Fatal Sepsis following Peripheral Intravenous Cannula Embolus*

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This is a case report of multiple septic complications of a peripheral intravenous cannula as a direct result of proximal embolization of a fragment of the cannula to the heart and major vessels.

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Bacteremia is a recognized complication of intravenous catheters, with the incidence much higher in central as opposed to peripheral intravenous catheters. Tricuspid valve endocarditis complicating an intracardiac catheter fragment and septic pulmonary emboli have recently been described in patients with central intravenous catheters. The following report is of a fatal case of sepsis in a patient in whom both these complications occurred. To our knowledge, this represents the first time either complication has been reported in a patient with a peripheral intravenous catheter.

CASE REPORT

A 74-year-old woman with a long history of schizophrenia and epilepsy was admitted to the hospital with an acute confusional state secondary to diuretic-induced hyponatremia. Her parenteral fluid replacement was complicated by peripheral cannula replacement on four occasions following forceful removal of these cannulas by the patient. The patient responded to medical therapy and was discharged from the hospital after 12 days. She was well for six months until she re-presented with a two-week history of a dry cough, dyspnea, fever, and confusion.

On examination the patient had a temperature of 39°C and coarse inspiratory crackles with diminished breath sounds at the left base. She was in sinus rhythm and had a systolic murmur at the left sternal edge. There were no signs of congestive cardiac failure or of bacterial endocarditis. The abdomen was unremarkable and she had features of a tachy dyskinesia and an acute delirium.

Chest roentgenogram showed left lower lobe consolidation and Staphylococcus aureus sensitive to flucloxacillin grew from blood cultures. Following ten days of intravenous therapy with this antibiotic, the patient remained unwell and febrile. A computed tomogram of the chest indicated further deterioration, with collapse and consolidation of the left lower lobe, an abscess in the right middle lobe, and bilateral pleural effusions. A dense linear opacity within the mediastinum, consistent with a foreign body, was also seen (Fig 1). Review of previous chest roentgenograms revealed on discharge six months earlier a foreign body visible in the right atrium; this had moved to the left pulmonary artery by the time of this hospital admission (Fig 2). There was no foreign body seen on a chest roentgenogram from two years earlier.

The foreign body was removed by a loop wire inserted via the right femoral vein under radiologic control. A 4-cm fragment of a venous cannula was retrieved and when cultured, S aureus grew profusely, having an identical sensitivity profile to the Staphylococcus grown from the blood. Despite initial clinical improvement, the patient died suddenly three days later.

Postmortem examination found a large vegetation on the atrial surface of the tricuspid valve, and there was a septic thrombus adherent to the wall of the left pulmonary artery. There was a wedge-shaped pulmonary infarct in the left lower lobe and a suppurative cavity within the right middle lobe.

DISCUSSION

This patient represents a particularly severe case of

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multiple septic complications of peripheral intravenous cannulation. These included staphylococcal septicemia, tricuspid valve endocarditis, septic thrombosis adherent to the left pulmonary artery, pulmonary emboli, infarction, and abscess formation.

Proximal embolization to the right atrium, and later to the left pulmonary artery, of a fragment of a peripheral intravenous cannula must account largely for the extent of cardiorespiratory sepsis. This is an uncommon complication and has been reported to occur from a central venous catheter, but to our knowledge, this patient represents the first reported case of this complication from a peripheral intravenous cannula. It is unlikely that this will occur without forceful removal of a cannula, a common occurrence in patients in confusional states. To avoid this, cannulas should be secured as firmly as possible in uncooperative patients. In the event of extraction, the cannula should be inspected and a report of this should be clearly recorded. If the cannula is found to be incomplete, a chest roentgenogram should be performed. Visualization of a fragment on the chest roentgenogram should be followed initially by an attempt at percutaneous removal. To aid detection of cannula fragments, we suggest a radiopaque marker along the length of all cannulas in routine use.

Staphylococcus aureus is one of the most common isolates grown from intravenous devices, and it has been postulated that for such growth to occur, microorganisms migrate from the catheter skin entry site. In view of staphylococcal infection manifesting six months after cannulation in this patient, an alternative explanation would be colonization of the foreign body fragment with a bacteremia, possibly resulting from a respiratory tract infection. Appropriate antibiotic therapy and removal of the infected cannula fragment did not prevent a fatal outcome in this patient. This is consistent with the known high mortality rates associated with serious S. aureus infections, even with adequate antimicrobial therapy.

This case reinforces the need to implement recommended practices to minimize cannula-associated sepsis, and this should include careful inspection of all cannulas forcefully removed.

REFERENCES

Critical Mitral Stenosis Causing Ischemic Hepatic Failure
Successful Treatment by Percutaneous Balloon Mitral Valvotomy

Michael B. Harding, M.D.; J. Keen Harrison, M.D.; Charles J. Davidson, M.D.; Katherine B. Kisslo; R.D.M.S.; and Thomas M. Bashore, M.D.

We report a 52-year-old patient with severe mitral stenosis who developed new onset atrial fibrillation, low output congestive heart failure and fulminant ischemic hepatic failure with subsequent severe coagulopathy. Percutaneous mitral valvotomy resulted in dramatic clinical improvement with complete resolution of liver function. This case illustrates the potential life-saving role for percutaneous balloon mitral valvotomy in treating critically ill patients who are unable to undergo thoracotomy due to coexisting medical illness.

Ischemic hepatic failure and coagulopathy due to critical mitral stenosis and low output cardiac failure are uncommon and an attempt at surgical correction of the underlying mitral stenosis in this situation may carry a prohibitive surgical risk. Our patient developed fulminant ischemic hepatic failure with new onset atrial fibrillation and critical mitral stenosis. The role of percutaneous balloon mitral valvotomy in this situation is described.

CASE REPORT

History and Findings

A 52-year-old white man was transferred to this institution for the evaluation of severe mitral stenosis, heart failure, and hepatic failure. Two years earlier, mitral stenosis had been incidentally diagnosed and mitral valve replacement was recommended but declined.

In the interim, the patient developed progressive dyspnea on exertion, orthopnea and pedal edema. Six weeks prior to admission, he became anorectic and developed resting dyspnea. After developing hemoptysis, he sought medical attention. There was no history of alcohol use or exposure to hepatotoxins.

On admission to the community hospital, the patient was hemodynamically stable in normal sinus rhythm. His condition dramatically worsened on the third hospital day with the development of atrial fibrillation and a ventricular response of 170 beats per minute. Metabolic acidosis with a lactate level of 9.6 mg/L was noted. Hepatic transaminases reflected acute hepatocellular necrosis. Failure of synthetic hepatic function and coagulopathy developed. No episode of hypotension or hypoxemia was documented.

Upon transfer, the blood pressure was 110/70 mm Hg, the resting pulse was 100 beats per minute and irregularly irregular, and the respiratory rate was 28. The patient was confused, lethargic and deeply icteric. Jugular venous pressure was above the angle of the jaw upright. The carotid upstroke was of low volume. Rales were noted in the midlung fields bilaterally. A right ventricular heave was obvious. The S1 and S2 were accentuated. A faint opening snap and a grade 1/6 diastolic rumble were audible. No regurgitant murmur was heard. The liver was enlarged and tender, and ascites

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Mitrail Stenosis Causing Ischemic Hepatic Failure (Harding et al)

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