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Sudden death after central venous cannulation

A previously fit 39-yr-old man was admitted with clinical findings suggestive of septicaemic shock. After routine insertion of a central venous catheter he developed electromechanical dissociation. Prolonged efforts to resuscitate the patient were unsuccessful. Post mortem examination showed a cyst arising from the tricuspid valve annulus which had prolapsed into the right ventricular outflow tract. The insertion of a central venous catheter probably caused the cyst to prolapse and cause cardiac arrest with electromechanical dissociation. This unique complication emphasises the importance of not allowing the Seldinger wire or central venous catheter to enter the heart during insertion. Correct positioning of central venous catheters during insertion is most safely confirmed by back flow of blood in the catheter and right atrial electrocardiography together with a follow up chest radiograph, rather than relying on the occurrence of cardiac arrhythmias during placement of the Seldinger wire or catheter. To avoid excessive lengths of Seldinger wire or catheter being inserted inadvertently during placement, both should be marked along their lengths.

Un homme de 39 ans jusque là bien portant est admis avec des signes de choc septique. L'insertion d'un cathéter de tension veineuse centrale provoque une dissociation électromécanique. Des efforts prolongés de réanimation s'avèrent inutiles. L'autopsie révèle dans la chambre de chasse du ventricule droit le prolapsus d'un kyste de l'anneau de la valve tricuspide. Il semble que l'insertion du cathéter veineux central ait provoqué ce prolapsus et la dissociation électromagnétique consécutive. Cette rare complication met en relief l'importance de ne pas laisser pénétrer dans le cœur un guide métallique de Seldinger ou un cathéter central. La bonne position du cathéter de tension veineuse centrale ou du guide de Seldinger est confirmée en

toute sécurité par un retour sanguin veineux et l'électrocardiographie auriculaire droite de même que par radiologie, alors qu'il est dangereux de provoquer des arrhythmies. Pour éviter une pénétration exagérée, le guide métallique et le cathéter veineux devraient porter des graduations.

Many complications have been described following the insertion of central venous catheters^{1,2} but, in general, relate to local difficulty at the site of insertion, e.g., haematoma formation, or distant complications, e.g., erosion through the right atrium or superior vena cava.

Serious cardiac complications are rare. A common cardiac complication during insertion of the catheter is the occurrence of short-lived unifocal ventricular arrhythmias which are often seen in patients who are hypoxic and acidotic.³ The diagnosis of more serious complications such as cardiac perforation or intramural thrombus formation is often delayed after insertion for 24 to 48 hr, and they are frequently only recognized at *post mortem*.⁴ It has been recommended that to minimize late complications the catheter tip be located in the lower superior vena cava.⁵

This paper reports a cardiac complication associated with insertion of a central venous catheter which occurred at the time of insertion and has not been previously described.

Case report

A previously fit and healthy 39-yr-old man presented with a two-day history of severe nausea and vomiting. Before admission he had complained of increasing breathlessness.

On admission he appeared dehydrated with poor peripheral perfusion and an axillary temperature of 34°C. Blood pressure was 80/40 mmHg. There was a pansystolic murmur, loudest at the apex and radiating to the left sternal edge, which had not been noted previously by the patient's family doctor. He had Kussmaul's type respiration. There was a marked pectus excavatum. An erythematous macular rash extended from the patient's upper abdomen to both groins. Abdominal examination was otherwise unremarkable.

Key words

COMPLICATIONS: cardiac arrest;
MONITORING: venous pressure, central.

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Initial investigations revealed Hb 15.2 g · dl⁻¹, WBC 26.4 · 10⁹ · L⁻¹, Na⁺ 152 mmol · L⁻¹, K⁺ 4.6 mmol · L⁻¹, and urea 24.2 mmol · L⁻¹. Arterial blood gas analysis whilst breathing 6 L · min⁻¹ oxygen via a Hudson mask showed a metabolic acidosis with a pH 7.09, PaCO₂ 2.02 kPa (15.5 mmHg), PaO₂ 27.5 kPa (206 mmHg), BC 4.7 mmol · L⁻¹. Chest radiograph was normal and a 12-lead ECG showed a normal axis, sinus rhythm of 76 min⁻¹, right bundle branch block and mild left ventricular hypertrophy.

A diagnosis of severe dehydration and metabolic acidosis possibly due to septicæmic shock was made and resuscitation was started with intravenous fluids. A decision was made to insert a central venous catheter to enable fluid replacement to be guided more accurately.

A 14 FG (200 mm) single lumen catheter (HydrocathTM, Viggo-Spectramed, Swindon, UK) was prepared to be inserted into the right subclavian vein. The 450 mm Seldinger wire was positioned in the right subclavian vein without difficulty but partially withdrawn because of the occurrence of numerous unifocal ventricular extra systoles. The ventricular extra systoles stopped immediately after repositioning the Seldinger wire. The catheter was then rapidly inserted over the Seldinger wire, which was withdrawn. Immediately after catheter insertion the patient became unconscious, stopped breathing and had no palpable central pulse.

Cardiopulmonary resuscitation was started immediately. The three-lead ECG continued to show normal shaped complexes after the cardiac arrest despite no central pulse being palpable, which supported a diagnosis of cardiac arrest due to electromechanical dissociation. Cardiopulmonary resuscitation was started. The patient's trachea was intubated and his lungs ventilated with 100% oxygen. One litre of Ringer's lactate solution, together with four doses of adrenaline hydrochloride (total dose 8 mg), 3 mg atropine sulphate, and 1 mg anhydrous calcium chloride (9 mmol) were given *iv* without improvement. The patient became cyanosed despite ventilation of the lungs with 100% oxygen. It was noted that the peripheral veins in the upper body and the internal jugular veins were distended throughout the resuscitation. A tension pneumothorax was excluded by bilateral pleurocentesis but cardiac tamponade could not be excluded by subxiphoid pericardiocentesis due to the severe pectus excavatum. Resuscitation was continued for 30 min but was eventually abandoned.

At *post mortem* the striking finding was of a normal heart but with a thin-walled fibrin cyst measuring 1.5 cm × 1.0 cm × 0.8 cm arising from the tricuspid valve annulus on the atrial aspect. The cyst was filled with fluid and could easily be moved down into the right ven-

tricle where it was able to prolapse into and completely obstruct the flow through the pulmonary artery outflow tract. There was no evidence of pneumothorax, cardiac tamponade or pulmonary emboli.

Discussion

The close temporal association between insertion of the central venous catheter and cardiac arrest strongly suggests a relationship between the two. Prolapse of the tricuspid valve cyst into the pulmonary artery outflow tract was well demonstrated at *post mortem* and could have been caused by insertion of either the Seldinger wire or the catheter. Unifocal ventricular extra systoles were seen at the time of insertion of the Seldinger wire which stopped after the wire had been withdrawn partially. The wire probably caused prolapse of the valve cyst, preventing ejection of blood from the right ventricle and causing cardiac arrest. The occurrence of electromechanical dissociation together with the clinical findings of persistent profound central cyanosis, despite adequate ventilation, and engorged peripheral veins suggests such a mechanism is likely. The chances of successful resuscitation following the cyst prolapse were low.

Unifocal ventricular arrhythmias are often seen during central venous catheterisation, are easily diagnosed at the bedside and, usually, are rapidly stopped by withdrawing the catheter or Seldinger wire.

The occurrence of cardiac arrhythmias caused by insertion of the Seldinger wire or catheter should not be used as a means of confirming correct placement. This is more safely achieved using right atrial electrocardiography⁶ and back flow of blood in the catheter during catheter placement, together with a chest radiograph after positioning.

To avoid excessive lengths of catheter from being introduced inadvertently most central venous catheters now have graduated markings along their length. Few Seldinger wires supplied with the catheters are similarly marked. Marking of Seldinger wires along their length might prevent accidental excessive insertion of the wire during central venous catheterisation and reduce the risk of intracardiac damage.

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