Case Report

Unsuspected cardiac tamponade following insertion of a haemodialysis catheter: a normal chest radiograph does not exclude a complication

Timothy B. L. Ho, Rebecca Cusack, Andrew Rhodes and R. M. Grounds

Intensive Care Unit, St. James’s Wing, St. George’s Hospital, Blackshaw Road, London, UK

Keywords: chest radiograph; haemodialysis catheter; pericardial tamponade; central venous cannulation; complications

Introduction

Central venous cannulation is a commonly performed medical procedure, widely used in general internal medicine and nephrology. It is used for both therapeutic and diagnostic indications. As with all invasive procedures, it is associated with a number of recognized complications [1], and strategies have been developed to minimize them. Cardiac tamponade is one such complication [2]. It is thought to arise from the guidewire, dilator or venous cannula piercing the myocardial wall. Suggestions made to minimize the risk of perforation include the use of soft j-tipped wires [3], ensuring that cannulae are not advanced too far or against resistance [2] and the use of post-procedure chest radiographs to confirm correct positioning of the catheter [4].

We report a case of an undetected pericardial tamponade associated with the insertion of a haemodialysis catheter, despite a normal post-insertion chest radiograph.

Case

A cachectic 42-year-old woman was admitted to the intensive care unit following the reversal of an ileal–jejunal bypass procedure which had been performed 11 years previously for morbid obesity. The initial operation had led to the desired weight loss but also eventually to hepatic failure manifested by malabsorption, peripheral oedema, a low serum albumin, hyperbilirubinaemia, elevated hepatic transaminases and a coagulopathy.

Before the reversal procedure, a pulmonary artery catheter sheath had been inserted in her left internal jugular vein and a triple lumen catheter in the right. Four days post-operatively, she developed a fever, hypotension, a worsening systemic acidosis and oliguria. Pulmonary artery catheter readings revealed a pulmonary artery wedge pressure of 23 mmHg, pulmonary artery pressure of 47/38 mmHg, cardiac index of 4.87 l/min/m², a systemic vascular resistance of 402 dyne.s/cm⁵ and a right atrial pressure of 24 mmHg. A diagnosis of septic shock was made. Inotropic support, in addition to intravenous broad spectrum antibiotics, was commenced. In view of progressive oliguria and the need for optimal fluid management, haemodialysis was planned. Accordingly, exchange over guidewire of the triple lumen catheter for a double lumen haemodialysis catheter was performed. Additionally, a new triple lumen line was inserted medial to the dialysis catheter. Soft j-tipped wires were used for both procedures, as were venous dilators, particularly as a larger bore haemodialysis catheter was to be accommodated. No resistance was encountered on insertion of guidewires, dilators or catheters. An immediate post-procedure chest radiograph indicated a satisfactory position of the catheters without obvious evidence of complications (Figure 1).

Post-procedure pulmonary artery readings (Table 1) initially remained relatively constant, although maintenance of the cardiac index eventually led to escalating adrenaline requirements. Despite increasing vasopressor support, the patient continued to deteriorate. She died 18 h after the change of central lines. A post-mortem examination revealed a large tense pericardial haematoma associated with a discrete 1 mm perforation of the right atrium. This was thought to be due to penetration of the myocardial wall by a venous catheter, dilator or guidewire.

Discussion

This case illustrates that an apparently normal post-insertion chest radiograph does not exclude a complication from a central line.

The true aetiology of this patient’s death was not suspected. Severe sepsis was thought to be the major contributor to this patient’s deterioration. Invasive
Fig. 1. An AP supine chest radiograph taken immediately after the insertion of the triple lumen and haemodialysis catheters in the right internal jugular vein. Note the monitoring cables lying over the left chest.

Table 1. Pulmonary artery catheter readings following the final central line insertion

<table>
<thead>
<tr>
<th></th>
<th>Pre-insertion</th>
<th>+1 h</th>
<th>+9 h</th>
<th>+17 h</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pulmonary artery pressure (mmHg)</td>
<td>47/38</td>
<td>54/42</td>
<td>52/40</td>
<td>44/34</td>
</tr>
<tr>
<td>Right atrial pressure (mmHg)</td>
<td>24</td>
<td>29</td>
<td>28</td>
<td>23</td>
</tr>
<tr>
<td>Pulmonary artery wedge pressure (mmHg)</td>
<td>23</td>
<td>31</td>
<td>29</td>
<td>25</td>
</tr>
<tr>
<td>Cardiac index (1/min/m²)</td>
<td>4.87</td>
<td>–</td>
<td>3.26</td>
<td>2.57</td>
</tr>
<tr>
<td>Systemic vascular resistance (dyne.s/cm²)</td>
<td>402</td>
<td>–</td>
<td>490</td>
<td>701</td>
</tr>
</tbody>
</table>

monitoring and measurement of cardiac physiology should have helped distinguish cardiac tamponade from septic shock. Although both conditions lead to profound hypotension and tachycardia, cardiac tamponade is usually associated with an elevated systemic vascular resistance (SVR) [5] and rising right atrial pressure, as opposed to the typical low SVR associated with sepsis-mediated vasodilatation. Both conditions are associated with rising fluid requirements and inotrope dependence, reflecting the general treatment for severe hypotension. This patient demonstrated a persistently low SVR throughout her deterioration, masking a typical feature of cardiac tamponade. We believe that the severity of her septic condition ameliorated the rise in SVR. Consequently, her persisting hypotension was assumed to be progression of her septic state.

Another typical feature of cardiac tamponade is the development of an enlarged and globular cardiac silhouette on chest radiography [6]. This patient’s post-procedure chest radiograph did not reflect these changes. Chest radiographs following procedures have usually been carried out to exclude complications such as pneumothoraces and catheter malpositions. Immediate post-procedure chest radiography, as performed in this case, does not necessarily reveal an abnormality if there is a small cardiac perforation secondary to malpositioning of the guidewire or dilator. The latter are only transiently in the patient (dyne.s/cm²) and are removed prior to post-procedure chest radiography. Accordingly, any abnormality in their positioning is not assessed. The typical globular appearance of the heart associated with a pericardial effusion can take time to develop, and hence may not be apparent on an early chest radiograph. Similarly, late presenting pneumothoraces have been reported following apparently normal chest radiographs [7].

The false reassurance from the radiograph and the pulmonary artery catheter readings inadvertently diverted attention from the true cause of this patient’s deterioration. It has been advocated that chest radiography is unnecessary following routine guidewire exchange of catheter in haemodynamically stable patients [8,9]. However, we suggest that chest radiography is insufficient if a complication is suspected or if the patient is unstable. Undue reliance on chest radiography may prejudice against the true diagnosis.
Additionally, we reiterate the warning not to insert either a guidewire or dilator too far. Therefore, in a hypotensive patient who recently has undergone central venous cannulation or catheter exchange, despite having a normal post-procedure chest radiograph, an iatrogenic complication such as a cardiac tamponade should still be considered. Repeat chest radiography and an urgent echocardiogram should be arranged.

References


Received for publication: 24.6.99
Accepted: 17.1.00