IDCases 20 (2020) e00751

Contents lists available at ScienceDirect

IDCases

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

Case report

Pulmonary thromboembolism – A rare complication in a scrub typhus infection

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ARTICLE INFO

Article history:
Received 21 March 2020
Accepted 21 March 2020

Keywords:
Scrub typhus
Vasculitis
Pulmonary thromboembolism
Endothelial dysfunction

ABSTRACT

We describe a rare haematological complication of pulmonary thromboembolism in a scrub typhus infection. There are very few case reports on scrub typhus infection causing deep venous thrombosis. Vasculitis and perivasculitis with endothelial dysfunction could be considered as the main pathologic mechanism causing this complication.

Introduction

Scrub typhus is a common re-emerging, acute febrile infectious disease, caused by Orientia tsutsugamushi, transmitted by bite of chiggers of thrombiculid mite, maintained in the environment by trans-ovarian and trans-stadial transmission in the mite's life cycle [1]. Orientia tsutsugamushi, is an obligate intracellular, gram negative bacteria, infecting endothelium and causing generalized vasculitis.

Scrub typhus has a latent period of 5–20 days, whose symptoms vary from mild to severe and include general malaise, fever, headache, rash and lymphadenopathy [2]. Severe scrub typhus manifestation includes: pulmonary edema, interstitial pneumonia, meningoencephalitis, glomerulonephritis, acute renal failure, hypotensive shock and coagulopathy. This report describes a case of scrub typhus with pulmonary thromboembolism.

Case report

A 10 years old healthy female patient for fever 14 days back treated at local hospital with azithromycin and injection ceftriaxone and was discharged, she got admitted this time with right sided lower lateral chest pain radiating to back, left sided neck pain, progressive dyspnea and haemoptysis since 3 days before admission. On admission, blood pressure: 108/60 mmHg, axillary temperature: 36.6 °C, heart rate: 120/ min and respiratory rate: 28/min.

There were no cervical lymph nodes palpable. Left external jugular vein was palpable and tender. Engorged superficial veins were seen over left upper part of anterior chest wall [Fig. 1]. On auscultation, right side air entry was diminished in axillary and infra-axillary area, S1 S2 normal, no murmur. Laboratory data revealed white cell count of 11,800/cumm with 49 % neutrophils, 40 % lymphocytes and platelet count 2.58 lakh/cumm. Erythrocyte sedimentation rate [ESR- 70 mm/hr] and C-reactive protein [CRP-10.8 mg/dl] were elevated. Base line investigations like liver function test, renal function test, arterial blood gas analysis and serum electrolytes were normal. Chest radiograph showed peripherally located wedge shaped opacity on the right side [Fig. 2]. ECG revealed sinus tachycardia [118/min] with incomplete right bundle branch block pattern. Montoux test and sputum for acid fast bacilli were negative. Scrub typhus immunoglobulin M [IgM] assay [InBios kit] was strongly positive [OD-1.44, cutoff value > 0.5 is positive]. USG Doppler scan documented absence of flow in left jugular vein and left external jugular vein. 2D ECHO with Doppler study documented thrombus blocking right main pulmonary artery [Fig. 3]. Chest computed tomography revealed microinfarcts with multifocal contrast filling defects in the right sided branch pulmonary arteries. Homocysteine levels, antiphospholipid antibodies [immunoglobulin G{IgG} and IgM], protein C and protein S assay, factor V [Leiden mutation], prothrombin gene mutation analysis were normal. Thrombolysis with intravenous Tenecteplase [100 units/kg], followed by continuous infusion of unfractionated heparin [loading dose: 75 units/kg over 10 min and maintenance dose: 20 units/kg/hour], was overlapped with oral warfarin for 5 days. Symptoms improved and patient was discharged with oral warfarin and is in regular follow-up consultations with INR monitoring.

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https://doi.org/10.1016/j.idcr.2020.e00751

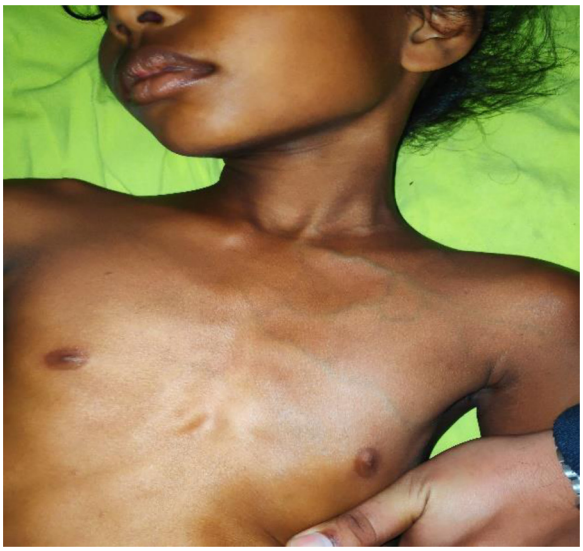

Fig. 1. Engorged veins over anterior chest wall.

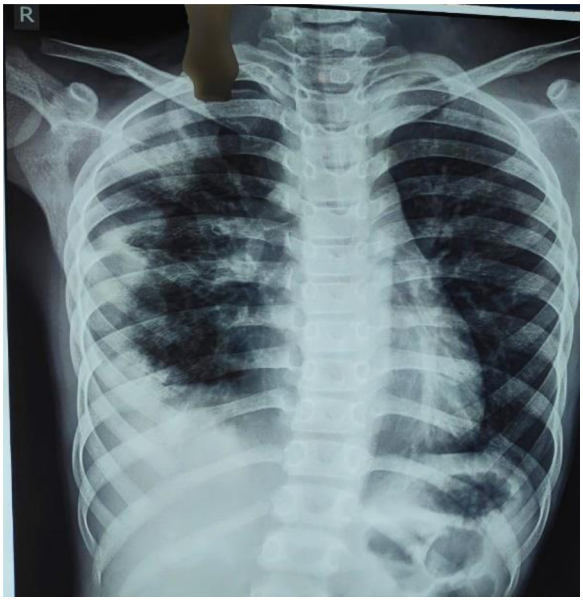

Fig. 2. Wedge shaped opacities on the right side.

Discussion

The proliferation of O, tsusugamushi in endothelial cells of the microvacular system is the main pathologic mechanism causing vasculitis and perivasculitis in scrub typhus infection [3]. This rickettsial vasculitis affects the microvascular system of skin, lungs, liver, kidneys, central nervous system and skeletal and cardiac muscles, which results in the severe clinical manifestations and includes non-cardiogenic pulmonary edema, interstitial pneumonia, acute respiratory distress syndrome, meningoencephalitis, seizures and coma [4].

Damage to the vascular endothelial cells causes perivascular infiltration of leukocytes, endothelial proliferation and microvascular thrombus formation [5]. Researchers have reported that the association between endothelial cell damage and systemic

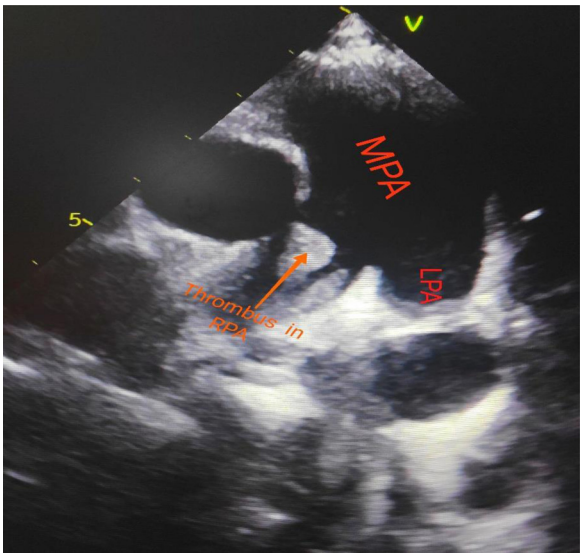

Fig. 3. Thrombus in RPA. Abbreviations: MPA-main pulmonary artery; LPA-left pulmonary artery; RPA-right pulmonary artery.

immune response is related to the pathogenesis of scrub typhus [6]. Paris et al. demonstrated that coagulation and inflammatory markers were increased and anti-coagulant factors were inhibited in patients with scrub typhus [7].

Considering our case, the scrub typhus infection may have affected the pro-coagulability state via inflammation causing jugular venous thrombosis with pulmonary thromboembolism.

Funding

The authors have not declared a specific grant for this research from any funding agency.

Patient consent for publication

Parental/guardian consent obtained.

Declaration of Competing Interest

None.

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