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Case report

Histiocytoid giant cellulitis-like Sweet's syndrome: case report and review of the literature

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Abstract

Background: Histiocytoid Sweet syndrome is an uncommon variant in which the dermal infiltrate is composed of mononuclear cells with a histiocytic appearance that represent immature myeloid cells. Giant cellulitis-like Sweet syndrome is a recently described variant characterized by relapsing widespread giant lesions.

Purpose: We report a unique patient with histiocytoid giant cellulitis-like Sweet syndrome and review the current literature on histiocytoid Sweet syndrome and giant cellulitis-like Sweet syndrome.

Material and Methods: We reviewed PubMed for the following terms and have reviewed the literature: histiocytoid, giant cellulitis-like, and Sweet syndrome.

Results: Six individuals, including our patient, have been reported with giant cellulitis-like Sweet syndrome; four had obesity, two had a hematologic malignancy, and one had breast cancer. Histiocytoid Sweet syndrome has been reported in association with autoimmune diseases, infection or inflammation, inflammatory bowel disease, malignancies, medications, and other conditions.

Conclusions: Histiocytoid Sweet syndrome is a rare variant of Sweet syndrome, often associated with malignancy. Giant cellulitis-like Sweet syndrome has been reported in six individuals; four of the patients were obese and three of the patients had an associated cancer. Our patient had histiocytoid giant cellulitis-like Sweet syndrome-associated myelodysplastic syndrome/myeloproliferative disorder. The diagnosis of histiocytoid Sweet syndrome or giant cellulitis-like Sweet syndrome should prompt the clinician to consider additional evaluation for a Sweet syndrome-associated malignancy.

Keywords: cellulitis, giant, histiocytoid, leukemia, Sweet, syndrome

Introduction

Giant cellulitis-like Sweet syndrome is a recently described form of acute febrile neutrophilic dermatosis [1]. Histiocytoid Sweet syndrome usually occurs in a paraneoplastic setting (Table 1) [2-26]. We describe a patient with histiocytoid giant cellulitis-like Sweet syndrome. We reviewed not only the previously reported patients with histiocytoid Sweet syndrome but also the previously described individuals with giant cellulitis-like Sweet syndrome.

Table 1. Characteristics of 64 patients with histiocytoid Sweet syndrome[$*]

AS	Symptoms	WBC (cells/uL)	Assoc disease	Treatment	Response [#]	Ref
14M	Fever, Malaise, Abdominal pain, Hematochezia	18.85	CD	Mpredn	Excellent	6
21M	Fever, Weight loss	2.61	None	Predn	Excellent	15
39M	Conjunctivitis, Lymphadenitis	NL	HL	Chemotx, filgrastim	Good	9
57M	Arthralgias, Myalgias, Dysesthesias	NR	MDS	Pred, Chemotx, Col, Dap, CP, PP, MMF	Good	11
57M	Pain	NL	Parotitis	Mpredn	Excellent	10
58M	Fever, Arthralgias	29.5	CML	Pred, nilotinib	Good	12
65M	Fever	+	None	Pred, CS	Excellent	2
67M	Fever, Pruritus	NL	MM, Bort	Dexa, tCS, AH	Excellent	13
69M	None	NL	MM, Bort	Mpredn, tCS	Excellent	16
71M	Malaise, Dizziness	6.78	MDS	Pred, Thal	Excellent	14
72M	Fever	1.4	MDS	Pred	Excellent	22
75M	Fever, Arth	4.2	MDS, PAN	Pred	Good	18
75M	Fever, Arth	11.1	PNA	Mpredn	Excellent	24
5F	None	NL	SLE	HQ	Excellent	4C2
9F	Abdominal pain	NL	SLE	Mpredn, Dap	Excellent	4C1
29F	Fever, edema	12.4	Preg	tCS	Excellent	3
42F	Fever, Arth, Diarrhea, Abdominal pain, Hematochezia	NL	CD	Pred, AZA, Dap	Excellent	20
44F	Fever	1.1	MDS Decitabine	tCS	Excellent with relapses	17
44F	Fever, myalgias	1.2	Sinusitis TMP-SMX	None	Excellent	25
59F	Fever	NR	RA	Pred	Excellent	23
68F	Fever, Altered mental status	1	MDS, AML	tCS, AH	Good	21
70F	Fever, Pain	13.4	LC	HU, HHT	Poor	26
72F	Fever, Foot pain	73.5	MDS/MPD	tCS	Excellent	CR

Abbreviations:

AH=oral anti-histamine, AML=acute myelogenous leukemia, Ane=anemia, AS=age (years) and sex, AZA=azathioprine, Bort=bortezomib, CD=Crohn's disease, Chemotx= chemotherapy, CML=chronic myelogenous leukemia, col=colchicine, CP=cyclophosphamide, CR=current report, CS=cyclosporine, dap=dapsone, dexa=dexamethasone, F=female, HHT= homoharringtonine, HL=Hodgkin's lymphoma, HQ=hydroxychloroquine, HU=hydroxyurea, LC=leukemia cutis, M=male, MDS/MPD=myelodysplastic syndrome/myeloproliferative disorder, MM=multiple myeloma, MMF=mycophenolate mofetil, mpredn=methylprednisolone, NL=normal, NR=not reported, PAN=polyarteritis nodosa, PNA=pneumonia, PP=plasmapheresis, pred=prednisone, predn=prednisolone, Preg=pregnancy, RA=rheumatoid arthritis, Ref=reference, SLE=systemic lupus erythematosus, tCS=topical corticosteroid, Thal=thalidomide, Thr=thrombocytopenia, TMP-SMX=trimethoprim-sulfamethoxazole, +=elevated

[$]The site of skin lesions and (number of patients) for the 23 individuals reported separately were:

Location (# of patients)	[References]
Upper extremity (16)	[2, 3, 4 (case 1), 6, 9, 10, 11, 12, 14, 17, 18, 20, 21, 22, 24, 26]
Trunk (13)	[2, 11, 12, 13, 14, 15, 16, 18, 20, 22, 23, 25, CR]
Lower extremity (10)	[2, 4 (case 1), 6, 11, 14, 17, 20, 21, 26, CR]
Head (10)	[3, 4 (case 2), 9, 10, 12, 15, 17, 18, 24, 26]
Neck (6)	[3, 9, 11, 13, 14, 20]
Buttock (1)	[4 (case 1)]

[*]These cases also include a series of 41 patients: 15 men and 26 women ranging in age from 29 years to 79 years. The site of skin lesions and (number of patients) were: trunk including abdomen and back (19), upper extremity including shoulder (19), hands including palms (12), lower extremity (7), face (2), feet including soles (1), and disseminated involvement of the whole body (1). Associated diseases included: B-chronic lymphocytic leukemia, breast carcinoma, chronic monocytic leukemia, conjunctivitis, erythema nodosum, eyelid edema, diabetes mellitus, immunosuppression, monoclonal gammopathy of undetermined significance, multiple myeloma, renal carcinoma, and ulcerative colitis [19].

[#]Excellent response defined as complete or near clearance of lesions, good response defined as lesion improvement without clearing, poor response defined as little or no response to treatment.

Case synopsis

A 72-year-old woman with a history of leukocytosis with marked neutrophilia, thrombocytopenia, daily fevers, and foot pain presented for evaluation of a new rash. Her unexplained foot pain had been treated with oral prednisone and her symptoms improved at higher doses. The prednisone dose was tapered to 20 mg daily and continued at this dose for the last two years; it was difficult to further taper the daily dose because of her foot pain. Nonetheless, another attempt to taper her prednisone dose to 17.5 mg daily was made three weeks prior to onset of rash. Two weeks after the taper was initiated, she developed fever with temperatures as high as 39.8 degrees Centigrade.

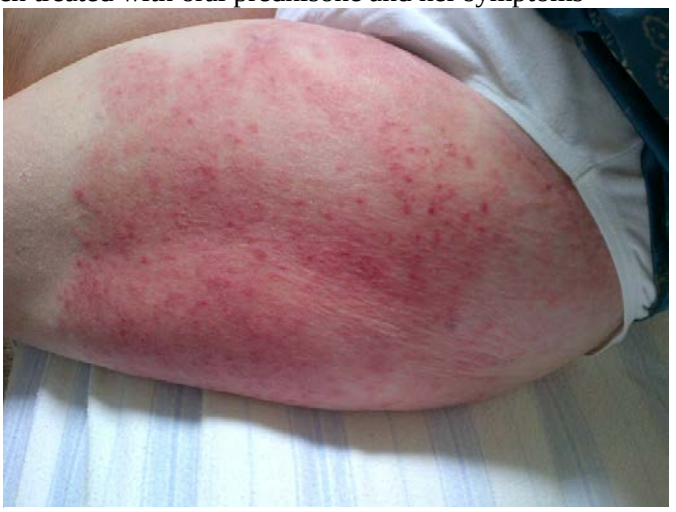
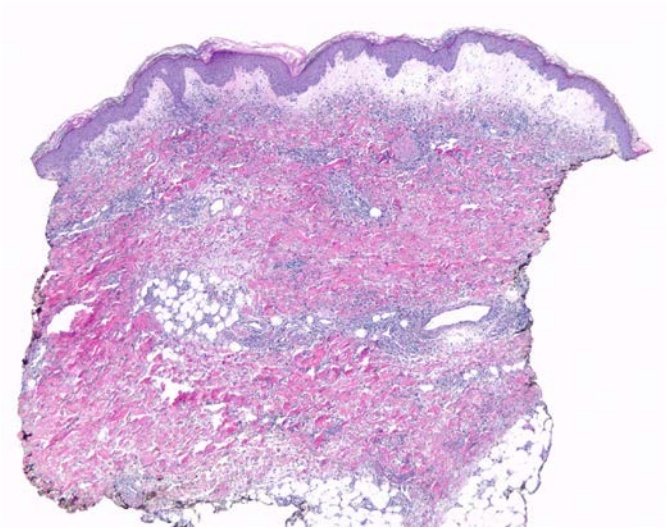
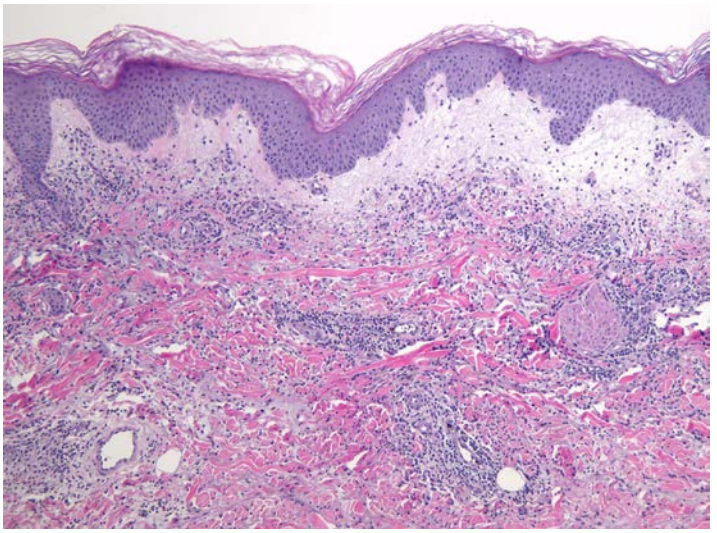


Figure 1.Clinical photograph of the erythematous plaque on her left lateral thigh.

Approximately three days prior to evaluation, the patient noticed the onset of a pink rash on her left upper leg that felt warm to the touch and slightly pruritic. Over the next few days, the rash extended in its involvement down the left leg and to the left flank. No preceding trauma occurred in the area and no topical treatments were attempted. No heating pads or cold packs were used. She had not experienced a recent streptococcal infection and she had no history of thyroid disease, lupus erythematosus, or inflammatory bowel disease.





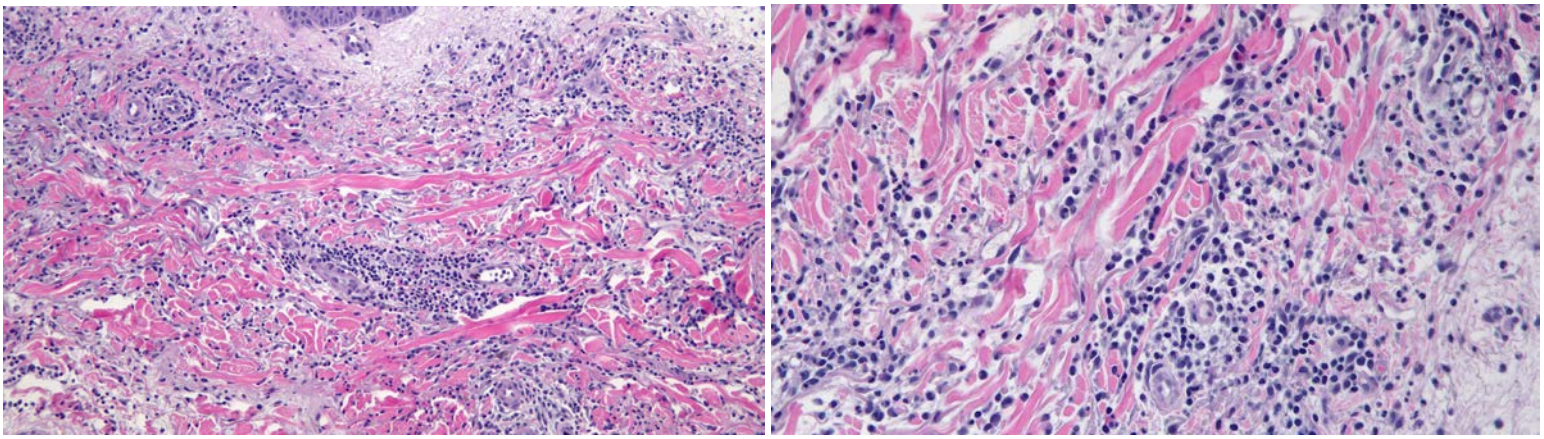


Figure 2. (a, b, c, d).Low (a), intermediate (b) and high (c, d) magnification views of the lesional skin biopsy. Prominent superficial dermal edema is noted, with a perivascular and interstitial inflammatory dermal infiltrate (a). Histiocytoid and immature granulocytic cells admixed with lymphocytes, eosinophils and occasional neutrophils are demonstrated on the higher magnification views (b, c, d) (hematoxylin and eosin: X4=a, X10=b, X40=c, X60=d).

Cutaneous exam revealed a 20 centimeter, non-tender, sharply demarcated erythematous plaque on her left lateral thigh containing a few scattered petechiae (Figure 1). Microscopic evaluation of the biopsy from the thigh plaque revealed a normal-appearing epidermis and prominent superficial dermal edema with a perivascular and interstitial inflammatory infiltrate of predominantly histiocytoid and immature granulocytic cells admixed with lymphocytes, eosinophils, and occasional neutrophils. Extravasated erythrocytes, hemosiderin-laden macrophages, and focal areas of vessel damage were also noted in the dermis (Figure 2).

Immunoperoxidase staining was performed to define the dermal infiltrate. CD68 staining (a marker for histiocytes) highlighted a majority of the interstitial cells (Figure 3). Myeloperoxidase staining (a marker for neutrophil granulocytes) also highlighted a majority of the interstitial cells, but with less intensity compared to CD68. CD117 staining (a marker for mast cells) highlighted rare interstitial cells.

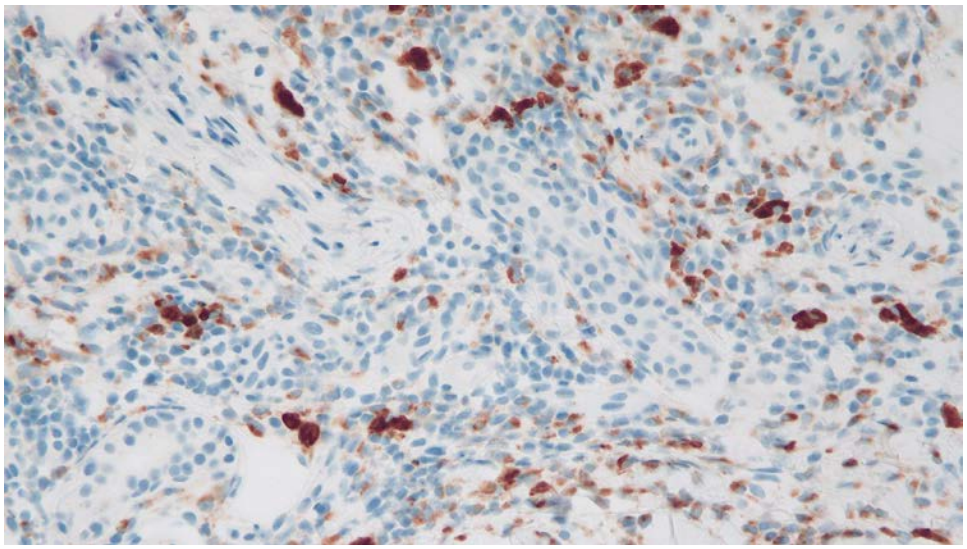


Figure 3. High magnification view show positive staining of the cells comprising the dermal infiltrate with CD68 (a histiocyte marker) (CD68: X40)

Tissue cultures from the left thigh were negative for bacterial, fungal, and mycobacterial organisms. Blood cultures were also negative. Urine culture grew *Klebsiella oxytoca* and she was treated with ciprofloxacin.

Laboratory studies showed that her white blood cell count was markedly elevated at 73.5 cells/uL (range: 4-10 cells/uL), showing predominantly neutrophils (66%) with an absolute neutrophil count of 58.1 cells/uL (range: 1.6-7 cells/uL). She was also found to be anemic with a hemoglobin of 10.6 gm/dL (range: 11.2-15.7 gm/dL) and platelets were decreased to 46 (range: 140-370 cells/mL). Prothrombin time, partial thromboplastin time, and international normalized ratio were normal. Lactate dehydrogenase was elevated at 362 U/L (range: 135-214 U/L). Complete metabolic panel was normal, as was her thyroid-stimulating hormone (0.84 uIU/ml, range: 0.27-4.2 uIU/mL).

Correlation of clinical presentation, lesion morphology, and histologic features led to a diagnosis of giant cellulitis-like (based on lesion features) and histiocytoid (based on pathology findings) Sweet syndrome. Topical clobetasol diproprionate 0.05% ointment was used twice daily, with significant improvement of her dermatosis in two weeks.

During the hospitalization, a bone marrow biopsy was also performed, which showed a hyper-cellular marrow with trilineage hematopoiesis, decreased and dysplastic megakaryocytes, but no evidence for an increased or aberrant immature cell population. In the setting of pancytopenia and with identification of several common myeloid malignancy genes on somatic mutation analysis,

she was diagnosed with an unclassified myelodysplastic syndrome/myeloproliferative disorder. She was treated with decitabine, with resolution of leukocytosis and dramatic improvement of fevers and foot pain. At time of report, there has been no recurrence of her Sweet syndrome for eight months.

Discussion

Sweet syndrome was described as an acute febrile neutrophilic dermatosis by Robert Douglas Sweet in 1964 [27]. The condition is characterized by the sudden onset of fever, leukocytosis, and tender, erythematous, well-demarcated papules and plaques. Sweet syndrome can be idiopathic. However, it can also follow an infection, including *Streptococcus* or *Yersinia*, or be associated with pregnancy, inflammatory bowel disease, medications, or malignancy [28].

Histology of lesional skin shows dense dermal neutrophilic infiltrates; vasculitis is usually minimal to absent. Classically, both the symptoms and skin lesions of Sweet syndrome have an excellent response to corticosteroids. Other treatments include colchicine, potassium iodide, and dapsone [27,28].

Clinical and histologic variants of Sweet syndrome have been described: neurologic Sweet syndrome [29], Sweet syndrome panniculitis [30], necrotizing Sweet syndrome [31] and Sweet syndrome concurrent with leukemia cutis [32]. More recently, histiocytoid Sweet syndrome and giant cellulitis-like Sweet syndrome have been reported [1,19].

Histiocytoid Sweet syndrome was initially described by Requena et al in 2005 [19]; subsequently, several additional observations of this unique variant of Sweet syndrome have been reported [5,7,25]. It is characterized by a dermal infiltrate composed of immature granulocytes that are histiocytic mononuclear cells, in contrast to the infiltrate of mature neutrophils typically seen in Sweet syndrome. Although the small cells of histiocytoid Sweet syndrome morphologically appear similar to neutrophils, they stain for CD15, CD43, CD45 (LCA), CD68, HAM56, lysozyme, and MAC 387, identifying a monocytic-histiocytic profile (Table 2) [19].

Table 2. Monocytic-histiocytic cell lineage antibody markers

Antibody	Specificity
CD15	Mature neutrophils, monocytes (promyelocytes)
CD43	Macrophages, myeloid cells
CD45 (LCA)	Granulocytes, macrophages, monocytes, all hematolymphoid cells
CD68	Basophils, macrophages, monocytes, myeloid precursors, neutrophils
HAM56	Macrophages, monocytes
Lysozyme	Granulocytes, histiocytes, macrophages, monocytes, myeloid cells
MAC 387	Granulocytes, monocytes, reactive macrophages

Abbreviations: LCA, leukocyte common antigen

Histiocytoid Sweet syndrome, to the best of our knowledge, has been described in 64 patients: 28 men and 37 women. Patients range in age from 5 years to 79 years (median: 59.5 years) at diagnosis; men range in age from 14 years to 79 years (median: 61.5 years) and women range in age from 5 years to 79 years (median: 55 years). The histiocytoid variant of Sweet syndrome has been associated with autoimmune diseases, malignancies, infections and inflammation, inflammatory bowel disease, medications, and other conditions (Table 3) [2-26]. The pathologic differential diagnosis of histiocytoid Sweet syndrome includes leukemia cutis and other inflammatory dermatoses histopathologically characterized by histiocytes interstitially arranged between dermal collagen bundles. Other diseases with this histologic picture include the interstitial type of granuloma annulare, interstitial granulomatous dermatitis with arthritis, and methotrexate-induced rheumatoid papules [19].

Table 3. Conditions and drugs associated with histiocytoid Sweet's syndrome

Autoimmune diseases
- Positive lupus erythematosus serologies [4,18,24]
- Rheumatoid arthritis [23]
- Systemic lupus erythematosus [4]

Infections/inflammation
- Conjunctivitis [19]
- Methacillin-resistant staphylococcus aureus (pneumonia) [24]
- Parotitis [10]
- Sinusitis [25]

Inflammatory bowel disease
- Crohn's disease [6,20]
- Ulcerative colitis [19]

Malignancies
- Acute myelogenous leukemia [21]
- Breast carcinoma [19]
- Chronic lymphocytic leukemia [19]
- Chronic monocytic leukemia [19]
- Chronic myelogenous leukemia [12]
- Hodgkin's lymphoma [9]
- Leukemia cutis (from acute myelomonocytic leukemia) [26]
- Lymphoma [19]
- Monoclonal gammopathy of undetermined significance [19]
- Multiple myeloma [13,16,19]
- Myelodysplastic syndrome [11,14,17,18,21,22]
- Myelodysplastic syndrome/myeloproliferative disorder [current report]
- Renal carcinoma [19]

Medications
- Bortezimab [13,16]
- Decitabine [17]
- Trimethoprim-sulfamethoxazole [25]

Other conditions
- Diabetes mellitus [19]
- Erythema nodosum [19]
- Eyelid edema [19]
- Glomerulonephritis [4]
- Hypertension [2]
- Immunosuppression [19]
- None [15, 19]
- Polyarteritis nodosa [18]
- Pregnancy [3]

Giant cellulitis-like Sweet syndrome is a morphologically distinctive clinical variant of Sweet syndrome characterized by relapsing widespread giant plaques. It was originally reported in three individuals with morbid obesity in 2013 [1]. Reports of two additional patients were published in 2014 [33,34]. To date, including our patient, giant cellulitis-like Sweet syndrome has been described in six individuals (Table 4) [1,33,34].

Table 4. Characteristics of patients with giant cellulitis-like Sweet's syndrome [*]

AS	Symptoms	WBC (cells/uL)	Other labs	Assoc dis	Tx	Response	Ref
62M	Fever Malaise	10.6 +	ANA – CRP +	Ob MM	Amox Pred	No effect from antibiotics; good response from oral prednisone	1C1
48F	Fever Malaise	24 +	CRP +	Ob	Pred	Good control	1C2
54F	Fever	4.1	Creat + TC –	Ob PBC Si	Pred	Excellent	33
60F^	Fever	10.3	TC –	–	Col Pred Dap	Response, but recurrence on colchicine and prednisone; excellent response to dapsone and	34

						prednisone	
68F	Fever Malaise	11.3 +	CRP + TC –	Br Ob	Surg Top	Transient improvement then recurrence with topical corticosteroid; remission after surgical treatment of breast carcinoma	1C3
72F	Fever Foot pain	73.5 +	LDH + TC –	MDS/ MPD	Top	Excellent	CR

Abbreviations: AS=age (in years) and sex, Amox=amoxicillin/clavulanic acid, ANA=antinuclear antibody, Assoc dis=associated disease, Br=breast carcinoma, Col=colchicine, CR=current report, CRP=C-reative protein, Creat=serum creatinine, Dap=dapsone, F=female, LDH=lactate dehydrogenase, M=male, MDS/MPD=myelodysplastic/myeloproliferative disorder, MM=multiple myeloma, Ob=obesity, PBC=primary biliary cirrhosis, Pred=prednisone, Ref=reference, Si=sicca syndrome, Surg=surgical treatment of breast carcinoma, TC=tissue culture, Top=topical corticosteroid, Tx=treatment, WBC=white blood cell, ^=patient was in her "60's", +=elevated, –=negative.

* The site of skin lesions and (number of patients) were:

Location (# of patients)	[References]
Lower extremity (6)	[1 (cases 1, 2, and 3), 33, 34, CR]
Trunk (5)	[1 (cases 1 and 2), 33, 34, CR]
Buttock (3)	[1 (cases 1 and 2), 33]
Upper extremity (2)	[1 (case 2), 33]
Head and neck (1)	[33]

Giant cellulitis-like Sweet syndrome has been observed in five women and one man. The median age at diagnosis was 62 years, ranging from 48 years to 68 years. The skin lesions most commonly occurred on the upper leg and buttocks. Four of the six patients were obese and three of the six patients had an associated malignancy: hematologic dyscrasia (multiple myeloma or myelodysplastic syndrome/myeloproliferative disorder), and breast cancer. The differential diagnosis of giant cellulitis-like Sweet syndrome includes not only cellulitis and other infections, but also periodic syndromes such as familial Mediterranean fever. Therefore, biopsy for histology, as well as tissue cultures, should be considered.

Conclusion

Histiocytoid Sweet syndrome is an uncommon variant in which histiocyte-like immature myeloid cells compose the dermal infiltrate. Giant cellulitis-like Sweet syndrome is a rare clinical variant characterized by relapsing widespread giant plaques on the leg and buttocks of middle-aged women. Our patient is unique in having both of these unusual variants. When the diagnosis of histiocytoid Sweet syndrome is entertained, leukemia cutis should be excluded and when the diagnosis of giant cellulitis-like Sweet syndrome is suspected, biopsy for histology and tissue culture should be considered. .

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