Respiratory Medicine Case Reports 26 (2019) 98–100

Contents lists available at ScienceDirect

Respiratory Medicine Case Reports

journal homepage: www.elsevier.com/locate/rmcr

Case report

Lethal left cardiac thromboembolism in infant: A rare complication of pulmonary tuberculosis

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ARTICLE INFO

Keywords:
Tuberculosis
Venous thromboembolism
Pulmonary embolism
Deep vein thrombosis

ABSTRACT

If acute or severe infectious are know that risk factors for venous thromboembolism (VTE), the role active tuberculosis is ill defined, despite different case reports and many small case series have proposed an association between VTE and chronic infectious such as tuberculosis. We are highlighting an exceptional phenomenon correlation between intra-cardiac thromboembolism and pulmonary tuberculosis and concluded that active tuberculosis should include in the physician's evaluation of intra-cardiac thromboembolism risk.

1. Introduction

Several risk factors are known for venous thromboembolism (VTE) and intra-cardiac thromboembolism [1] but the role for chronic infections such as tuberculosis infectious is less clear in the mechanism of thrombus formation. There have been reports of intra-cardiac thrombus related to pleuro-pulmonary tuberculosis in non immunocompromised infant and clarify the etiopathogenesis association between these two entities.

2. Case report

A 15-year-female presented in our department with complaints of low-grade fever, swelling and pain in right hemithorax since one week and sudden onset of breathlessness of one month duration. She had a 10 kg weight loss over a 2-month period. The patient had been on anti-TB therapy (ATT) initiated two weeks prior hospitalization.

On admission, the patient presented an acute respiratory distress and she was found to be tachycardiac, tachypneic, hypotensive and febrile. On examination, she was a right-sided pleural effusion syndrome. Rest of the systemic examination was unremarkable. A 12-lead ECG revealed sinus tachycardia with lateral ST segment elevation. Transthoracic echo was done which revealed a large mobile left ventricular thrombus with a size of 40,8 × 22,9 mm (Fig. 1). Enhancement computed tomography (CT) imaging showed a left ventricular thrombus (Fig. 2).

Bacteriology examination of broncho-alveolar liquid confirmed the presence of acid fast bacilli. Results of percutaneous needle pleural biopsy confirmed tuberculosis. Cytology examination of pleural fluid revealed lymphocyte predominant. Coagulability workup returned negative protein C, protein S, antithrombin and factor V deficiency. Antituberculous therapy was started Isoniazid (300 mg/day), rifampicin (600 mg/day), pyrazinamide (1000 mg/day) and ethambutol (800 mg/day). Simultaneously, the patient was treated with low-molecular-weight heparin and 3 days later, warfarin was started with a target International Normalised Ratio (INR) of 2.0–3.0. Antituberculous therapy was going on. Even well-managed treatment, the patient remained persistently a cardigenic shock syndrome, and on day 5 of admission she developed ventricular fibrillation unresponsive. However, the patient did not respond to resuscitation measures and was expired.

3. Discussion

Our case shows that intra-cardiac thromboembolism may complicate severe pulmonary tuberculosis. Pulmonary tuberculosis is very prevalent in developing countries as Madagascar but its thrombogenic potential is a rare entity [2]. Haemostatic complications are very rare and thrombogenic potential of tuberculosis is not frequently

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https://doi.org/10.1016/j.rmcr.2018.11.016
Received 10 November 2018; Accepted 23 November 2018

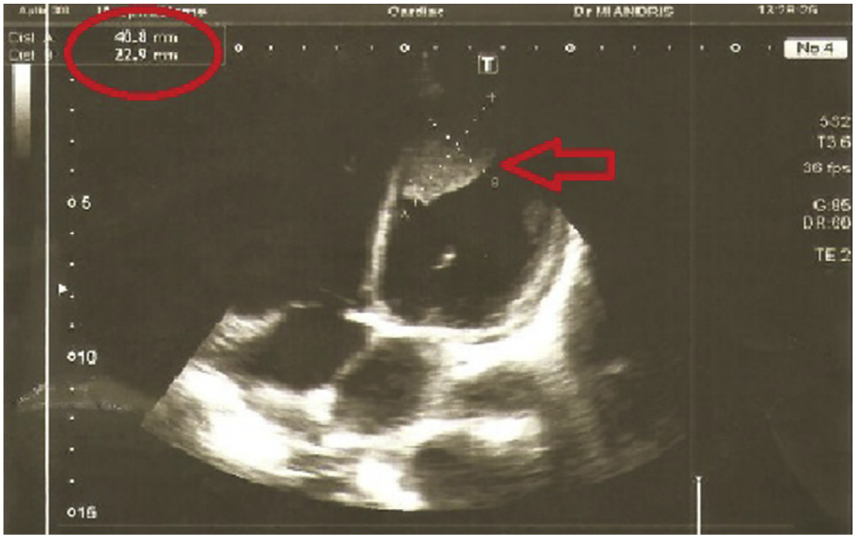

Fig. 1. Transthoracic echo was done which revealed a large mobile left ventricular thrombus with a size of 40, 8 × 22, 9 mm and showing miliary tuberculosis.

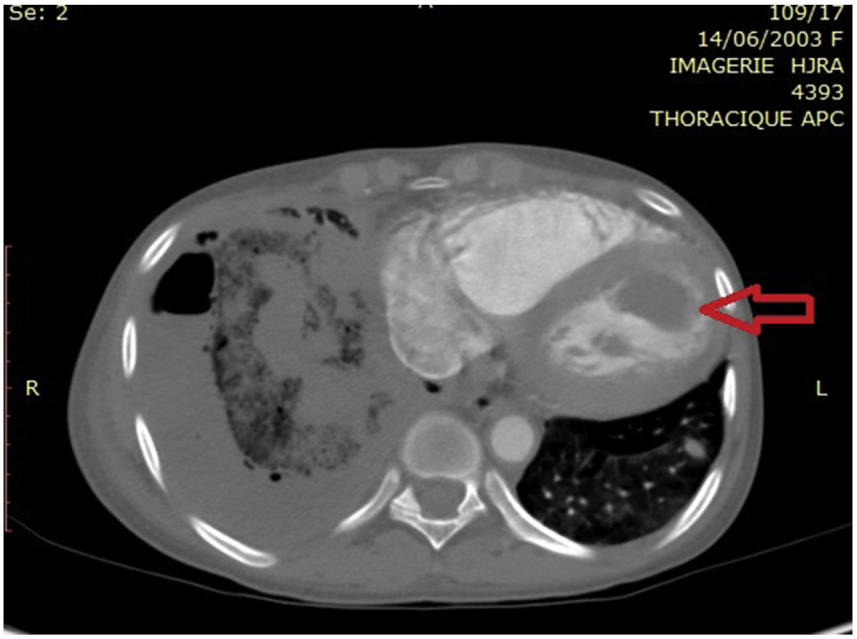

Fig. 2. Enhancement computed tomography (CT) imaging showed a left ventricular thrombus.

documented in literature, especially with reference to intracardiac thrombus. Classically, Deep Vein Thrombosis (DVT) are more common in patients with pulmonary tuberculosis. Other unusual sites have been reported in the literature specifically splenic vein thrombosis [3], pulmonary venous thrombosis with atrial extension [4], left Ventricular Thrombus similar to our case [5]. DVT is clinically observed and can be confirmed with laboratory methods in 3–4% of patients with pulmonary tuberculosis [2]. It has been established that severe tuberculosis can be responsible for a transitory hypercoagulable state [1].

Firstly, Deficiency of Antithrombin III, protein C and protein S and elevated plasma fibrinogen levels, increased platelet aggregation seems to induce hypercoagulable state [2]. On the other hand, local compression of veins by the enlarged reactive lymph nodes and injury to endothelium can lead to thromboembolic complication in tuberculosis [6]. All these factors described are not present and no other predisposing cause of DVT was found in our case.

The pathophysiological then leading to the formation of the left ventricular thrombus include:

(a) Extension of pulmonary venous thrombosis to the left ventricular
(b) Direct endocardial tissue injury caused by Koch's bacillus (Endocardial miliary tubercles) and alteration in the normal blood flow secondary to disseminated tuberculosis infection. Cardiac tuberculosis was recognized and described by Laennec in 1826, assigning the heart as the 13th organ affected in order of frequency [7].
(c) Or the use of rifampicin [6,8].

A multivariate analysis to assess the specific VTE risk associated with tuberculosis concluded that tuberculosis must be considered as a pertinent risk factor for VTE and should be included in thromboembolism risk evaluation similar to any acute and severe infection (odds ratio, 1.55 [95% CI, 1.23–1.97], $P < .001$) [1]. Turken et al. showed that patients with active pulmonary tuberculosis displayed thrombocytosis and increased fibrinogen, factor VIII, and plasminogen activator inhibitor 1 plasma levels, associated with depressed antithrombin III and protein C levels in the first month of treatment, resulting in

activated coagulation and inhibited fibrinolysis [9].

4. Conclusion

In summary, association between pulmonary tuberculosis and Intracardiac thrombus is rare condition in clinical practice. This exceptional association should be in mind of clinicians among haematological tuberculosis complications which in theory improve with antitubercular drugs and avoid with anticoagulant drugs on the prevention. Rapid action should be taken to ensure a timely, proper management.

Competing interests

None.

Acknowledgement

The author is very grateful to Razafimanjato A. Esteban for his help in preparing this article.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.rmcr.2018.11.016.

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