Soft J-tipped Guide Wire-induced Cardiac Perforation in a Patient with Right Ventricular Lipomatosis and Wall Thinning

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Abstract

Cardiac tamponade caused by perforation is a rare but potentially lethal complication of central venous catheter (CVC) insertion. We herein report a case of cardiac perforation associated with the use of a soft J-tipped guide wire. Twenty minutes after the insertion of a CVC, the patient developed unexpected cardiac arrest. An autopsy revealed 400 mL of pericardial blood. The right ventricular wall was 1 mm thick with about 10 myocyte layers, which is one-third that of the normal heart. A histological analysis revealed widespread fatty infiltration of the right ventricular wall (right ventricular lipomatosis).

Key words: cardiac tamponade, cardiac perforation, central venous catheter, right ventricle, fatty infiltration, soft J-tipped guide wire

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Introduction

Cardiac tamponade caused by perforation is a rare but potentially lethal complication of central venous catheter (CVC) insertion. Cardiac tamponade associated with CVCs is caused by a puncture of the cardiac wall, superior vena cava (SVC) or inferior vena cava (IVC) by guide wires, dilators, or catheters (1). It was predicted that the incidence of lethal complications of cardiac perforation could be reduced by using soft J-tipped guide wires (2). Only two previous cases of cardiac tamponade due to soft J-tipped guide wires have been reported (3, 4). These two cases differed from ours in two important respects: 1) in both of them, the CVCs were inserted via the right jugular vein, whereas in our case, the point of entry was the right subclavian vein, and 2) in neither of these cases was a pathological reason for the perforation identified (3, 4). We herein report a case of fatal cardiac perforation by a soft J-tipped guide wire after insertion via the right subclavian vein, and describe the abnormal histological features of the myocardium in this patient that were concluded to be responsible for the perforation. We detected not only widespread fatty infiltration but also thinning of the right ventricular wall to one-third to one-fifth of the normal thickness. These histological features suggest that there was myocardial fragility. Therefore, in order to prevent the unpredictable lethal complication of cardiac perforation by CVCs, it is important to establish a medical standard for performing every CVC insertion under videofluoroscopy.

Case Report

Our patient was a 73-year-old man with a history of polycythemia vera. He was treated with intermittent methyl-6-[[2-chloroethyl) nitrosoamino] carbonyl]-amino]-6-deoxyalpha-D-glucopyranoside (MCNU) from 1991 to 1998, and with hydroxycarbamide from 1999 to 2010. In 2011, he developed a fever, severe anorexia, and progressive pancytopenia. We found no palpable lymphadenopathy, but noted the presence of hepatosplenomegaly. The first bone marrow aspirate showed marked hemophagocytosis but no obvious
lymphoma cell infiltration. When the patient first came to our hospital, his alcohol consumption was 30 g/day, but his past alcohol consumption was unknown.

We treated the patient with steroids (prednisolone, 1 mg/kg/day) with no resolution of his febrile syndrome. On the fourth day of admission, we commenced the cyclophosphamide; hydroxydaunorubicin, vincristine, and prednisolone (CHOP) regimen to control the hemophagocytosis, administering the drugs via a CVC [smooth manipulation advanced catheter (SMAC) plus Safe Guide Microneedle Seldinger kit, Covidien Group, Tokyo, Japan] inserted through the right subclavian vein. A histological examination of the heart revealed widespread myocardial fatty infiltration in the bone marrow and liver. No lymphoma cells were detected in the myocardium.

The clinical findings and course strongly suggested the patient to have lymphoma associated with hemophagocytic syndrome. Therefore, we decided to insert a second CVC using the Seldinger technique to administer chemotherapy and nutrition. The patient’s laboratory values included a white blood cell (WBC) count of 8.0×10^9 cells/L (neutrophils, 92.7%; lymphocytes, 2.1%; eosinophils, 0%; basophils, 0.3%; and monocytes, 4.9%); red blood cell count, 287×10^6 cells/L; Hb, 8.5 g/dL; Hct, 23.3%; Platelet count, 40×10^9 cells/L; prothrombin time, 15.1 sec; international normalized ratio, 1.28; activated partial thromboplastin time, 33.5 sec; blood fibrinogen, 230 mg/dL; D-dimer, 24.89 μg/mL (reference value: 0.1-0.9 μg/mL); serum LDH, 1,732 IU (reference value 119-229 IU); serum C reactive protein, 5.15 mg/dL; and serum ferritin, 13,167 ng/mL. We identified no bleeding tendency. The patient received a transfusion of 10 units of platelet concentrate before the insertion of the CVC.

The CVC insertion procedure was successfully performed without incident, except that the patient complained of palpitations while the soft J-tipped guide wire was being inserted. Slight retraction of the guide wire resulted in an immediate improvement in the palpitations. We encountered no resistance to the insertion of the guide wire and inserted a double lumen catheter to 13 cm. Immediately after the insertion procedure, the patient was stable, doing well, and fully conscious. A chest radiograph showed that the catheter tip was in the right atrium. There was no evidence of pneumothorax. Twenty minutes later, the patient suddenly went into cardiac arrest, and we suspected that he had developed a lethal arrhythmia. A second chest radiograph indicated no obvious evidence of progressive cardiomegaly. In spite of attempts at cardiopulmonary resuscitation, the patient died before we were able to make a definitive diagnosis.

A postmortem examination revealed 400 mL of blood and hematoma in the right pericardial sac. A histological analysis revealed widespread myocardial fatty infiltration in the right ventricle (RV) without fibrosis (Fig. 1). The fatty myocardial replacement extended from the epicardium (Fig. 1). Only about 10 myocyte layers were in the stratum compactum layer of the right ventricular wall, which is one-third to one-fifth that found in the normal heart. The perforation near the RV apex was through a gap between the trabeculae carneae where the stratum compactum was very thin (Fig. 1B, 2). An immunohistochemical analysis of autopsy specimens revealed peripheral T-cell lymphoma in the bone marrow and liver. No lymphoma cells were detected in the myocardium.

Discussion

This is the first reported case of cardiac perforation caused by a soft J-tipped guide wire via a right subclavian vein approach. A histological examination of the heart revealed widespread fatty infiltration in the RV wall and only 10 myocyte layers, which is about one-third to one-fifth that found in the normal heart. Unfortunately, the perforation site was through a gap between the trabeculae carneae where the wall was much thinner. It is likely that both the thinning and the fat replacement of the RV wall made the myocardium fragile, thereby facilitating perforation and precluding hemostasis.

Previous studies have described fatty infiltration of the right ventricle under non-pathological conditions (5-7). Tansley et al. showed 50% of the lateral RV wall to be replaced by adipose tissue in 10% of men over 40 years of age, and
Cardiac tamponade has been widely recognized as a rare complication of CVC insertion (1, 10). The rate of tamponade is reportedly 0.2% (10). In most cases, this complication is thought to be caused by the catheter. There have been five reported cases of similar complications caused by Seldinger guide wires (2-4, 11, 12) and two by soft J-tipped guide wires (3, 4). In the first reported case of tamponade caused by a soft J-tipped guide wire, the wire was inserted via the right jugular vein under echographic guidance, and it perforated the right atrial wall. The patient died before a definite diagnosis could be made (3). In the second case, a soft J-tipped guide wire was also inserted via the right jugular vein under echographic guidance, and it perforated the inferior vena cava (4). Both of these patients had end-stage renal failure requiring hemodialysis (3, 4). Renal failure has been recognized as a cause of hemorrhagic diathesis (13). Therefore, a small perforation by a guide wire would more likely lead to hemorrhagic complications in such patients. However, no histological features of the patients’ myocardium that could have contributed to a bleeding tendency were discussed in these reports (3, 4).

No systemic hemorrhagic bleeding disorder was identified during the pre- or postmortem examination in our case. However, we cannot exclude the possibility of local or latent hemostatic dysfunction. Malpositions and complications following CVC largely depend on the site of venous approach (14, 15). There are few reports on prospective comparisons of internal jugular versus subclavian catheter insertion (14, 15). In one of these reports (14), there were more arterial punctures, but fewer catheter malpositions with the jugular compared with subclavian approach. Another report showed a statistically lower risk of mechanical complications using the jugular approach (15). However, these data were not from randomized studies, and no valid randomized studies have been performed.

Although the soft J-tipped guide wire was introduced to reduce the risk of serious complications, no data are available to document any reduction in the incidence of perforations. In at least three cases, including the present one, lethal cardiac tamponade was caused by soft J-tipped guide wires. In these cases, the guide wires seem to have been inserted too far and migrated into the heart, inducing lethal complications. However, the precise mechanisms responsible for these injuries remain unclear.

Several policies for avoiding serious lethal complications of CVCs have been suggested (1-4, 10-12). The most effective and reliable strategy seems to be CVC insertion under videofluoroscopic guidance. Although the present situation of daily medical practice makes this impossible in many hospitals, it is necessary to establish a medical standard for performing every CVC insertion under videofluoroscopy in the near future.
The authors state that they have no Conflict of Interest (COI).

References


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