



ANTI-TUBERCULAR TREATMENT INDUCED DEEP VEIN THROMBOSIS- A CASE REPORT

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ABSTRACT Deep vein thrombosis (DVT) is a serious condition that occurs when a blood clot forms in a vein located deep inside our body. Tuberculosis may induce at the peripheral blood - activation of mononuclear cells, and the interaction of these cells activated with mycobacterial products will induce increase in synthesis of factor tumour necrosis alpha and interleukin-6. A 26-year-old Male patient was brought to hospital with complaints of Shortness of Breath -Grade III, ass. with palpitations and dry cough since a week. 2months before current admission He was diagnosed for tuberculosis and began treatment with combination of anti-tubercular treatment. The patient was on multidrug therapy. Multidrug therapy was withdrawn on the day of admission and diagnosis of ATT induced DEEP VEIN THROMBOSIS was made based on history of intake of anti-tubercular treatment, followed by impression of thrombosis.

KEYWORDS : Anti Tubercular drug, Deep vein thrombosis,

INTRODUCTION

India reports about 9 million cases per year out of which 1.3-5% are associated with deep vein thrombosis (DVT).

Deep vein thrombosis (DVT) is a serious condition that occurs when a blood clot forms in a vein located deep inside our body. A blood clot is a clump of blood that turns into solid state (Mary cushman 2007)

Venous thrombosis, comprising deep vein thrombosis (DVT) and pulmonary embolism (PE), occurs with an incidence of approximately 1 per 1000 annually in adult populations. Rates are slightly higher in men than women. About two-thirds of episodes manifest as DVT and one-third as PE with or without DVT (H Kouismi 2013)

Tuberculosis may induce at the peripheral blood - activation of mononuclear cells, and the interaction of these cells activated with mycobacterial products will induce increase in synthesis of factor tumour necrosis alpha and interleukin-6 (O. Turken 2002)

Case report:

A 26-year-old Male patient was brought to hospital with complaints of Shortness of Breath -Grade III, ass. with palpitations and dry cough since a week. 2months before current admission He was diagnosed for tuberculosis and began treatment with combination of anti-tubercular treatment (rifampicin 150mg, isoniazid 75mg, ethambutol 275mg, pyrazinamide 400mg).

Vitals on admission were BP: 110/80mmHg, PR: 130/min, SPO2:88% with RA, Currently on CPAP with Fio2:100%, Physical examination revealed Pallor +, B/L Pedal edema +(L>R), Facial Puffiness +.

Laboratory evaluation revealed- Blood urea: 43.53mg, Sr. Creatinine: 1.27mg, ALP: 122.6IU/L, TSB: 0.95mg/dl, Albumin: 2.66g/dl, Proteins: 5.56g/dl, GRBS:135mg/dl.

His liver function tests were normal with decreased levels of albumin and increased levels of blood urea. Colour Doppler Study: Impression: Chronic Partial thrombosis in Left CFV, SFV, left external iliac vein, popliteal vein, tibial vein.

Well Score :06

The patient was on multidrug therapy. Multidrug therapy was withdrawn on the day of admission and prescribed with Inj. Augmentin

1200mg thrice a day, Inj. UFH 18 IU/kg/hour, Tab. Apixaban 2.5mg twice a day, and other topical applications for symptomatic relief.

A diagnosis of ATT induced DEEP VEIN THROMBOSIS was made based on history of intake of anti-tubercular treatment, followed by impression of thrombosis.

After 25 days of complete treatment patient was discharged with following Prescription:

3. Tab. Rifampicin 150mg
4. Tab. Ethambutol 275mg
5. Tab. Pyrazinamide 400mg
6. Tab. Apixaban 2.5mg

DISCUSSION

Association of deep vein thrombosis with tuberculosis infection is seen in about 1.5% - 3.4% of the infection cases reported. The treatment is generally accompanied with anti-coagulants to decrease the risk of deep vein thrombosis (Binal Nithin Lodaria 2020)

General mechanism associated with it may be the haemostatic disturbances caused by severe infection. Decreased antithrombin III levels and protein C levels along with the elevated plasma fibrinogen and platelet aggregation levels might be the reason behind DVT in patients (Jayesh Sangani 2015)

Various studies conclude that significant increase in the level of plasma fibrinogen, along with impaired fibrinolysis associated with a decrease in factor antithrombin III, protein C and platelet aggregation. These contribute to induce a hypercoagulable state promoting the development of DVT. Cytokines activates the vascular intima and make thrombogenic endothelium. There by leading to a stimulation of hepatic synthesis of coagulation proteins.

First line anti tubercular drug – Rifampin may also increase the risk of developing DVT in patients. Since rifampin is an enzyme inducer it may cause disturbances in the coagulation and anti-coagulation proteins there by increasing the risk of DVT (H Kouismi 2013)

Use of rifampicin in the treatment regimen relatively increased the risk of developing DVT by 4.74% in the patients. A possible mechanism behind developing venous thrombosis might be due to enlarged lymph nodes where the haemostatic disturbances do not play any role. In such cases controlling DVT might be difficult (O. Turken 2002).

CONCLUSION

In patients diagnosed with pulmonary tuberculosis, early immobilization and physiotherapy can prevent the development of DVT

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