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IJV Thrombosis Complicating Pulmonary Tuberculosis with Cervical Lymphadenopathy: A Rare Association

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INTRODUCTION

IJV thrombosis is a rare occurrence in children. It is found in hospitalised patients with central venous catheters, hypercoagulability associated with malignancy, head and neck surgery, polycythaemia, hyperhomocysteinaemia and deep neck infection. It is a serious and potentially life-threatening occurrence. It can lead to pulmonary embolism and intracranial propagation of thrombus.

The pathophysiology behind the inherited and acquired factors leading on to development of thrombosis is based on Virchow's triad of stasis, hypercoagulable state and vascular injury(2)

Tuberculosis has been postulated to be a hypercoagulable state that appears to develop secondary to the acute phase response(3). The production rate of fibrinogen, which is an acute-phase reactant, may increase greatly secondary to inflammation(3,4). Reduced antithrombin III and protein C levels, due to hepatic dysfunction secondary to tuberculosis, have been reported and may contribute to the hypercoagulable state(5). Vascular injury-induced increased platelet activity may be another mechanism for vein thrombosis in tuberculosis. In children with pulmonary tuberculosis, platelet activation occurs and has a good correlation with the extent of the disease (6).

Also in tuberculosis, large collective matted lymph node mass or mediastinal enlargement causing mechanical venous obstruction may cause stasis to blood flow(7). Lymphadenopathy can cause extensive vessel wall damage with endothelial damage predisposing to thrombosis(8)

USG is a reliable radiological investigations for detecting IJV thrombosis, along with cervical lymph nodes. It is inexpensive and readily available for monitoring response to treatment. Doppler scan shows patency of remaining IJV lumen. Early detection of IJV thrombosis is must to avoid complications like raised intracranial pressure, cerebral venous sinus thrombosis, pulmonary embolism, and septic emboli.

Pulmonary Tuberculosis with cervical lymphadenopathy with IJV thrombosis is extremely rare complication and very few cases are reported in pediatric age group.

CASE SUMMARY

7 year female child of weight 13 kg presented with complain of low grade intermittent fever for one month associated with cough, loss of appetite, weight loss and new onset of respiratory distress for 1 wk. child was on ATT (HRZE) for 15 days. There was no known contact with tuberculosis.

On admission child was sick looking, pallor present, HR 137/min, RR 45/min. on auscultation air entry was reduced on left side of chest, subcostal and suprasternal retraction were present. Child was found to have diffuse non tender swelling on left side of neck. Multiple matted non tender suprasternal lymph nodes were palpable not fixed to underlying structure or overlying skin.

Investigation: Hb 10.0 gm/dl, TLC 19000/ cumm, 83% PMN cells, 12% lymphocytes. Platelet count- 3 lac / cumm, ESR Tuberculin test as per previous report was 12*11mm.

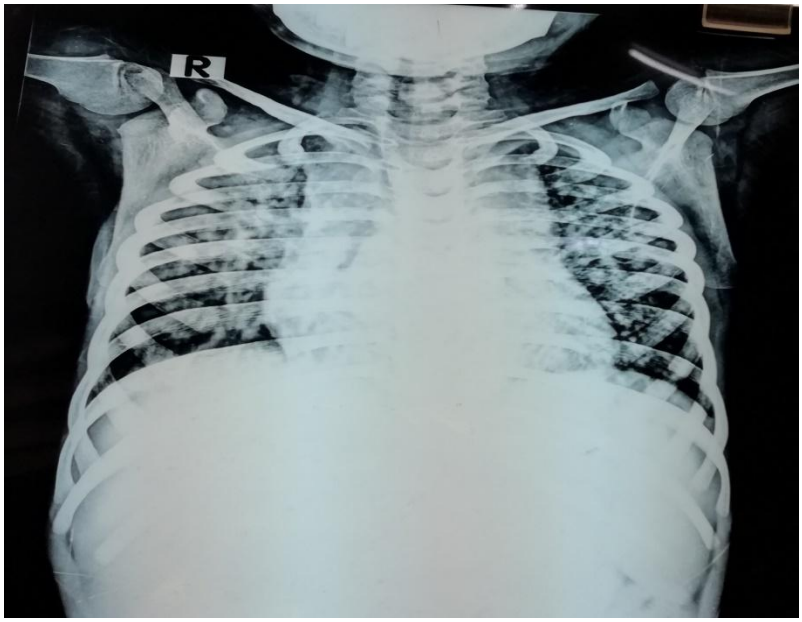

Figure 1: X- ray chest at the start of ATT

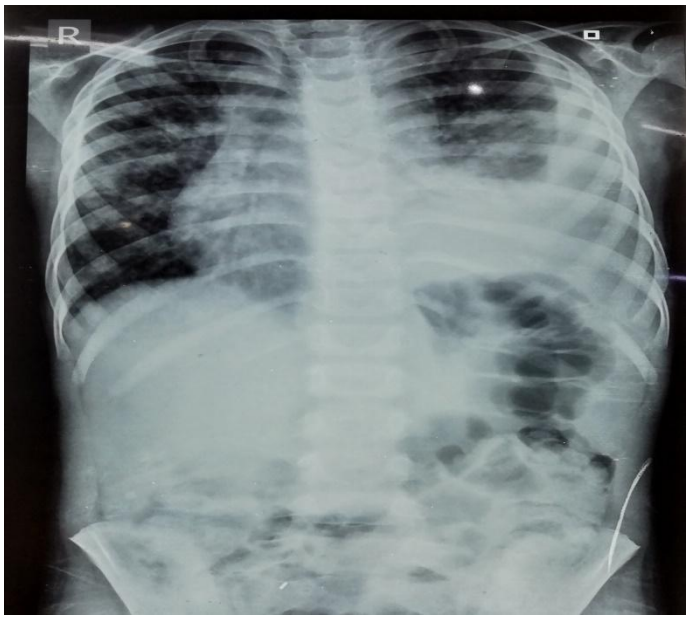

Figure 2: X-ray chest at the time of presentation

Usg chest showed mild pleral effusion on right side with moderate left sided pleural effusion with few internal echoes.
Usg neck showed multiple hypodense cervical lymph nodes with loss of cortico medullary differentiation. Left sided IJV was partially compressible and patent flow in color Doppler and showed echogenic thrombus in it. Right sided IJV was normal.

HIV serology was negative.

Blood urea 32, s.creat 0.6, s.uric acid 6.6, s.AST 33, s.ALT 16, s. protein 7.8, s. albumin 3.5, A/G 0.8, s. bilirubin 0.2.
PT 17.6, INR 1.37, PTT 32.0,

Based on these reports child was diagnosed as pulmonary tuberculosis with cervical lymphadenopathy with IJV thrombosis.
Protein C 77.5, Protein S 90.4, Factor V leiden 2.51, Fibrinogen C 4.7g/l.
CBNAAT of pleural fluid was positive for Tuberculosis.

Treatment - ATT continued and the child started on enoxaparin.

DISCUSSION

The mechanism responsible for development of venous thrombosis in TB is unclear. All the three parts of Virchow's triad, i.e., hypercoagulability, venous stasis, and endothelial dysfunction, may play a role in pathogenesis of the disease. Patients with active PTB have anemia, reactive thrombocytosis, elevations in plasma fibrinogen degradation products, tissue plasminogen activator, and inhibitors with depressed antithrombin III levels which may favor the development of venous thrombosis in patients with TB(9).

In the present case, all these three factors may be in operation

1. cervical lymph node enlargement causing mechanical venous obstruction that may cause stasis to blood flow

2. hypercoagulable state secondary to tuberculosis as explained earlier.

3. Lymphadenopathy that may cause extensive vessel wall damage with endothelial damage predisposing to thrombosis
Also in present case there was no co-existing thrombophilia state.

In conclusion, although rare, IJV thrombosis may complicate pulmonary tuberculosis.

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