Case Report

A case report of scarlet fever: An uncommon diagnosis for fever and rash

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Abstract

Streptococcus pyogenes is a pathogen capable of causing a wide spectrum of diseases including pharyngitis and streptococcal toxic shock syndrome, with a range of complications and outcomes. Even though it is not an uncommon pathogen, reports of scarlet fever are scarce in Sri Lanka. We describe a case of scarlet fever in a seven-year-old boy with typical features. The child presented with high fever, body aches, vomiting, loss of appetite and a rash and developed pharyngitis and a strawberry tongue during the ward stay. A pure growth of group A *Streptococcus* was isolated from a throat swab taken on admission. He improved with appropriate treatment and recovered without complications. The importance of throat swab culture and clinical correlation is illustrated in this case report

Keywords : Scarlet fever, Fever, Rash, Streptococcus pyogenes, Children

Introduction

Streptococcus pyogenes is a known cause of both invasive and non-invasive diseases.[1] Scarlet fever is caused by pyrogenic exotoxin producing strains of *S. pyogenes*. Though this is a well-known presentation of *S. pyogenes*, it is rarely recognized in Sri Lankan children as patients do not present with the typical clinical picture and throat swab cultures are not taken in most instances. We present a case of scarlet fever in a seven-year-old child.

Case report

A 7-year-old boy with no known drug or food allergies presented to the paediatric ward with high fever, body aches, vomiting and loss of appetite for 2 days and an itchy rash throughout the

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body for 1 day. The patient had no contact history with anyone with fever. He had a history of bronchial asthma and was on inhalers. He was an only child

On admission, the child was ill looking, afebrile, not pale, with no cervical lymphadenopathy and was haemodynamically stable. There was a fine erythematous punctate eruption throughout the body (Figure 1), mostly on the back, that gave a rough texture to the skin. Ear and nose examination were found to be normal while the throat was inflamed with pustules over the tonsils. Cardiovascular, respiratory, and abdominal examination were normal.

Figure 1
Fine erythematous punctate eruption

On admission his white cell count was $21.49x10^3$/L with a neutrophil predominance ($16.01x10^3$/L) and eosinophils $2.43x10^3$/L. Platelets were 217000/L and CRP was 57.9 mg/L. Throat swab taken on admission grew beta-haemolytic streptococci. Grouping was done using the latex agglutination kit (Oxoid) and the isolate was identified as group A *Streptococcus* (GAS). Antibiotic sensitivity test was done using CLSI (2018) method.[2]

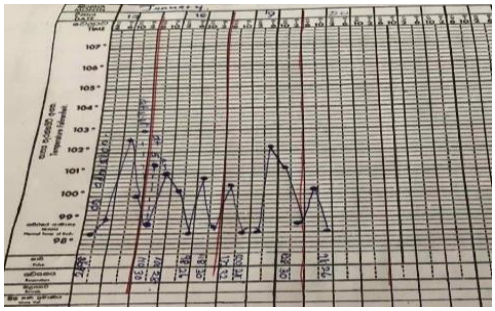

Figure 2: fever chart

The patient developed a sore throat after admission (on day 3 of fever – Figure 2) and a mild non-productive cough. While in the ward he also developed a red inflamed tongue (Figure 3) on day 4 of fever followed by desquamation of the white coating on day 5 (Figure 3).

The child was initially given amoxicillin 500 mg 8 hourly for 3 days orally. It was then converted to intravenous co-amoxiclav 1.2 g 8 hourly for another 4 days. In addition, the child was given paracetamol, domperidone and chlorpheniramine for supportive care. He was discharged with amoxicillin 500 mg 8 hourly for 7 days. He had a complete recovery and was without complications in the follow-up up at 2 weeks and one month.

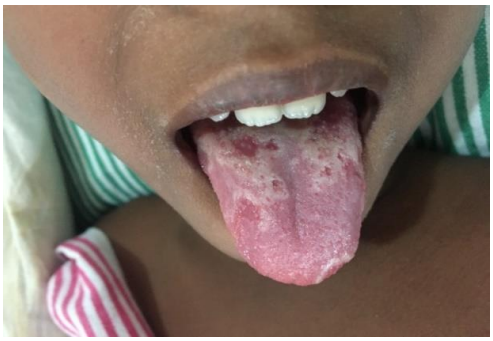

Figure 3: red inflamed tongue

The timeline of the patient's illness is shown in Figure 4

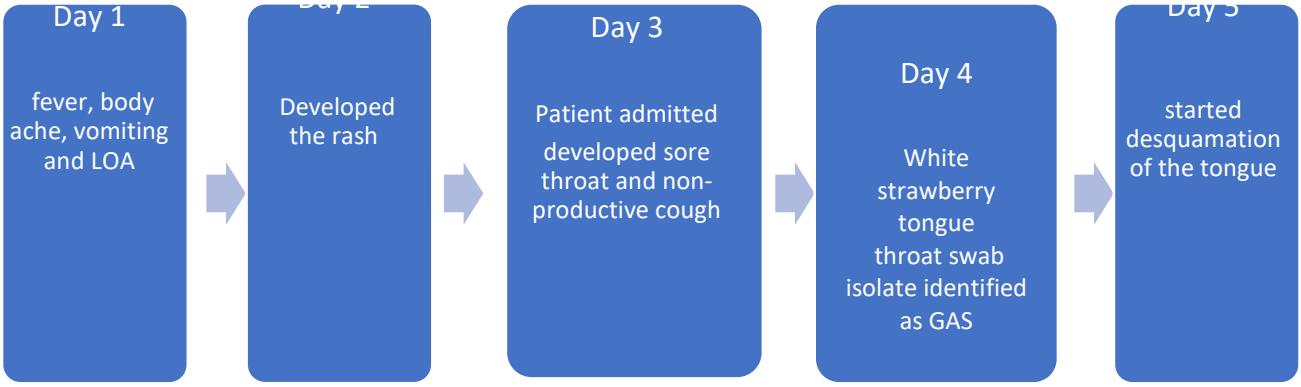

Figure 4 – Time line

Discussion

Erythrogenic exotoxin producing streptococcal strains give rise to scarlet fever. Pharyngeal infections usually precede scarlet fever though it can also be a result of infected wounds and puerperal sepsis. The scarlatina rash makes the syndrome different from the disease caused by non-toxigenic strains.[3]

The rash typically appears on the second day of the disease as a spread out erythema which blanches on pressure. It has a centripetal distribution, commonly sparing the palms, soles and face. Skin creases appear dark red and scattered petechiae can be observed. In dark skinned patients it is difficult to elicit these features of rash other than the appearance of the sandpaper texture on skin as a result of the sweat gland occlusion. Circumoral pallor is noted on the flushed face. Pharyngitis and tonsillitis are also noted in some with enanthem. The white strawberry tongue noted in the early part of the disease appear as white yellowish coating with red papillae. Later in the disease, the tongue turns into a red strawberry tongue which is beefy red in colour. The skin rash usually fades over a week, resulting in wide-spread desquamation that can last for weeks. Sometimes a degree of eosinophilia is noted in early disease.[3]

Septic scarlet fever and toxic scarlet fever are severe forms of scarlet fever resulting from haematogenous spread and severe toxaemia respectively. Those patients get high fever and significant toxicity. Scarlet fever can rarely result in complications like jaundice, arthritis and hydrops of the gallbladder. These severe presentations are uncommon with antibiotic usage.[3]

To the best of our knowledge, there are no published data or case reports on scarlet fever in Sri Lanka. This may be due to poor reporting as well as early antibiotic usage that alter the typical clinical picture.

Hong Kong had a scarlet fever epidemic in 2011 which was associated with emm12 strain of *S. pyogenes*.[4] In England, the scarlet fever incidence has risen from 8.2 per 100,000 in 2013 to 33.1 per 100,000 in 2016, and 2.5% required hospitalization, which suggested rising virulence in common strains[5] and strain was emm12.[6] In the South Korea outbreak in 2011, emm4, emm28,

emm1, were the commonest strains[7] and emm1 and emm12 were noted in Shandong province, China in 2013.[8] Complete resistance to erythromycin and clindamycin was noted in a Chinese study.[6] SSA and SpeC superantigen genes were noted in another Chinese study .[9]

Our patient did not give a history of contact with a person suspected of streptococcal infection. His rash and tongue showed typical clinical features of scarlet fever and the isolation of group A *Streptococcus* with typical colony morphology confirmed the diagnosis. He responded to the antibiotic therapy and remained without complications during the follow up visit. Strain identification and toxigenicity were not carried out due to unavailability of resources. Bacitracin sensitivity was not carried out due to unavailability of discs. Not all non-Group A Streptococcus are resistant to bacitracin, only about 80 – 90% are resistant to bacitracin[3].

Take home message

It is important to suspect scarlet fever though it is said the rash is difficult to identify in dark skinned children. Delayed or untreated group A *Streptococci* lead to significant complications[10]. Increased incidence of virulent Group A strains are being reported worldwide. Sri Lanka needs to establish facilities for culture, identification and sensitivity testing which are accessible to general practioners as well as in hospital settings. Non-availability of rapid bedside tests may hinder the diagnosis in community settings.

Declarations

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N. S. Chandrasiri and S. Ganeshan, corrected the manuscript.

References

1. CDC, Scarlet Fever. Available at: *https://www.cdc.gov/groupastrep/diseases-hcp/scarlet-fever.html*. Accessed on 27/12/2021.
2. CLSI supplement M100. 30th ed. CLSI guideline Wayne, PA: Clinical and Laboratory Standards Institute; 2019.
3. Bennett JE, Dolin R, Blaser MJ. Principles and Practice of Infectious Diseases. 9th Edition. Philadelphia, PA. Elsevier Ltd. 2020 ISBN: 978-0-323-48255-4
4. Luk EYY, Lo JYC, Li AZL, et al. Scarlet fever epidemic, Hong Kong 2011. *Emerg Infect Dis*. 2012; 18:1658–1661. *doi: http://dx.doi.org/10.3201/eid1810.111900*
5. Carapetis, J.R., Steer, A.C., Mulholland, E.K., Weber, M. The global burden of group A streptococcal diseases. *Lancet Infect Dis*. 2005; 5:685–694. *doi: http://dx.doi.org/10.1016/S1473-3099(05)70267-X*
6. Chen M, Yao W, Wang X et al. Outbreak of scarlet fever associated with emm12 type group A Streptococcus in 2011 in Shanghai, China. *Pediatr Infect Dis J*. 2012; 31:e158–e162. *doi: http://dx.doi.org/10.1097/INF.0b013e31825874f3*
7. Park DW, Kim SH, Park JW, et al. Incidence and Characteristics of *Scarlet Fever, South Korea, 2008-2015. Emerg Infect Dis. 2017; 23(4):658-661. doi*: *10.3201/eid2304.160773*
8. Liu Z, Fang M, Hu B, et al. Molecular types of group A Streptococcus isolated from scarlet fever patients and asymptomatic carriers in Shandong province, 2013. 2014; 35(12):1375-8. *No doi* PMID: 25623458

9. Davies MR, Holden MT, Coupland P, et al. Emergence of scarlet fever *Streptococcus pyogenes* emm12 clones in Hong Kong is associated with toxin acquisition and multidrug resistance. *Nat Genet.* 2015; 47:84–87. *doi: http://dx.doi.org/10.1038/ng.3147*
10. Pardo S, Perera TB. Scarlet Fever. [Updated 2022 Jan 17]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: *https://www.ncbi.nlm.nih.gov/books/NBK507889/*