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UNCOMMON COMPLICATION OF A COMMON DISEASE (ENTERIC FEVER)

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A B S T R A C T

A 42year old lady, without any previous comorbidities presented with complaints of fever with chills and rigor for 8 days and loose stools. She was diagnosed with Salmonella paratyphi A septicemia and was treated with appropriate antibiotics. But her clinical condition worsened, and her fever continued. On PET-CT whole body, she was detected to have a pulmonary embolism. Her thrombophilia panel check was negative. She was treated with a therapeutic dose of low molecular weight heparin Enoxaparin and appropriate antibiotics. Thus, we present a rare case of in-situ pulmonary embolism formation following enteric fever in an Indian lady

Keywords: Septicemia; Pulmonary embolism; Thrombophilia; Enoxaparin.

INTRODUCTION:

Enteric fever is a common but serious infectious disease in tropical countries. Salmonella typhi is the most common organism, although Salmonella paratyphi A and B are the emerging organisms [1]. Fever characteristically comes in a stepladder-wise pattern [2]. Common complications of enteric fever include hepatitis, cholecystitis, typhoidal intestinal perforation, anemia, etc.
Here, we present an uncommon complication of Enteric fever.

CASE REPORT:

A 42year old lady, without any previous comorbidities presented with complaints of fever with chills and rigorfor 8 days and loose stools for 1 day.
She was well 8days backwhen she developed a fever which was insidious in onset and gradually progressive in nature. It was initially intermittent in nature but during the presentation was a continuous high grade.It was associated with chills and rigor. There was no history of headache, nausea, vomiting, chest pain, cough, palpitations, urinary symptoms, or leg pain. There is no history of recent vaccination or oral contraceptive pill intake.

On examination, she was alert conscious, and cooperative and was mobilizing in the wards. Heart rate 84/minute, regular in rhythm, normal volume, and palpable peripheral pulses. Respiratory rate 18/minute, regular and thoracoabdominal in nature. Blood Pressure 100/60 mm of Hg. SpO_2 95% in room air. Temperature 101^0 Fahrenheit.Pallor was present. There was no cyanosis, icterus, clubbing,oredema. No lymph node was palpable. Jugular venous pressure was not elevated.

On abdominal examination,the mouth and oral cavity were normal. On inspection, there was no obvious deformity, scar mark, or pulsation. The liver was palpable 3 cm below the right coastal margin in the right mid-clavicular line and the margins were palpable, the surface was smooth, soft inconsistency, no tenderness, moving up and down with respiration, liver span 18 cm. The spleen was palpable 2 cm below the left coastal margin in thesplenic axis, margins were well palpable, surface smooth, soft in consistency, splenic notch was not palpable and moving up and down with respiration. Shifting dullness was present. Peristaltic sounds were normal. Other systemic examinationswere within normal limits.

She underwent all the relevant investigationswhich showed Haemoglobin 9.2gm/dl[13-16 gm/dl], TLC 3800/cu mm [4000-11000/ cu mm], Platelet 1.2lac/cu mm [1.5-4lac/cu mm],ESR 14mm in 1st hour [0 – 30mm in 1st hour],Procalcitonin 3.4 ng/ml [less than 0.5ng/ml], CRP 16.7mg/dl [less than 0.33 mg/dl], Creatinine 0.8mg/dl [0.6-1.3 mg/dl],SGOT 99 U/lit [0 -40 U/lit],SGPT 67 U/lit [0 -40 U/lit], ALP 67 IU/lit [45-145 IU/lit],Ferritin 1096 mg/lit [24-150 mg/lit],LDH 1409 U/lit [140 -280 U/lit], TG 171mg/dl [100 – 150 mg/dl], fibrinogen 241mg/dl [200 -400 mg/dl],Widal test/ Dengue serology/Malarial parasite dual antigen/Scrub typhus IgMall negative. Urine routine and culture were normal. Chest X-ray was normal.A blood culture was sent. The stool infection panel detected Salmonella and the Blood culture showed growth ofSalmonella paratyphiA. Antibiotics were adjustedas per sensitivity and she was continued on IVCefepime. She improved initially with complete resolution ofdiarrhea and fever intensity started to reduce and a spike came down from 99^0F - 100^0F from the initial 102^0F – 104^0F. But her fever never came down below 99^{0F}- Fever of 99^0- 101^0F was persisting although repeat Procalcitonin came down to normal range and CRP came down to almost normal range.Repeat chest X-ray was normal. Repeat blood cultures were negative.ANA and ANCA were negative.COVID 19 RT PCR was negative. Repeat Ferritin was 2000mg/dl. Because ofthe non-resolution of fever,even on sensitive antibiotics and the apparent absence of any other cause she underwent PET-CT whole body which detected Pulmonary embolism in the main right pulmonary artery at the bifurcation, proximal part of the right lower lobe and middle lobe pulmonary artery and their segmental branches.

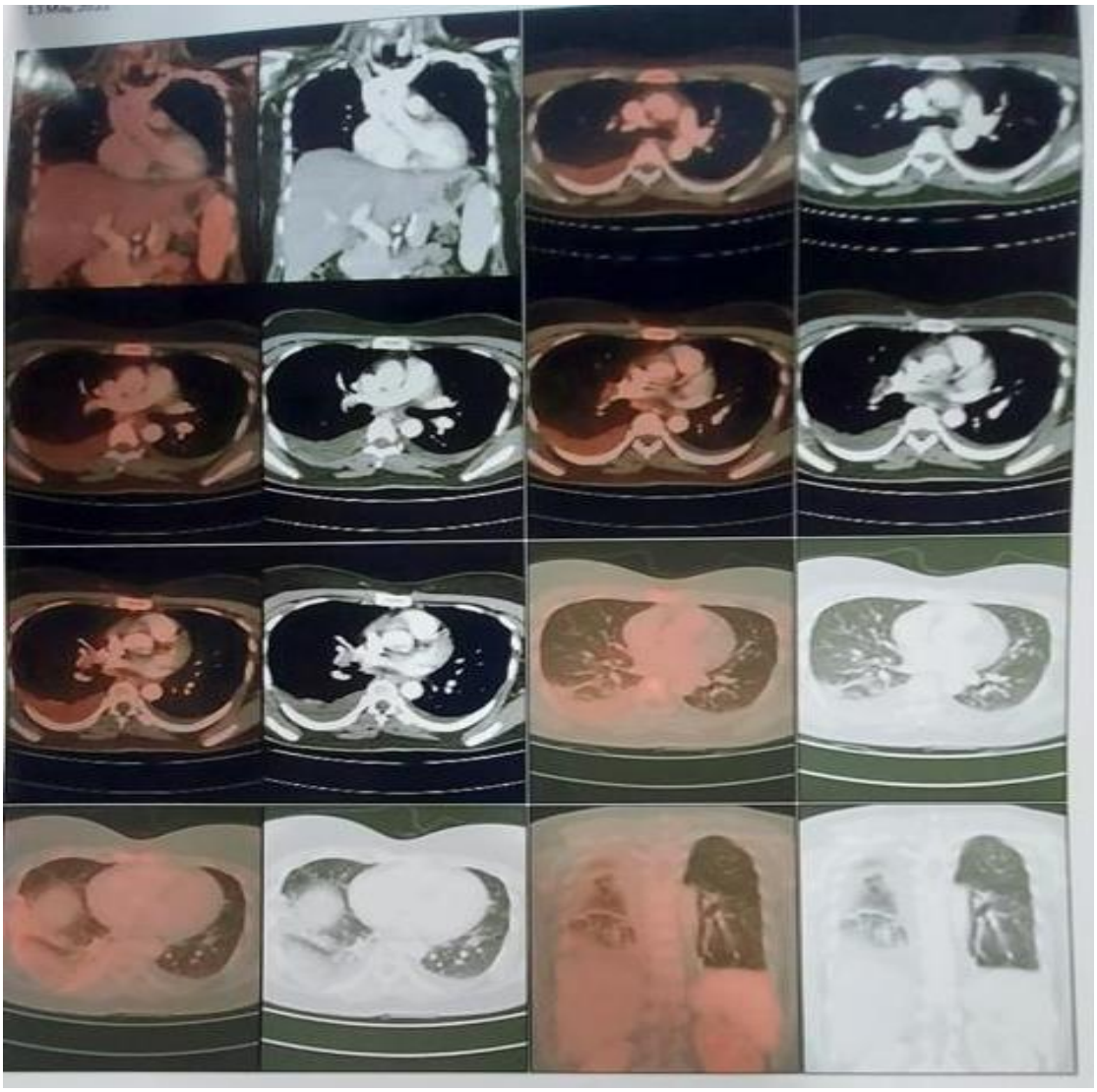

Figure 1

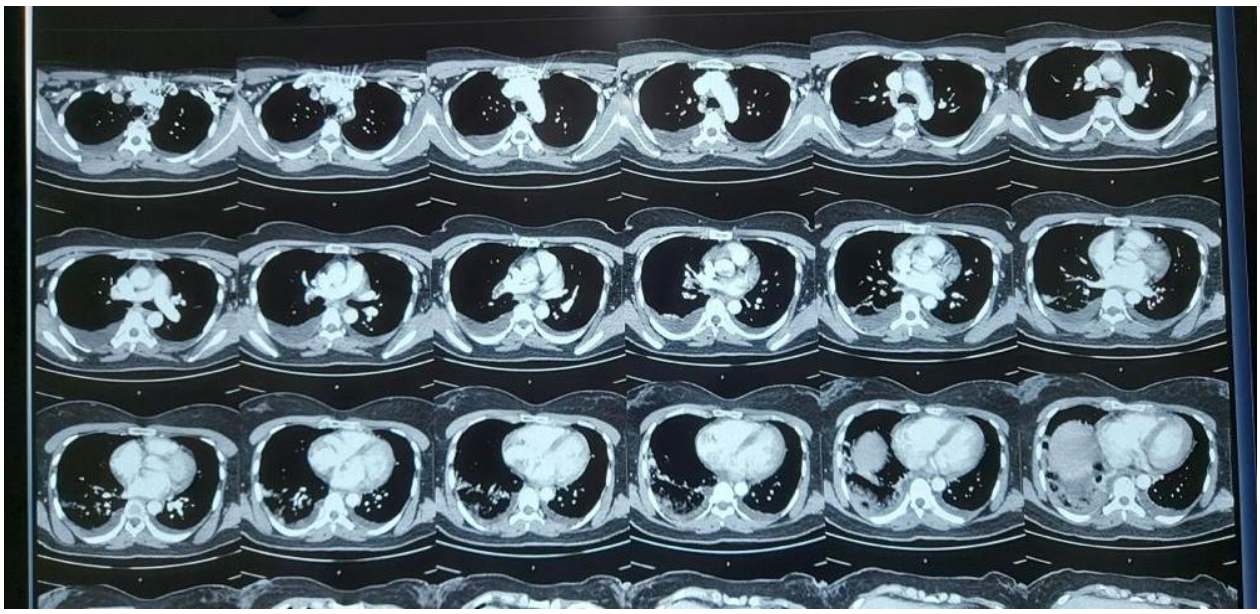

Figure 2

FIGURE 1 & 2: PET-CT WHOLE BODY IMAGES

She was initiated on a therapeutic dose of Low Molecular Weight HeparinEnoxaparin and intravenous antibiotics were continued. She underwent extensive thrombophilia panel checkswhich included: Protein C, Protein S, anti-Thrombin III, von Willebrand factor assay, Factor V Leiden, anti-phospholipid antibody IgG, and IgM, Beta-2GP1 IgG and IgM, Lupus coagulant, APTT, PT and INR which all were normal. She improved and her fever was remitted in 3 days and was discharged on oral anticoagulants. On follow-up after 1 month, she returned to her normal life and is on the oral anticoagulant Apixaban.

DISCUSSION:

Common complications of enteric fever include gastrointestinal perforation and bleeding, pneumonitis, myocarditis, hepatitis cholecystitis, encephalopathy, and shock. Treatment of them requires immediate recognition and treatment, or else they can turn fatal [3]. In a study from Iran by Esmailpour et al [4], it was found that 4.6% of patients with enteric fever can have cardiac complications which include myocarditis, pericarditis, and pulmonary embolism. In another study from India by Dutta et al [5], there were no incidences of cardiovascular complications.

In an extensive study done by Jong et al [6], it has been found that in enteric fever there is an enhancement of coagulation cascade by activation of vascular endothelium, downregulation of anticoagulation pathways, and activation and inhibition of fibrinolysis. Thrombocytopenia was also noted. In the study, Disseminated Intravascular Coagulation was noted in some patients with severe enteric fever. However, the incidence of Pulmonary embolism was not noted.

Pulmonary embolism is caused by inherited or acquired causes of hypercoagulability, is caused by atherosclerotic diseases and rarely idiopathic spontaneous venous thrombosis happens. Common inherited causes include Protein C, Protein S deficiency, von Willebrand factor deficiency, and so on; acquired causes include smoking, trauma, immobilization, oral contraceptive pill intake, and so on [7]. Pathophysiological mechanisms include inflammation, hypercoagulability, and endothelial injury. The gold standard of investigation is CT Pulmonary Angiography [8].

In our case, it might be a case of septic pulmonary embolism,but confirmation could not be possible.

CONCLUSION:

Hence, we report a rare presentation of idiopathicIn-situPulmonary Embolism formation or septic embolismin an Indian lady following an episode of Enteric fever without any other risk factors and was treated with Low Molecular weight Heparin, oral anticoagulant, and prolonged course of intravenous antibiotic.

PATIENT CONSENT: Taken.

CONFLICT OF INTEREST: None.

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