ASSOCIATION DEEP VEINOUS THROMBOSIS WITH PULMONARY TUBERCULOSIS

Leila El Fekih, Imen Oueslati, Hela Hassene, Souraya Fenniche, Dalenda Belhabib, Mohamed Lamine Megdiche.

Service de pneumologie Ibn Nafiss. Hôpital Abderrhmane MAMI de L'ARIANA

L. El Fekih, I. Oueslati, H. Hassene, S. Fenniche, D. Belhabib, M. L. Megdiche.

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RÉSUMÉ

Pré-requis : La tuberculose pulmonaire est considérée comme un facteur de risque de maladies veineuses thromboemboliques.

But : Le but de ce travail est de rappeler les particularités physiopathologiques, épidémiologiques, cliniques et thérapeutiques de l'association tuberculose pulmonaire et thromboses veineuses profondes.

Méthodes: Il s'agit d'une étude rétrospective colligeant tous les dossiers de tuberculeux hospitalisés dans notre service entre Janvier 2000 et Décembre 2007 et ayant développé une thrombose veineuse profonde au cours de leur hospitalisation.

Résultats: Il s'agit de 14 hommes d'un age moyen de 40 ans. La tuberculose pulmonaire était confirmée par la présence du bacille acido-alcoolo-résistant à l'examen direct des crachats dans 12 cas (81, 8%) et par la fibro aspiration dans 2 cas (18%).La phlébite s'est constituée en moyenne dans les vingt jours suivant le diagnostic de la tuberculose et était confirmée par une échographie doppler veineuse des membres inférieurs. Tous nos patients étaient sous traitement antituberculeux associé à des anticoagulants. Le bilan étiologique a montré la présence d'anticorps antiphospholipides chez un malade et un déficit en protéines S et C chez 2 malades ayant en plus une embolie pulmonaire. Nous avons eu des difficultés à contrôler le taux de prothrombine chez 4 malades et nous avons gardé un patient sous héparine de bas poids moléculaire pendant 6 mois.

Conclusion : La maladie thrombo embolique est à rechercher systématiquement chez les tuberculeux vu le risque de survenue de cette complication en particulier dans les formes étendues et sévères. Le traitement anticoagulant prophylactique trouverait ses indications dans ces formes.

MOTS-CLÉS

Tuberculose, thrombose, anticoagulant, traitement.

SUMMARY

Background : Pulmonary tuberculosis has been reported as a risk factor for deep venous thrombosis.

Aim: In the present study we reported, physiopathological, epidemiological, clinical and therapeutic features of the association of deep venous thrombosis and pulmonary tuberculosis.

Methods: This is a retrospective study done in our department between January 2000 and December 2007 .It is about 14 cases of confirmed pulmonary tuberculosis associated with deep venous thrombosis.

Results: It is about 14 men. The mean age was 40 years old. Pulmonary tuberculosis was confirmed by the presence of acido-alcoolo-resistant bacillus on the sputum at direct exam in 12 cases (81, 8%) and in the bronchial aspiration in 2 cases (18%). Phlebitis occurred within a mean of 20 days after the diagnosis of tuberculosis. It was confirmed by doppler deep venous ultrasound of inferior members. All patients received anti-tuberculosis drugs in association with anticoagulant treatment. Etiologic investigations showed positive anti-phospholipids antibodies in one case, and decrease in C and S proteins for 2 patients in which phlebitis was complicated by arterial pulmonary embolism. We had difficulties for controlling prothrombin level in 4 cases and we must prescribe low molecular weight heparin for 6 months in one case.

Conclusion: A lot of attention should be done, in the follow up of pulmonary tuberculosis especially in severe presentation; because of the deep venous thrombosis's risk occurrence. Prophylactic anticoagulant treatment should be done in some cases, when the risk is higher.

KEY-WORDS

hereditary hemolytic anemia, splenectomy,transfusion, complications

من التخثر الأوردة العميقة والإصابة بمرض السل الرئوي

الباحثون : الفقيه. ل - وسلاتي . إ - حسان . هـ - فنيش . ث - بالحبيب . د - مقديش . م . ل.

الهدف من هذه الدراسة هو التذكير بالخصائص الفيزيولوجية المرضية والوبائية والسريرية والعلاجية لتزامن مرض السل الرئوي مع تخثر الأوردة العميقة . آستملت دراستنا على 14 مريضا خضع كل مرضان إلى علاج يتمثل في مضادات لمرض السل ومضادات للتخثر . نستنتج أنه يجب البحث عن الإصابة بالتخثر عند كل المرضى المصابين بالسل ويمثل العلاج بمضادات التخثر الوقائي أحسن الدواعي.

الكلمات الأساسية : مرض السل - تخثر - مضادات التخثر علاج

Vascular complications associated with mycobacterium tuberculosis infection had been reported in the literature and occurred in 1, 5 to 3, 4 % of tuberculosis infection (1).

Deep venous thrombosis occurs in pulmonary tuberculosis because of hypercoagubility. Other hypothesis are evoked this study aimed to report clinical features and therapeutic

management of the association deep venous thrombosis with pulmonary tuberculosis..

PATIENTS AND METHODS

This is a retrospective study done in our department between January 2000 and December 2007. It is about 14 cases of confirmed pulmonary tuberculosis associated with deep venous thrombosis.

RESULTS

The average was 40 years old with extremely going to 32 from 62 years. In our study, all patients are male. Three of them had antecedent history of pulmonary tuberculosis. In the current episode, pulmonary tuberculosis was confirmed by the presence of acido-alcoolo-resistant bacillus on the sputum at direct exam in 12 cases (81, 8%) and in the bronchial aspiration in 2 cases (18%). Chest x ray showed an extensive disease, with bilateral lesions in 5 cases. All patients received Isoniazid, rifampicin, pirazinamid and streptomycin. Phlebitis occurred within a mean of 20 days with extremely going to 1 from 60 days of the diagnosis of pulmonary tuberculosis. It was revealed by clinic signs and high level of dimmers and then confirmed by doppler deep venous ultrasound of inferior members. In 2 cases, phlebitis was complicated by arterial pulmonary embolism.
No patient had signs evocating Behçet syndrome or systemic disease. Etiological investigations (level of fibrinogen, protein S, protein C, level of antithrombinIII, level of antiphospholipid antibody) had be done in the four youngest patients and in two cases in front of non extensive lesions. These investigations done before starting anticoagulant treatment showed positive anti-phospholipids antibodies in one case, and defect in C and S proteins in 2 cases whose phlebitis was complicated by arterial pulmonary embolism. For the other patients, no abnormality had notified at the clinical exam or biological investigation. Oral anticoagulant drugs were associated with low molecular weight heparin treatment after a mean of 4 days. Low molecular weight heparin was stopped when a monitoring of the prothrombin level was correct within a mean of 10 days. In all cases Streptomycin was stopped and replaced by Ethambutol.
Well clinical, bacteriological and radiological evolutions were noted in the term of six months for pulmonary tuberculosis.
We had difficulties for controlling prothrombin level in 4 cases and we must prescribe low molecular weight heparin for 6 months in one case. In 11 cases oral anticoagulant treatment was stopped at the term of 3 months; in the 3 other cases, oral anticoagulant treatment was prescribed in long term without recurrence of any thrombosis.

DISCUSSION

Severe pulmonary tuberculosis is often complicated by deep venous thrombosis, because of the association between inflammation and haemostatic changes that can result in a hypercoagulable state. The factors commonly associated with the pathogenesis of thrombosis are: alteration in the wall of the vein, alteration in the blood constituents and slowing of the blood stream. No intimal changes had been described in tuberculosis except in those relatively infrequent instances in witch the disease process actually involves the wall of the vein(1). Tuberculosis has several mechanisms that induce a hypercoagulable state and can lead thromboembolic complications. Different studies concluded that elevated plasma fibrinogen level, with impaired fibrinolysis coupled with a decrease in thrombin III, protein C and increased platelet aggregation appear to induce a hypercoagulable state to favour the development of deep venous thrombosis in pulmonary tuberculosis (2, 3).Two patients were presented this problem.
Some authors mentioned the high frequency of antiphospholipid antibodies detected in the tuberculosis as we found in one case of our patients, and the potential relationship between these and defect on protein S. Although the studies on prothrombin activity in tuberculosis are not many, they indicate that hypoprothrombinemia rather than prothrombin hyperactivity exists in appreciable number of cases. Different studies indicate that a prothrombin deficiency occurs in about one-third of tuberculosis patients (4, 5) Of all the factors involved in the production of venous thrombosis, circulatory stasis has been given greatest emphasis. It has been demonstrated that the reflex inhibition of respiration will temporarily obstruct the return flow of blood to the heart. Although the mechanics of respiration in many tuberculosis patients are hampered by collapse measures, this is probably more than offset by their vigorous coughing (4, 6). The act of coughing is followed by deep inspiration; repetition of this act several times daily with its increase in the negativity of intrathoracic pressure furthers the flow of blood from the periphery to the heart. Although these data, the physiopathology mechanism is still not yet clear and other tuberculosis inflammation mediators as TNF· could be involved in deep venous thrombosis occurrence.The difficulty of management of this association is due to interference between anti-tuberculosis and oral anti-coagulant drugs because of enzymatic induction Rifampicin, resulting in difficult anticoagulant level adjustment and prolonged hospitalisation (6).

CONCLUSION

A lot of attention should be done, in the follow up of pulmonary tuberculosis especially in severe presentation, because of the deep vein thrombosis's risk occurrence. Prophylactic anticoagulant treatment should be use in some cases, when the risk is higher. A close monitoring of prothrombin level in necessary with the oral anticoagulant when Rifampicin is associated.

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