

Simple Site, Serious Sequelae: Septic Emboli from a Peripheral Cannula

A. Walker, E. Keelan.

Internal Medicine Department, Ulster Hospital, South Eastern Health and Social Care Trust,
Upper Newtownards Rd., Dundonald, Belfast, Northern Ireland, United Kingdom.

Abstract

Presentation

A young female patient presented with right arm pain and pleuritic chest pain following recent hospitalisation. Investigations demonstrated marked inflammation (CRP 296 mg/L, WCC $13.7 \times 10^9/L$).

Diagnosis

She was diagnosed with multiple septic pulmonary emboli (SPE) secondary to cephalic vein thrombophlebitis and cellulitis at a previous cannula site.

Treatment

Despite prolonged intravenous antibiotics, she later developed culture-proven septic arthritis requiring washout and further treatment.

Discussion

Peripheral venous cannula-associated SPE remain rarely reported.

Introduction

Septic pulmonary emboli (SPE) is a severe infectious process in which an infected thrombus embolises to the pulmonary vasculature, causing septic infarcts and systemic complications. It is historically associated with right-heart infective endocarditis (IE), intravenous (IV) drug use and infected central venous catheters (CVC).

Peripheral venous cannulas (PVCs) are commonly used invasive devices in healthcare and are traditionally considered low risk for catheter-related bloodstream infection (BSI). SPE secondary to PVCs is exceedingly rare, with only three published cases in the English literature.

Case Report

A female patient in her early 20s presented complaining of right arm pain, swelling, and pleuritic chest pain. She had recently been admitted with an eczema flare during which a short PVC was inserted into the right cephalic vein for IV antibiotics. Prior to discharge, she reported erythema and purulent exudate at the PVC site, prompting removal.

The patient had been receiving ciclosporin for eczema, which was discontinued two weeks prior to admission. She was, therefore, not considered immunosuppressed at presentation, however, impaired skin integrity secondary to eczema may have increased her susceptibility to infection.

Upon examination, she was pyrexia (38.0°C) and tachycardic (116 bpm). Erythema and tenderness were present in the right ante-cubital fossa. Inflammatory markers were significantly elevated (CRP 296 mg/L, WCC $13.7 \times 10^9/L$), with a mildly raised D-dimer of 0.786 µg/mL (reference range 0.0–0.5 µg/mL). The patient's Wells score placed her at moderate risk for pulmonary embolism (16.2% probability).

CT pulmonary angiogram demonstrated multiple bilateral SPE. Upper limb Doppler ultrasound showed superficial thrombophlebitis in the cephalic vein, corresponding to the previous PVC site. A single set of blood cultures on day 3 of admission grew *Staphylococcus Epidermidis*, felt clinically relevant by microbiology given the patient's history and imaging. Transthoracic Echocardiography (TTE) showed no vegetations suggestive of IE, (the authors acknowledging the limitations of TTE for this purpose). Transoesophageal echocardiography (TOE) was not pursued at this point as superficial thrombophlebitis was considered the likely source.

The patient was discharged under the outpatient parenteral antimicrobial team to complete a 6-week course of IV antibiotics.

Two weeks post antibiotic completion, she re-presented with a right ankle effusion. Arthroscopic washout yielded *Staphylococcus aureus* from joint aspirate cultures. Immunosuppressive therapy had not been recommenced, and the patient was considered immunocompetent at re-presentation.

She required a further 6-week course of IV antibiotics for septic arthritis. Although presumed secondary to haematogenous spread from the original PVC-related infection, the discordant organism does mean an alternative source cannot not be excluded. TOE was performed at this point after discussion with Cardiology and excluded IE.

Discussion

PVCs are ubiquitous in hospital medicine and are perceived as lower-risk devices than CVCs. Although the incidence of PVC-related BSI is substantially lower than that of CVCs (0.5 vs 2.7 infections per 1000 catheter-days),¹ the widespread use of PVCs means their overall contribution to healthcare-associated infection may be underestimated.²

Only three previous cases of PVC-associated SPE have been reported in the literature,³⁻⁴ with one resulting in death⁵, highlighting both the rarity and potential severity of this complication. This case illustrates how even a short PVC can act as the source of severe metastatic infection with significant morbidity.

Several factors may have contributed to infection risk in this case. Although the patient was not considered overtly immunosuppressed at the time of presentation, she had recently discontinued ciclosporin therapy and suffered from active eczema, both of which may have impaired skin barrier integrity and host defence mechanisms. In addition, the presence of visible local site infection prior to discharge raises the possibility that earlier recognition and intervention may have reduced progression to thrombophlebitis and septic embolisation.

This case reinforces the importance of meticulous PVC care, including strict aseptic insertion technique, secure dressing fixation to reduce cannula micro-motion, routine inspection and prompt removal of devices when no longer clinically indicated.

Particular caution should be exercised in patients with impaired skin integrity or recent immunomodulatory therapy, even when they are not considered formally immunosuppressed.

In conclusion, PVCs should not be regarded as benign devices and localised cannula-site inflammation or discharge may precede severe invasive complications including septic thrombophlebitis, SPE, and metastatic infection. Early recognition of PVC-related infection, vigilant site monitoring, and prompt device removal are essential to preventing serious morbidity and potential mortality.

Declarations of Conflicts of Interest:

None declared.

Corresponding Author:

Andrew Walker,
Internal Medicine Department,
Ulster Hospital,

South Eastern Health and Social Care Trust,
Upper Newtownards Rd.,
Dundonald,
Belfast,
Northern Ireland,
United Kingdom.

E-Mail: awalker32@qub.ac.uk

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