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Case Report

Atypical case of mycoplasma pneumoniae presenting as Stevens-Johnson syndrome

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ABSTRACT

Mycoplasma pneumoniae causes atypical pneumoniae in children and adolescents. *M. pneumoniae* associated Stevens-Johnson Syndrome (SJS) is also known as atypical SJS. SJS is characterized by generalised exanthema associated with fever, mucositis and history of numerous medications and certain agents of atypical pneumonia. We describe the case of a 7-year-old boy with bilateral atypical pneumonia along with multiple skin and mucosal exanthema that required intensive care unit admission due to respiratory distress. He was diagnosed with SJS associated atypical pneumonia caused by *Mycoplasma pneumoniae* confirmed by serological test. Since, there are few similar cases in literature; a treatment protocol is yet to be established. Our case is an example evidence-based treatment and hopefully in future there might be a definite protocol for Atypical Stevens-Johnson Syndrome.

Keywords: Steven Johnson syndrome, Atypical pneumonia, Mycoplasma pneumoniae

INTRODUCTION

Mycoplasma pneumoniae (MP), an important pathogenic microorganism in clinical practice, is a frequent cause of respiratory tract infections in school-going children. Mycoplasma belongs to class Mollicutes (Mollis, soft; cutis, skin), order Mycoplasmatales.[1] Stevens-Johnson syndrome (SJS) and Toxic Epidermal Necrolysis (TEN) are characterized by blisters and mucosal/epidermal detachment resulting from full thickness epidermal necrosis in the absence of substantial dermal inflammation.

Stevens-Johnson syndrome (SJS) is described in a case where the total body surface area of blistering and eventual detachment is less than 10%. The term SJS/TEN overlap is used to describe cases with 10-30% epidermal detachment and toxic epidermal necrolysis is used to describe case with greater than 30% detachment. Mycoplasma and other respiratory infections in children cause a clinically distinct presentation with prominent mucositis and limited cutaneous involvement. Patients with SJS/TEN presents with fever >39 ºC, sore throat, conjunctivitis, and acute onset of painful dusky, atypical, target-like lesions. Drugs that most commonly cause SJS/TEN are sulphonamides, allopurinol, antiepileptics, oxicam, NSAIDs, β-lactum and other antibiotics.[2]

CASE REPORT

A 7-year-old male child presented to Department of Otorhinolaryngology and Head and Neck surgery, at a tertiary health care centre at emergency department on January 2024 with oral mucocutaneous lesions along with target lesions all over the body and purulent discharge from eye for 1 day. It was preceded by an episode of high-grade fever for which acetaminophen was given by his primary care giver. It was sudden in onset with painful and difficulty in opening of mouth and eyes. There was also difficulty and pain on swallowing foods and liquids. It was associated with cough and shortness of breath. On physical examination, patient was drowsy and lethargic, febrile

with temperature 37.94°C, tachypnea RR=24/min, wheeze noted in bilateral upper lobes of lung, pulse 110/min felt at left radial artery, BP recorded as 90/60 mmHg, transcutaneous oxygen saturation showing 94% at room air. On local examination haemorrhagic crusting were noted over lips and intra-oral peeling of buccal mucosa and mucosal surfaces of hard palate along with a coated dirty tongue and drooling of saliva noted.

On ocular examination bilateral conjunctivitis with widespread eyelid margin necrosis and meibomitis were noted. Discharge and crusts were found at bilateral nasal vestibule. Multiple erythematous nummular lesions along with blisters noted in the upper and lower limbs and few on the trunk and face. Blood investigation taken upon arrival showed normal cell counts and haemoglobin, decreased albumin (3.2 g/dl), decreased sodium levels (124 mEq/l), and increased levels of CRP, ESR. Chest X-ray reporting shows- haziness in both upper zones with prominent broncho vascular markings.

Patient was admitted under ENT department at pediatric ICU. Initially patient was started on co-amoxiclav 670 mg intravenously thrice daily along with parenteral nutrition, intravenaous dexamethasone 3 mg twice daily. He was put on parenteral nutritional support with combination of albumin, multivitamin infusion, dextrose, lipid infusion and amino acid. Oxygen support was provided via face mask maintained at 5l/min, transcutaneous O2 showed 92-96% on O2 support.

Temperature and input output monitoring done. Oral swabs sent for culture and sensitivity that showed no growth of micro-organism after 48 hours of incubation period. There was no improvement in oral lesions and pulmonary status after 2 days hence he was upgraded from co-amoxiclav to intravenous Linezolid 200 mg twice daily. For eye lesions, moxifloxacin eye drops were applied locally 1 drop 4 hourly. Department of dermatology was consulted and they came to a provisional diagnosis of Stevens-Johnsons syndrome on clinical findings and advised to continue systemic steroids.

On the fifth consecutive day of starting i.v Linezolid oral and ocular lesions were progressively improving and there were no new cutaneous lesions but pulmonary symptoms worsened as there were crepitus noted in bilateral upper and lower lobes and SPO2 wasn't maintained in room air. Oxygen support was continued.

Regular dressing of oral and cutaneous lesion was done. Dressing was initially done with saline followed by local application of metronidazole gel 1.5% w/w. The skin lesions were treated with local application of mupirocin ointment. For the cutaneous lesions Condy's compress was advised from department of dermatology i.e., potassium permanganate solution soaked in sterile gauze and applied locally. The healing of the oral and dermo cutaneous lesions progressed promptly. Department of paediatrics was consulted and they opined to rule out mycoplasma pneumoniae infection by sending blood samples to test for IgM and IgG titres for the same and advised to change antibiotic to azithromycin on empirical basis and syrup azithromycin (200 mg/5 ml), 2.5 ml once daily started.

Patient's pulmonary symptoms began improving 2 days later and the patient was weaned from oxygen support and eventually shifted to general ward. Patients IgM titres were 30.92 suggestive of acute infection of Mycoplasma pneumonia.

On testing positive IgM antibodies for infection with *Mycoplasma pneumoniae*, the diagnosis was made to be "Atypical mycoplasma infection". Patient was reviewed by department of paediatrics and pulmonary medicine. He was started on azithromycin suspension 5 ml once daily for 15 days.

With the start of macrolide, the pulmonary and oral lesions started improving at a very satisfying rate. Within 5 days of starting macrolide, lung crepitus decreased and by 7th day bilateral vesicular breath sounds were heard. The oral, ocular and skin lesions completely resolved.

Patient was discharged after completing course of antibiotics and getting clearance for discharge from both paediatrics and pulmonary medicine department. Patient was reviewed in outpatient basis after 2 weeks and he was clinically and hemodynamically stable.

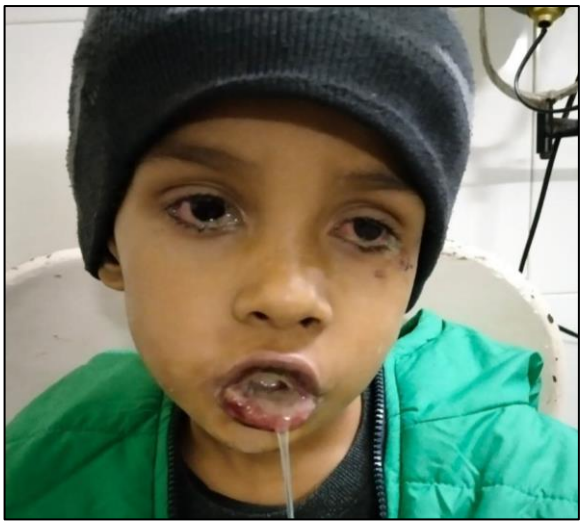


Figure 1: Oral lesions.

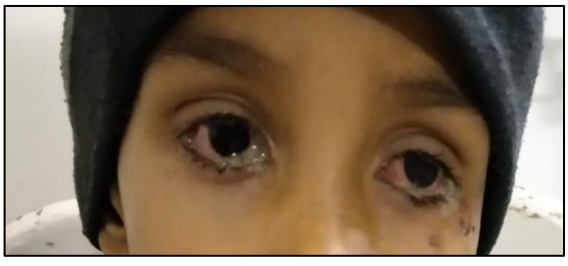


Figure 2: Ocular lesions at day 1.

Figure 3: Ocular and oral lesion on day 7.

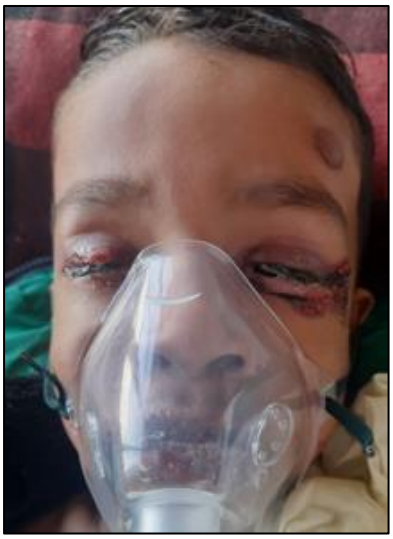
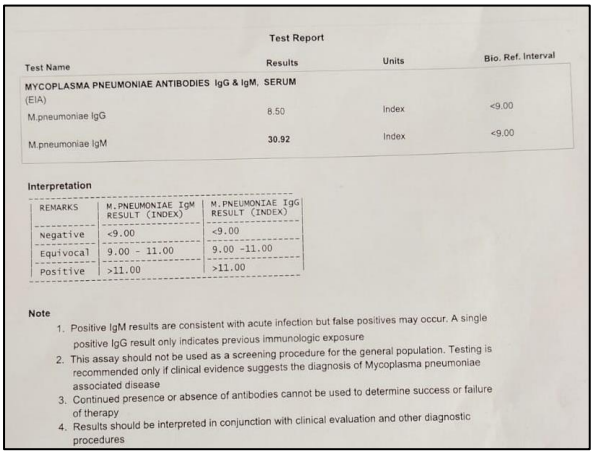


Figure 4: Patient on continuous moist oxygen inhalation.



Test Report

Test Name	Results	Units	Bio. Ref. Interval
MYCOPLASMA PNEUMONIAE ANTIBODIES IgG & IgM, SERUM (EIA)			
M.pneumoniae IgG	8.50	Index	<9.00
M.pneumoniae IgM	30.92	Index	<9.00

Interpretation

REMARKS	M.PNEUMONIAE IgM RESULT (INDEX)	M.PNEUMONIAE IgG RESULT (INDEX)
Negative	<9.00	<9.00
Equivocal	9.00 - 11.00	9.00 -11.00
Positive	>11.00	>11.00

Note

1. Positive IgM results are consistent with acute infection but false positives may occur. A single positive IgG result only indicates previous immunologic exposure
2. This assay should not be used as a screening procedure for the general population. Testing is recommended only if clinical evidence suggests the diagnosis of Mycoplasma pneumoniae associated disease
3. Continued presence or absence of antibodies cannot be used to determine success or failure of therapy
4. Results should be interpreted in conjunction with clinical evaluation and other diagnostic procedures

Figure 5: Report of IgM and IgG antibodies for *M. pneumonia.*

Figure 6: Oral, cutaneous and ocular lesion improved on the day of discharge.

DISCUSSION

Mycoplasma pneumoniae is a common respiratory pathogen. It is short, rod-shaped bacteria that lacks cell wall, bounded by triple layered membrane and causes respiratory infections. As the organism lacks cell wall detecting it through gram staining is difficult. These were pleomorphic and were called PPLO (pleuropneumonia like organism). The colony of mycoplasma on solid agar medium appears as just like fried egg under stereomicroscope.

The colony shows spherical or hemispherical portion in centre, surrounded by surface growth towards the periphery.[3] Culture can confirm their presence but it takes 2-6 weeks. Serology testing like fixation assays, and indirect immunofluorescence sensitivity and specificity, 80%-90% and 92%-100%, respectively.

Extrapulmonary manifestation of *M. pneumoniae* are rare but have been noted in previous literature. Extrapulmonary manifestation like haemolytic anaemia, myocarditis, myringitis, encephalitis, Miller Fischer syndrome, Stevens-Johnson syndrome and others are reported. The extrapulmonary manifestations are mainly reported in previous literature in children and young adults aged 6-19 years.[4]

The use of systemic steroids in *M. pneumoniae* associated SJS showed satisfactory improvements. In this case, serological testing confirmed our suspected diagnosis and guided the course of treatment. The pulmonary as well as the extra pulmonary symptoms resolved with the combination of systemic steroids (dexamethasone) and oral antibiotics (azithromycin).

CONCLUSION

Atypical SJS differs from classical SJS with respect to clinical presentation and aetiology. *M. pneumoniae* associated SJS mimics various other autoimmune and infectious conditions, which results in delayed diagnosis and treatment. At present there is no established treatment

regimen that shows reduced hospital stay and/or reduced mortality. This case summarizes the diagnostic serology testing to diagnose *M. pneumoniae* infection, with atypical extra pulmonary manifestation that delayed the diagnosis. The case showed reduced disease progression and fast improvement following initiation of dexamethasone and azithromycin. This case is a fine example of evidence-based treatment and hopefully will provide guidance on how to care for patients presenting with *M. pneumoniae-associated* Stevens-Johnsons syndrome.

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