CASE REPORT

Venous thrombosis in subclavian, axillary, brachial veins with extension to internal jugular vein, right sigmoid sinus and simultaneous pulmonary embolism

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We present a rare case of Venous Thrombosis in Subclavian, Axillary, Brachial Veins with extension to Internal Jugular vein, right sigmoid sinus and simultaneous Pulmonary embolism during the treatment with low molecular weight heparin.

Key words: Cavernous sinus thrombosis, upper extremity deep vein thrombosis, venous thromboembolism

INTRODUCTION

Since 150 years ago, the pathophysiology of venous thrombosis has been explained by Virchow's triad: Endothelial injury, change in blood flow, and hypercoagulability of blood. Deep vein thrombosis (DVT) is an important clinical dilemma with an estimated incidence of 500,000 cases per annum in the United States.[1,2] About 300,000 hospital admissions reported annually, with an estimated length of stay between 1 and 7 days.[1]

Upper extremities DVT accounts for less than 5% of all venous thromboembolism (VTE) events and its incidence has recently increased. It may be due to the development of more precise diagnostic methods or use of indwelling central venous catheters.[3]

Here, we present a case of both upper extremities together with jugular vein thrombosis who developed sub-segmental pulmonary embolism during his treatment with low molecular weight heparin (LMWH). This association of simultaneous venous thromboembolism is rare. There have reported only few cases reports in this regard.[3-5]

CASE REPORT

A 79-year-old man admitted in Alzahra University Hospital, Isfahan, Iran for left upper extremity swelling since 2 weeks ago. He also complained of sudden onset dyspnea and cough. After 2 days, he experienced a generalized tonic colonic seizure with left-side hemiparesia and sudden onset impairment in his visual field. He had a history of chronic obstructive lung disease, chronic history of cigarette smoker and systemic hypertension. At the time of admission, he had no respiratory distress with stable vital signs but decreased respiratory sound on auscultation in bilateral lower lung fields. Laboratory tests showed peripheral white blood cells 7,600/mm^3, (4000-8000) hemoglobin 11 g/dL (normal level between 14 and 16), and platelets increased to 157,000/mm^3 (150,000-450,000) Arterial blood gas analysis under 2 L/min oxygen supply showed pH 7.459, $PaCO_2$ 38.9 mmHg, and PaO_2 79.5 mmHg. Serum D-dimmer value was increased to 9.02 µg/mL (Normal <4 µg/mL). Color duplex sonography of the neck veins and proximal veins of left arm was revealed hypoechoic and mobile thrombosis occluded the left Internal Jugular Vein (IJV) which extended to sub-clavian, axillary, brachial veins [Figure 1]. There was no evidence of thrombosis in the lower extremities. At first, Contrast-enhanced multi-slice multi-detector computed tomography (MSCT) revealed no thrombus in pulmonary vasculatures. During the first week, his symptoms were gradually improved with full anticoagulation by LMWH, (Enuxaparin 60 mg Subcutaneous q12 hours hospital after 5 days, he suddenly developed respiratory distress, progressive dyspnea with hypoxemia without any changes in hemodynamics. Repeated MSCT showed right segmental pulmonary embolism with involvement of sub-segmentary vessels. He was transferred to intensive care unit and the patient's management continued with

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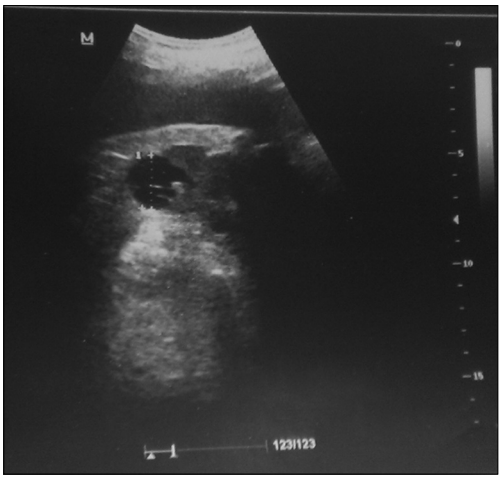

Figure 1: Hypoechoic and mobile thrombosis occluded the left internal jugular vein

anticoagulation and his symptoms improved within 1 week. After 2 months repeated venous duplex revealed evidence of resolution following appropriate anticoagulation.

DISCUSSION

Cerebral sinus thrombosis

Superior sagittal (62% of patients) and the transverse sinus (40-45% of patients) are the most frequent locations for thrombosis at the cerebral veins.[1,2] According to the site of involved venous structure, clinical presentations are different. If thrombosis located in the cortical veins, localized brain edema and parenchimal infarction may develop.[1] Stupor or coma (in 15%) may occur due to large intracranial infarcts or hemorrhages.[1,2] The most common clinical presentations are headache and papilledema due to intracranial hypertension, seizures, focal neurological deficits and altered consciousness. Headache occurs in a majority of patients (>90% cases), papilledema (about 30%) that may cause visual loss and occasionally diplopia.[2] Seizures (Focal or generalized) may develop in up to 40% of patients, with motor deficits.,[2] The onset of symptoms is sub-acute, developing from 2 days to 1 month in many of these patients. Prognosis of cerebral sinus thrombosis is favorable in more than 80% of cases. Death in the acute phase is results from cerebral herniation and underlying diseases (e.g., malignancy).

Prognosis of superior sugital vein thrombosis (SSVT) is favorable owing to the early identification of the disease that allow treatment in the acute stage. Early anticoagulant treatment with fixed doses of subcutaneous LMWH (according to the patient's body weight) or adjusted doses of intravenous or subcutaneous unfractionated heparin (maintaining the activated partial thromboplastin time ratio between 2.0 and 3.0) is also crucial to limit thrombus extension.[1,2]

Upper-extremity deep vein thrombosis (UEDVT)

The most common cause of UEDVT is the use of indwelling central venous catheter or pacemakers.[2] Central catheterization is complicated by thrombosis, often asymptomatic, in more than two thirds of adults.

It accounts for only 5-10% of all cases of venous thrombosis. Malignancy is another strong risk factor for UEDVT. Recently, a case-control study showed an approximately eight-fold increased risk of UEDVT in patients with cancer. Catheter and malignancy related UEDVT are secondary events and account for about 70% of all UEDVT. The remaining cases are primary UEDVT, including those due to Paget-Schroëtter syndrome or effort thrombosis, which are triggered by strenuous muscular activity and hyperabduction of the arms. The main complications of UEDVT are pulmonary embolism and post-thrombotic syndrome.[3]

The clinical presentation of UEDVT is similar to that of the lower limbs deep-vein thrombosis, with swelling (most common sign), functional impairment, weakness and discomfort of the arm and/or hand. Paresthesia, cyanosis and pain may be present.

Duplex ultrasound examination is the first diagnostic investigation. Its diagnostic accuracy is so much high, with sensitivity and specificity ranging from 78% to 100% and 82-100%, respectively.

Treatment of primary UEDVT does not differ from that recommended for deep-vein thrombosis of the lower limbs, i.e., subcutaneous LMWH followed by oral anticoagulation with Vitamin K Antagonists (VKAs) adjusted to maintain an international normalised ratio range between 2.0 and 3.0.

Internal jugular vein thrombosis

Internal jugular vein thrombosis is a rare condition. Its prevalence was 1.5% in patients presenting DVT.[4] In 72.4% of patients with IJV thrombosis exhibited concurrent involvement of suclavian/axillary/brachial vein. The most common causes of IJV thrombosis are cancer, central venous catheter and ovarian hyperstimulation syndrome.

Among cancer-associated IJV thrombosis, patients mainly exhibited pulmonary cancer and lymphoma/leukemia. Our findings underscore that the search for cancer should be made routinely in patients exhibiting IJV thrombosis, especially in the group with bilateral IJV thrombosis.

DISCUSSION

This case of internal jugular, upper extremity, sigmoid sinus thrombosis, without a central catheter subsequently developed

pulmonary emboli, in spite of receiving anticoagulation is extremely rare. We did not find any similar case report by searching in pubmed. Our investigation did not find any sign of malignancy and patient discharged from the hospital 10 days after admission. Three months after discharge he was in regular follow-up and was in good general condition.

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