Miliaria Scarlatinosa, A Peculiar and Rare Form of Scarlet Fever – A Case Report

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ABSTRACT Scarlet fever typically presents with distinctive erythematous papular rash following pharyngitis. Atypical forms may develop, making the diagnosis difficult. We present the case of a girl with feve, and unusual vesicular skin eruption (miliaria scarlatinosa) preceded by a skin infection, without mucosal changes. Leukocyte count, C-reactive protein, and antistreptolysin O-titer were elevated. Bacteriological swabs of the skin injury revealed *Streptococcus pyogenes*. Histopathology was compatible with scarlet fever exanthema. Intramuscular penicillin and topical wound care induced complete remission. It is of great importance to be aware of uncommon clinical presentations of scarlet fever in order to establish a timely diagnosis and prevent potential complications.

KEY WORDS: scarlet fever, scarlatina, miliaria scarlatinosa, girl

INTRODUCTION

Scarlet fever (SF) or scarlatina is an acute disease caused by *Streptococcus pyogenes* (SP) or Group A *Streptococcus* (GAS) (1). GAS produces erythrogenic toxins types A, B, and C, responsible for the skin manifestations (2,3). Erythrogenic toxins initiate local inflammatory response or delayed-type hyper-sensitivity reaction in persons that have been previously exposed to GAS (4,5). GAS can cause a variety of infections (pharyngitis, impetigo, severe soft-tissue infections), postinfectious immune syndromes (post-streptococcal glomerulonephritis, acute rheumatic fever) and toxin-mediated diseases (toxic shock syndrome, scarlet fever) (1).

Scarlet fever presents as exanthema, usually associated with tonsillitis and pharyngitis (6). It is a disease of childhood, occasionally seen in adults (7), typically presenting as a blanching, papular "sandpaper" rash with no vesicles or pustules (6). However, SF can manifest after wounds ("surgical scarlet fever"), burns, and pelvic or puerperal infections (6). Whether an infected patient develops SF or a septic illness depends on the level of antitoxic immunity, acquired by previous exposure to the bacteria (3). Given that SF may present with variable severity and different clinical manifestations, it might be difficult to diagnose in its early stages.

We present the case of an unusual form of scarlet fever, the so-called miliaria scarlatinosa, in a child.

CASE REPORT

A 9-year-old girl was admitted because of a rash that had developed during the previous day. Several days before the eruption, she had scratched her left lower leg, and the wound had been left untreated.

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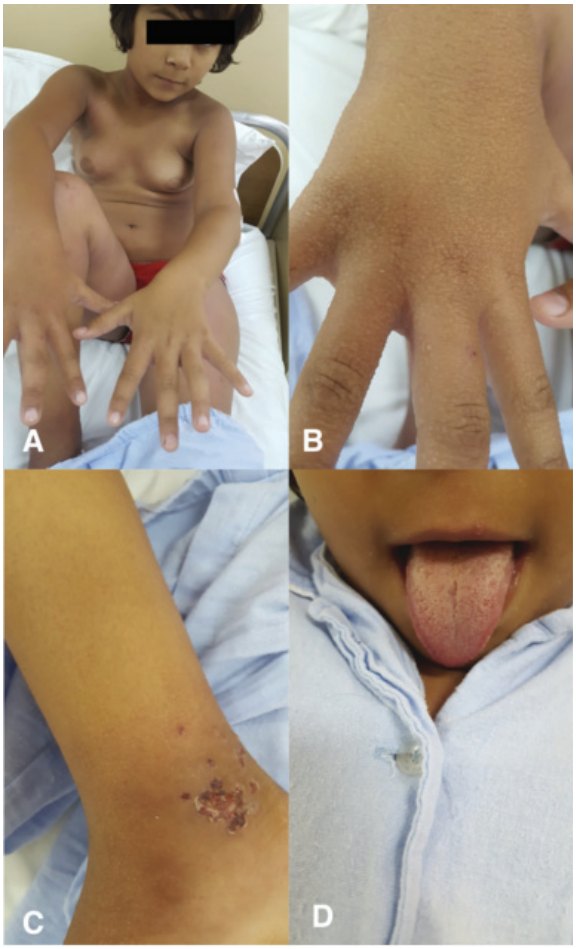

Figure 1. Clinical presentation: (A) Slight erythema on the trunk and extremities. (B) Close-up of numerous tiny vesicles, grouped on the dorsa of the hands, without background erythema. (C) Ulceration covered with yellowish crusts in the perimaleolar region. (D) Whitish tongue, without bright red papillae expressed; no signs of "strawberry tongue".

The rash started on the injured leg and subsequently spread to the other leg, trunk, and upper extremities, followed by fever, malaise, and fatigue.

Clinical examination revealed mild erythema on the girl's neck, trunk, and prominently on the lower extremities. More noticeable were numerous tiny vesicles disseminated on the trunk and extremities, which were grouped in some regions. region An ulceration with pus and yellowish crusts was observed in the perimalleolar tissue, with pustules on the surrounding skin (Figure 1). Lymphadenopathy, conjunctival, tongue, and throat mucosa changes were absent. The girl was febrile (38.6 °C), but in good general condition. Laboratory analyses revealed leukocytosis (18.4×10^9/L with neutrophilia 84%, absolute number of neutrophils 15.5×10^9/L), elevated C-reactive protein (9.4 mg/L, n<5) and antistreptolysin O-titer (258 U/mL, n<200). Urinalysis revealed mild leukocyturia (15 leucocytes/L). Other routine laboratory analyses were normal. After biopsies and bacteriological swabs, oral cephalexin at 100 mg/kg BM/day was introduced, together with wound cleansing and topical gentamycin ointment. When the skin swab culture revealed SP, intramuscular penicillin was administrated, 1.6 MU/day, for 10 days. Skin histopathology showed acute spongiotic dermatitis with rare eosinophils and neutrophils as well as focal hemorrhage, compatible with miliaria scarlatinosa (Figure 2, A, B). Direct immunofluorescence test was negative. One day after the introduction of systemic antibiotics, the girl was afebrile, and three days afterwards the characteristic desquamation occurred (Figure 3). Control urinalyses were normal.

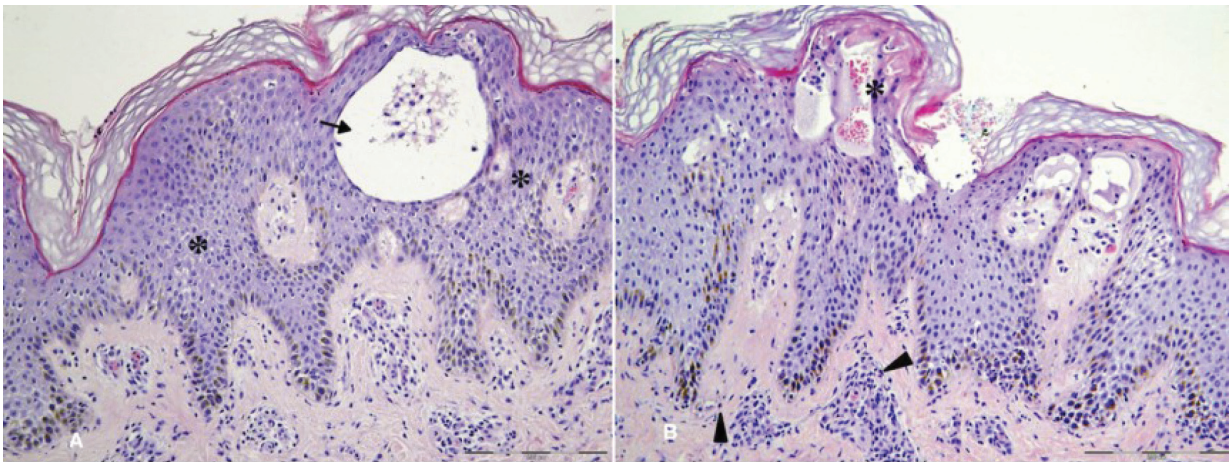

Figure 2. Histopathological features: (A) Prominent spongiosis (asterisk) was noted in the epidermis along with a spongiotic vesicles (arrow) containing rare Langerhans cells. (B) Some vesicles contain red blood cells (asterisk). Superficial capillaries surrounded by dominantly lymphocytic infiltrate with rare eosinophils and neutrophils (arrowheads) (hematoxylin and eosin stain, magnification ×200).

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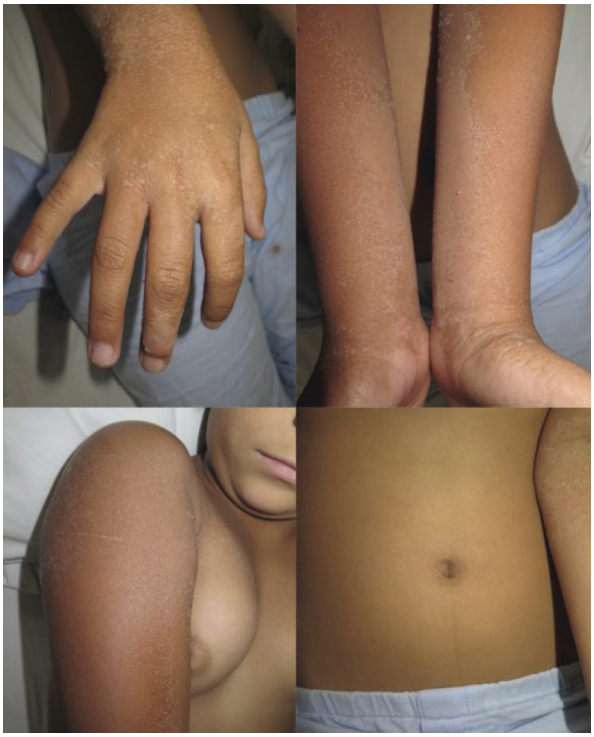

Figure 3. Clinical regression: Characteristic desquamation, more prominent in regions previously covered by numerous vesicles.

DISCUSSION

SF is a well-known entity with a characteristic clinical presentation. After an incubation period of 2-5 days, erythrogenic toxin-producing strains of GAS, most commonly cause fever, malaise, headache, nausea, and vomiting (8). More severe presentation such as abdominal pain and convulsions may be observed in young children (7). Most frequently, SF is characterized by typical cutaneous presentation, initially called "scarlatina papulosa" (9). In such cases, the diagnosis is easy. However, in a small number of cases there are no signs of pharyngitis and the source of infection is GAS-affected skin (6), or cutaneous manifestations are atypical (9).

The rare cutaneous presentation characterized by myriad of dewdrop-like vesicles with a thin roof at the end of sudoriferous dust, as in the present case, has been described and termed "sudamina" (9). Vesicles represent drops of sweat retained by an obstructive epithelial pellicle of hot and dry skin, with no tendency towards sweating. This presentation is also called "scarlatina miliaris", "scarlatina vesicularis", or "miliaria scarlatinosa" (9,10). Vesicles are commonly disseminated, less often in a group, and usually appear on the chest and abdomen, less frequently on the extremities. Vesicles are conical with turbid content; their size is compared with millet seeds, pin-points, or pin-heads, and they may be easily overlooked. Rarely, they can coalesce, forming blebs, and then the condition is called "scarlatina pemphigoidea". If the eruption is more severe, vesicles may be more numerous and prominent. The intensity of residual desquamation is directly proportional to vesiculation (9). The histopathological background of the vesicles in SF is leukocyte infiltration either in the epidermis, the deeper layer of rete ridges, or intrafollicular infiltration. Sweat duct lumen can also be filled with endothelial cells (9).

The other rare presentations of SF are the toxic and septic forms, with multiorgan involvement. Recurrent scarlatiniform erythema, repeated attacks of a somewhat similar rash, followed by exfoliation, can also occur without discoverable cause (3).

It has not yet been elucidated why some patients develop atypical clinical forms of SF. It is hypothesized that microbial determinants of GAS are involved, such as specific strains with different virulence capacities. It is possible that host-related factors are also involved (4). The probable explanation is that different GAS genotypes cause different biological behavior and consequently different clinical presentations (1,4). The wider availability of bacterial whole genome sequencing would allow a better understanding of the pathogenesis of infections due to *S. pyogenes* (1).

Scarlet fever remains far from being completely understood, and we may still be missing the key host and environmental factors which determine the clinical presentation. Since the incidence of SF has been found to be increasing and outbreaks may appear in the future, it is essential to be aware of atypical forms of this disease.

References:

1. Wong SSY, Yuen KY. The Comeback of Scarlet Fever. E Bio Medicine. 2018 Feb;28:7-8.
2. Mahajan VK, Sharma NL. Scarlet fever. Indian Pediatr. 2005;42:829-30.
3. Hay RJ, Adriaan BM. Bacterial Infections. In: Rook's Textbook of Dermatology (Burns T, Breathnach SM, Cox NH, Griffiths CEM, eds.), 8th edn. Oxford: Blackwell Publishing, 2010; 30.1-30.74.
4. Andrey DO, Posfay-Barbe KM. Re-emergence of scarlet fever: old players return? Expert Rev Anti Infect Ther. 2016;14:687-9.
5. Brinker A. Scarlet Fever. N Engl J Med. 2017;376:1972.
6. Pardo S, Perera TB. StatPearls [Internet]. Treasure

Island (FL): StatPearls Publishing; 2018 Jan-2018 Jun 13.

7. Phillips R, Martin-Bates AJ, Withnall R. Unusual case of suspected recurrent scarlet fever in a UK serviceman. J R Army Med Corps. 2018 May;164:130-1.
8. Abeck D. Staphyloccocal and Streptococcal Diseases. In: Burgdorf WHC, Plewig G, Wolf HH, Landthaler M, eds. Braun-Falco's Dermatology, 3rd ed. Springer Medizine Verlag Heidelberg; 2009. pp. 114-38.
9. Shulman ST, Bisno AL, Clegg HW, Gerber MA, Kaplan EL, Lee G, *et al.* Clinical practice guideline for the diagnosis and management of group A streptococcal pharyngitis: 2012 update by the Infectious Diseases Society of America. Clin Infect Dis. 2012;55:1279-82.
10. Schamberg JF. A clinical and pathological study of the rash of scarlet fever with especial reference to the origin and character of the desquamation. JAMA. 1900;XXXV:1199-205.