Case Report
Sweet syndrome associated with mycobacterium tuberculosis

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Abstract: Mycobacterium tuberculosis can induce cutaneous reactions such as erythema nodosum and erythema induratum. However, tuberculosis associated with Sweet’s syndrome is extremely rare. Here we presented a 58-year-old tuberculosis meningitis patient with Sweet’s syndrome.

Keywords: Mycobacterium tuberculosis, tuberculousis, meningeal, sweet syndrome

Introduction
Sweet’s syndrome (SS) was named after Dr. Robert Douglas Sweet from Plymouth, England, who first described this condition in 1964 [1]. It is characterized by fever, cutaneous erythematous plaques, diffuse mature neutrophil infiltration in the upper dermis. Sweet’s syndrome is identified as a reactive phenomenon to various factors and it should be considered a skin manifestation of systemic disease. Drugs, infections, malignancy and autoimmune disease are the often mentioned reasons for it. Sweet’s syndrome has been reported following infections of the upper respiratory tract infection (e.g. viral, streptococcal) [2, 3] and the gastrointestinal tract infection (e.g. Yersinosis) [4]. But mycobacterium tuberculosis associated cases is extremely rare. We presented a 58-year-old tuberculosis meningitis patient with Sweet’s syndrome. To our knowledge, this is the first report about tuberculosis meningitis associated Sweet’s syndrome.

Case report
A 58-year-old male patient was admitted to our hospital with complains of a high-grade continuous fever (up to 38.8°C) for 15 days. His fever accompanied with the development of multiple reddish elevated skin lesions (Figure 1A-C). Half a month ago, the patient developed red papules on his twists. The lesions enlarged quickly to form non-pruritic, painful plaques with blisters, blood blisters on the surface. The lesions developed to the back of his hands quickly. They progressed and presented with rupture, exudate and crusting.

Two months ago, the patient was diagnosed as tuberculous meningitis. He had received a regimen of four anti-tubercular drugs (isoniazid, rifampicin, pyrazinamide and ethambutol). His previous medical history included nephrotic syndrome, recurrent laryngeal nerve and chronic atrophic gastritis. He had been given methylprednisolone 20 mg/d for his optic neuritis.

Laboratory investigations revealed hemoglobin 9.9 g/dl, total leukocyte count 16.1×10^9/L (normal 4.0~11.0×10^9/L), with 71.5% polymorphonuclear leucocytes, platelet count 434×10^9/L, erythrocyte sedimentation rate 102 mm in the first hour and C-reactive protein 63.3 mg/L. Biochemical examinations including blood sugar, renal profile and liver function test were within normal limits. Tumor marker, ultrasonic test and chest computed tomography scan were within normal limits. Secretion cultivation was negative. We took a biopsy from a lesion of the right hand showed dermal papillae and subepidermal fissure, dense upper dermal infiltration with neutrophils. The histopathological figures suggest a diagnosis of Sweet’s syndrome (Figure 2A-C).
The patient continued his anti-tuberculosis therapy strategy. We added the dose of methylprednisolone to 28 mg daily for her skin lesions and optic neuritis. After five days of treatment, the patient’s fever returned to normal and skin lesions improved rapidly. The methylprednisolone was tapered to 20 mg. The patient was followed up after 3 months, the lesions resolved completely (Figure 3).

**Discussion**

Sweet’s syndrome is identified as a reactive phenomenon. Sweet’s syndrome can be classified based upon the clinical setting: infection associated Sweet’s syndrome, malignancy associated Sweet’s syndrome, drug-induced Sweet’s syndrome, autoimmune disease associated Sweet’s syndrome and others [5]. The often mentioned reason for infection associated Sweet’s syndrome is the upper respiratory tract infection (e.g. viral streptococcal) and the gastrointestinal tract infection (e.g. yersinosis). Recently, *mycobacterium tuberculosis* associated Sweet’s syndrome have been reported. Such as *mycobacterium cervical lymphadenitis* associated Sweet’s syndrome [6, 7] and pulmonary tuberculosis associated Sweet’s syndrome [8]. So *mycobacterium tuberculosis* infection cannot be ignored as the reason of infection associated Sweet’s syndrome. In our case, we did not find any evidence of malignancy and autoimmune disease. Before and after the onset of Sweet’s syndrome, common medications reported to cause Sweet’s syndrome were not taken. But it is notable that the patient was diagnosed as tuberculous meningitis two months ago. And clinic features and laboratory results did not prompt other infection. So mycobacterium tuberculosis infection is the most suspect reason for this case. Our patient’s lesions were controlled with methylprednisolone without interruption of anti-tubercular drugs. Sweet’s syndrome is cure in some anecdotal case reports with conventional anti-inflammatory and/or immunosuppressive therapy without interruption of anti-tubercular drugs strongly goes against this hypothesis of anti-tubercular drugs induced Sweet’s syndrome [8, 9]. So the anti-tubercular drugs were not the routine reason of Sweet’s syndrome.

**Figure 1.** A-C. There are red plaques with blisters, blood blisters, rupture, exudate and crusting on the surface.
Our patient’s lesions haven’t had a relapse when we decrease the dose of methylprednisolone to 20 mg/d, the basic dose for his optic neuritis. So we tend to believe our case is associated with *Mycobacterium tuberculosis* infections instead of anti-tubercular drugs.

*Mycobacterium tuberculosis* can induce several cutaneous reactions such as erythema nodosum, and erythema induratum. But the association of Sweet’s syndrome with tuberculosis is extremely rare, only few cases have been described in literature [6-8, 10]. We presented an a tuberculosis meningitis patient with Sweet’s syndrome. In clinic work, we should be on high alert for the Mycobacterium tuberculosis infection is the reason of Sweet’s syndrome.

**Disclosure of conflict of interest**

None.

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**References**

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