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CASE REPORT

LUNG MALIGNANCY PRESENTING AS BILATERAL INTERNAL JUGULAR VEIN THROMBOSIS

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Abstract

Venous thrombosis may be fatal, for example by a pulmonary embolism and right or left atrial thrombosis. Alternatively, deep vein thrombosis may follow a benign pattern such as femoral and popliteal vein thrombosis. Internal Jugular vein (IJV) thrombosis is a rare entity. Internal jugular vein thrombosis is a serious event with potentially fatal outcome, where the clinical symptoms may be vague or absent. It is usually secondary to various etiologies such as catheter, malignancy, trauma, infection and hypercoagulable status. Associated malignancies, either known or occult, are also uncommon and not well documented in the etiology of IJV thrombosis. Spontaneous internal jugular vein thrombosis may occur in connection with a neoplasm, termed Trousseau's syndrome. Although the true incidence of IJV thrombosis is unknown it appears to be increasing.

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Introduction

Venous thrombosis a common problem usually involves extremities. Its pathophysiology is described by Virchow's triad i.e. endothelial injury, change in blood flow and hypercoagulability (1). Internal jugular vein thrombosis (IJVT) is a rare but potentially fatal condition and was a well known complication of head and neck infections in pre-antibiotic era (2). In this era the leading causes for IJV thrombosis are central vein catheterization (3) and repeated injections in large neck veins (4). Head and neck vein thrombosis without any obvious cause may be the initial manifestation of an occult malignancy (5). However, IJVT may be a part of a superior vena cava (SVC) syndrome, which mostly occurs secondary to malignancy (6, 7). Bilateral

internal jugular vein thrombosis is a rare condition and can be a sign of metastasis.

CASE REPORT:

Our case was a 65 year old male patient who is a chronic smoker but normotensive and nondiabetic and presented with pain and swelling neck and breathlessness on and off of one month duration. Pain was insidious in onset but progressive and was associated with swelling of the neck and puffiness of face. Patient also had history of hoarseness of voice, weight loss and decreased appetite of same duration. Rest of the history was insignificant. On examination patient was conscious and oriented to time, place and person. Patient was afebrile but had mild pallor. On chest examination patient had dull percussion note on right infra axillary area and bronchial breathing in right infrascapular area on auscultation. There was

decreased air entry in the base of right lung. Rest of the systemic examination was normal. On local examination there was swelling on the right side of neck measuring 5×7 cm which was tender to touch but compressible. Visible veins were present on the chest and neck. Ultrasonography (USG) abdomen showed right sided moderate pleural effusion while USG neck revealed normal thyroid gland and no features suggestive of thyroid or parathyroid malignancy. Chest radiograph showed opacity on right side involving middle and upper lobe of lung suggestive of malignant lesion with right sided moderate pleural effusion (**FIG. 01**).

CECT chest and neck revealed irregular speculated 5.3×5.3×6 cm central right lung mass causing abrupt cutoff of right upper lobe bronchus with presence of central necrosis. The mass was compressing superior vena cava (SVC) with confluent paratracheal and subcarinal lymphadenopathy. Moderate pleural effusion was present on right side and right upper lobe showed scattered alveolar infiltrates. Bilateral internal jugular vein were partially thrombosed (**FIG. 02**). Subclavian, axillary and bronchocephalic veins were normal. Impression of right central upper lobe mass with SVC compression with bilateral partial IJV thrombosis and mediastinal lymphadenopathy was made.

ABG revealed pH of 7.39/7.43, sodium 145/128 mEq/l, potassium of 3.3/3.8 mEq/l, pCO₂ of 41/34 mmHg, pO₂ of 55/60 mmHg, bicarbonates of 18.3/22.6 mEq/l measured two times respectively. Complete blood count showed Hb 9.9g/dl, TLC $7.18 \times 10^3 /\mu\text{l}$, DLC 67% neutrophils and 23% lymphocytes, platelets 1.48 lac/ μl and ESR 31mm/hr. KFT showing urea of 25mg/dl and creatinine of 0.8mg/dl. LFT showed bilirubin of 0.98mg/dl, ALT of 37IU/l, AST of 28IU/l, ALP of 74IU/l, total protein of 6.64g/dl and albumin of 4.6g/dl. Other investigations showed calcium of 9.2mg/dl, phosphorus of 2.71mg/dl, CPK of 33 IU/L and LDH of 287 IU/L. Coagulogram revealed protombin time of 10.6 seconds, international normalized ratio of 1.0, activated partial thromboplastin time of 34.0 seconds. ANA, ANCA, anticardiolipin antibodies, protein C, protein S were all negative. Pleural fluid analysis showed lymphocytic exudate. M-cells were positive in pleural fluid two times. Then CT guided biopsy was done and histopathological examination revealed adenocarcinoma.

Fig. 01: Right Sided Lung Mass with Effusion

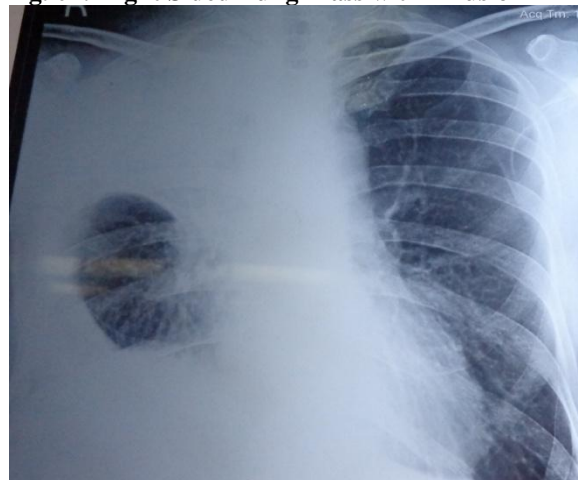


Fig 02: CT neck with bilateral internal jugular vein thrombosis



DISCUSSION:

Internal jugular vein thrombosis a rare condition has varied etiology from head and neck infections in pre-antibiotic era to trauma due to central catheterization, intravascular drug use, and occasionally cervical traction related and ovarian hyperstimulation syndrome (8). Several studies have shown occult malignancy as a cause for jugular vein thrombosis. The association of malignancy and coagulation disorders is well known; 50% of patients with tumours and over 90% of patients with metastatic disease will have some detectable coagulation abnormality and up to 15% of patients are symptomatic (9,10). The mechanism of tumour-induced coagulopathy remains ill-understood. It may involve the production of coagulation activators, such as tissue thromboplastins and platelet activating factors, or the direct increase of circulating levels of

clotting factors, inhibitors of fibrinolysis or platelet numbers (11,12). Trousseau first observed the association of cancer and thrombophlebitis (13). A 3 to 19-fold increase in prevalence of concomitant cancer has been reported in patients presenting with an idiopathic venous thromboembolism (VTE). The prevalence of occult cancer in patients with secondary VTE is comparable with the prevalence of cancer in the general population, while the prevalence of occult cancer in patients with idiopathic VTE is 4-10% (14). Reported complications associated with jugular vein thrombosis include septic emboli, pulmonary embolism, elevated intracranial pressure, facial edema, intracranial venous thrombosis and loss of vision (15). The incidence of pulmonary embolism after upper extremity DVT is approximately 14% (16).

Accurate diagnosis of deep venous thrombosis is very difficult and imaging plays a crucial role in the diagnosis or exclusion of DVT. Although contrast venography is the gold standard for the demonstration of venous occlusion, there are many inherent risks of this modality. Noninvasive investigations, such as ultrasound, CT and MR, can establish the diagnosis of IJV thrombosis easily and have replaced the jugular venography, which carried significant risks, such as dislodgement of the clot and dissemination of septic emboli or trauma to the vein (17, 18). Suspected internal jugular vein thrombosis can be rapidly diagnosed using duplex ultrasonography (19). Ultrasound has the key advantage of providing a bedside diagnosis, with high sensitivity and specificity, and may achieve superior resolution to CT in superficial areas. A CT scan and magnetic resonance image (MRI) can also be used for diagnosis. These methods are especially useful for excluding a local mass effect from an unsuspected malignancy. Furthermore, these methods can help identify the possible cause of internal jugular vein thrombosis.

The treatment, although difficult, must be initiated with heparin; however, it is often unsuccessful and long-term therapy with either warfarin or heparin is typically not satisfactory. Patients with JVT may need to be on long-term oral anticoagulation therapy. Treatment of the underlying malignancy is the most definitive therapy, but usually in these particular diseases is also unsuccessful (20).

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