresis. He was subsequently fluid-resuscitated even though he became tachypnoeic. Blood pressure was 80/50 mmHg by cuff and blood gas analysis showed: pH 7.0, PO₂ 125 mmHg, PCO₂ 16 mmHg and HCO₃ 4.4 mmol · L⁻¹ breathing 30% O₂ by mask. His course deteriorated and he was transferred to the ICU where a femoral arterial line was inserted, arterial blood gases were drawn and his trachea was intubated. Initially, the blood pressure could not be measured manually or by automatic cuff and no peripheral pulses were palpable. Blood from the femoral cannula was cyanotic and pulsatile and the measured pressure was 60/40 mmHg (Figure 1). Blood gas analysis showed pH 6.9, PO₂ 44 mmHg, PCO₂ 42 mmHg and HCO₃ 8.5 mmol · L⁻¹. A pulmonary artery catheter was inserted through a left subclavian venous sheath. The PAOP was 24 mmHg and cardiac index 2.6 L · min⁻¹. The lungs were ventilated with 100% O₂ and the patient was given dopamine (2.5 µg · kg⁻¹ · min⁻¹) and norepinephrine (up to 40 µg · min⁻¹). Despite pressor support, the pressure increased only slightly to 70/40 mmHg although the pulse rose from 85 · min⁻¹ to 120 · min⁻¹. The serum lactate concentration was 13.9 mmol · L⁻¹ and the mixed venous blood gas showed pH 7.0, PO₂ 51 mmHg, PCO₂ 34 mmHg, and HCO₃ 8.5 mmol · L⁻¹. It was inferred at this point that the blood from the femoral cannula sent for gas analysis was a venous sample. A right femoral pulse was palpated and cannulated yielding pulsatile

Key words

COMPLICATIONS: monitoring;
EQUIPMENT: arterial catheter;
HEART: tricuspid valve.

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FIGURE 1 The upper tracing is the femoral line pressure and the lower tracing the central venous pressure (CVP). The femoral line tracing is narrower and appears to have a dicrotic notch. The peak pressures were similar.

FIGURE 2 The upper tracing is the true femoral arterial pressure waveform and the lower tracing the CVP. The arterial tracing is much narrower than the venous tracing and the femoral line in Figure 1.

bright red blood and pressure of 130/60 mmHg (Figure 2). Blood gas analysis from this sample showed: pH 7.1, PO_2 242 mmHg, PCO_2 25.4 mmHg, and HCO_3^- 8.5 mmol $\cdot$ L $^{-1}$. The norepinephrine infusion and the FiO_2 were appropriately reduced. The haemodynamic state stabilized and the lactic acidosis resolved. An echocardiogram showed moderate tricuspid regurgitation. The trachea was extubated 72 hr later and the patient was discharged to a medical ward five days after admission. The presumed cause of the patient's deterioration was myocardial ischaemia with no evidence of infarction.

Discussion

In this patient, marked venous pulsations secondary to tricuspid regurgitation led to the erroneous placement of a femoral arterial cannula. Resuscitation, as guided by erroneous venous readings, led to the use of excessive doses of norepinephrine and oxygen and overall confusion of the clinical picture. Venous placement of this can-

nula was not initially suspected because severe hypotension and hypoxia were consistent with the clinical picture. Also, the blood pressure was not measurable with a non-invasive cuff which also prevented early detection of the problem.

Administration of norepinephrine produced an apparent pressor response which could be explained by two mechanisms: (i) an increase in pulmonary vascular resistance which increased tricuspid regurgitation and transmitted higher pressures to the femoral vein, and (ii) a decrease in systemic venous compliance which increased venous pulsatility.⁸ As well, arterial pulsations from the adjacent femoral artery may have been transmitted to the cannula in the femoral vein. The venous pressure waveform seen in Figure 1 is wider and exhibits a delayed upstroke compared with that of the true arterial waveform (Figure 2). In retrospect, this provided a clue which should have been used to suspect the problem. The measurement of mixed venous O_2 saturation finally led to detection of the problem since the pulmonary O_2 saturation was similar to the PO_2 from the "arterial" femoral line.

The aetiology of tricuspid regurgitation in this patient was not clear. There was no history of congenital or rheumatic heart disease; however, ischaemic cardiomyopathy with associated congestive failure and pulmonary hypertension likely played a role. Although not noted during resuscitation in this acute setting, physical findings that should alert the clinician to the possibility of tricuspid regurgitation include: a prominent right ventricular impulse, a holosystolic murmur at the left lower sternal border, distended neck veins, an enlarged and/or pulsatile liver, ascites and peripheral oedema.⁹

Pulsations in the systemic veins secondary to tricuspid regurgitation have been previously reported.¹⁻⁷ Although this may be a rare occurrence, the clinician should be aware of this possibility when using invasive monitoring in a patient with tricuspid regurgitation.

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