Sweet’s syndrome in a diabetic patient
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Citation

Abstract
The onset of Diabetes mellitus in middle aged individuals is by and large with indistinct symptomatology. In this case report a 47 year old female presented with Sweet’s syndrome - a relatively uncommon illness. Sweet’s syndrome or acute neutrophilic dermatosis is a neutrophil mediated hypersensitivity reaction in response to systemic illnesses that may be hematological disease, infection, inflammation, malignancy or drug induced. However in many cases the etiology is idiopathic. Knowledge about this entity and its early recognition and prompt treatment is important to the internist to prevent devastating outcomes.

INTRODUCTION
Sweet’s syndrome is an uncommon disease. More unusual is its presentation as a diabetes cutaneous manifestation at its very onset. An extensive review of literature on subject of dermatological profile in diabetes reveals that its presentation as Sweet’s syndrome is extremely rare. Here we report the case of a middle aged lady presenting with this unusual dermatological expression with a concurrent diagnosis of new onset diabetes.

CASE REPORT
A 47 yr old woman presented to the Medical out patient department with history of low grade fever, headache and malaise of ten days duration. She had mild redness in bilateral conjunctiva at presentation. There was no evidence of sore throat, cough, coryza, diarrhea, burning micturition or arthralgia. A thorough physical examination did not reveal any abnormality. Four days later she had appearance of tender reddish blue papulonodular lesions with a vesicular centre on palmar surfaces of both the hands (Figure 1).

Figure 1: Palmar surfaces of hands showing asymmetrically distributed papulo-nodules suggestive of Sweet’s syndrome

Dermatologist opinion suggested differential diagnoses of erythema multiforme, drug eruptions or Sweet’s syndrome. A skin biopsy was performed. Over next two days few papules coalesced and some turned pustular in the course of a week and she continued to run mild fever. The patient did not have any genital or oral mucosal lesions and there was no history of any diarrhea. The past history and current history and clinical examination was inconclusive for underlying malignancy. The hematological investigations revealed hemoglobin of 12.1 g%, TLC of 9500/ ul, DLC N 92, L 8, M0, E0, Platelet count 1.8 lacs/ ul and ESR 60. Malarial parasite was negative, blood and urine cultures were sterile. There was no proteinuria or pyuria. Chest radiograph was normal.

At the same visit she was detected to be diabetic. She had a
BMI of 26 kg/m². Her fasting and post prandial plasma glucose was 220mg/dl and 315mg/dl. She had no evidence of hypertension, proteinuria, retinopathy or neuropathy. Her renal and liver functions were normal. She had a normal lipid profile. Patient was not keen on being started on insulin therapy. Patient was told about lifestyle modification, diabetic diet regime and was started on glimepride 2 mg od half hour prior to breakfast.

Patient’s glycemic control continued to worsen despite glimepride treatment. She was advised admission, was started on insulin and glimepride was stopped. Her symptoms and glycemic status improved during hospital stay. The histopathological examination showed a diffuse dermal neutrophilic infiltrate, edema of the papillary dermis, spongiform pustules; consistent with the diagnosis of Sweet’s syndrome. Within a week she became afebrile and the lesions started regressing and resolved completely over the next two weeks. Patient is under careful follow up and there is no evidence of disease recurrence, malignancy or vasculitis.

**DISCUSSION**

The entity of Sweet’s syndrome or Acute neutrophilic dermatosis was named after Dr Robert Douglas Sweet from Plymouth, England, who first illustrated this condition in 1964. It is neutrophil mediated hypersensitivity reaction in response to systemic illnesses. In a study from Iran the incidence of Sweet's syndrome was found to be 3 / 10,000 amongst new dermatologic patients². Middle aged women are more likely to develop this illness.

Sweet’s syndrome is distinguished by a group of clinical symptoms, signs and histological findings. These include abrupt onset of tender, red-violet papules, nodules and circinate or arcuate plaques asymmetrically located on upper limbs, face and neck. Usually they are accompanied by fever, flue like illness, conjunctivitis, arthalgias and peripheral neutrophilia. Histologically the hallmark is a diffuse infiltrate consisting predominantly of mature neutrophils typically located in the upper dermis and no evidence of leukocytoclastic vasculitis.

Sweet's syndrome presents in three clinical settings: classical (or idiopathic), malignancy-associated, and drug-induced³. Extracutaneous manifestations in the form of alveolitis, sterile osteomyelitis, renal, hepatic, and central nervous system involvement have also been reported⁴. Although it is not one of the common life-threatening dermatoses, Sweet’s syndrome can potentially cause significant pulmonary involvement and respiratory compromise and internists need to be aware of this condition.

Systemic corticosteroids are the mainstay of treatment. Other first-line options are potassium iodide and colchicine. Second-line drugs include indomethacin, clofazimine, cyclosporine, dapsone, pentoxifylline and doxycycline⁵. However spontaneous resolution is not uncommon as seen in our case. Recurrence may occur in one third patients despite appropriate treatment.

**References**

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