CENTRAL VENOUS CATHETER INDUCED SEPTIC THROMBOSIS OF THE SUPERIOR VENA CAVA: A CASE REPORT

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Abstract

The development of septic thrombosis due to central venous catheterization is relatively rare and difficult to diagnose. The associated morbidity and mortality is high if medical therapy fails. We report a 51-year-old male who presented with fever and persistent Staphylococcus aureus septicemia. The same pathogen was also isolated from the tip of his Hickman catheter. Enhanced computed chest tomography (CT) demonstrated thrombosis of the superior vena cava and septic pulmonary embolization. The treatment included early removal of the infected catheter and intravenous broad-spectrum antibiotics. However, further complications including infective endocarditis of the tricuspid valve, septic pulmonary embolization and acute hypoxic respiratory failure were encountered. The patient ultimately expired due to refractory septic shock and multiple organ failure. This case reminds us that medical therapy may be insufficient and surgical thrombectomy and removal of the infected vein may be necessary.

Key words: Septic thrombosis, Superior vena cava, Staphylococcus aureus, Pulmonary embolization

Introduction

Catheter-induced septicemia is a common nosocomial infection and central venous catheterization is responsible for large portion of these bloodstream infections.¹ The common causative organisms are *Staphylococcus* species, *Enterococci*, gram-negative aerobic bacilli, and *Candida* species.² Successful treatment is often achieved with immediate removal of the infected catheter and administration of appropriate antibiotics. Septic thrombosis of the superior vena cava (SVC) is a rare but serious clinical entity with significant mortality and morbidity. The disease is usually a critical complication in patients who require central venous catheterization for prolonged dwell times. It may progress to infective endocarditis and septic pulmonary embolization.³ Chest CT scans with contrast medium enhancement usually confirm the diagnosis.⁴ In addition to medical intervention, the best strategy for managing septic thrombophlebitis, especially when medical therapy fails, will be discussed.⁵

Case Report

A 51-year-old man with a history of chronic renal failure received regular hemodialysis via
a right subclavian Hickman catheter for one month. Pulmonary tuberculosis was diagnosed two months prior to this hospitalization and isoniazid, rifampin, ethambutol and pyrazinamide were prescribed. He visited our emergency department due to fever for two days. On arrival, he appeared acutely ill. His body temperature was 38.4°C; pulse rate 110 beats/min; respiratory rate 21 breaths/min, and blood pressure 153/100 mmHg. A chest radiograph (Fig. A) showed a Hickman catheter in the SVC. There was no purulent discharge from the catheter insertion site. Intravenous amoxicillin/clavulanate was prescribed under the impression of sepsis with an uncertain source of infection. Methicillin resistant Staphylococcus aureus (MRSA) was isolated from a blood culture on the 8th day of hospitalization and intravenous amoxicillin/clavulanate was shifted to vancomycin 1 gm twice a week. The same species of organism was isolated from a blood specimen withdrawn through the catheter. Because catheter-related MRSA septicemia was highly suspected, the Hickman catheter was removed immediately. The semiquantitative culture of the catheter tip grew MRSA ≥ 15 colony-forming units, and the diagnosis of catheter-related MRSA septicemia was established. To exclude infective endocarditis, we arranged transthoracic echocardiography, which disclosed left ventricular dysfunction (ejection fraction 30%) and moderate mitral regurgitation (MR). There was no evidence of vegetation formation. However, clinical improvement was not achieved after the above medical management. Signs of sepsis including sustained fever, hypotension, and thrombocytopenia were found even under an adequate serum vancomycin trough level (11.22 mg/L). Persistent MRSA bacteremia was noted after the Hickman catheter was removed. On the 10th day of vancomycin administration, a chest radiograph (Fig. B) showed multifocal peripheral opacities in both lungs and the patient suffered from severe hemoptysis at the same time. Chest CT with contrast medium enhancement showed a filling defect (1.5 x 1 cm) in the SVC and multiple cavitary lesions over both lungs (Figs. C1 and C2), which indicated septic thrombosis of the SVC and pulmonary embolization. We shifted the
medication to teicoplanin 400 mg every 72 hours. An anticoagulant was not administered due to concerns of the active hemoptysis. Surgical intervention including thrombectomy and debridement of the adjuvant infected vessel was strongly suggested, but the patient refused the invasive procedure. On the 22nd day of antibiotic treatment for MRSA bacteremia (vancomycin 10 days and teicoplanin 12 days), low blood pressure accompanied by aggravation of a heart murmur was noted. Repeated transthoracic echocardiography showed one huge tricuspid vegetation (4 cm in diameter) with severe tricuspid regurgitation and moderate pulmonary hypertension (Figs. D1 and D2). Acute hypoxic respiratory failure developed on the 30th hospitalized day, and the patient received endotracheal intubation and mechanical ventilation. The patient expired on the 34th hospitalized day due to refractory septic shock and multiple organ failure.

Fig. C. CT scan of the chest. An intraluminal filling defect (1.5 x 1 cm) in the SVC (C1) and septic embolic lesions over both lungs (C2) were found.

Fig. D1, D2. Cardiac echo showed one huge tricuspid valve vegetation (4 cm in the long axis).
Discussion

Central venous catheterization is essential for many clinical situations, such as establishing venous access, hemodynamic monitoring, and parenteral nutrition. However, it can lead to severe complications, such as pneumothorax, hemothorax, catheter-related septicemia, infective endocarditis and thrombosis. Among the complications, septic thrombophlebitis of the central venous system is an infrequent entity, but often leads to a lethal outcome. The catheter may be infected at the time of, or shortly after, insertion. The principal infection source is the microbes on the skin that migrate along the subcutaneous tract created by the indwelling catheter. Other possible mechanisms include hematogenous seeding or if the catheter is contaminated during skin puncture.

Thrombosis formation around the catheter is another complication and it may be precipitated by catheter-related septicemia. Once the thrombosis has become infected, further severe complications such as septic pulmonary emboli and other metastatic infections can be seen.

The incidence of catheter-related septic thrombophlebitis of the central venous system may be underestimated. The reported frequency of septic thrombosis due to central venous catheterization ranges from 1 to 14%.

Clinical manifestations of catheter-related septic central venous thrombosis vary from fever (83%), leukocytosis (78%), sepsis syndrome (39%) and pleuropulmonary complications (28%), and it may easily be confused with other sepsis sources. In addition, thrombotic venous occlusion could result in swelling of the involved ipsilateral neck, chest or limbs. Our patient had a similar presentation with fever, leukocytosis, septic shock and septic pulmonary embolism during his hospital course.

The establishment of the correct diagnosis usually relies on clinical suspicion. Persistent septicemia despite catheter removal and appropriate antimicrobial therapy are clues to the diagnosis. Prompt diagnostic modalities include CT with contrast medium enhancement, gallium scans, digital vascular imaging, series of blood cultures, and catheter tip culture with various sensitivity and specificity. Chest CT with enhancement is the most useful investigative tool and may reveal an intraluminal filling defect of the SVC and inflammatory infiltrations of the surrounding soft tissue. The presence of gas within the thrombus can be found in some cases, and it has been found to be a diagnostic CT feature of SVC septic thrombosis. In addition, chest CT can easily detect septic pulmonary emboli.

There have not been any randomized controlled studies to guide the management of SVC septic thrombophlebitis. Based on previous experience, immediate removal of the involved catheter, intravenous antibiotics and anticoagulation are the currently recommended management. Since this disease is an intravascular infection, prolonged antibiotic therapy (4-6 weeks) at high doses is needed. This approach has been reported to result in 50% successful treatment. There is currently no role for thrombolytic agents as adjuvant treatment. Surgical intervention including thrombectomy and drainage should be applied for failure of medical treatment or suppurative focus around the SVC. The complex nature and high risk of surgery should not preclude this approach in such life-threatening conditions.

Conclusion

With more frequent use of central venous catheterization, septic thrombosis is a lethal complication that should not be underestimated. In addition to prompt diagnosis and aggressive medical treatment, surgical thrombectomy may be necessary.

References

中心靜脈導管引起之上腔靜脈敗血性血栓--病例報告

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摘要

放置中心靜脈導管所導致的上腔靜脈敗血性血栓是相對地罕見和困難診斷。假使內部治療失敗，其相關的併發症及死亡率就非常的高。在此我們報告一個51歲男性以發燒和持續性的金黃色葡萄球菌敗血症為其臨床表現。胸部電腦斷層掃描顯示上腔靜脈血栓併發敗血性肺栓塞。其治療包含立即移除中心靜脈導管及廣效性抗生素的使用。然而，卻併發三尖瓣性心內膜炎、敗血性肺栓塞及急性呼吸衰竭。最後這個病患因敗血性休克及多器官衰竭而死亡。此病例提醒我們針對上腔靜脈敗血性血栓內部治療反應不良的病人應考慮合併外科手術治療。

關鍵詞：敗血性血栓，上腔靜脈，金黃色葡萄球菌，肺栓塞