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Case Report

Splenic Findings in A Pediatric Case of Concurrent Mycoplasma Infection and Sickle Cell Anemia: A Case Report

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ABSTRACT

Mycoplasma pneumoniae (MP) is a frequent cause of community-acquired respiratory infections in children and adults, especially in school-aged children. It is also responsible for a wide spectrum of non- pulmonary manifestations including hematological, gastrointestinal, renal, cardiac and central nervous system involvement. Pneumonia in young adults, serum cold hemagglutinins in a titre of 1:64, a positive IgM MP antibody, polymerase chain reaction or effective treatment with macrolides can be helpful for confirming the diagnosis.

Keywords: Mycoplasma pneumonia; Nervous system; Sickle cell anemia; Nebulization

Introduction

We present a case of a 11 year 7-month-old male child who was brought to the hospital with history of moderate to high grade fever (1 spike per day) in the evening which subsided with administration of oral antipyretics. In the inter-febrile period of 14 days there was associated non spasmodic, non-productive cough not severe enough to disturb his sleep. He was admitted to an outside hospital on the second day of illness, where he received IV antibiotics, a pint of packed cells, round the clock nebulization and symptomatic treatment.

However, symptoms persisted and he was shifted to a tertiary care hospital on day 9 of illness and was started on IV antibiotics (Inj Piperacillin tazobactam, Inj Vancomycin) and oral Oseltamivir for 5 days as well as Oral Doxycycline for 3 days along with round the clock nebulization.

His fever and cough reduced in intensity, however as symptoms persisted, he was referred to our hospital.

The patient was admitted and started on Inj. Ceftriaxone and was continued on oral Doxycyxline.

Based on the clinical presentation, a comprehensive radiological evaluation was initiated.

Chest radiograph revealed an ill-defined opacity mid zone **(Figure 1)**.

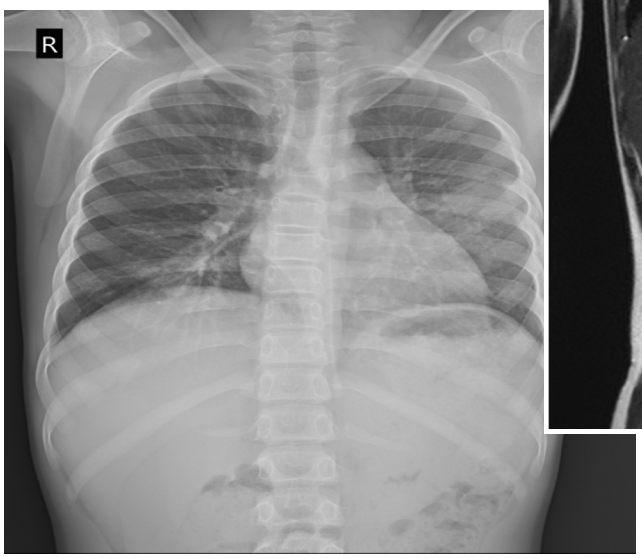


Figure 1: Chest radiograph revealing an ill-defined opacity in the left middle zone (arrow).

Ultrasonography revealed splenomegaly with coarse echotexture of the spleen which showed an overall increase in echotexture. Multiple focal hypoechoic lesions were seen in the spleen. Possibility of an infective or neoplastic lesion was suggested **(Figure 2)**.

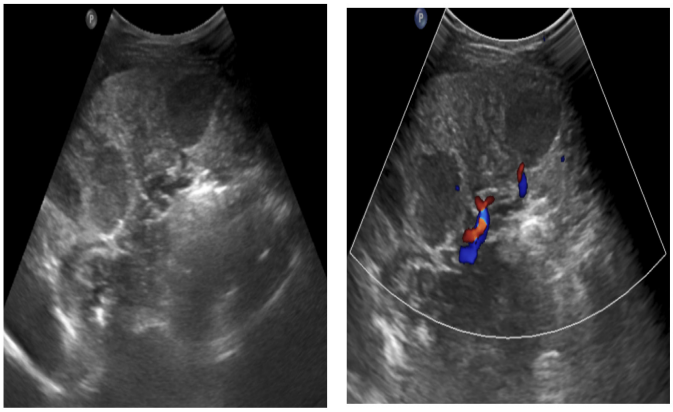


Figure 2: A- USG of spleen showing enlarged spleen showing coarse echotexture and multiple well defined hypoechoic lesions in the spleen
B- Colour Doppler of spleen showing normal splenic artery and lesions not showing vascularity

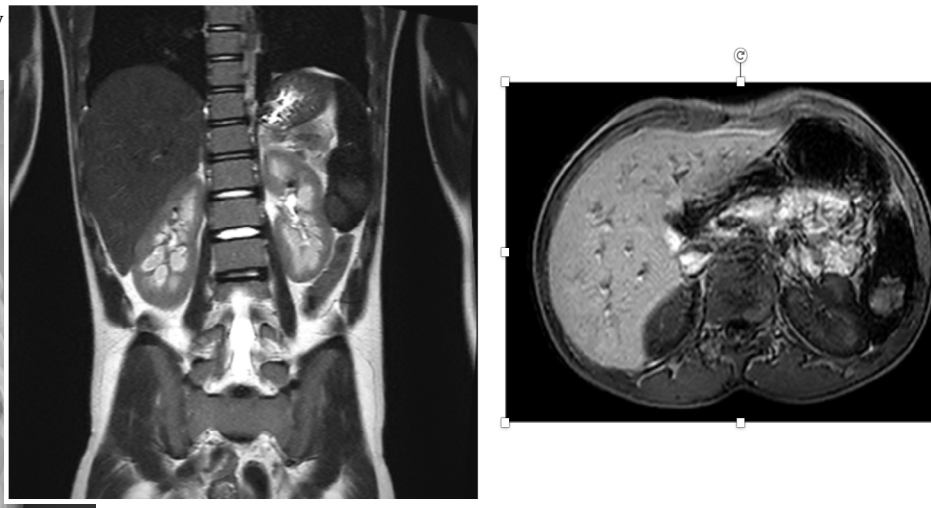
Based on the USG images, MRI was performed and revealed a spleen which was T1 and T2 hypointense with a coarse appearance with presence of multiple well defined minimally hyperintense lesions as compared to the rest of the splenic parenchyma on the T1 and T2 weighted images **(Figure 3)**.

CT done in an outside hospital was suggestive of ground glass opacities in the left upper and right middle lobes and lingula. Consolidation was noted in the right lower lobe and lingula. Mediastinal lymphadenopathy was also noted **(Figure 4)**.

Microbiology revealed raised titers of IgM Mycoplasma (> 27).

Peripheral blood smear revealed sickling of the red blood cells.

Parents' HPLC : positive : heterozygous (HbS = 38.5%) **(Figure 5)**.



(A) **(B)**

Figure 3: A- T2 coronal image showing coarse echotexture of spleen with multiple hyperintense lesions
B- Fat saturated axial T1 weighted image showing no suppression of signal.

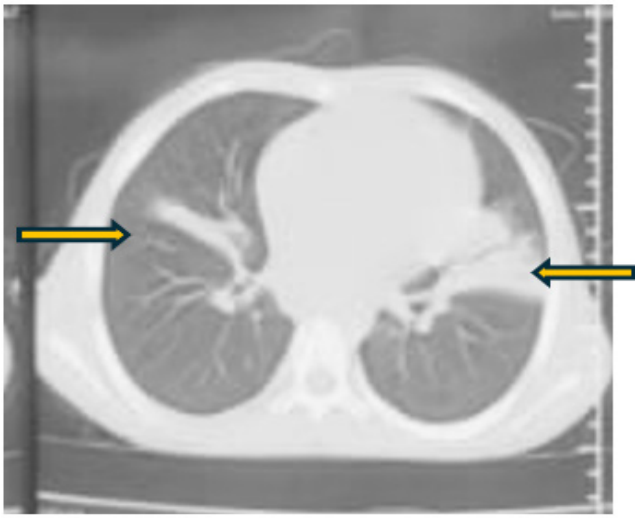


Figure 4: Revealing consolidation surrounded by ground glass opacities in the right middle and left lingula.

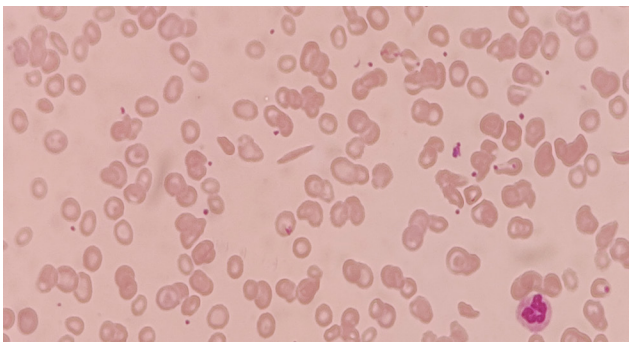


Figure 5: Peripheral blood smear image revealing sickling of the red blood cells

Discussion

Mycoplasma pneumoniae is a common cause of atypical pneumonia in children, typically presenting with mild to moderate respiratory symptoms. However, its course and

complications can be more severe in patients with underlying chronic diseases, such as sickle cell disease (SCD). This case highlights the unique diagnostic and clinical challenges when Mycoplasma infection occurs in a child with SCD.

Children with SCD are predisposed to a range of pulmonary complications, including acute chest syndrome (ACS), pneumonia, and pulmonary infarction[1]. Distinguishing Mycoplasma pneumonia from other causes of lung infiltrates in these patients can be challenging because clinical and radiological features often overlap. ACS, for example, is a leading cause of morbidity and mortality in SCD and is commonly precipitated by infections, fat embolism, or pulmonary infarction. Mycoplasma species have been identified as one of the infectious triggers for ACS, underlining the importance of accurate diagnosis and prompt treatment.

Radiologically, Mycoplasma pneumonia often presents with round pneumonia, patchy, peribronchial or interstitial infiltrates, which may be unilateral or bilateral. However, in children with SCD, differentiating findings of pulmonary infarction from bacterial / round pneumonia can be difficult.

Chest radiographs and high-resolution CT may reveal consolidation, ground-glass opacities, or atelectasis - findings that overlap significantly with other pulmonary complications of SCD. In our patient, imaging demonstrated splenic lesions which, along the diagnosis.

Young children with sickle cell anaemia are at risk for acute splenic sequestration crises[2]. In paediatric sickle cell anaemia patients, the spleen undergoes a continuum of changes. Initially, there can be splenomegaly due to acute splenic sequestration. Repeated episodes of sickling can cause atrophy, fibrosis and ultimately asplenia. The coarse echotexture observed on ultrasound in this patient was suggestive of chronic irreversible changes within the splenic parenchyma reflecting areas of fibrosis, hemosiderin deposition and previous microinfarctions.

While mycoplasma is not typically recognized as a primary cause of direct splenic abscesses or lesions, the compromised immune function inherent in SCA patients makes them susceptible to secondary infections[2].

This case underscores the importance of a comprehensive approach to paediatric patients with sickle cell anaemia where imaging findings must be interpreted in light of both the underlying haematological disorder and any concurrent acute infections.

This case emphasizes the need for a high index of clinical suspicion, careful radiological evaluation, and early microbiological testing in SCD patients presenting with respiratory symptoms. It also emphasizes the radiologist's role in recognizing imaging patterns that raise suspicion for atypical infections in the setting of an underlying haematological disorder. It is important to communicate these findings to the clinical team to guide prompt management.

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