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Deep venous thrombosis during tuberculosis cases of 19-year guinean

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Abstract

Observation: This is I S S 19 years admitted pupil for pain of the lower right limb, swelling of the right lower limb, fever and physical asthenia under anti tuberculosis treatment. At the heart: Regular heart sounds at 105bpm, no added noise, blood pressure 110 / 70mmhg. There is a red, feverish swelling in the lower left limb with a positive Homans sign with no detectable entryway. Temperature 38.70c. The rest of the exam is peculiar. Treatment: Lovenox 0.6UI, Sintrom 4mg, Tramadol 50mg.

Conclusion: Tuberculosis is a factor favoring the occurrence of venous thromboembolic disease. Any time the anticoagulant treatment should be started as soon as the signs of MTVE appear preferably heparinotherapy. In the future the scientific world should floor on the interaction Rifampicin and anti vitamin K in order to find an alternative apart from heparin therapy for the purpose of achieving the therapeutic target whenever tuberculosis is associated with thromboembolic disease.

Keywords: Young, Thrombosis, TB

Introduction

In 2011, 8.7 million new cases were identified by the World Health Organization and TB is responsible for 1.4 million deaths per year [1]. Tuberculosis is considered a risk factor for thromboembolic venous diseases (VTE). The prevalence of this association varies between 0.6% and 10% [2]. Some authors have estimated that the actual prevalence of VTE may be greater than 10% but it is unknown in two-thirds of cases [3].

Observation

This is I S S 19 years admitted pupil for pain of the right lower limb, swelling of the right lower limb, fever and physical asthenia under anti tuberculosis treatment. At the heart: Regular heart sounds at 105bpm, no added noise, blood pressure 110 / 70mmhg. There is a red, feverish swelling in the lower left limb with a positive Homans sign with no detectable entryway. Temperature 38.70c. The rest of the exam is peculiar. Treatment: lovenox 0.6UI, Sintrom 4mg, Tramadol 50mg.

Result of the examinations carried out

Electrocardiogram: normal appearance.

Facial Telethorax: Lung opacities in the cavern located in the right upper lobe.

Venous doppler echo of the lower limbs: shows the presence of an acute deep veinous thrombosis extended from the sural vein extended to the popliteal vein and the superficial femoral vein with thrombus head at the level of the right iliac vein.

Biology: THB at 13g/l; VS=50mm; TP=49%; 1st INR=1.61; 2nd INR=1.50; Coagulation time=17 seconds, negative SRV; Platelet count=600000/mm^3 (**Figures 1-4**).

Discussion

We report the case of a young man of 19 years in whom deep vein thrombosis of the lower right limb was diagnosed on pulmonary tuberculosis under anti tuberculosis treatment.

As early as 1856, R. Virchow described in his triad the mechanisms that can contribute to the formation of a thrombus: the

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alteration of coagulation, the lesion of the vascular wall and the venous stasis, these 3 mechanisms being often entangled. Tuberculosis acts at these 3 levels. Indeed, during TBC several mechanisms can induce a state of blood hypercoagulability causing thromboembolic complications. Tuberculosis, in its various localizations, can induce in the blood an activation of the mononuclear cells whose interaction with Mycobacterium tuberculosis induces an increased synthesis of the interleukin 6 (IL6), the interleukin 1 and the tumor necrotif alfa factor [**4**] IL6 stimulates the synthesis of proteins of the acute phase of inflammation including fibrinogen and C4bBP complexing protein S and factor VIII, which promotes a procoagulant state [**6**]. Some studies have shown that during the course of tuberculosis the platelet count was increased with not only a decrease in their size, but in addition increase in their aggregability [**7**,**8**,**9**]. In our case the platelet count was 600mil / mm3. Data from the literature remains controversial regarding the role of rifampicin in the genesis of [**10**]. Thromboembolic disease Our patient has been on anti-tuberculosis treatment for 2 months and the local signs of deep vein thrombosis have appeared 2 after the start of treatment. The administration of rifampicin is accompanied by a proliferation of the endoplasmic reticulum of the hepatocyte with an enzymatic induction of cytochrome P450, this induction can alter the balance of coagulant and anticoagulant proteins synthesized by the liver thus favoring a state of hypercoagulability [**11**]. From the therapeutic point of view, although the rifampicin and anti-vitamin K interaction is well demonstrated, no consensus was established, despite the difficulties encountered in the management of the two associated pathologies. Rifampicin, a bactericidal anti-infective agent used primarily as an antituberculous agent, has a potent cytochrome P450 enzymatic inducing potential. Interactions between rifampicin and anti-vitamin K are described, with rifampicin inducing an accelerated catabolism of antis vitamin K and thus a decrease in the efficacy of oral anticoagulants [**12**]. For the present case the patient is under acenocoumarol an anti vitamin K (Sintrom 6mg per day). Low molecular weight heparins may be a good alternative; but their high cost is a cause of poor compliance of patients with treatment and therefore of stopping it. Due to the high cost of heparin therapy, the patient received only 3 days of heparin therapy of the type Enoxaparin 0.6 IU twice daily. Despite this anticoagulant treatment the therapeutic range that is between 2 and 3 for MVTE has not been reached. Failure to reach the therapeutic target is justified not only by the coagulation effect of tuberculosis but also by the action of rifampicin on the reduction of anticoagulant efficacy of anti-vitamin K. Local signs have completely disappeared within a week. anticoagulant treatment.

Conclusion

Tuberculosis is a factor that promotes the occurrence of venous thromboembolic disease. Any time the anticoagulant treatment should be started as soon as the signs of MTVE appear preferably heparinotherapy. In the future the scientific world should floor on the interaction Rifampicin and anti vitamin K in order to find an alternative apart from heparin therapy for the purpose of achieving the therapeutic target whenever tuberculosis is associated with thromboembolic disease.

Figure 1. Before treatment.

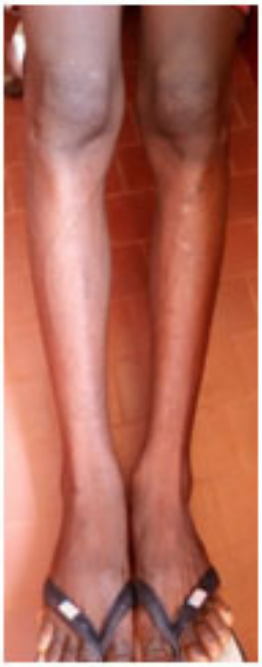

Figure 2. One week of treatment.

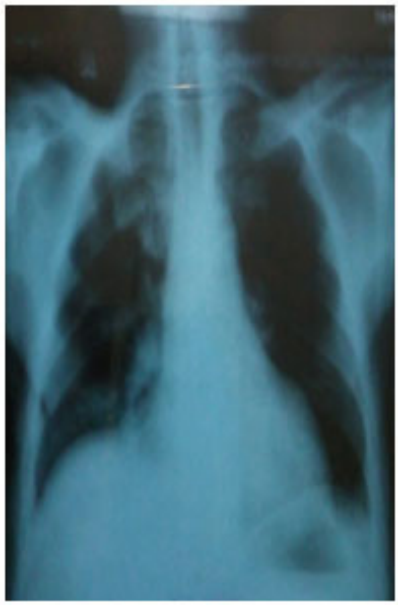

Figure 3. Tele-thorax on the face showing cave opacities located in the right upper lobe.

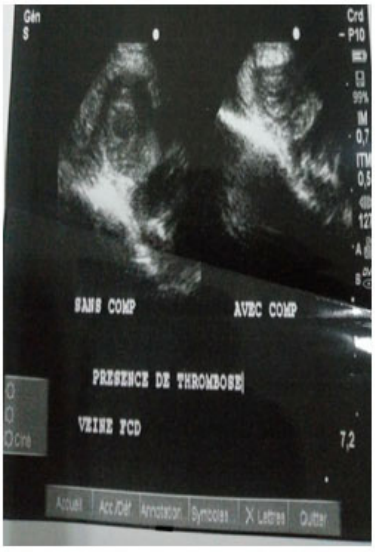

Figure 4. Venous doppler echo shows the presence of acute extensive deep vein thrombosis of the sural vein extended to the popliteal vein and the superficial femoral vein with thrombus head at the level of the right iliac vein.

Competing interests
The authors declare that they have no competing interests.

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