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Case Series

Dermatology Section

A Case Series of Amicrobial Pustulosis of Folds: An Eye-opener for the Diagnosis of Autoimmune Disorders

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ABSTRACT

Amicrobial Pustulosis of Folds (APF) is a rare, chronic, relapsing cutaneous disease seen exclusively in younger women with a history of autoimmune disease, most commonly Systemic Lupus Erythematosus (SLE), or who simply have circulating autoantibodies. This case series highlights the occurrence of an unusual manifestation of APF as a marker of autoimmune disorders and emphasises the importance of double-stranded Deoxyribose Nucleic Acid (dsDNA) positivity in such cases. A total of five female patients with APF were included in this case series. All five cases presented with pustular lesions lasting 10-12 weeks, involving conchal bowls, eyes, perineal, and perianal regions, either as an initial presentation or as a flare-up of pre-existing autoimmune disease. All patients underwent screening for autoimmune disorders, which revealed dsDNA positivity and met the diagnostic criteria for APF. Therefore, a diagnosis of APF with SLE was made. All patients showed significant improvement with oral steroids. Although SLE is a complex multisystem disorder where patients may not always present with malar rash, photosensitivity, arthritis, and arthralgia, APF could serve as an eye-opener for diagnosing underlying autoimmune diseases.

Keywords: Neutrophilic dermatosis, Pustular dermatosis, Sterile pustules, Systemic lupus erythematosus

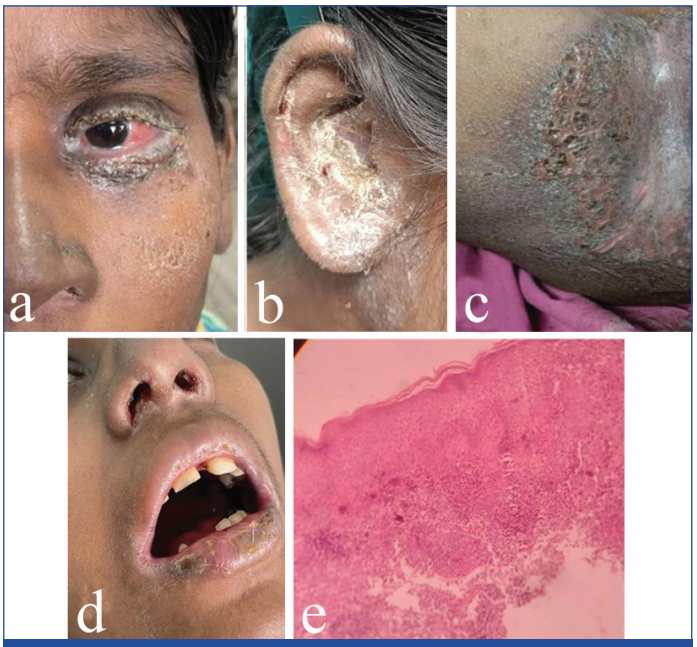
INTRODUCTION

Amicrobial pustulosis of the folds is a rare neutrophilic dermatosis seen predominantly in young adult female patients with underlying autoimmune or rheumatic disease. APF is characterised by recurrent crops of pustules primarily in the skin folds and periorificial regions that eventually coalesce into plaques [1]. It is characterised by the sudden onset of chronic, relapsing small follicular and non follicular pustules on an erythematous base, coalescing to form erosive plaques in patients with underlying autoimmunity. Associated with a wide variety of dermatoses, this entity was first reported in 1991 by Antille C et al., [2]. In 2008, Marzano AV et al., outlined diagnostic criteria for APF that included major and minor criteria. The major criteria include pustulosis affecting one or more of the major folds or one or more minor folds in the anogenital area, histopathology showing intraepidermal spongiform pustules and a predominantly neutrophilic infiltrate in the dermis, negative culture of an intact pustule. The minor criteria include association with one or more autoimmune disorders, Antinuclear Antibody (ANA) titres of 1/160 or more, presence of autoantibodies such as Extractable Nuclear Antigens (ENA), anti-DNA, antismooth muscle, antimitochondrial, antigastric parietal cell, or endomysial antibodies [3]. Clinically, these patients experience constitutional symptoms such as fever, arthritis, arthralgia, myalgia, sudden appearance of pustules, along with signs of flare-up of mucocutaneous disease manifestations like bullous SLE, acute palmar telangiectasia. The present case series entails five cases of APF associated with underlying autoimmune disorders, which can be an eye-opener for diagnosing such diseases.

Case 1

A 31-year-old female came to the Dermatology OPD with a chief complaint of fluid-filled vesicles and pustules over the genital region, gluteal cleft, and axilla. She also had a painful erosion over the lip for the past four months, along with intermittent gingival bleeding and discharge from the left ear and left eye for the past three months. There was no history of drug intake, topical application of any drugs, or any medical co-morbidities. No family history of similar complaints was noted. The patient had previously been evaluated at another institution three months ago and was diagnosed with mucosal pemphigoid, acute mucopurulent conjunctivitis, and Chronic Suppurative Otitis Media (CSOM). She was treated with antibiotics and other supportive measures for two weeks, but without any relief. Since the lesions recurred, the patient sought further evaluation at the Outpatient Department (OPD).

On examination, the patient had open pustules that were forming oozing erosions in both the groin area. Redness and crusted plaques were observed on the lower left eyelid, and oozing from both conchal bowls was noted. Oral mucosal erosions, nasal erosions, and lip erosions with crusting were also observed [Table/Fig-1a-e]. A culture swab taken from the groin area tested positive for Staphylococcus aureus. Lab investigations revealed pancytopenia,



[Table/Fig-1]: a) Blepharoconjunctivitis of left eye; b) Pustules opened up to form crusts in right ear; c) Opened pustules with crusted erosions in groin; d) Oral and nasal erosions; e) Histopathological examination showing neutrophilic infiltrates in dermis (H&E, X10).

elevated Erythrocyte Sedimentation Rate (ESR) and C-Reactive Protein (CRP), low C3 and C4 levels, normal renal and liver function tests, normal 24-hour urine protein, and spot Polymerase Chain Reaction (PCR). Antinuclear Antibodies (ANA) (1:100 titre) and dsDNA showed positivity.

Since the eczematous lesions resembled late-onset acrodermatitis enteropathica in adults, the patient's serum zinc levels were measured, which were found to be low-49 µg/dL [Normal range: 52-286 µg/dL]. A skin biopsy was performed from the pustules in the inguinal region, which showed psoriasiform acanthosis and spongiotic neutrophil collections in the papillary dermis, leading to a diagnosis of APF. Based on the major and minor criteria, including pustules in the inguinal folds and conchal bowls, intraepidermal spongiotic pustules, and positive ANA and dsDNA, a final diagnosis of cutaneous lupus erythematosus with amicrobial pustulosis of the folds was made.

The patient was treated with Mycophenolate mofetil 500 mg BD and Dexamethasone 8 mg OD for a period of 4-6 weeks until the last pustule of APF resolved. Then, the treatment was slowly tapered to Prednisolone 5 mg every two weeks. Currently, the patient is in remission with Prednisolone 20 mg OD and Mycophenolate mofetil 500 mg BD as a steroid-sparing agent. The patient has been under follow-up for the past six months and has shown no new lesions. During the follow-up period, the patient's lab parameters remained within normal range.

Case 2

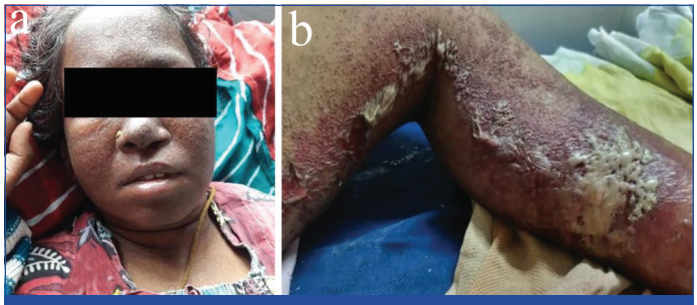
A 31-year-old female presented with chief complaints of persistent lip erosions, photosensitivity, fever, and arthralgia lasting for four months. There was no significant medical or family history. Dermatological examination revealed a malar rash and coalesced pustules with discharge from both conchal bowls [Table/Fig-2a-c]. Swabs taken from unopened pustules from the concha were negative for any organisms. Lab investigations showed pancytopenia and elevated inflammatory markers (ESR, CRP). The ANA level was 6.6, and dsDNA was positive. Renal function tests revealed high urea creatinine, and C3 and C4 levels were below normal. Skin biopsy showed vacuolar degeneration in the epidermis with inflammatory infiltrates in the papillary dermis, consistent with the diagnosis of lupus erythematosus. Additionally, renal biopsy revealed Class II Mesangioproliferative glomerulonephritis.

[Table/Fig-2]: a) Oral erosions,malar rash; b) Pustules coalesced to form plaques in left conchal bowl; c) Photomicrograph showing basal vacuolar degeneration with inflammatory infiltrates in papillary dermis (H&E, x40).

Based on the presence of bacteriologically sterile pustules involving minor skin folds, and positive ANA, dsDNA, and associated SLE, a final diagnosis of SLE with lupus nephritis and APF was made. The patient was treated with Inj. Dexamethasone 8 mg OD and Tablet Mycophenolate mofetil 500 mg BD for five weeks. Subsequently, the patient was maintained on Tablet Prednisolone 30 mg OD and Tablet Mycophenolate mofetil 500 mg BD for eight months. All mucocutaneous lesions and pustules of APF resolved after eight months.

Case 3

A 35-year-old female, known to have SLE with Class V Lupus nephritis, presented to the Dermatology OPD with swelling of the face and extremities, as well as pus along both her popliteal folds and oral mucosal erosions that developed within 24-48 hours. The patient had been prescribed tablet prednisolone 20 mg per day but had stopped taking it for one month. Examination revealed anasarca, facial edema with redness, and pustules that had coalesced into sheets on both her lower limbs, extending from the popliteal fossa to the ankle. Oral mucosal erosions were seen in her buccal mucosa and palate [Table/Fig-3a,b]. A swab taken from a discrete pustule was negative for organisms. Blood investigations showed a positive ANA with a titre of 1:160, positive dsDNA, and elevated ESR and CRP. A diagnosis of amicrobial pustulosis of folds was made.



[Table/Fig-3]: a) Malar rash and facial puffiness; b) Pustules in the popliteal fossa extending upto the ankle in left leg.

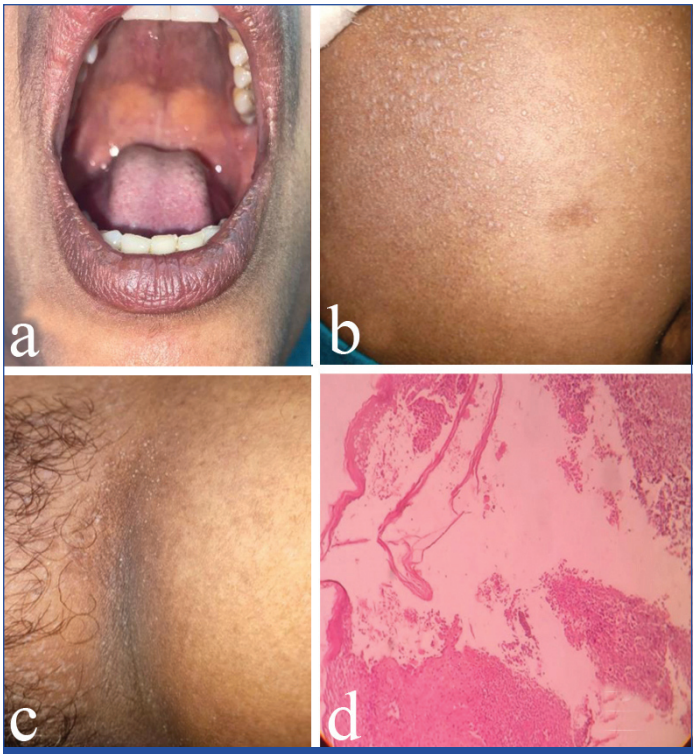
During this disease flare-up, the patient was treated with Inj. Dexamethasone 8 mg OD and Tablet Mycophenolate mofetil 500 mg BD for 4-6 weeks. Over the course of the eight-month follow-up period, the pustules resolved and the disease activity gradually decreased.

Case 4

A 24-year-old female presented with chief complaints of fever, arthralgia, facial puffiness, tenderness, and swelling in the right breast, as well as tense fluid-filled blisters over the trunk for a duration of four months. She also reported a history of tiny pustules in both her inguinal folds for two months. The patient had been previously treated as a case of Rheumatoid arthritis with Hydroxychloroquine, Sulfasalazine, and Ivabradine for the past one year. Two months ago, she had undergone incision and drainage for right breast swelling.

On examination, tiny pustules were observed in both inguinal folds, along with vesicles on the abdomen and erosions on the palate [Table/Fig-4a-d]. Cultures from the inguinal pustules showed no bacterial growth. Further screening for autoimmune disorders revealed a speckled pattern of ANA, positive dsDNA, and chest X-ray findings consistent with lupus pneumonitis. Histopathology revealed subcorneal spongiotic pustules and intraepidermal neutrophil-rich infiltrates, suggestive of APFs. Given that the patient met two major and two minor criteria, a diagnosis of SLE with APF and Lupus pneumonitis was made.

Treatment was initiated with Inj. Dexamethasone 8 mg for four weeks, which was gradually tapered to oral prednisolone 20 mg per



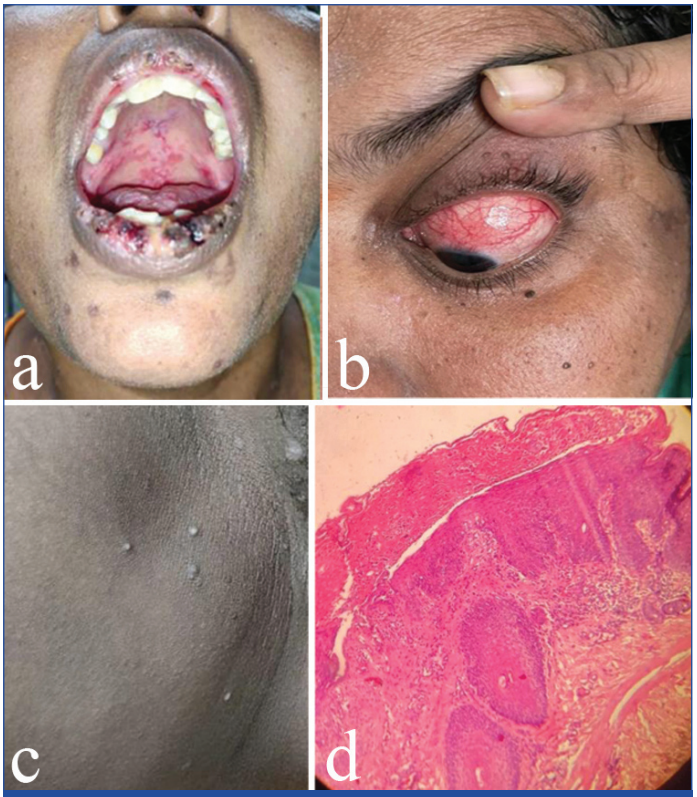
[Table/Fig-4]: a) Palatal erosions; b) Tense blisters in trunk; c) Tiny pustules in inguinal folds; d) photomicrograph showing neutrophilic collection in dermis (H&E, x10).

day. During a six-month follow-up, the patient's condition resolved, and no new lesions were observed.

Case 5

A 41-year-old female presented to the OPD with complaints of lip erosions, painful oral ulcers, redness of the eyes, vaginal erosions, and pustular lesions in her groin, gluteal cleft, and both axilla for a duration of four months. There was no significant medical or family history, and the patient denied any history of drug intake.

On examination, multiple pustules were noted in her inguinal folds, axilla, and gluteal folds. The oral mucosa showed erosions on the lip, buccal mucosa, and palatal ulcers with an erythematous floor. Conjunctival congestion was observed in both eyes [Table/Fig-5a-d]. Swabs and cultures from the axillary pustules showed no organisms. The patient's ANA and dsDNA were negative. Biopsy of the pustules in the groin revealed a subcorneal pustule containing neutrophils and red blood cells, along with capillary dilation and collections of neutrophils and mononuclear cells in the papillary dermis. These findings were suggestive of amicrobial pustulosis of the folds. Direct immunofluorescence of perilesional skin was negative for immunoreactants. Biopsy of the buccal mucosa revealed a lesion consistent with mucosal pemphigus vulgaris. Therefore, a final diagnosis of mucosal pemphigus vulgaris and APF was made. This documented a new case of autoimmune disorder association between amicrobial pustulosis of the folds and mucosal pemphigus vulgaris, which was the first of its kind.



[Table/Fig-5]: a) Lip and palatal erosions; b) Conjunctival congestion; c) Pustules in groin; d) Photomicrograph showing neutrophillic aggregation in dermis in (H&E, x10).

Treatment was initiated with Inj. Dexamethasone 8 mg once daily and tablet Cyclophosphamide 100 mg once daily as a steroid-sparing agent for five weeks. The patient achieved remission and was maintained on tablet Prednisolone 20 mg OD and tablet Cyclophosphamide 100 mg OD for the past four months. There have been no new pustules, and the mucosal erosions have healed at the four-month follow-up [Table/Fig-6] shows the profiles of all the patients included in the case series.

Profile of patients	Pus culture sensitivity	ESR	CRP	ANA	dsDNA	C3;C4	Associated disorders	System involvement	Treatment given after final diagnosis	Current status of patient/ post discharge follow-up duration (in months)	Role of APF
Case 1	Opened up pustules-Staphy-lococcus aureus	49	17	1:100, spindle pattern	Positive	C3:0.7 (Normal: 0.9 to 1.8) C4: 0.08 (Normal: 0.1 to 0.4)	SLE	Nil	Inj. Dexamethasone 8 mg OD and Tablet Mycophenolate mofetil 500 mg BD for 4-6 weeks	On remission with Tab. Prednisolone 20 mg OD and Tablet Mycophenolate mofetil 500 mg BD for 6 months	EOD
Case 2	Negative from unopened pustules	118	12	6.6 IU (Normal: <1.0 IU)	Positive	C3: 0.8 C4: 0.08	SLE	Class II mesangio-proliferative nephritis	Inj. Dexamethasone 8 mg OD and Tablet Mycophenolate mofetil 500 mg BD for 5 weeks	On remission with Tab. Prednisolone 30 mg OD and Tablet Mycophenolate mofetil 500 mg BD for 8 months	EOD
Case 3	Negative from unopened pustule	120	20	1:160	Positive	C3: 0.7 C4: 0.09	SLE	Class V Lupus nephritis	Inj. Dexamethas one 8 mg OD and Tablet Mycophenolate mofetil 500 mg BD for 4-6 weeks	On remission with Tab. Prednisolone 40 mg OD and Tablet Mycophenolate mofetil 500 mg BD for 8 months	MDA

Case 4	Negative from unopened pustule	107	100	1:40 Nuclear speckled 4+	Positive	C3: 0.8 C4: 0.07	SLE	Lupus pnemonitis	Inj. Dexamethasone 8 mg OD for 4 weeks	On remission with Tab Prednisolone 20 mg OD and for 6 months	EOD
Case 5	Negative from unopened pustule	129	11	Negative	Negative	C3-1.1 C4-0.2	Pemphigus vulgaris	Nil	Inj. Dexamethasone 8 mg one daily and Tablet Cyclophosphamide 100 mg once daily for 5 weeks	On remission with Tab. Prednisolone 20 mg OD and Tablet Cyclophosphamide 100 mg OD for 4 months	A new association-Pemphigus vulgaris

[Table/Fig-6]: Profile of patients.
OD: Once daily; BD: Twice daily; EOD: Eye opener of diagnosis; MDA: Marker of disease activity

DISCUSSION

The condition known as APFs is also referred to as 'pustular dermatosis,' 'follicular impetigo,' 'pyodermatitis vegetans,' and 'amicrobial pustulosis associated with autoimmune diseases' (APF). It is a neutrophilic dermatosis caused by autoimmune dysregulation.

To date, a total of 78 cases of APF have been reported in the literature, with a female preponderance of nearly 90% [4-8]. This case series also reports five cases of APF, all of whom were females.

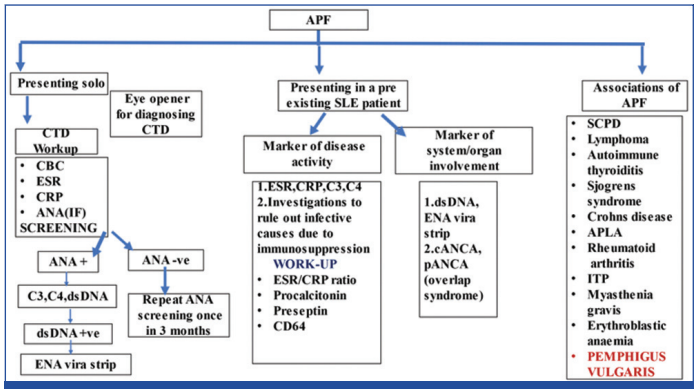
Similar to the findings reported by Wang MZ et al., all the cases in this series presented with involvement of the major and minor folds, as well as the genital area, with erythematous plaques, erosions, and coalesced pustules in the conchal bowls and popliteal fossa [9]. As mentioned by Márquez Balbás G et al., cultures for bacteria and fungi collected from unopened pustules before antibiotic treatment were either negative or positive for Staphylococcus aureus [10]. Histopathology showing subcorneal pustules with neutrophil-rich dermal infiltrates was a common finding in most of the cases.

In previously reported cases, associations were found with conditions such as Lupus erythematosus, Psoriasis, Autoimmune thyroiditis, Sjogren's syndrome, Crohn's disease [11], and Rheumatoid arthritis [12]. In the present case series, an association with Lupus erythematosus was established in four of the cases, and one case was associated with mucosal pemphigus, which is the first of its kind.

Although the exact pathogenesis of amicrobial pustulosis of the folds is unclear, possible explanations for its occurrence could be high levels of neutrophils, including low-density granulocytes seen in SLE and other autoimmune disorders. These abnormal neutrophils have an increased capacity to synthesise Neutrophil Extracellular Traps (NETs), and the aberrant and/or excessive formation of NETs seems to play a role in the development and perpetuation of autoimmune disorders [13]. The affected skin and kidneys are infiltrated with the netting neutrophils. Additionally, the antimicrobial components exposed by NETosis can serve as an immunostimulatory signal that facilitates the recognition of self dsDNA. Activated neutrophils in amicrobial pustulosis of the folds influence damaged organs like the kidneys to increase the uptake of dsDNA. This mechanism ultimately results in organ damage in patients with SLE and other autoimmune disorders.

It has been noted that zinc restriction can lead to reduced autoantibodies, anti-dsDNA titres, and reduced lymphoproliferation [14,15]. Therefore, zinc supplements are not recommended for patients with SLE. Additionally, matrix metalloproteinase, a zinc-containing proteinase that plays a role in remodelling the extracellular matrix, has been reported to be related to SLE activity and Lupus nephritis due to its high levels [16,17].

In young females with a prior history of Connective Tissue Disorder (CTD) or individuals with new symptom onset and pustules in the intertriginous areas, skin folds, and conchal bowls, APF should be considered as a differential diagnosis. In the present cases, the clinical and morphological picture raised a high degree of suspicion, which was supported by investigations such as high



[Table/Fig-7]: Approach to Amicrobial pustulosis of the folds.
CTD: Connective tissue disorder; CBC: Complete blood count; ESR: Erythrocyte sedimentation rate; CRP: C-reactive protein; ANA: Anti nuclear antibody; IF: Immunofluroscence; ENA: Extractable nuclear antigen; SCPD: Sub corneal pustular dermatosis; APLA: Antiphospholipid antibody; ITP: Immune thrombocytopenic purpura

ANA, ESR, CRP, positive dsDNA, neutrophil-rich cellular infiltrate in histopathology, and negative culture for microorganisms. All of these findings were compatible with the criteria for APF [Table/Fig-7] shows the approach to amicrobial pustulosis of the folds. The most effective treatment for managing APF is still systemic corticosteroids. Systemic antibiotics are not effective in treating this disease unless there is secondary impetiginization of the lesions. New therapeutic alternatives are emerging, such as anakinra and anti-TNF-alpha drugs [18]. Doxycycline has been tried for its anti-inflammatory action. Other drugs that have been tried include cimetidine and ascorbic acid [19]. A good clinical response to hydroxychloroquine (400 mg/d) in combination with prednisone (0.5 mg/kg/d) has also been described in a patient who failed to tolerate treatment with oral sulfone [2]. Other treatments include zinc [20], cyclosporine, methotrexate, levamisole, and colchicine [21], although the results are variable.

Given its rarity, a high degree of suspicion is necessary to diagnose amicrobial pustulosis of the folds. This can lead to the identification and prompt treatment of the underlying autoimmune condition.

CONCLUSION(S)

The presence of neutrophilic leukocytosis is a clue that patients may have amicrobial pustulosis of the folds. Cutaneous lupus erythematosus with histopathological features of a neutrophil-rich inflammatory infiltrate in the perivascular and perifollicular areas is a strong predictor of cutaneous lupus erythematosus progressing to SLE, thus acting as a prognostic indicator. APF can serve as a marker of disease activity or organ involvement, and dsDNA is the most common antigen among ENA associated with APF. Due to the frequent association of APF with lupus erythematosus and other immunologic disorders, all cases of APF need to be closely monitored with the aim of making an early diagnosis of any autoimmune disorder at the time of presentation or in the future.

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