Case Series

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Neutrophilic Dermatoses: A Case Series of Unusual Adverse Effects with Commonly Administered Drugs

Dermatology Section

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ABSTRACT

Neutrophilic Dermatoses (NDs) are inflammatory conditions characterised by sterile neutrophilic infiltrates on histopathology. Multiple clinical presentations can occur in a single patient with ND. The location of the neutrophilic infiltrate (in the skin's superficial, deeper, or subcutaneous layers), the clinical features, and the duration of the disease help to identify ND. Robert Douglas Sweet introduced the term neutrophilic dermatoses in 1964 to describe febrile neutrophilic dermatoses, now known as Sweet syndrome. The major conditions in this group include Pyoderma Gangrenosum (PG), Sweet Syndrome (SS), subcorneal pustular dermatoses, Generalised Pustular Psoriasis (GPP), and Inflammatory Bowel Disease (IBD)-associated neutrophilic dermatoses. The present case series described five female patients, aged 32 to 85, who developed painful, red, pus-filled skin lesions after taking common medications such as aceclofenac, diclofenac, and isotretinoin. The cases included Neutrophilic Eccrine Hidradenitis (NEH), Sweet syndrome, and PG, with symptoms such as fever, sore throat, and arthralgia. Skin biopsies confirmed the diagnoses, showing inflammatory changes, dermal oedema, and features such as vascular injury. Blood tests consistently showed neutrophilia and elevated inflammatory markers {Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP)}. Management consisted of stopping the suspected offending medications and initiating corticosteroid therapy, which led to improvement in symptoms. This case series highlights the importance of promptly identifying drug-induced neutrophilic disorders to avoid misdiagnosis, inappropriate treatment, and potential complications. Heightened awareness among clinicians is essential to facilitate timely diagnosis and effective management of these rare yet significant dermatological reactions. Hence, the case series aimed to summarise therapeutic innovations related to the principal neutrophilic dermatoses.

Keywords: Autoinflammatory, Pyoderma gangrenosum, Sweet syndrome

INTRODUCTION

Neutrophils are critical inflammatory cells, accounting for 40-70% of the total white blood cell population. They promptly accumulate at sites of infection or injury, where they ingest, eliminate, and degrade pathogens using specialised proteins and reactive oxygen species [1]. Nonetheless, the accumulation of neutrophils without a discernible cause can damage healthy tissue. Neutrophilic dermatoses (NDs) are a group of skin conditions in which mature neutrophils invade the skin in the absence of infection [1,2]. The clinical presentation may vary and include vesicles, papules, nodules, or ulcerations, as well as extracutaneous involvement. Because there are many similarities in their history, symptoms, and tissue features, diagnosing the different types of ND can be difficult, especially in hospitalised patients who have other inflammatory issues or related conditions [3]. In this case series, authors aimed to illustrate and analyse the occurrences of ND.

Case 1

Neutrophilic Eccrine Hidradenitis (NEH): An 85-year-old female was referred to the Outpatient Department (OPD) with painful red lesions that had persisted for three days on both palms and difficulty sleeping. The patient had a history of intermittent back pain lasting eight years, with aggravation noted over the past month. She had been treated with aceclofenac 100 mg for one month. The patient reported that this was her first exposure to aceclofenac and indicated no prior lesions. Twelve days after starting treatment, she presented with painful, reddish lesions on the tips of her fingers, which subsequently progressed to involve the palms of both hands. The lesions progressed to pustules within three days. The patient reported an inability to flex the interphalangeal joints, accompanied by fever and neck pain lasting three days. She reported no other medical problems. Her mother had leukaemia. The preliminary examination revealed multiple plaques and vesicles filled with pus, along with erythema and oedema on both palmar surfaces [Table/Fig-1]. Inflammation was present. No scales or discharge were noted, and the nails, mucous membranes, and genitalia were unaffected. The physical examination revealed painful pustules. Blood tests, including C-Reactive Protein (CRP), Absolute Eosinophil Count (AEC), and Complete Blood Count (CBC), showed that the neutrophil count was 86.6% and the AEC was 470 cells/µL [Table/Fig-2]. The Tzanck smear showed predominantly neutrophils with occasional lymphocytes [Table/Fig-3a].

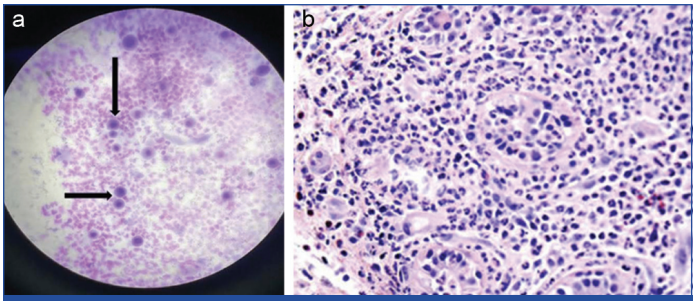
[Table/Fig-1]: Multiple erythematous plaques and vesicles with pustular lesions on the palmar surfaces, exhibiting erythema and oedema.

Degenerated cells were observed in certain regions against a proteinaceous background. A 3 mm punch biopsy was performed on the left palm.

Complete Blood Count (CBC)	Case 1	Case 2	Case 3	Case 4	Case 5
Haemoglobin (Hb)	10.3 g/dL	11.1 g/dL	10.7 g/dL	12.4 g/dL	11.1 g/dL
RBC (Red Blood Cells)	4.25 million cells/micro liter	4.03 million cells/micro liter	4.08 million cells/micro liter	4.36 million cells/micro liter	4.03 million cells/micro liter
WBC (White Blood Cells)	20,000/µL	27,500/µL	19,000/µL	22,000/µL	19,400 cells/µL
Neutrophils	86.6%	85.6%	78.9%	83%	78.6%
Monocytes	7.1%	7.2%	7%	7.3%	72%
Eosinophils	2.9%	2.8%	2.9%	2.1%	2.8%
Basophils	0.1%	0.2%	0.1%	0.2%	0.2%
Lymphocytes	12%	11.2%	10.2%	10%	11.2%
Platelet count	3.1 lac/ microliter	3.6 lac/ microliter	3.0 lac/ microliter	3.3 lac/ microliter	3.6 lac/ microliter
Erythrocyte Sedimentation Rate (ESR)	50 mm/1st hour	51 mm/1st hour	48 mm/1st hour	49 mm/1st hour	52 mm/1st hour
C-Reactive Protein (CRP)	111.4 mg/L	126.1 mg/L	102 mg/L	118 mg/L	112.3 mg/L
Absolute Eosinophil Count (AEC)	320 cells/µL	450 cells/µL	390 cells/µL	460 cells/µL	400 cells/µL

[Table/Fig-2]: Blood investigations of study subjects.

Given the diverse clinical presentations, a provisional diagnosis of NEH was established. Differential diagnoses include erythema nodosum and erythema multiforme. Histopathological examination {Haematoxylin and Eosin (H&E)} demonstrated inflammatory neutrophilic infiltration around the deeper regions of the eccrine glands, accompanied by dermal oedema [Table/Fig-3b]. Periodic Acid-Schiff (PAS) staining yielded a negative result for infection. The data supported a diagnosis of NEH.



[Table/Fig-3]: a) Tzanck smear showing neutrophils (Leishman stain 200x); b) Inflammatory neutrophilic infiltrate surrounding the deep part of the eccrine glands and oedema of the dermis (H&E 200x magnification).

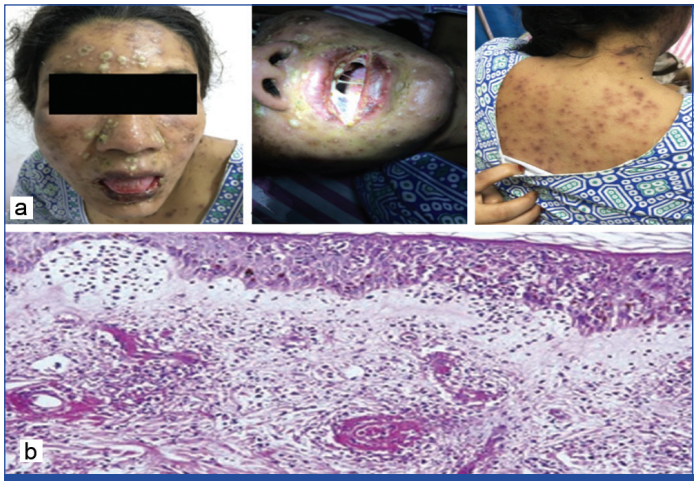
The patient was treated with dapsone 100 mg daily and followed for four weeks. The patient showed significant clinical improvement with complete resolution of skin lesions and achieved full recovery during the follow-up period.

Case 2

Sweet syndrome: A 32-year-old female presented to the outpatient clinic with painful facial pustules/lesions characterised by purulence, which had been ongoing for approximately one week. The patient reported an initial burning sensation in the lesions, which was subsequently accompanied by fever and pharyngitis lasting one week.

In reviewing her medical history, she revealed that isotretinoin had been prescribed for nodulocystic acne by a local clinician a month earlier. Three days after starting isotretinoin, the skin lesions became visible. The patient indicated that the lesions deteriorated quickly with continued use of the medication, and the oral cavity and trunk were subsequently affected. The patient has no history of other medications and no family history of Sweet syndrome.

Upon physical examination, multiple distinct pustules accompanied by erythematous plaques and nodules were noted on the face and within the oral cavity [Table/Fig-4a]. The lesions varied in size, ranging from 1 cm to 2 cm. Systemic examination revealed fever and pharyngitis. The patient was diagnosed with Sweet syndrome. The provisional diagnosis was supported by the acute onset of painful, erythematous, pustular and nodular skin lesions, systemic symptoms including fever and pharyngitis, and recent exposure to isotretinoin, a known drug trigger. Differential diagnoses encompassed dermatomyositis and thyroiditis. Histopathology revealed intraepidermal pustules, leukocytoclastic vasculitis, and dermal oedema, which are typical features of Sweet syndrome [Table/Fig-4b]. Laboratory findings showed neutrophilia and elevated CRP, reinforcing the inflammatory aetiology. The diagnosis of Sweet syndrome was made.



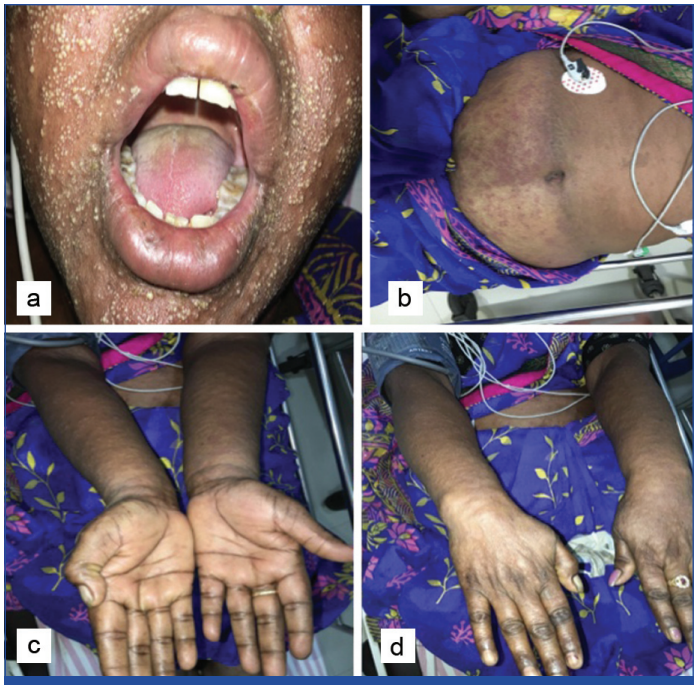
[Table/Fig-4]: a) Multiple, discrete, pustules with erythematous plaques and nodules over the face; oral cavity and upper back; b) Intraepidermal pustule formation with leukocytoclastic vasculitis and dermal oedema (H&E, 200x magnification).

Prednisone was initiated at a dose of 40 mg daily and gradually tapered over four weeks to minimise potential complications. The patient was closely monitored through regular physical examinations and demonstrated complete recovery following treatment.

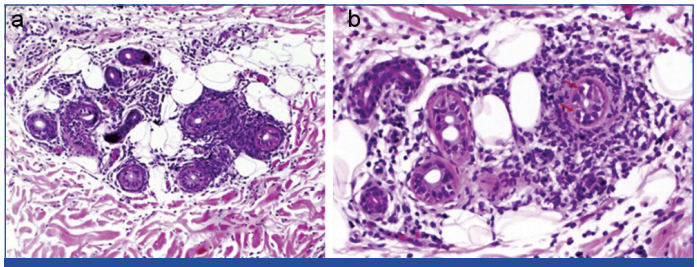
Case 3

Sweet syndrome: A 52-year-old female presented to the OPD with painful, pus-filled lesions on the face, abdomen, and hands that had been present for approximately five days. She also reported pain in the lesions and had fever and sore throat for five days. Review of her medical history revealed that she had been prescribed diclofenac 100 mg to address fever and joint pains by a local healthcare unit. The skin lesions appeared two days after starting the medication and progressively deteriorated despite treatment. The condition subsequently involved the abdomen and oral mucosa. The patient reported no family history of Sweet syndrome.

Upon physical examination, multiple discrete pustules and erythematous plaques were observed on the face, as well as the extensor and flexor aspects of both arms and forearms, and the abdomen. Systemic examination revealed fatigue [Table/Fig-5a-d]. The provisional diagnosis of Sweet syndrome was established based on distinct clinical features, including the sudden appearance of painful erythematous skin lesions (such as papules, nodules, or plaques), frequently accompanied by fever and additional symptoms such as malaise, arthralgia, and myalgia. The differential diagnosis included lymphoma cutis and Behçet's disease. Laboratory findings included leukocytosis with neutrophilia, and elevated ESR and CRP. The histopathological examination demonstrated neutrophilic infiltration around the eccrine glands, with partial necrosis of the secretory epithelium [Table/Fig-6a,b]. The conclusive diagnosis was Sweet syndrome.



[Table/Fig-5]: Multiple, discrete, pustules and erythematous plaques present over the: a) face; b) abdomen; c) flexor; and (d) extensor aspect of both arms and forearms.



[Table/Fig-6]: a) Neutrophils are surrounding eccrine secretory cells; b) Partial necrosis of the secretory epithelium indicated by arrows.

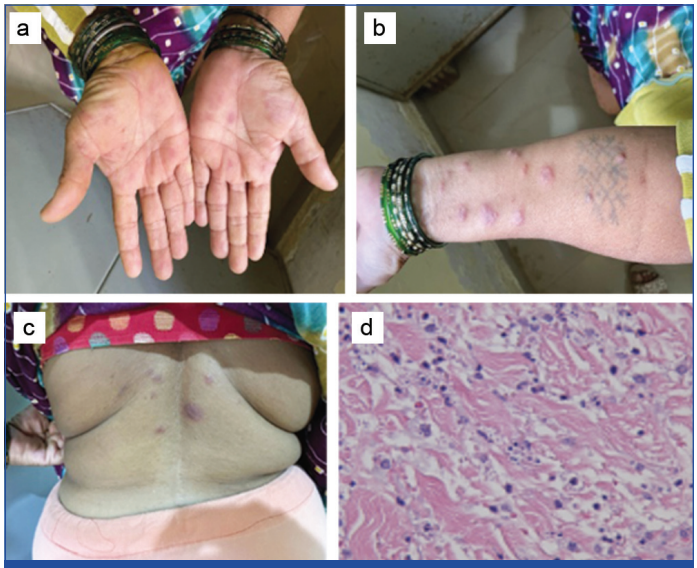
Prednisone was initiated at a dose of 60 mg daily and gradually tapered over four weeks to reduce the risk of potential complications. The patient was regularly monitored through physical examinations and showed complete clinical recovery following treatment.

Case 4

Sweet syndrome: A 48-year-old female presented to the Dermatology department with a complaint of painful red lesions on her hands for the past three days. She reported a burning sensation in the areas of the lesions and had been prescribed Aceclofenac 100 mg tablets for joint pains by a local healthcare unit, after which the skin lesions began to manifest two days after initiation of the medication. The patient reported that the lesions progressively worsened with ongoing medication use and ultimately spread to the hands and lower back. The patient had no prior history of adverse habits and a family history of Sweet syndrome.

Upon physical examination, multiple discrete erythematous plaques and papules were noted on both palms, the flexor surfaces of both forearms, and the lower back [Table/Fig-7a-c]. A provisional diagnosis of Sweet syndrome was made based on the clinical characteristics, including the sudden appearance of painful erythematous skin lesions (such as papules, nodules, or plaques), frequently accompanied by fever and additional symptoms such as malaise, arthralgia, and myalgia. A raised CBC, ESR, and CRP were among the laboratory findings. By ruling out other neutrophilic dermatoses and infections, the data were consistent with Sweet syndrome.

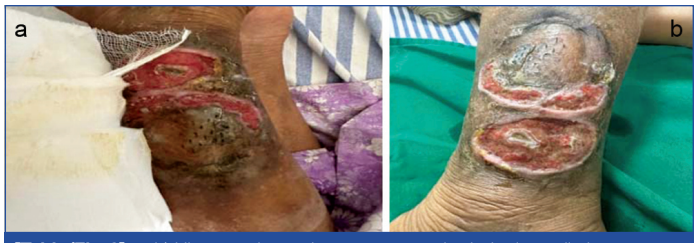
Histopathological examination revealed superficial and deep perivascular and interstitial infiltrates, primarily composed of neutrophils and eosinophils [Table/Fig-7d]. A diagnosis of drug-induced Sweet syndrome was established on the basis of the clinical presentation and the skin biopsy. Prednisone at a dose of 60 mg daily was given, and with each subsequent dose, the severity of the symptoms was effectively reduced. In the course of follow-up sessions, the patient made a full recovery.



[Table/Fig-7]: Multiple, discrete, erythematous plaques and papules present over: a) both palms; b) the flexor aspect of both forearms; and (c) the lower back; d) Histopathological examination revealed superficial and deep perivascular and interstitial infiltrates, primarily composed of neutrophils and eosinophils (H&E stain, 400X).

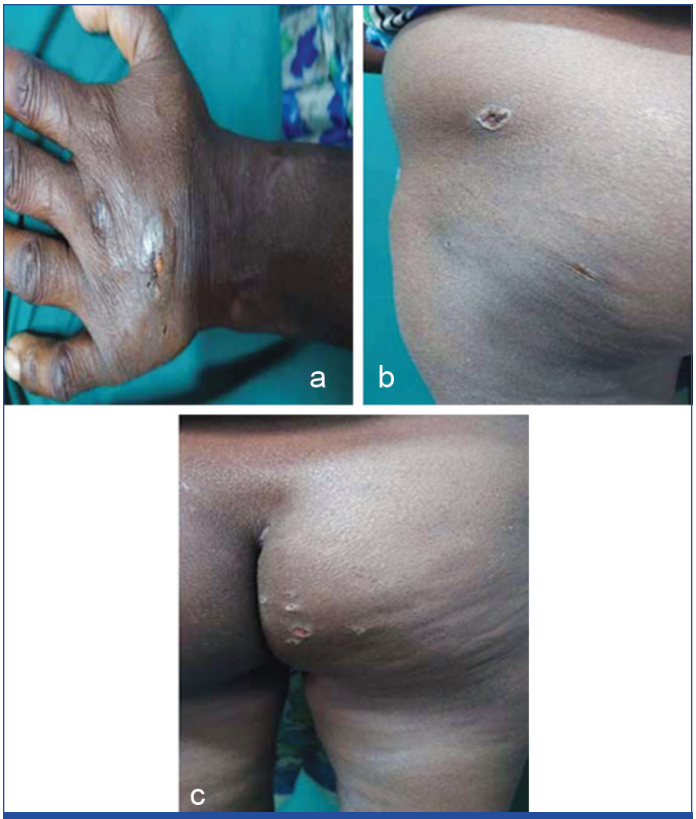
Case 5

Pyoderma Gangrenosum (PG): A 59-year-old female presented to our Dermatology department with a painful ulcer on the right lower limb. One month earlier she was well and took aceclofenac 100 mg for three days for joint pains; following this treatment, she developed a low-grade, continuous fever that was not associated with chills or rigors. She experienced a rapidly progressive and painful destructive ulcer on the right lower limb within two days. On physical examination, two ulcers measuring 5×4 cm and 6×5 cm were observed, characterised by defined margins and violaceous undermined borders. The floor of the ulcers was covered with yellowish slough and granulation tissue, located on the anterolateral aspect of the right lower limb [Table/Fig-8a,b]. Numerous hyperpigmented macules of differing sizes were observed on both lower limbs. Swollen patches with a sore and crusty center were seen on the dorsum of the left hand {where (intravenous) i.v. access could have been placed but wasn't)} and on both buttocks (sites of multiple injections), showing a positive pathergy phenomenon [Table/Fig-9a-c]. No local increase in temperature was observed. No significant past medical history was reported.

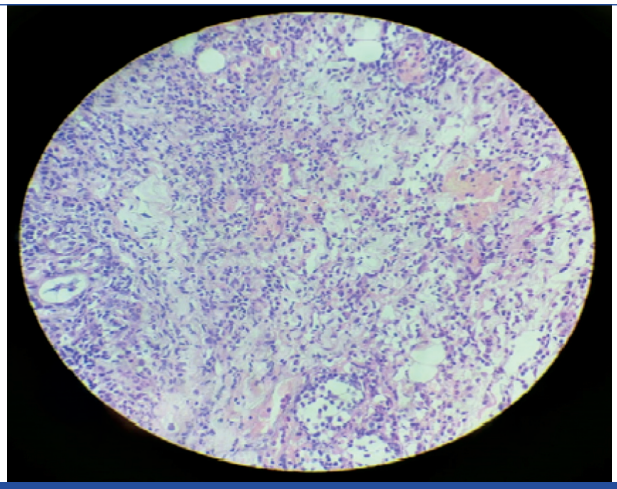


[Table/Fig-8]: a,b) Ulcers and macules present over botht he lower limbs.

A provisional diagnosis of PG was made based on the characteristic clinical presentation of a rapidly progressing, painful ulcer with a violaceous undermined border. The differential diagnosis included bacterial, mycobacterial, fungal, and viral infections. CRP and ESR levels were significantly elevated. A biopsy obtained from the margin of the ulcer, which also encompassed a healthy segment of the lower lip, revealed orthokeratosis, parakeratosis, and glycogen within epidermal cells, while the underlying tissue showed a significant infiltrate of inflammatory cells [Table/Fig-10]. Dense inflammatory



[Table/Fig-9]: a-c) Pathergy phenomenon.



[Table/Fig-10]: Histopathological examination showing orthokeratosis, parakeratosis, glycogenation of cells and sub epithelial connective tissue showed dense mixed inflammatory cell infiltrate (H&E stain, 200X).

cell infiltrates were noted infiltrating mucinous glands and ducts as well. A concentrated aggregation of neutrophils resulting in microabscess formation was also observed. The identified characteristics suggested PG after ruling out other conditions. The diagnosis of PG was made on the basis of clinical presentation, biopsy findings, and guidelines from Maverakis E et al., [4]. The patient was treated with saline compresses on the ulcer areas, intravenous antibiotics, intravenous steroids, topical triamcinolone paste, and chlorhexidine mouthwash to prevent mouth sores. The patient recovered completely after treatment.

DISCUSSION

Neutrophilic dermatoses (ND) exhibit variability in clinical presentation and underlying causes, making histological evaluation essential for accurate diagnosis. Studies have clarified the multifactorial aetiology of ND, which includes aberrant neutrophil activity, inflammasome activation, malignant transformation involving dermal neutrophils, and genetic predisposition, all contributing to the condition. With emerging data, novel therapies for ND are forthcoming [1-3]. ND may be induced by several factors, including infections, underlying systemic diseases (including inflammatory bowel disease or haematologic cancers), and certain drugs [5]. Medications have the potential to induce severe manifestations of ND. Drug-induced ND has been documented, including cases linked to aceclofenac; some reports also describe ND in relation to granulocyte colony-stimulating factor therapy [6]. These dermatoses can present with complex symptoms, making diagnosis challenging since they may resemble other inflammatory and infectious skin disorders [6]. Timely diagnosis and intervention are crucial, since delayed treatment may result in significant distress, morbidity, and chronicity.

The treatment of neutrophilic dermatoses often involves immunosuppressive medications, such as corticosteroids, and, where applicable, the elimination of known triggers, including certain medications or underlying conditions [7]. Understanding the pathogenesis, diagnostic criteria, and potential precipitating factors of ND is essential for appropriate patient management. The predominant type of ND identified in this study was Sweet Syndrome, which was characterised by tender, erythematous skin lesions accompanied by fever and leucocytosis [8]. A skin biopsy is typically required for the diagnosis of Sweet Syndrome. Histology shows widespread neutrophilic infiltration in the skin, with varying degrees of dermal oedema. Neutrophils can extend through the dermis and may involve hair follicles and sweat glands, as well as perivascular and periadnexal areas [9].

Erythema elevatum diutinum, PG, eccrine neutrophilic hidradenitis, neutrophilic panniculitis, Sweet Syndrome (acute febrile neutrophilic dermatosis), and Sneddon-Wilkinson disease were initially distinguished from this category [10]. Marshall syndrome is an extremely uncommon condition that affects children and is characterised by loss of elastic tissue (cutis laxa) as a result of acquired, localised neutrophilic dermatitis. This condition does not affect internal organs. Marshall syndrome, also known as acquired cutis laxa type II, has been reported in only a small number of patients [11]. A rare case report describing acquired cutis laxa (Marshall syndrome) secondary to Sweet Syndrome in a child has been reported from Gujarat, India [12]. Further investigation by Nithin Rajan R et al., identified a rare case in Thiruvananthapuram, Kerala, subsequently diagnosed as Sweet Syndrome. Histology revealed the epidermis with focal mild acanthosis, parakeratosis, and spongiosis. The reticular dermis showed neutrophilic infiltration in both the perivascular and interstitial regions. Areas of oedema with leukocytoclasis were observed. Some sections demonstrated perivascular lymphohistiocytic infiltration, with occasional eosinophils. Mild neutrophilic exocytosis was also observed. This case differed clinically from those observed in authors' study cases [13].

Screening for Sweet Syndrome is recommended due to various associated pathologies. This encompasses assessment of blood pressure, along with laboratory evaluations such as haemogram, ESR, Lactate Dehydrogenase (LDH) and uric acid levels. Additionally, testing for Human Immunodeficiency Viruse (HIV), Antinuclear Antibodies (ANA), and serum immunoglobulin levels is recommended. A wide range of medications has also been implicated in the development of Sweet Syndrome, including antibiotics, antineoplastic agents, antiepileptics, and colony-stimulating factors [14]. The current case series also observed a role for antibiotics in the development of Sweet Syndrome. As a treatment for Sweet Syndrome, systemic steroids and dapsone have been shown to be very effective. Dapsone can be used in the acute phase of Sweet Syndrome to control swelling, although no therapy is curative for the underlying condition [15].

The other type of ND in this study was PG. The first description of PG was by Brocq in 1916 as "phagedenisme geometrique," and later named by Brunsting et al.; PG has since been considered the dissemination of a distinct focus of infection [4,16-20]. Clinically, PG can mimic or complicate other cutaneous ulcerative diseases

such as vasculitic and venous ulcers, infections, lymphoma, leukaemia and other ND such as atypical Sweet Syndrome. The histopathological distinction of PG from ulcerative diseases is a neutrophilic infiltrate at the edge of the ulcer [21]. PG may show fibrosis, vascular proliferation, and ulceration regardless of the cause. Histopathological findings vary by type, duration, and biopsy site. PG primary lesions can resemble Sweet Syndrome [22]. This investigation found severe dermal infiltration, leukocytoclastic vasculitis, and vasculopathy in both Sweet Syndrome and PG. However, vascular proliferation and fibrosis can indicate PG. Dermal oedema was occasionally seen in PG specimens. Previous investigations have shown that PG can cause localised vasculitis in well-developed lesions [23].

CONCLUSION(S)

This series emphasises that commonly used medications can induce ND. Increased awareness among dermatologists is crucial to effectively recognise and manage these rare adverse effects.

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