# Thrombosis of Internal Jugular and Subclavian Veins: A Possible Complication of Cough

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## **Abstract**

Increased use of internal jugular and subclacian vein as a venous access route has recently caused an increase in prevalence of thrombotic complications of these veins. Cancers, hypercoagulable conditions, infection, peripheral venous lines, intravenous drug abuse and trauma are other well known risk factors for thrombosis of upper extremity and neck veins. Here, we describe a patient who had chronic obstructive pulmonary disease and

developed combined thrombosis of left internal jugular and subclavian veins just following prolonged and vigorous coughing bouts. The patient was treated initially with intravenous continuos heparin infusion and subsequently with oral coumadin with no early complications or late sequels.

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### Introduction

Upper extremity deep venous thrombosis (UEDVT) is characterized by pain, edema, and functional impairment, however, it may be completely asymptomatic. In the past, upperextremity DVT was considered as an uncommon condition accounting for less than 2% of all DVT's as a result of increased use of central venous catheters but it has recently been recognized as a more common and less benign disease than previously reported (1-5). Both, isolated or combined thrombosis of internal jugular vein and subclavian vein may occur (6). Central venous catheters, underlying neoplastic diseases, hypercoagulability conditions, radiation are the most frequent risk factors (2,4,5). Furthermore, internal jugular DVT may be associated with neck dissection, ovulation induction with gonadotropins, trauma, infection (Lemierre's syndrome) (7-10) and axillo-subclavian vein DVT may be related with effort (thoracic outlet syndrome), intravenous drug abuse or peripheral venous line. Herein, we report development of combined thrombosis of left internal jugular and subclavian veins just following vigorous and prolonged

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coughing attacks in a patient with chronic obstructive pulmonary disease (COPD).

# Case Report

A 42- year-old male patient was admitted to the hospital with the complaints of left upper extremity swelling, shortness of breath, productive cough with purulent sputum and lower extremity edema. The patient had a medical history of COPD for more than ten years, which resulted in development of core pulmonale six weeks ago. Three days ago following a common cold, he began to have vigorous coughing attacks due to exacerbation of his COPD. Further questioning revealed that the patient had felt some pain on the left side of his neck and distention of the neck vessels where his ache was, after a vigorous and long lasting coughing attack in the night. He also had became aware of numbness on left arm a few hours later. The next afternoon, he noticed a swelling on his left arm and hand. Swelling on his left arm had gradually increased on the next two days. His history was remarkable with a 20-pack-year of smoking, which he has given up six weeks ago. The patient had no history of DVT, pulmonary embolism, surgery, hypercoagulability, recent central or peripheral venous catheterization and the family history was also negative for thrombotic diseases.

Physical examination revealed a bloated, bruised man with a swelling on his left arm. Vital signs were: pulse rate 106 beats/min; blood pressure, 100/70 mm Hg; respiratory rate 26 breaths/min; temperature, 37°C. He was cyanotic on lips and tongue. There was bilaterally jugular venous distention on his neck, however, left jugular venous distention was manifested in association with a painful and palpable venous cord.



Figure 1. The patient's picture, showing digital clubbing and peripheral cyanosis at fingertips and non-pitting swelling at left arm from shoulder to fingers.

On chest examination, prominent venous vessels (collaterals) could be seen on the left anterior chest wall. Bilateral coarse crackles and expiratory wheezes and rhonchi were audible on both lung areas. Cardiac examination revealed tachycardia and loud  $P_2$ . The liver was tender and palpable 10 cm below the right costal margin. The lower extremities were both edematous (+++). He had digital clubbing and peripheral cyanosis at the fingertips and non-pitting swelling at the left arm from the shoulder to the fingers.



Figure 2. The patient's chest x-ray, showing mild cardiomegaly and hyperlucent lungs.

Laboratory results were hematocrit, 53%; WBC count, 8400/mm<sup>3</sup> with a normal differential; platelet count, 182000/mm<sup>3</sup> and erythrocyte sedimentation rate, 4 mm/h. Arterial blood gas values while the patient was breathing room air: pH, 7.44; pO<sub>2</sub>, 50 mm Hg; pCO<sub>2</sub>, 47 mm Hg. Blood biochemistry results were BUN, 19 mg/dl; creatinine, 0.9 mg/dl; ALT, 22 IU/l; LDH, 485 IU/l (normal range 0-450); serum D-dimer level, 0.88 mcg/ml (normal range 0-0.50). Prothrombine time was 13.6 sec (INR:1.1) and aPTT 26 seconds. Electrocardiogram showed right ventricular hypertrophy. There were mild cardiomegaly and hyperlucent lungs on chest radiography. The patient's history and physical and laboratory findings suggested diagnosis of deep venous thrombosis (DVT) of left upper extremity and jugular veins. A color Doppler sonography was performed and showed thrombosis of left internal jugular vein extending from two centimeters below the angulus mandibulare to the end of the left subclavian vein. Left subclavian vein was also found to be thrombotic without



Figure 3. Computed tomography of the neck showing thrombosis of the left internal jugular vein (arrow).

any currency. Color Doppler sonography of right upper extremity and bilateral lower-extremity did not provide any evidence suggesting DVT. Contrast-enhanced computed tomography of the neck and chest revealed thrombosis of left internal jugular and subclavian veins consistent with the findings of color Doppler sonography (Figure 3). A ventilation-perfusion lung scanning was performed and interpreted as low probability of pulmonary embolism. Abdominal sonography showed diffuse hepatomegaly. Serum levels of tumor markers of CA-19-9, CA-72-4, carcinoembriogenic antigen, alpha-fetoprotein and prostate specific antigen were within normal limits. Fiberoptic broncoscopic examination was negative for lung malignancy. Sputum cytology was negative for malignant cells.

The patient was treated with antibiotics and bronchodilators for COPD exacerbation. Our preference in establishing anticoagulation was to administer a loading dose of heparin, and then maintain activated partial thromboplastin times at twice the control values with a continuous heparin infusion. Heparin was continued for eight days and oral anticoagulation using coumadin is started keeping INR between 2-3, before discontinuing heparin. A week later palpable jugular venous cord disappeared and left arm swelling has improved. Two months later, a control color Doppler sonography showed reestablishment of venous flow in subclavian and internal jugular veins.

#### Discussion

DVT is usually a vascular disorder of the lower limbs, which is very rare at the level of the upper limbs and the neck, particularly as a consequence of a central venous catheterization, infectious disease or trauma. Acute symptoms of DVT of the upper extremity and neck veins include pain, swelling, and a sense of coldness in the hand and forearm. Edema is typically non-pitting. A tender cord along the

course of the involved vein also may be palpable as in this patient. Collateral veins may be seen over the shoulder and chest wall. Clinical diagnosis of deep venous thrombosis of the arm may be difficult. Phlebography is the most reliable diagnostic procedure for axillo-subclavian vein thrombosis, however, it is not useful for the evaluation of internal jugular veins. Color Doppler sonography has been established as a rapid, noninvasive and useful technique for diagnosis and follow up of venous disease in the upper extremity (3-6,11). In our case, the diagnosis of combined thrombosis of subclavian and internal jugular veins was made by contrastenhanced computed tomography of the neck and chest and color Doppler sonography.

Cough is an important defense mechanism to help clear excessive secretions and foreign material from airways. It is one of the most common complaints in patients with COPD and may be vigorous during acute exacerbation. Subjective perceptions of exhaustion, symptoms of insomnia, hoarseness, musculoskeletal pain and sweating are the most common troublesome symptoms of persistent and vigorous cough. Besides these subjective symptoms, very high intrathoracic pressures of up to 300 mm Hg, produced during vigorous coughing can cause development of a variety of serious complications in nearly all organ systems such as cough syncope, rib fractures and abdominal hernias (12,13). Transmission of this very high intrathoracic pressure through blood vessels may cause damage in both venous and arterial vessels. Rupture of subconjunctival, nasal and anal veins and stroke due to vertebral artery dissection have been reported as complications of vigorous coughing (13,14). In our patient, shearing stress produced by high intrathoracic pressure may have resulted in endothelial damage of upper limb and neck veins. Intimal vascular damage initiates activation of coagulation cascade by releasing thromboplastic substances that activate the extrinsic coagulation pathway and by directly activating the intrinsic coagulation pathway. In this case, besides activation of coagulation cascade due to vascular endothelial injury, venous stasis due to temporary cessation of venous flow during coughing bouts, pulmonary hypertension, heart failure and high hematocrit level may be other contributing factors for development and propagation of fibrin formation. Hilzenrat and et al. reported a 35-year-old woman with chronic cystic lung disease who developed spontaneous thrombosis of right internal jugular vein presenting with sudden and severe right-sided neck pain (15). The authors postulated that the main risk factor for the development of internal jugular vein thrombosis was stasis resulting from severe pulmonary hypertension and high blood viscosity. We suggest that, besides stasis, vigorous cough may have played a role in the development of internal jugular vein thrombosis in their patient.

Pulmonary embolism, the post-thrombotic syndrome and rarely venous gangrene are the main complications of thrombosis of subclavian and internal jugular veins. The most serious aspect of immediate morbidity and mortality in DVT is related to the development of pulmonary emboli. The clinical diagnosis of acute pulmonary embolism in patients with COPD is often difficult, because the presentation of an acute embolic event may closely mimic the symptoms of acute airway obstruction (16). The patient also had COPD for ten years, however, we believe that, V/Q lung scan findings were sufficient to exclude the presence of a major pulmonary embolism. The goal of therapy in UEDVT patients is to alleviate the acute symptoms, prevent pulmonary emboli and avoid disabling late sequel. Anticoagulant therapy is the mainstay of the treatment, alone or in association with fibrinolytic treatment or surgical approach. Anticoagulation using initially intra venous heparin infusion and subsequent oral coumadin was successful in the patient. Oral anticoagulation should be continued at least three months to maintain patency of venous collaterals, allowing for recanalization and reendothelization of thrombosis.

Our observation suggests that, prolonged and vigorous coughing bouts may cause development of thrombosis of subclavian and/or internal jugular veins in patients who had an underlying disease such as chronic obstructive pulmonary disease. Detailed history of the patients interpreted as having spontaneous or idiopathic UEDVT may clarify the role of cough in pathogenesis of UEDVT and facilitate the recognition of new cases.

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