A case report of severe mycoplasma pneumonia with autoimmune haemolytic anaemia

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SUMMARY

Mycoplasma pneumonia is a common cause of respiratory disease and more so in school going children. The spectrum of the manifestations range from haematological, dermatological, neurological, musculoskeletal, renal, cardiac and also gastrointestinal. The treatment approach has varied over time. In this report we would like to share our experience in a case of M.pneumonia with autoimmune haemolytic anaemia (AIHA).

INTRODUCTION

Mycoplasma pneumonia is a bacteria that belongs to the class Mollicutes class and is the smallest self-replicating bacteria pathogenic to humans, the only known host.1 The manifestations of M.pneumoniae infection can be broadly classified into respiratory tract infection and extrapulmonary disease. The most common presentation among this two groups are tracheobronchitis and pneumonia. The extrapulmonary manifestation encompasses a different spectrum which includes haematological, dermatological, neurological, musculoskeletal, renal, cardiac and gastrointestinal manifestations.1 The haematological presentation ranges from anaemia, thrombocytopenia, as well thrombotic thrombocytopenic purpura and haemophagocytosis. We report a case of severe M.pneumoniae associated with mild autoimmune haemolytic anaemia (AIHA) with minimal sign and symptoms of haemolysis in a 5-year-old girl.

CASE REPORT

A 5-year-old Indian girl presented recently to the general ward in Hospital Sultan Abdul Halim (HSAH) with fever, cough, runny nose, sore throat associated with reduced oral intake. A nasopharyngeal (NPA) swab was taken and was found negative for the SARS-CoV-2 infection. Prior to her presentation to HSAH she had visited a general practitioner and was prescribed antibiotic a total of three doses which she completed.

Examination on admission noted an alert girl with bilateral cervical lymphadenopathy with the lung examination resulting in stony dullness over the right lower zone coupled with decrease air entry. The liver was palpated and measured at 4cm. A chest x-ray was done on admission and showed right lung opacity with meniscus sign seen until the middle zone (Fig 1a).

Fig. 1a: Chest X-ray on admission showing right lung pleural effusion; 1b: Chest X-ray prior to discharge showing resolving right-sided pleural effusion. The right sided chest tube was previously removed.

The initial investigation revealed a reduced white cell at 7700 which with a raised C-reactive protein (CRP). The liver function was also deranged with the aminotransferase (ALT) at 432 and the aminotransferase (AST) 560. An initial mycoplasma screening was done which resulted in a titre of 1:40.

She was supported with 5L/min of oxygen via face mask and was covered with high dose intravenous broad-spectrum antibiotics (Ceftriaxone) as well as penicillin (Cloxacillin) and oral macrolides (Syrup Clarithromycin) was initiated. We proceed for an ultrasound thorax and was reported as right middle and lower zone lung consolidation with rightsided pleural effusion.

In view of the ultrasound (USG) thorax findings we inserted a 10Fr chest tube that initially drained 200cc of straw-coloured fluid. However, the culture and sensitivity for organisms were negative with the biochemical tests not yielding any pathological findings.

Her fever did not abate by the 6th day of admission and the intravenous medication were upgraded to carbapenem (Meropenem) group. We also repeated the Mycoplasma titre and the was increment of the titre to 1:320. A full blood picture (FBP) was sent to rule the possibility of malignancy. The FBP was reported as underlying infection associated cold autoimmune haemolytic anaemia. We started to monitor the full blood count (FBC) every alternate day for the period of her admission (2 weeks). We also initially investigated for the presence of any haemolysis however the direct Coombs test was positive and reticulocyte percentage was 5%. In addition to blood investigations we monitored the appearance of the urine of the patient which was normal and not blood stained. As the patient showed clinical improvement coupled with a settling fever a series of FBC showed a sudden drop of the haematocrit level to 3.5% followed by a repeated sample showing 4.5%. The haemoglobin was 10.5 and 11.2q/dL respectively. We had a discussion with our haematologist and was informed in view of the underlying AIHA the blood sample for this patient should be warmed prior to the reporting. Subsequently all the FBC sample for this patient that was sent were pre-warmed by to reporting.

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	8/4/2020	14/4/2020	18/4/2020	18/4/2020 (Repeated sample)	19/4/2020
White Cell Count					
(4000-1100u/L)	7.7	10.3	10.4	7.5	10.9
Haemoglobin					
(12.5-15.5g/dL)	14.9	11.8	10.2	10.9	11.0
Haematocrit					
(37-47%)	44.3	20.6	3.3	4.9	20
Platelet					
(15,000- 40,000uL)	181	260	734	739	715
C-Reactive Protein					
(0-5mg/L)	365.5	165.2	45		
Mycoplasma Titre	1:40	1:320			

Table I: Blood investigation trend during the patient's stay in the ward



Fig. 1: a: Chest X-ray on admission showing right lung pleural effusion; b: Chest X-ray prior to discharge showing resolving right-sided pleural effusion. The right sided chest tube was previously removed.

By the 5th day of the chest insertion the drainage for the right chest cavity was nil. We were able to wean down the patient from the oxygen support. A repeated CXR was done prior to the removal of the chest drain showed a persistent right, middle and lower zone opacity as compared to the previous CXR. We continued the carbapenem antibiotics however switched the oral macrolides (clarithromycin) to intravenous and intravenous macrolide formulation (azithromycin). The oral azithromycin completed a 2-week duration.

The patient was also referred for physiotherapy for spirometry and she showed a gradual improvement and was able to lift up to two balls prior to discharge.

The patient was clinically well with the latest CXR increased lung marking (Fig 1b). After about 20 days of admission the patient was discharged with a plan to see in the clinic after two weeks. We discharged her with oral Augmentin and to review the CXR in clinic to decide the further duration for antibiotics.

Table I show the serial laboratory workup that guided our team as well as aided us in the viewing of the effectiveness of the treatment.

DISCUSSION

The age of the patient, symptoms as well as laboratory investigations indicated a case of *M.pneumoniae* infection. However, the severity of the symptoms made the treatment and the diagnosis a challenge to us. As the blood investigations in table shows in the infective stages of the disease there was no evidence of any ongoing autoimmune haemolysis.

At the point of admission, it was not noted by our team that the mild hepatomegaly was in relation to an already started autoimmune haemolytic process that had already started. The most common presentation of autoimmune haemolysis in a post-infectious condition is the cold form @ cold agglutinin.² In this condition the IgM autoantibody is reactive at lower temperatures and has a higher tendency to bind to red cells in the cooler parts of the body (e.g., distal extremities) and this leads to increase fixation of complement (C3) on these cells. The complement (C3) + re cells are sensitised cells and are removed from the circulation primarily by the hepatic Kupffer cells.²

We proceeded for an FBP in view of the pleural effusion and hepatomegaly however with an aim of ruling out a malignancy. The FBP however was reported as AIHA to which we adjusted our subsequent management and investigations.



Fig. 2: A flowchart of the path to establishing the diagnosis of Cold AIHA secondary to Mycoplasma Pneumonia.

In addition to the disease progress and also the sensitisation of the red blood cells, there have been reports of antibiotics especially high doses of 2nd and 3rd generation cephalosporins responsible for immune and auto immune AIHA.³

Our patient did not exhibit any sign or symptoms of haemolysis. As can be observed from table I, the blood parameters especially the FBC began showing abnormalities during the afebrile phase. This can be explained as during the febrile phase most of the complement factors steadily being consumed. We become alerted to the fact there was a sudden drop of the haematocrit percentage without consequent fall in the haemoglobin levels. It was also noted in addition to a sudden fall in the percentage of the haematocrit there was also a steep rise in the MCHC, MCV and MCH values. However, after discussion with the in-house haematologist, a suggestion to incubate the sample at 37°C prior to reporting and this leads to a decrease in the potency of immunoglobulins and causing them to dissociate from the RBC.⁴

It is worth mentioning here that we did screen the patient for haemolysis parameters (Coomb's test, reticulocyte count). The reticulocyte was within normal parameter and the Direct Coomb's test was positive.

The other blood investigation that has been recommended by a number of studies is the direct antiglobulin test (DAT). However, this test was not available in our facility. The flow chart to establish the diagnosis of AIHA is as shown in Fig 2.

The reason for publishing a report of a case of Cold Autoimmune Haemolytic Anaemia secondary to M.pneumoniae is mainly to highlight the challenges and share the experiences faced in managing as well diagnosing a case of Cold Autoimmune Haemolytic Anaemia secondary to *M.pneumoniae*.

CONCLUSION

The presence of an autoimmune haemolytic process along with M.pneumoniae infection has been reported previously. The management approach for a patient with this infection is well established. However, a high degree of suspicion and awareness should be maintained as to not miss this serious and fatal complication. The DAT blood test is the mainstay in diagnosis of AIHA. This test is not readily available, more so in the district setup. Based on our experience and research we have come with a flow chart that may help others in the management and approach of type of cases.

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REFERENCES

- 1. Krafft C, Christy C. Mycoplasma Pneumonia in Children and Adolescents. Pediatr Rev 2020; 41(1): 12-19.
- Kurugol Z, Onen SS, Koturoglu G. Severe Hemolytic Anemia Associated with Mild Pneumonia Caused by Mycoplasma pneumonia. Case Rep Med 2012; 2012: 649850.
- 3. Gu L, Chen X, Li H, Qu J, Miao M, Zhou F, et al. A case of lethal hemolytic anemia associated with severe pneumonia caused by Mycoplasma pneumoniae. Chin Med J (Engl) 2014; 127(21): 3839.
- Katharia R, Chaudhary RK. Removal of antibodies from red cells: Comparison of three elution methods. Asian J Transfus Sci 2013; 7(1): 29-32.