

Case Study

THROMBOTIC COMPLICATION OF CENTRAL VENOUS CATHETERISATION IN SUBCLAVIAN VEIN

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Abstract: *Central venous catheterization (CVC) has become an integral part for management of patients in intensive care units. It enables invasive monitoring, facilitate atrial pacing and permit delivery of parenteral alimentations and medications. Notwithstanding, CVCs are associated with numerous complications such as mechanical complications, infections and thrombotic complications. We presented a case of 17-year-old male patient who developed massive central venous thrombosis causing superior vena cava (SVC) syndrome after sequential placement of a right and left subclavian vein central venous catheter. Colour Doppler and Computed tomography (CT) angiography showed thrombosis of bilateral subclavian, internal jugular and axillary veins with minimal blood flow. Patient was treated with low molecular weight heparin (LMWH) and warfarin with partial recanalization of veins and resolution of symptoms.*

Keywords: *Central venous catheterization, Central venous thrombosis, superior vena cava syndrome.*

Introduction

Central venous thrombosis is a serious and life-threatening complication in patients with an indwelling catheter and a source of considerable morbidity [1]. CVC has become an important secondary cause of upper limb deep vein thrombosis (ULDVT). Incidence of venous thrombosis associated with CVC has been varied in different studies [2,3]. However catheter-related thrombosis is often under-diagnosed and this can have disastrous outcomes.

Case report

A 17 year male patient of road traffic accident with head injury was admitted in emergency department. Right subclavian vein catheterisation was done with triple lumen catheter as a part of initial management and resuscitation. Insertion of central venous catheter (CVC) took multiple attempts. The correct position of the catheter tip was checked by chest radiography after CVC insertion. Computed tomography (CT) scan of head showed multiple contusions. Patient was intubated and kept on mechanical ventilation. Patient was tracheostomised on day 10. On day 15 CVC got blocked. Patient also developed slight swelling of right arm. A second CVC was inserted in left subclavian vein and right CVC was removed. Swelling of right side reduced gradually over days. On day 30 left CVC also got blocked and patient developed swelling over left arm, face and upper thorax with reappearance of swelling of right arm (Fig.1). A clinical diagnosis of superior vena cava (SVC) syndrome was made. Investigations showed normal platelet count and coagulation profile. Colour flow doppler (Fig.2a) showed intraluminal thrombus with minimal blood flow in both left and right subclavian , internal jugular(IJV) and proximal part of axillary veins with blockage more on right side as compared to left side. CT angiography (Fig.3) yielded similar results. Femoral vein was secured for intravenous access and left CVC was removed.



Figure 1:

Patient treated with enoxaparin (LMWH) 0.6ml twice daily for 2 weeks till swelling subsided (Fig.4). Physical therapy in form of limb elevation and hot fomentation was also provided. Complete blood count, blood gases, and clotting profile were monitored. Repeat colour doppler showed partial recanalization of right axillary left subclavian and left internal jugular vein with right subclavian vein and IJV thrombosis, and normal blood flow in left axillary veins (Fig.2b). With resolution of symptoms oral warfarin was started. Patient was discharged on request after tracheostomy site closure with advice to continue warfarin for 3 months and periodic follow ups.

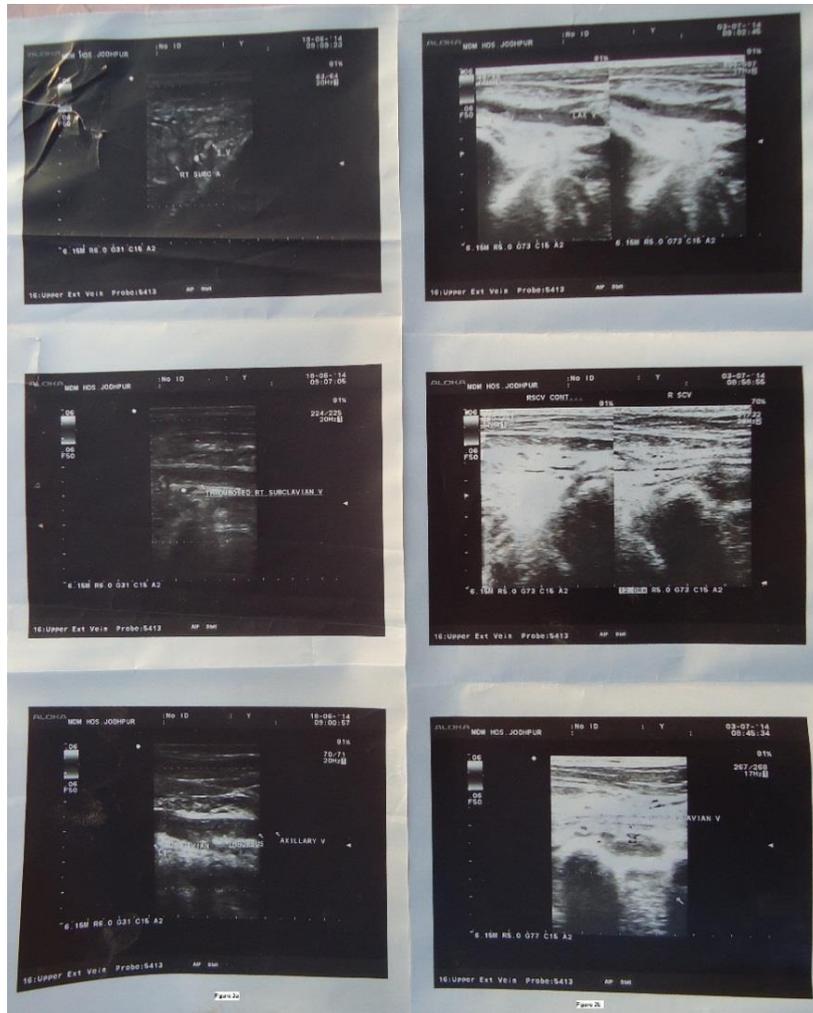


Figure 2:



Figure 3:



Figure 4:

Discussion

With the growing number of central venous cannulations of subclavian and internal jugular veins, thrombosis of these deeper veins is not an unusual occurrence^[4]. Not all patients with subclavian vein or internal jugular vein thrombosis are symptomatic. Mild-to-moderate non-pitting edema or dilatation of subcutaneous collateral veins over the upper arm and chest may be the only clue to the clinical diagnosis. In symptomatic patients, the clinical features of acute ULDVT (swelling and pain) are nonspecific. More often, minor symptoms and signs are overlooked and the diagnosis is missed or delayed resulting in extension of the thrombus with involvement of other major vessels. This may also be reason for reported variable mean time to thrombosis after insertion of the central venous catheter.

Unilateral swelling indicates an obstructive process at the level of the brachiocephalic, subclavian, or axillary veins whereas head, neck, or bilateral upper-extremity swelling suggests involvement of either the superior vena cava or both the subclavian and brachiocephalic systems^[5] (as with this case). Thrombosis in this patient initiated at the right subclavian vein with manifestation of slight edema of right arm. It gradually extended to the right axillary vein and the internal jugular vein. Venous drainage from collaterals may not have allowed the onset of venous obstructive symptoms. Left IJV and subclavian vein thrombosis challenged the compensatory mechanisms resulting in edema of both upper limb and chest. Thus such huge thrombosis of veins produced clinical picture of superior vena cava syndrome in absence of involvement of SVC.

The thrombosis of the veins with indwelling catheter may be related to local trauma to the vein wall, turbulent flow around the catheter (with consequent release of procoagulants) and stasis of blood stream. The catheter, being a foreign body, also increases the likelihood of serving as a site for platelet adherence^[6]. Duration of catheterization^[7], physical properties of the catheter and nature of infused fluid also influence catheter associated venous thrombosis. Larger catheters increase the likelihood that stasis can develop. This may be the reason for higher risk of developing thrombosis with the use of triple-lumen CVC than with double or single lumen catheters^[8]. Multiple insertion attempts and a second CVC also increases risk of ULDVT^[9].

Color flow Doppler examination is the screening technique of choice^[10]. Contrast venography is considered the gold standard in the evaluation of ULDVT. Venography is used when thrombosis is highly suspected despite a negative ultrasonography (USG) study.

Because large series of patients with this condition are lacking, the therapeutic approach to such massive thrombosis is mainly anecdotal. The initial treatment includes rest, elevation of the limb, and application of heat or warm compresses. Physical therapy may decrease swelling and pain. Thrombolytics (streptokinase, urokinase, recombinant tissue-type plasminogen activator) therapy is found to be ineffective in chronic venous thrombosis and in patients with massive organizing thrombi^[11]. Hence anticoagulation therapy (heparin, oral anticoagulants) or surgical repair remain the mainstay of treatment in patients with catheter-related thrombosis^[12]. Removal of the catheter should be combined with anticoagulation to avoid embolization.

Anticoagulants maintain patency of the venous collaterals and prevent recurrent thrombus formation, pulmonary embolism and permanent pathologic changes, such as venous valvular dysfunction and postphlebotic syndrome. The therapy is initiated with heparin. Low molecular weight heparin (LMWH) is just as effective as unfractionated heparin, but the former is associated with a significant decreased incidence of venous thromboembolism. Prothrombin time and activated partial thromboplastin time should be monitored every 6 hours to prevent adverse effects of excessive anticoagulation. The heparin therapy is followed by warfarin therapy, with an international normalized ratio goal of 2-3. Warfarin is continued for at least 3 months. Patients with severe persistent symptoms despite anticoagulation can be considered for surgical thrombectomy or endovascular stenting^[13].

Current evidence does not recommend routine use of thromboprophylaxis for CVC or a specific anticoagulant for thromboprophylaxis. Thus until information from large clinical trials is obtained, the best prophylaxis of upper limbs deep vein thrombosis is to prevent it by limiting the use of central venous catheters for extended periods of time.

Conclusion

Despite the presence of many factors predisposing to local thrombosis around catheter tips, massive thrombosis causing SVC syndrome, as seen in our case, is not common. Although central venous catheter was the main cause of thrombosis in our patient, multiple attempts at insertion and delayed diagnosis must have contributed to the extent of thrombosis. Not all patients with deep vein thrombosis are symptomatic. Hence clinical suspicion should initiate investigations. Early detection of venous thrombosis by colour doppler prevent clot extension and fatal complication of thrombus.

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