DRUG INDUCED SWEET’S SYNDROME – CASE PRESENTATION

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Abstract

Reports of Sweet’s syndrome associated with drug eruption are uncommon. We report a 50 years old female patient presenting numerous erythematous swollen papules and plaques on her upper extremities and sacral area for 10 days and drug-induced Sweet’s syndrome is discussed here.

Keywords:
drug eruption,
neutrophilic dermatosis,
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Introduction

Sweet’s syndrome (SS) was first described by Dr. Robert Douglas Sweet in 1964 (1). It can be classified upon the clinical setting in which it occurs: classical or idiopathic SS, malignancy associated SS, and drug induced SS (2). The drug-induced variant is quite rare. Less than 5% of all cases present with this type (3). The drug-induced variant of SS is frequently observed by administration of granulocyte-colony stimulating factor (GCSF) (3). Trimethoprim-sulfamethoxazole (4), carbamazepine, diazepam (5), furosemide (6) and nonsteroidal anti-inflammatory drugs such as diclofenac and celecoxib (7) commonly cause SS.

Case report

We are presenting a case report of a 50 years old female patient suffering from red plaques on her sacral region together with arthritis in her elbows and knees for 10 days. Her finger joints were swollen and painful. She had fatigue and hyperesthesia at her back. She didn’t have any similar rash in the past. She also had red painful plaques on her hands and arms. She didn’t have any history of diabetes, hypertension, contact with pets or insect bite.

On physical examination, the patient was found to have good nutritional status, a pulse rate of 98 beats per minute, blood pressure of 130/90 mmHg, a respiratory rate of 25 breaths per minute and temperature of 36°C. Laboratory tests disclosed an elevated erythrocyte sedimentation rate and anti-nuclear antibody positivity. All other systemic investigations were within normal limits. The patient was on treatment with acetylsalicylic acid as pain killer for two weeks together with naproxen sodium, doxycycline and colchycine. After 15 days there was no change in clinical findings. The drugs the patient had been taking regularly were: acetylsalicylic acid and rosuvastatin. On her dermatological examination there were erythematous, edematous, urticarial plaques located on her upper extremities, dorsal of the hands, palms, fingers, lower extremities, abdominal region and at her back. Her finger joints were painful with palpation (Figures 1-4). A skin biopsy was performed from the lesions located over the dorsal of the hands. Histopathologic examination revealed a normal squamous epithe-
lum with diffuse perivascular neutrophilic infiltrate, edema of the dermis with leukocytoclasia and diffuse infiltration with eosinophils and histiocytes. The diagnosis was acute febrile neutrophilic dermatosis or Sweet’s syndrome (Figures 5-6). Prednisolone 60 mg/day was prescribed. One week after the initiation of systemic steroid treatment the symptoms were resolved. After the treatment, because she had headache, she had had flurbiprofen 100 mg/day intake and then she referred to our clinic with the erythematous plaques again. When she totally stopped getting pain killers her skin lesions rapidly resolved on her follow up.

Discussion

Sweet’s syndrome or acute febrile neutrophilic dermatosis is characterized by fever, tender erythematous papules, nodules or plaques, neutrophilic leukocytosis, and infiltration of polymorphonuclear neutrophils in dermis (8, 9).

Sweet’s syndrome presents in several clinical settings: classical (or idiopathic) Sweet’s syndrome, malignancy associated Sweet’s syndrome, and drug-induced Sweet’s syndrome (2). Sweet’s syndrome predominantly affects women and usually is reported at the age of 30-60 years. There is no racial predilection reported so far.

The diagnosis of Sweet’s syndrome requires at least two major and two minor criteria. The major criteria are: abrupt onset of painful erythematous plaques or nodules and histopathologic evidence of a dense neutrophilic infiltrate without evidence of leukocytoclastic vasculitis. The minor criteria are: pyrexia >38°C, association with an underlying hematologic or visceral malignancy, inflammatory disease, pregnancy, upper respiratory or gastrointestinal infection or vaccination, relationship between drug ingestion and clinical presentation, or temporally-related recurrence after oral challenge,
excellent response to treatment with systemic corticosteroids or potassium iodide (2).

The syndrome may be associated with cancer as a paraneoplastic accompaniment, appearing as a first sign of malignancy, usually meaning a poor prognosis, infection (streptococcosis, salmonellosis and yersiniosis), inflammatory bowel disease, medications (G-CSF, lithium), autoimmune and collagen vascular disease (rheumatoid arthritis, Behcet’s disease), sarcoidosis, and pregnancy (2, 10).

Our case is a rare form of drug-induced Sweet’s syndrome with no relation of rheumatoid arthritis, tuberculosis or Crohn’s disease. The clinical differential diagnosis comprised flubipofen-induced Sweet’s syndrome (FISS), flubipofen-induced hypersensitivity syndrome and erythema multiforme (EM). Both FISS and flubipofen-induced hypersensitivity syndrome are rare idiosyncratic reactions in clinical presentation, such as malaise, neutrophilia, acute rash and other organ involvement. In our case, abrupt onset of painful erythematous plaques and a dense neutrophilic infiltrate in the absence of leukocytoclastic vasculitis demonstrated in histology which was consistent with Sweet’s syndrome. Absence of annular targetoid plaques and absence of interface dermatitis or keratinocyte necrosis excluded the possibility of EM.

Drugs are one of the main causes of Sweet’s syndrome. Among them, proteosome inhibitors, several anticancer agents, and dapsone are the most frequent drugs reported as a causative factor. Other potential drugs are indomethacin, clofazimine, cyclosporine, furosemide, and colchicine (3-10).

We report a rare form of drug-induced Sweet’s syndrome. FISS is a rare paraneoplastic phenomenon (11). The first case of flubipofen-induced Sweet’s syndrome has been recently described (12) and was associated with a paraneoplastic presentation (13).

Purpose of this report is to help clinicians recognize this syndrome.

Conclusion

An extensive literature search showed that only a small number of drug-induced Sweet’s syndrome cases occurred. Thus, we reported this case of drug-related Sweet’s syndrome to help clinicians recognize this syndrome.

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Patient consent was obtained.

Conflicts of interest: none declared.

Bibliography