CASE REPORT

Catheter-related right atrial thrombus and pulmonary embolism: A case report and systematic review of the literature

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Central venous catheters (CVCs) are commonly used in clinical practice. One of the foremost complications associated with their use is the potential for symptomatic or asymptomatic thrombosis. CVC thrombosis, in turn, may not only result in vascular catheter occlusion but also infection, pulmonary embolism, and formation of right heart thromboemboli. Thrombi within cardiac chambers are associated with an increased risk of mortality due to their potential for embolization to the pulmonary vasculature. We describe the case of a 77-year-old man, who was successfully thrombolysed following detection of a right atrial thrombus and hemodynamically significant pulmonary embolism resulting from thrombus formation on the tip of a peripherally inserted central catheter (PICC). The present article is the first report of a PICC-related right atrial thrombus in an adult treated with thrombolysis. A systematic review of the literature suggests that the true incidence of this complication may be underestimated because the diagnosis may not be considered in asymptomatic and symptomatic patients, or may be missed by transthoracic echocardiography. The present case highlights the importance of maintaining a high index of suspicion for thromboembolic complications and heparin-induced thrombocytopenia in patients with CVCs or a PICC. It also underscores the important role of transesophageal echocardiography and thrombolysis in the diagnosis and management, respectively, of right heart thromboemboli with associated pulmonary embolism.

Key Words: Catheter; Echocardiography; Embolism; Heart; Thrombolysis

CASE PRESENTATION

A 77-year-old man with a medical history of chronic myelogenous leukemia, stable angina and chronic obstructive pulmonary disease was admitted for elective repair of a 5.8 cm juxtarenal abdominal aortic aneurysm, bilateral common iliac artery aneurysms, and a right femoral thrombectomy. On postoperative day 10, the patient suffered a respiratory arrest that required intubation. Despite aggressive fluid resuscitation, he remained hypotensive, requiring hemodynamic support with noradrenaline and milrinone. Central venous vascular access was changed from a right-sided internal jugular central venous catheter (CVC) to a left-sided subclavian introducer, through which a pulmonary artery catheter was inserted. Initial hemodynamic parameters on milrinone demonstrated a central venous pressure of 10 mmHg, a pulmonary artery pressure of 42/26 mmHg, pulmonary capillary wedge pressure of 12 mmHg, a cardiac index of 4.2 L/min/m² and a systemic vascular resistance of 941 dynes/cm². A complete blood count revealed a white blood cell count of 17.9×10^9/L and platelets at 284×10^9/L. After obtaining cultures, antibiotics for presumed sepsis were initiated. At this time, a complete blood count revealed a hemoglobin of 66 g/L, a white blood cell count of 17.9×10^9/L and platelets at 284×10^9/L.

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had risen to 766×10^9/L and hydroxyurea was initiated. By postoperative day 24, the patient was reintubated and vasopressor support was reinitiated. His platelet count was now 73×10^9/L. Oxygen and vancomycin was initiated. A left-sided internal jugular catheter and new arterial line were inserted. The patient’s clinical status improved, and he was extubated on postoperative day 14, at which time his platelet count was 87×10^9/L. Oral coumadin was initiated the following day. A transthoracic echocardiogram (TTE) on postoperative day 15 showed a moderately to severely dilated right ventricle (RV) with moderate RV dysfunction and mild tricuspid insufficiency. On postoperative day 17, he was transferred to the ward and a new right subclavian CVC was inserted. The patient was noted to have increased swelling of his right arm two days later, and his international normalized ratio was 2.8. A 4 Fr double-lumen peripherally inserted central catheter (PICC) was inserted in the left cephalic vein on postoperative day 21 with its tip in the superior vena cava.

On postoperative day 22, the patient’s oxygen requirements increased. The following day, his tachypnea worsened and he was noted to have a left lower lobe infiltrate on repeat chest radiography. At that time, his platelet count was 187×10^9/L, with an international normalized ratio of 2.6 on coumadin and a prothrombin time of 48 s. A repeat chest CT scan confirmed a new PE in the right upper lobe. Unfractionated heparin was resumed and an inferior vena cava filter was inserted. On postoperative day 24, the patient was reintubated and vasopressor support was reinitiated. His platelet count was now 73×10^9/L.

A transesophageal echocardiogram (TEE) revealed a 2 cm serpiginous thrombus adherent to the tip of the PICC in the right atrium (Figure 2). The thrombus was rotating circumferentially within the right atrium (RA). A total of 100 mg of recombinant tissue plasminogen activator was administered to the patient over 2 h through the PICC line initially (30 min), and subsequently through a peripheral intravenous line. A repeat TEE performed the following day showed resolution of the RA thrombus (RAT). The patient’s platelet count was 85×10^9/L and a diagnosis of heparin-induced thrombocytopenia (HIT) was made by ELISA; fondoparinux was initiated. Oxygen and vasopressor support were discontinued over 48 h by postoperative day 27 and platelets had normalized the following day. The PICC line was retained and coumadin was reinitiated on postoperative day 41. By postoperative day 43, his platelet count had risen to 766×10^9/L and hydroxyurea was initiated.

DISCUSSION

Thrombosis is a relatively common complication associated with CVC use. Inconsistent incident rates of CVC-related thrombosis are reported in the literature and vary according to host factors, catheter characteristics, cannulation site and the infusates administered. Estimates of CVC-related thrombosis vary depending on the site of insertion, with the incidence of PICC-related thrombosis, in general, ranging from 2% to 4%. PE occurs in approximately 15% of individuals with CVC-related upper extremity DVT.

Right heart thromboemboli (RHTE) are embolized DVTs lodged in the right heart chambers, predominantly the RA. RHTE also include thrombi that develop within the cardiac chambers at anastomotic foci or on injured endothelium, implanted devices or foreign bodies including tumours, pacemakers and CVCs. Classifications of RHTE are based on morphology. Type A thrombi are highly mobile and may prolapse through the tricuspid valve. Conversely, type B thrombi are attached to the right atrial or ventricular wall and may originate in association with foreign bodies or in structurally abnormal chambers. The incidence of right heart mural thrombi (innominate-superior vena cava, superior vena cava-RA and RA) was noted to be 29% in 141 patients with CVC undergoing postmortem examination (1). In a prospective study of 55 patients with CVCs who were followed for up to eight weeks, Gilon et al (2) noted the incidence of RHTE to be 12.5% and significantly associated with a catheter tip in the right atrium, malignancy, concurrent infection, procoagulant states and structural abnormalities. RHTEs are associated with PE in approximately 4% to 6% of cases (3,4) and increase the three-month mortality rate from 16% to 29% (3).

While management strategies for hemodynamically significant PE have been well delineated, strategies for the management of RHTE, with and without PE, have not been well described. For patients with suspected PE, anticoagulation, in the absence of contraindications, should be initiated. Doppler ultrasonography of the lower and upper extremities, especially in the presence of a CVC, should be obtained. A confirmatory spiral CT or perfusion scan is desirable but may not be feasible in unstable patients. In hemodynamically unstable patients and patients with a CVC, an echocardiogram should be...
obtained to assess for RV dilatation and dysfunction, and RHTETEE is preferable to TTE because of its improved ability to detect RHTET and characterize clot morphology.

We considered three options in the management of our patient with RHTET, PE and hemodynamic compromise: surgery, interventional radiology-guided clot retrieval and thrombolysis. A surgical consult deemed that the patient was not a candidate for thoracotomy and cardiopulmonary bypass given his age, recent major surgery and hemodynamic instability. We subsequently approached interventional radiology to inquire about the possibility of clot and/or catheter removal; however, local expertise was not available. Consequently, we considered both systemic and catheter-directed thrombolysis for RHTET with PE as treatment strategies in our patient with RV dysfunction and hemodynamic instability. While several thrombolytic agents can be used to treat massive and submassive PE, we selected recombinant tissue plasminogen activator, which has a greater affinity for plasminogen in the presence of fibrin and a shorter infusion time than streptokinase or urokinase. We considered the risks associated with thrombolysis including bleeding, hematoma formation at puncture sites, intracranial hemorrhage, and the potential for proximal clot dissolution and subsequent embolization. While successful thrombolysis has been described for type A clots, type B clots may dislodge and cause PE following lysis of their attachments.

We considered the first author of a recent meta-analysis (3) of RHTET and PE to summarize the current experience using thrombolytic agents to treat CVC-related RAT, with or without PE, is limited to case reports. To summarize the current experience using thrombolytic agents to treat CVC-related RAT in adults, we systematically reviewed English language publications from Ovid MEDLINE (January 1966 to November 2004) using the following search terms: “exp catheterization/central venous” or “exp catherization peripheral, exp thrombosis”, “exp heart atria”, “exp vena cava”, “superior/ or exp vena cava”, “inferior”, ‘right atria*.ml’, “exp fibrinolytic agents”, “exp thrombolytic therapy/ or exp tissue plasminogen activator”, “exp urinary plasminogen activator/ or exp streptokinase”, “exp recombinant proteins”. Additionally, we contacted the first author of a recent meta-analysis (3) of RHTET and PE to identify cases of CVC-related RAT treated with thrombolysis in their registry. Table 1 summarizes the case reports of thrombolytic treatment for RAT associated with CVCs in adults (3,7,8). Limited information regarding the assessment of complications associated with thrombolysis was available. To our knowledge, the present article represents the first report of CVC-related RAT in an adult patient treated with thrombolysis.

The present case highlights several important considerations. First, CVCs expose patients to potential complications that, in turn, are associated with increased morbidity and mortality. Second, a decrease in platelets by 50% or more after day 4 and before day 14 should increase suspicion for HIT, even in the presence of concurrent sepsis. Third, several factors related to the host (e.g., underlying chronic myelogenous leukemia), instrumentation (e.g., PICC) and treatment (heparin exposure) may have acted synergistically and resulted in thrombus formation. Fourth, HIT in the presence of PE and a PICC-related thrombus presents diagnostic and management challenges. Fifth, the literature may underestimate the incidence of thromboembolic complications related to CVC and PICC because the diagnosis may not be considered in symptomatic and especially, in asymptomatic patients, or may be missed by TTE. Finally, the present case underscores the important role of TEE and thrombolysis in the diagnosis and management, respectively, of RHTET with associated PE.

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REFERENCES

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