BŁASZCZYK, Agnieszka, TROJAN, Sara, FUSSEK-STYGA, Urszula, KWIECIŃSKI, Jakub, MISZUDA, Sławomir, BASIAGA, Bartosz, BEDNARZ, Krzysztof, SZWEDKOWICZ, Agata, LEŚNIAK, Marek & HELUSZKA, Jakub. Sweet Syndrome - A Review of Pathogenesis, Clinical Features, Diagnosis and Treatment. Journal of Education, Health and Sport. 2023;30(1):45-56. eISSN 2391-8306. DOI http://dx.doi.org/10.12775/JEHS.2023.30.01.004
https://apcz.umk.pl/JEHS/article/view/43684
https://zenodo.org/record/7937706

The journal has had 40 points in Ministry of Education and Science of Poland parametric evaluation. Annex to the announcement of the Minister of Education and Science of December 21, 2021. No. 32343.
Has a Journal's Unique Identifier: 201159. Scientific disciplines assigned: Physical Culture Sciences (Field of Medical sciences and health sciences); Health Sciences (Field of Medical Sciences and Health Sciences).
Punkty Ministerialne z 2019 - aktualny rok 40 punktów. Załącznik do komunikatu Ministra Edukacji i Nauki z dnia 21 grudnia 2021 r. Lp. 32343. Posiada Unikatowy Identyfikator Czasopisma: 201159.
Przypisane dyscypliny naukowe: Nauki o kulturze fizycznej (Dziedzina nauk medycznych i nauk o zdrowiu); Nauki o zdrowiu (Dziedzina nauk medycznych i nauk o zdrowiu).

The authors declare that there is no conflict of interests regarding the publication of this paper.
Received: 24.04.2023. Revised: 10.05.2023. Accepted: 15.05.2023. Published: 15.05.2023.

Sweet Syndrome - A Review of Pathogenesis, Clinical Features, Diagnosis and Treatment

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Abstract

Sweet syndrome is a rare, inflammatory, non-infectious skin disorder characterized by an acute onset of skin leasions such as painful, erythematous plaques, nodules and papules, most frequently located on the upper extremities, trunk, neck and face. Generally, dermatological symptoms are accompanied by fever, headaches, arthralgia and leukocytosis. The syndrome belongs to the group of febrile neutrophilic dermatoses. Skin biopsy reveals a diffuse, neutrophilic infiltrate in the upper dermis. Sweet syndrome presents in three clinical subtypes: classical, malignancy-associated and drug-induced. The etiology of this disease still remains unclear, but it seems that can be associated with dysfunction of the immune system, genetic predispostion and neoplastic process. Sweet syndrome has been reported in relation to pregnancy, drug administration, vaccinaton and infection of the respiratory or digestive system. Systemic glucocorticosteroids still remain the first line treatment for most patients, nevertheless new therapeutic options against reccurent Sweet syndrome have been investigated.

Aim of the study

The aim of this literature review was to summarize the current state of knowledge about the etiopathogenesis, clinical features, diagnosis and treatment of Sweet syndrome. It is particularly important to make the proper diagnosis and implement the appropriate management due to possible association between Sweet syndrome and development of cancer.

Materials and methods

Standard criteria were applied to review the literature data. The search of articles in the PubMed and Google Scholar database was performed with using the following keywords: Sweet Syndrome, Acute Febrile Neutrophilic Dermatosis, Drug-induced Sweet's syndrome, Malignancy-associated Sweet's syndrome, Sweet's syndrome treatment, Pathergy Phenomenon, Neutrophilic dermatosis of dorsal hands. We have selected the articles that are most relevant to the topic.

Key words: *Sweet Syndrome, Acute Febrile Neutrophilic Dermatosis, Drug-induced Sweet's syndrome, Malignancy-associated Sweet's syndrome, Sweet's syndrome treatment, Pathergy Phenomenon, neutrophilic dermatosis of dorsal hands.*

Introduction

Sweet Syndrom, also known as ***„Gomm-Button disease"*** or ***„Acute Febrile Neutrophilic Dermatosis"***, is a rare, inflammatory, non-infectious skin disorder, first described in 1964 by Robert Douglas Sweet [1]. Dr Sweet investigated the case of eight women, who presented very similar clinical picture of a uknown disease. As a result of his research, it was possible to define the diagnostic criteria of the disease.

It is currently known that Sweet syndrome is characterized by a sudden onset of clinical symptoms and physical finding, which include pyrexia, neutrophilia and tender skin leasions such as plaques, papules and nodules. The classic histopathologic pattern of Sweet's syndrom reveals a diffuse infiltrate, consisting mainly of mature neutrophils, that are typically located in the reticular dermis. Sweet's syndrome usually presents in three clinical subtypes: classical (idiopathic), malignancy-associated and drug-induced. The exact prevalence of this disease is unknown.

Etiopathology

The etiopathogenesis of Sweet's syndrome may be multifactorial and still remains unclear. Clinical and laboratory evidence suggests that autoimmune diseases, neoplastic processes, infections and medication may have an etiologic role. There are multiple molecular mechanisms involved in development of Acute Febrile Neutrophilic Dermatosis, such as altered expression of inflammatory effector molecules, abnormal neutrophil function and genetic predisposition. In patients with an active phase of the disease there is reported higher level of cytokines and chemokines such as G-CSF, GM-CSF, IL-1, IL-2, IL-17 and interferon-gamma [5]. Elevated G-CSF levels have been shown to be associated with more severe course of the disease [25]. The significant role of cytokines in pathogenesis is confirmed by cases of Sweet's syndrome induction after administration of G-CSF and GM-CSF preparations [22-24].

It is suspected that same genetic factors increase susceptibility to the disease. It has been observed that HLA-B54 positive Japanese are more likely to develop neutrophilic dermatosis of the dorsal hands [26]. Furthermore, a connection between Sweet Syndrome and Familial Mediterranean Fever (FMF), caused by mutation in the MEFV gene, has been reported [27, 28]. Currently, there are many theories regarding the pathogenesis of acute febrile neutrophilic dermatosis. The most consistent version is that bacterial or tumor antigens trigger the activation and infiltration of neutrophils, leading to the onset of Sweet syndrom.

Clinical features

The typical clinical manifestations of Sweet syndrome are acute, tender, violaceous or erythematous, oedematous papules, nodules and plaques of different sizes in an asymmetric distribution. Sweet syndrome may present as a single lesion or multiple lesions, most frequently involving the upper extremities, neck and face [6]. However, various clinical variants have been reported in the course of this dermatosis, including localized neutrophilic dermatosis of dorsal hands, cellulitis, necroting lesions and blisters [29]. Neutrophilic dermatosis of the dorsal hands is an infrequent version of Gomm-Buttom disease, which responds very effectively to the treatment with glucocorticoids and is typically treatable [30].

Involvement of the oral cavity, mucous membranes and genitals is rare. The most common associated symptom is fever, which may precede the appearance of skin findings. The coexistance of pathergy, in which skin lesions develop even in areas of minor trauma, and photodistribution of skin leasion has been described in the literature [7].

Additional symptoms of Acute Febrile Neutrophilic Dermatosis include headaches, arthralgia, myalgia and fatigue. Occasionally, neutrophilic infiltration may involve central nervous system, heart, lungs, liver, intestines and muscles, which significantly increasing the risk of complications and mortality [8]. An example of respiratory involvement is the case of a 36-year-old woman diagnosed with Sweet's syndrom, who developed respiratory failure during the course of disease, requiring bilateral lung transplantation [9]. Further example of serious complication of this disease is myocardial involvement, including coronary artery occlusion, aortitis, cardiomegaly and aoratic stenosis [3].

Classical Sweet 's Syndrome

Classical or idiopathic Sweet's syndrom is the most common subtype of the disease and has a marked predilection for women in middle age. The female to male ratio of idiopathic SS is 4:1 [10]. Initial symptoms most frequently appear in people in their sixth decade of life. Rarely, it may occur in the pediatric population. Several cases of Sweet's syndrome in children have been reported in the literature [11][12].

Classical Sweet's syndrom is associated with infections, especially of the upper respiratory tract due to streptococci or gastrointestinal tract caused by salmonella and yersinia. It may also develop during pregnancy as a result of cellular, vascular and immunological changes associated with increased levels of estrogen and progesterone. Inflammatory bowel

disease and systemic lupus erythematosus are often related with the onset of Sweet's syndrome. Cases of acute febrile neutrophilic dermatosis have been also reported after vaccinations against SARS-COV-2 [13,14].

Drug-induced Sweet's syndorme

Drug-induced Sweet's syndrome develops mainly in patients treated with G-CSF (granulocyte-colony stimulationg factor), azathioprine, retinoids, anti-cancer medicines (bortezomib, imatinib, ipilimumab) and antibiotics (tetracyclines, trimethoprim-sulfamethoxazole) [5]. Predominatingly, skin lesions appear 5-7 days after initiation of treatment. However, after discontinuation of the causative drug, the symptoms of the disease often improve.

Paraneoplastic Sweet's syndrome

Malignancy-associated Sweet's syndrome accounts for approximately 20% of the disease cases and most frequently is associated with a poor prognosis for the patient. It may be the first symptom of a cancer, occur simultaneously or appear after oncological diagnosis. This subtype is mostly related with hematological disorders, especially acute myeloid leukemia (AML), Hodgkin's disease and polycythemia vera [3]. Less frequently it appears in case of patients with solid tumors, such as breast, gastrointestinal cancer or cervical cancer [31][32].

The paraneoplastic variant is characterized by a higher incidence of extracutaneous manifestations including those involving the eyes, kidneys, lungs and liver. This form of the disease may present with atypical, more severe and recurring cutaneous lesions such as blisters, ulceration and necrosis [33][34][35].

Diagnosis

The diagnosis of classial Sweet syndrom is based on diagnostic criteria proposed by Su and Liu in 1968 and later modified by von den Driesch (Table1) [16]. We distinguish two major criteria and four minor criteria. It's necessary to fulfil both major criteria and minimum two minor criteria to make the diagnosis. The diagnostic criteria in drug-induced cases of the disease were proposed by Walker and Cohen (Table2) [17]. All five criteria listed in table no. 2 must be met for the diagnosis of drug-induced Sweet's syndrome.

In order to establish the diagnosis, it can be essential to perform additional laboratory and imaging tests. Laboratory findings in patients with Acute febrile neutrophilic dermatosis

may reveal elevated markers of inflammation, including C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), peripheral leukocytosis with neutrophilia and hepatic and renal impairment. It is crucial to exclude the presence of infection, autoimmune disease, neoplasm and to obtain a precise drug-therapy and vaccination history. Additionally, to extend the differential diagnosis of Sweet's syndrome it may be reasonable to determine an antistreptolysin-O antibody titre, thyroid function and rheumatoid factor [3]. Sweet's syndrome requires differential diagnosis including viral, bacterial and fungial infections, autoimmune diseases such as systemic lupus erythematosus or sarcoidosis and also inflammatory and neoplastic diseases. In some cases, a skin biopsy can be necessary to establish the diagnosis. The skin biopsy should present the diffuse, dense neutrophilic infiltrate in the superficial dermis and specific edema of the dermal papillae [2].

In order to exclude the presence of a malignant disease imaging test such as chest radiographs, computerized axial tomography and SPECTs should be performed.

Table 1. Diagnostic criteria for classical Sweet's syndrome (by Liu and Siu, modified by von den Driesch) [16].

Major criteria	**Minor criteria**
1. Abrupt onset of painful erythematous plaques or nodules	1. Pyrexia > 38 °C
2. Histopathologic findings of a dense neutrophilic infiltrate without evidence of leukocytoclastic vasculitis	2. Association with an underlying hematological or visceral malignancy, inflammatory disease, or pregnancy, or preceded by an upper respiratory or gastrointestinal tract infection or vaccination
	3. Excellent response to treatment with systemic corticosteroids or potassium iodide
	4. Abnormalities in laboratory values (three of four): • erythrocyte sedimentation rate > 20 mm/h • positive C-reactive protein • leukocytosis > 8000 • neutrophilia > 70%

Table 2. Diagnostic criteria for drug-induced Sweet's syndrome (by Walker and Cohen) [17].

Diagnostic criteria - drug-related Sweet's syndrome
1. Abrupt onset of painful erythematous plaques or nodules
2. Histopathologic evidence of a dense neutrophilic infiltrate without evidence of leukocytoclastic vasculitis
3. Pyrexia > 38 °C
4. Temporal relations between drug ingestion and clinical presentation or temporally related recurrence after oral challenge
5. Temporally related resolution of lesions after drug withdrawal or treatment with systemic corticosteroids

Treatment

In general, systemic glucocorticosteroids remain the first line treatment for most patients. Usually, rapid resolution of skin lesions is observed after oral administration of prednisone at a daily dose of 40 mg to 60 mg (0,5-1,5 mg/kg/day) [2]. Treatment with topical corticosteroids of medium or high potency may also be effective in the local subtype of the diseases. It is important to emphasize that Sweet syndrome may resolve spontaneously. Rare examples of pregnancy-associated Sweet's syndrome, that resolved after delivery, have been reported in the literature [18]. In case of relapses of the disease or contraindications to corticosteroids, the administration of potassium iodide, colchicine, dapsone, doxycycline, indomethacinb and cyclosporine may prove effective. [19][20]. Human interleukin-1 receptor antagonists, including anakinra, can be used in treatment-resistant patients therapy. [21]

Conclusion

Sweet syndrome is rare dermatological disease belonging to the group of neutrophilic dermatoses. Making a proper diagnosis and promptly implementing appropriate treatment is of crucial importance for patients. The rapid resolution of disease manifestations contributes to a significant improvement in the quality of life. Moreover, Sweet syndrome may be the first sign of a malignant disease, whose early diagnosis improves the patient's prognosis. For these reasons every case of the disease should be considered individually. Systemic glucocorticosteroids still remain the main line of treatment, however therapeutic options should be personalized as much as possible. It is essential to take into consideration patient's

past medical history, coexisting diseases, contraindications, preferences and safety of treatment.

Disclosures

The authors received no financial support and declare no conflict of interest.

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