CASE REPORT

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Successful treatment of superior vena cava syndrome due to internal jugular vein thrombosis in a 55-year-old man with comorbid diabetes mellitus type 2 and pneumonia

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ABSTRACT

Introduction: Superior vena cava syndrome (SVCS) associated with jugular internal vein thrombosis can be dangerous. Diagnosing SVCS and venous thromboembolism (VTE) must be done precisely and accurately to minimize the risk of pulmonary embolism.

Case Presentation: Male, 55 years old, complaining of shortness of breath, the feeling of choking in the neck, and a lump in the neck for one month. The patient has a history of type 2 diabetes mellitus and does not routinely take medication. The patient experienced tachypnea and tachycardia, and there was an enlargement in the submental to the submandibular region; the boundaries were not firm, supple, or fixed, approximately ten centimeters in diameter, and the collateral veins were enlarged and felt pain. Chest examination revealed telangiectasia, crackles, and bronchovesicular breath sounds over both lungs at the base and medial side. The patient had mild anemia and leukocytosis, increased HbA1c levels, total cholesterol levels, and increased D-dimer and Lactate dehydrogenase (LDH). Antero-posterior (AP) lateral cervical radiograph shows a widening of the soft tissue in the retro tracheal space at the level of the second cervix. The patient given warfarin, enoxaparin, methylprednisolone, and heparin. Improvement of the condition was shown.

Conclusion: Superior vena cava syndrome due to internal jugular vein thrombosis is a challenging case. Careful diagnostic measurement based on history and physical examination, in addition of additional investigation may lead to the diagnosis. Management with warfarin, enoxaparin, methylprednisolone, and heparin show clinical benefit.

Keywords: Vena cava syndrome, internal jugular venous thrombi, DM type 2, warfarin, enoxaparin, heparin
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INTRODUCTION

The internal jugular vein (IJV) originates from the jugular foramen and travels alongside the lateral neck, positioned medially to the sternocleidomastoid muscle within the carotid triangle. It terminates at the brachiocephalic vein. The IJV is one of the four components that form the blood vessels in the neck, together with the Common and Internal carotid arteries, vagus nervous system, and deeper cervical lymph nodes. IJV thrombosis is a potentially life-threatening condition caused by the formation of a blood clot within the vein. Thrombosis can occur anywhere from the jugular foramen to the point where the IJV meets the subclavian vein, which forms the brachiocephalic vein.[1-3]

Any level of thrombosis is possible, from the jugular foramen to the confluence of the IJV and subclavian vein, which together form the brachiocephalic vein. Chylothorax, superior vena cava syndrome, pulmonary embolism (PE), lower airway swelling, superior sagittal sinus thrombosis, intracranial hypertension, cerebral edema, septic emboli, and intracranial hypertension are only a few of the severe life-threatening consequences that have been reported during IJV thrombosis.[3-7]

Thrombosis is the formation of a blood clot in a blood vessel.[8] This thrombus or blood clot can form in a vein, artery, heart, or microcirculation and cause complications due to obstruction or embolism.[9-11] Deep vein thrombosis is known as deep vein thrombosis (DVT).[9] Examinations to detect DVT include D-Dimer and imaging (such as ultrasound, venography, computed tomography scan, or magnetic resonance venography). In the case report below, we describe a case of DVT in the bilateral internal jugular veins.

CASE PRESENTATION

A man (55 years old) came to the emergency room (ER) with dyspnea—difficulty breathing and tightness and choking on the neck. Shortness of breath is not affected by activity. Shortness is also felt at rest. This complaint has been getting worse since the last few days before going to the emergency room hospital. The patient also complained of swelling in

the front of the neck, under the chin, and jaw (Figure 1). The swelling has formed an uneven lump since one month ago. The lump was initially the size of a marble, then grew slowly. This lump is sometimes painful and sometimes not. The patient has had diabetes mellitus since one month ago but does not take medication regularly. The patient has gone to a general practitioner and was given clindamycin. There was no diabetes or hypertension before. The patient worked as a farmer.

On physical examination, the patient looked ill, with dyspnea; vital signs were 120/80 mmHg, heart rate 102 times/minute,respiratory rate 25 times/minute, and saturation 98%. His palpebra was edema, and on his neck, found a mass in the submental for the submandibular region; the submandibular border was not clear with firm, solid consistency, fixed with diameter ±10 cm, tenderness, and telangiectasia on the thorax. Auscultation on his lung found crackles without wheezing. Bronchovesicular found in basal and medial bilateral lung, crackles found at basal et medial bilateral lung. His motor and sensory examination was regular. On workup examination, leukocytes were 13,810, with elevated blood glucose (239 gr/dl) and cholesterol 232 gr/dl.

On radiological examination (Figure 2), it appears that the retro tracheal space widens, a suspected mass with an abscess, straight cervical. His x-ray found a bilateral pleural effusion, pneumonia, pneumonia, and right lung fibrosis caused by former inflammation. The diagnosis was given when the patient underwent a Doppler ultrasound of the cervical veins (Figure 3). A thrombus was seen in the right jugular vein, which caused severe stenosis (77% stenosis) along ± 6.39 cm, some with total stenosis. The partial outflow of the right jugular vein is still visible with peak systolic velocity (PSV) 16.5 cm/s. A thrombus was seen in the left jugular vein, which caused severe stenosis (89.8% stenosis) along ± 4.7 cm, some with total stenosis. The partial outflow of the right jugular vein is still visible with PSV=13.4 cm/s. Conclusion of the Doppler ultrasounds supports the picture of DVT in bilateral jugular veins.

The patient was given oral warfarin 5 mg, increased the dose to 6 mg, then

Figure 1. Clinical examination

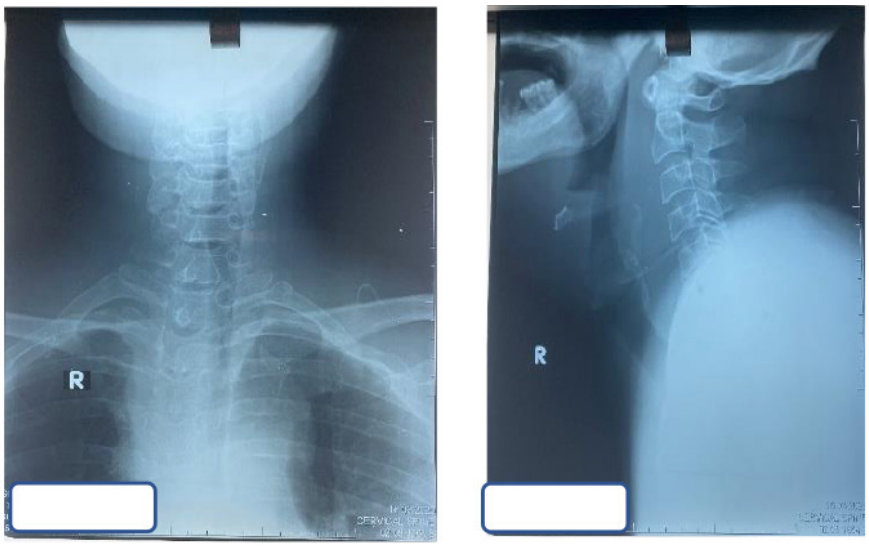

Figure 2. Cervical antero-posterior lateral

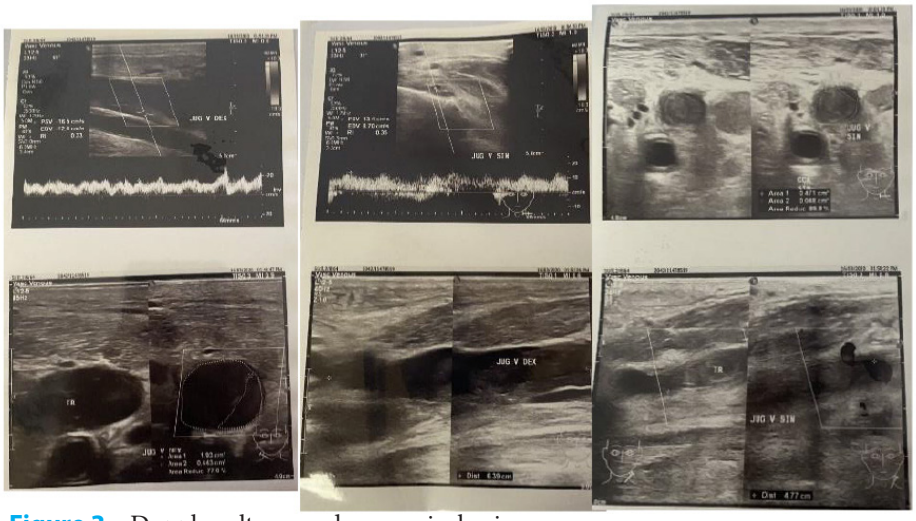

Figure 3. Doppler ultrasound on cervical vein

given subcutaneous enoxaparin 2 x 0.6 cc for five days, followed by intravenous methylprednisolone 3 x 62.5 mg and given drip heparin for several days and adjusted the dose according to the aPTT results. At the time of discharge, the patient found the lump had decreased in size and tightness, and the cough had also decreased. We gave oral warfarin 6 mg and basal and prandial insulin to control blood sugar.

DISCUSSION

Deep vein thrombosis (also known as deep-vein thrombosis or venous thrombosis and usually abbreviated as DVT) is the formation of a blood clot ("thrombus") in a deep vein.[12-14] DVT is a form of thrombophlebitis, which is the inflammation of a vein with the formation of a blockage. Blood clots (thrombus) in the deep venous system of the legs are not dangerous.[12,15] The situation becomes life-threatening when a piece of the blood clot breaks off (embolus, pleural embolism), travels downstream through the heart into the pulmonary circulation system, and lodges in the lung.[11,16,17] Diagnosis and treatment of DVT are intended to prevent PE.[18] Causes of DVT include immobility conditions such as prolonged travel and sitting, such as long airplane flights (economy class syndrome), car, or train travel, hypercoagulabilities such as the use of estrogen pills and smoking, and trauma to the veins.[10,12] The symptoms of DVT are related to the obstruction of blood returning to the heart and causing backflow in the legs. Classically, the symptoms of DVT include pain, swelling, warmth, and redness. None of the above symptoms appear; maybe just one could be all, or maybe no symptoms at all.[10,12] Symptoms may mimic infection or cellulitis of the feet. It is not enough to diagnose DVT based solely on clinical symptoms because each individual's symptoms or signs are neither specific nor sensitive for making a diagnosis. Clinical judgment can determine a patient's pretest probability of DVT. Combining the Well's Rule for assessing pretest probabilities with noninvasive test results is expected to increase the accuracy of the diagnosis to reduce the need for further investigation.[18]

Compression vein ultrasonography (CUS) is currently the most widely used noninvasive test in cases of suspected DVT because of its precise detection of thrombus in the popliteal vein or its proximal area.[19] In the absence of compressibility of the proximal lower leg veins, it has a sensitivity of 97% and a specificity of 96% in symptomatic patients with suspected DVT. Therefore finding an uncompressed portion of the vein, particularly in the popliteal or femoral vein, has a high positive predictive value for symptomatic DVT patients and is an indication for therapy. A 'two-point' (popliteal and common femoral) or 'three-point' (two-point plus calf) ultrasound should be performed and, if expected, can be repeated one week after the initial examination. This approach will identify 20–25% of patients with a clot extending from the distal calf vein proximally. D-dimer testing or clinical and ultrasonographic assessment may limit the need for repeat testing. Another examination is venography.[19,20] Also called phlebography, ascending contrast phlebography, or contrast venography; it is an invasive diagnostic examination that provides constant images of the leg veins on fluoroscopy. Venography identifies the location, spread, and severity of blood clots and assesses the condition of the deep veins. Venography is explicitly used in cases of high suspicion of DVT, but noninvasive tests fail to identify DVT. Venography is the most accurate test for detecting DVT. Its sensitivity and specificity are close to 100% in making the diagnosis of DVT.[21]

D dimer is a blood test used as a screening test to determine if a blood clot is present. D-dimer is a chemical that is produced when blood clots in the body gradually dissolve/break down. Other than D dimer, neutrophil-lymphocyte ratio (NLR) and platelet lymphocyte ratio (PLR) also useful as DVT diagnostic tools. Magnetic resonance imaging (MRI) is commonly used to diagnose DVT in pregnant women or in DVT in the pelvic, iliac, and vena cava areas where duplex scanning of the lower extremities is negative. The sensitivity and specificity of MRI are as low as duplex ultrasound in diagnosing venous thrombosis. The advantage of MRI is that it can evaluate the entire venous system, including the pelvic area veins. MR angiography is as sensitive as multidetector computed tomography (MDCT) in diagnosing emboli. The disadvantages of MRI are the high cost and difficult access.[20,22-25]

The management of DVT includes stopping the spread of the clot or thrombus and preventing recurrent thrombus, PE, and the development of pulmonary hypertension, which can lead to multiple and recurrent PE. Unfractionated heparin administered as a bolus of 80 I.U./kg body weight intravenously followed by an infusion of 18 1U/kg body weight per hour before administration, activated partial thromboplastin time (APTT), prothrombin time (PT), and platelet count should be checked, especially in patients over 65 years of age. Low molecular weight heparin (LMWH) is combined with warfarin for four to five days. Enoxaparin is FDA-approved (U.S. Food and Drug Administration) for the treatment of DVT at a dose of 1 mg/kg twice daily or 1.5 mg/kg once daily. The dose of warfarin is 5 mg daily, adjusted every three to seven days to get an INR value between 2.0 and 3.0. Trying to maintain the INR between 1.5 and 2.0 is still a contradiction. In one study, consecutive outpatients treated with LMWH for DVT or P.E. were randomized to receive 5 or 10 mg of warfarin. An INR of more than 1.9 was obtained on average 1.4 days immediately after the patient received 10 mg. Thrombolytic therapy aims to quickly lyse the thrombus by activating plasminogen into plasmin. Thrombolytic drugs include streptokinase, recombinant tissue plasminogen activator (rt-PA), and FDA-recommended urokinase.[14,16,26]

In 2004 the American College of Chest Physicians stated that thrombectomy therapy is recommended for routine use in acute DVT. Two things need to be considered in thrombectomy. First, thrombectomy is associated with bleeding during the procedure. Second, recurrent thrombosis may occur in some patients despite postoperative anticoagulation. This may be due to damage to the venous endothelium, either from the clot itself or from mechanical damage in an attempt to remove the thrombus. Prevention of DVT includes using Compression stockings (socks compression) routinely. Surgery patients walk out of bed early, and low

doses of heparin or enoxaparin are used for DVT prophylaxis (steps taken to prevent DVT).[27]

CONCLUSION

A 55-year-old man had difficulty breathing and shortness of breath and seemed to choke on his neck. This complaint has been getting worse since the last few days before admission. The patient lumped the submental area and was diagnosed as a thrombus on Doppler Cervical Vein ultrasound examination. The patients assessed with bilateral internal jugular vein thrombosis as well as diabetes mellitus and community pneumonia. The patient was given oral warfarin then given subcutaneous enoxaparin for five days, followed by intravenous methylprednisolone. The patient also received heparin for several days and adjusted the dose according to the aPTT results. Superior vena cava syndrome due to internal jugular vein thrombosis is a challenging case which need attentive examination and management.

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PATIENT CONSENT

Written informed consent was obtained from the patient to be included in the case report.

CONFLICT OF INTEREST

The author stated that there is no conflict of interest.

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AUTHOR CONTRIBUTION

RT, SOW contributed to the study's conceptual, data acquisition, clinical data assessment, follow-up of the patient, and during manuscript preparation. RT and ARN contributed to the study's conceptual and data validation during the manuscript revision.

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