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CASE REPORT

A Case of Cutaneous Dirt-Adherent-Like Sweet Syndrome in Pregnancy

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Abstract: Pregnancy-associated Sweet syndrome (PASS), is a diagnostically challenging entity, especially in atypical presentations. Delayed management may result in significant maternal-fetal risks. We describe a 24-year-old woman with PASS presenting like cutaneous dirt-adherent disease, characterized by abrupt-onset facial erythematous plaques with adherent dirt-like crusts. Histopathology revealed superficial dermal edema and dense perivascular neutrophilic infiltrates without vasculitis. Low-dose systemic corticosteroids (prednisone 20mg/day) achieved rapid resolution, underscoring their efficacy and safety in pregnancy. This case explores the potential pathogenesis of PASS and underscores the necessity for awareness among clinicians about PASS, contributing valuable insights into the diagnosis and management of this rare condition in pregnancy.

Keywords: sweet syndrome, dermatosis neglecta, pregnancy, neutrophilic dermatosis, treatment

Introduction

Sweet syndrome (SS) is the prototypical neutrophilic dermatosis, characterized by the abrupt onset of painful, tender erythematous papules, nodules, and plaques accompanied by dense neutrophilic infiltration.[1] SS is typically classified as classic, malignancy-associated, or drug-induced, with rare variants including bullous, cellulitis-like, histiocytoid, and subcutaneous forms.[1,2] Its pathogenesis involves dysregulated inflammatory mediators, abnormal neutrophil function, and genetic susceptibility. Pregnancy-associated Sweet syndrome (PASS) is rare, accounting for only about 2% of all SS cases.[1] It necessitates differentiation from other pregnancy-associated autoinflammatory neutrophilic dermatoses (AINDP), a group of disorders characterized by neutrophilic infiltration but diverse clinical manifestations, often leading to diagnostic and therapeutic difficulties. For instance, pyoderma gangrenosum typically exhibits a mixed neutrophilic and lymphocytic infiltrate on histopathology and often responds slowly to corticosteroid therapy.[3] Behçet's disease is characterized by the classic triad of recurrent oral ulcers, genital ulcers, and uveitis, with histopathological findings that may include neutrophilic infiltration around eccrine structures.[3] Neutrophilic figurate erythema presents with distinctive arciform erythematous plaques accompanied by pustules and associated tenderness.[4] Furthermore, infectious dermatoses such as tinea incognito and cutaneous tuberculosis should also be excluded clinically, for which microbial culture or PCR techniques are valuable. Given these complexities, unfamiliarity with PASS may easily result in misdiagnosis. Here, we report a rare case of PASS presenting with cutaneous dirt-adherent-like lesions and discuss its potential mechanisms.

Case Presentation

A 24-year-old woman, G1P0, presented at 17^{+3} weeks of gestation with a 2-month history of erythematous plaques. Lesions first appeared on her forehead and left cheek, gradually enlarged and thickened, involving the right face, and entire left cheek with extensive dirt-like adherent yellow crusts (Figure 1), without pain and itching. She reported no fever, family history, autoimmune diseases, or respiratory symptoms. Laboratory results, including blood tests (leukocytosis 6.9×10^9/L, neutrophils

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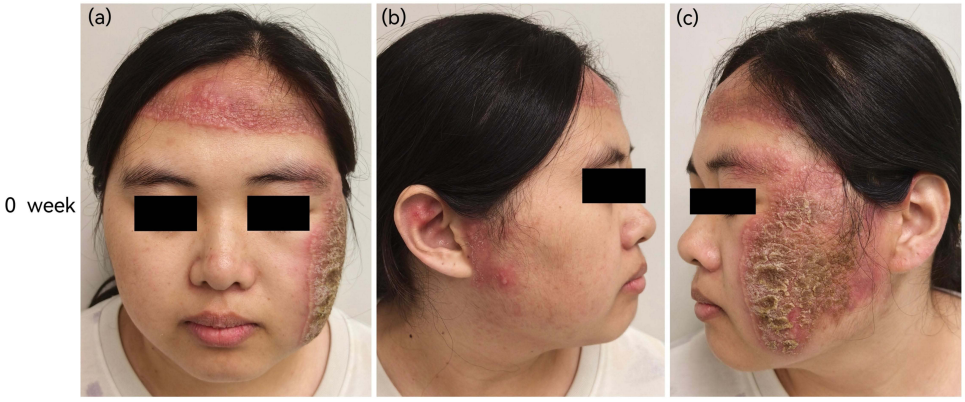

Figure 1 Clinical presentation of the patient's facial lesions before treatment (**a**–**c**).

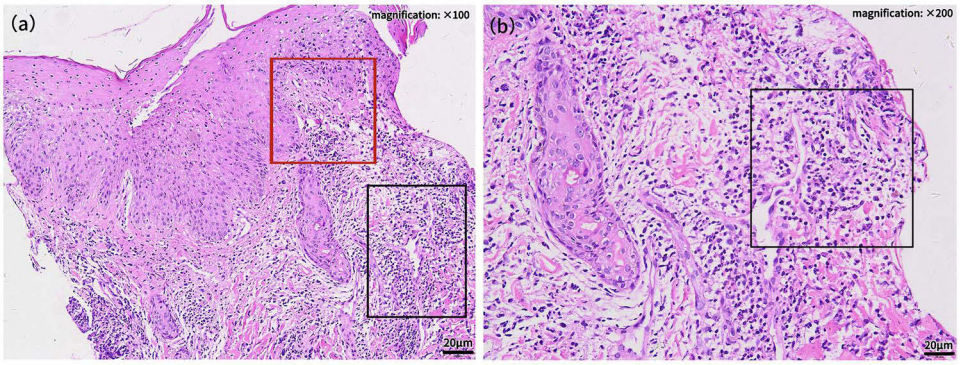

Figure 2 Histopathological features. (**a**) Partial dermal papillary edema (red square) and dense dermal neutrophilic infiltrate (black square) without leukocytoclastic vasculitis (HE, ×100). (**b**) Higher magnification of the neutrophilic infiltrate with some nuclear fragmentation (black square) (HE, ×200).

62.8%, lymphocyte 24.5%, erythrocyte 4.33×10^{12}/L, hemoglobin 137g/L, platelet 150×10^{9}/L), liver/kidney function (alanine aminotransferase 21U/L, aspartate aminotransferase 18U/L, creatinine 65umol/L), lipid profile (triglyceride 1.65mmol/L, low-density lipoprotein 2.43mmol/L), cardiac enzymes (creatine kinase 60U/L, creatine kinase Isoenzymes 2.12ng/mL, lactate dehydrogenase 96U/L), C-reactive protein (6mg/L), erythrocyte sedimentation rate (ESR: 15mm/h), antinuclear antibodies, and peripheral blood smear, were normal. Fetal ultrasound revealed a live fetus without abnormalities. Fungal microscopy of the face was negative. Histological examination from the forehead showed partial dermal papillary edema and superficial perivenular infiltrates of lymphocytes and neutrophils without vasculitis (Figure 2), supporting a diagnosis of PASS. Oral prednisone (20mg/day for 2 weeks) led to marked improvement, allowing discontinuation. At 6 weeks, lesions almost disappeared (Figure 3a–c). By 12-weeks, only patchy hyperpigmentation remained (Figure 3d–f). At 40^{+1} weeks, she delivered a healthy neonate. At the 11-month postpartum follow-up, the patient exhibited only residual facial hyperpigmentation with no recurrence. Following an uneventful delivery and the absence of breastfeeding contraindications, the infant was breastfed routinely and has demonstrated normal development.

Discussion

Currently, research on SS in pregnancy remains relatively limited. The onset of PASS may be associated with increased levels of estrogen and progesterone, which can result in excessive neutrophil activation targeting unknown self-components.[1] During pregnancy, naive $CD4^{+}$ T cell differentiation shifts toward a Th2 phenotype, increasing Th2-associated cytokines such as IL-6 and IL-1β.[5] Furthermore, elevated maternal serum alpha-fetoprotein (AFP) may further enhance IL-6 secretion, stimulating granulocyte colony-stimulating factor (G-CSF) release and neutrophil proliferation.[6] Additionally, higher lipopolysaccharide (LPS) levels in pregnancy can augment IL-8 production, facilitating neutrophil recruitment and secondary inflammation, potentially driving PASS pathogenesis (Figure 4).[6,7]

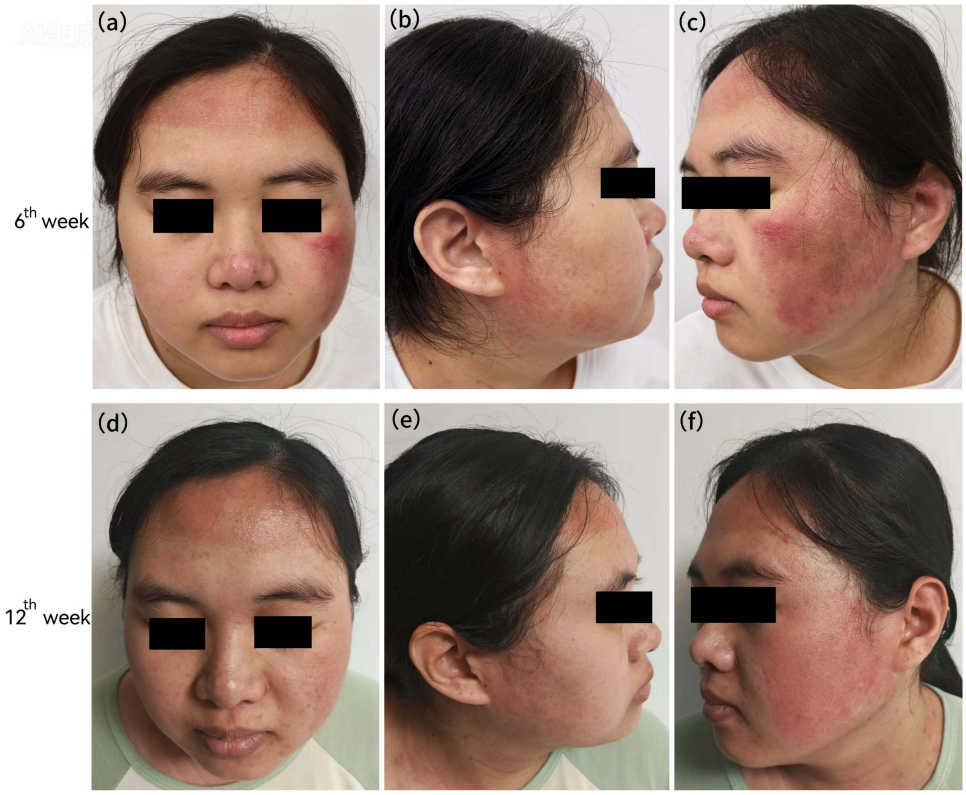

Figure 3 Facial clinical presentation after treatment. (**a–c**) At the 6-week follow-up after initiation of systemic prednisone, the patient demonstrated significant improvement in facial lesions. (**d–f**) By the 12-week follow-up, there was complete resolution of plaques, crusting, and pseudo-vesicles, with only residual patchy hyperpigmentation remaining.

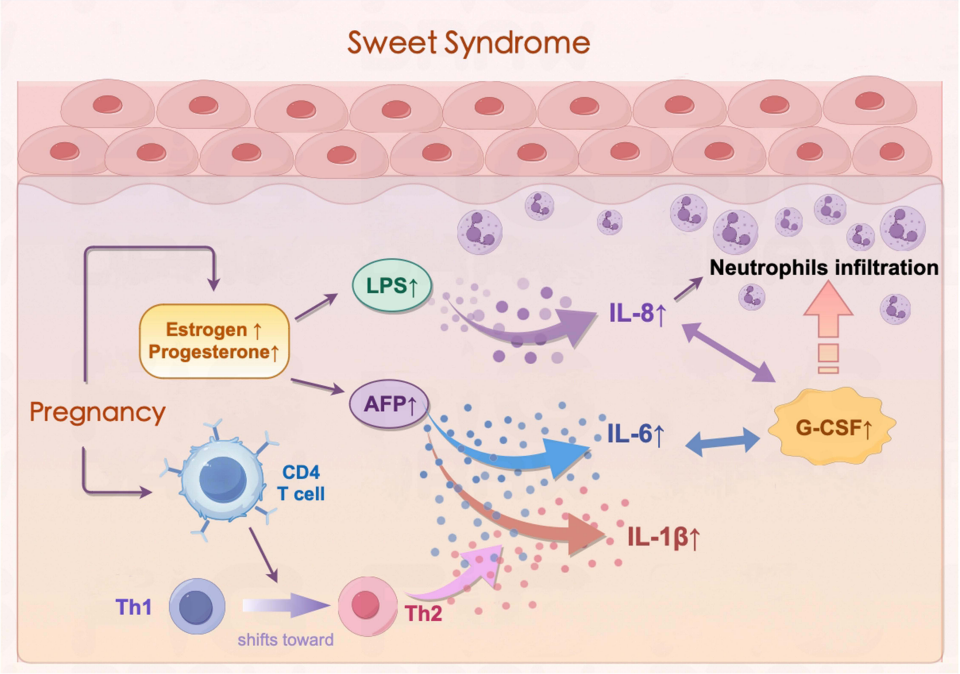

Figure 4 The potential mechanisms through which pregnancy-specific cytokines may contribute to PASS pathogenesis. Upward arrow (↑): increased expression; Single-headed arrow (→): promotion or activation; Double-headed arrow (↔): interaction.

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PASS has been reported in all trimesters, most commonly in the first and second trimester, and may recur in subsequent pregnancies.[1] Approximately 36% of patients are primigravidae, 64% multigravida, with 26% of the latter experiencing recurrence in multiple pregnancies.[1,8] A prior history of SS or comorbidities such as inflammatory bowel disease or autoimmune disorders may increase the risk of developing PASS. The histopathology resembles other SS types, but it is essential to evaluate for extracutaneous involvement, particularly of the uterus and adnexa. A severe PASS case with dense neutrophil infiltration of the endometrium, serosa, and fallopian tubes has been reported, progressing to abdominal wall necrosis post-cesarean.[9] Therefore, early recognition and appropriate evaluation are essential.

Cutaneous dirt-adherent disease(COAD), also known as dermatitis neglecta, is a rare psychodermatological disorder characterized by localized, persistent, dirty-appearing adherent material on the skin.[10] Clinically, it presents as thick, yellow-brown, verrucous crusts with sharply demarcated borders that are resistant to routine cleansing. A positive "alcohol wipe test" can assist in diagnosis.[10] In this case, pregnancy-associated cutaneous hypermetabolism and increased sebaceous gland activity, compounded by inadequate cleansing, likely predisposed to COAD-like changes. Additionally, delayed intervention due to pregnancy may have prolonged disease, expanded lesions, and created conditions for dirt-like adherent crust formation. Integrating the abrupt onset of erythematous plaques, histopathological characteristics, pregnancy history, and rapid glucocorticoid response fulfill the 1994 Von den Driesch diagnostic criteria for SS (two major and two minor criteria; Table 1).[11] This case may represent a rare COAD-like variant of PASS.

The treatment of PASS is challenging. In our case, lesions resolved rapidly with glucocorticoid, without adverse fetal effects. It is important to note that corticosteroid safety in pregnancy is dose-dependent. High-dose oral prednisone may increase risks of intrauterine growth restriction and prematurity.[12] Consequently, although our case demonstrated efficacy and safety, the use of glucocorticoids in pregnancy requires prudent management, where the maternal benefit must be weighed against potential fetal risks. In this context, a short-course, low-dose regimen remains a recommended first-line treatment for PASS. Furthermore, recent research advancements suggest that biologics, including TNF-α inhibitors (such as infliximab and etanercept), IL-1 receptor antagonists (anakinra), IL-6 inhibitors (tocilizumab) and Janus kinase inhibitors, demonstrate efficacy in treating refractory neutrophilic dermatoses.[13,14] While the safety of biologics during

Table 1 Diagnostic Criteria for Sweet Syndrome

	Von den Driesch in 1994*	**Our Case**
Major criteria	① Abrupt onset of painful erythematous plaques or nodules ② A dense neutrophilic infiltration in the dermis without leukocytoclastic vasculitis	✓ Abrupt-onset facial erythematous plaques with adherent dirt-like crusts. ✓ Superficial dermal edema and dense neutrophilic infiltration in the dermis without leukocytoclastic vasculitis
Minor criteria	①Preceded by a nonspecific respiratory or gastrointestinal tract infection or vaccination or associated with: ■ Inflammatory diseases such as chronic autoimmune disorders, infections ■ Hemoproliferative disorders or solid malignant tumors ■ Pregnancy ② Pyrexia>38°C ③ Laboratory values during onset (three of four): ■ ESR>20mm/h ■ CRP elevated ■ Neutrophils>70% ■ Leukocytosis>8.0×10^9/L ④ Excellent response to treatment with systemic corticosteroids or potassium iodide	/ / ✓ Pregnancy 36.5°C ESR: 15mm/h CRP: 6mg/L Neutrophils: 62.8% Leukocytosis: 6.9×10^9/L ✓ Excellent response to treatment with systemic corticosteroids

Note: *Both major and two minor criteria are needed for diagnosis.
Abbreviations: ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; ✓, meeting diagnostic criteria.

pregnancy varies by agent, current evidence from case series and registries supports cautious use in selected cases. For example, etanercept has a lower placental transfer rate compared to infliximab and adalimumab, and may be considered until 30–32 weeks of gestation if maternal disease is stable.[15] In contrast, JAK inhibitors and other small molecules should be avoided due to limited human safety data and preclinical evidence of fetal risk.[12] Prospective studies are needed to evaluate maternal and fetal outcomes.

We acknowledge the limitations of this work, particularly its nature as a single case report, which limits the generalizability of the findings. Larger, multicenter studies or cohort analyses are needed to further investigate the disease mechanisms and validate the efficacy and safety of therapeutic interventions, thereby providing evidence with greater clinical relevance.

Conclusion

The hormonal changes specific to pregnancy and placental immune privilege affect the maternal immune function and the course of inflammatory. This case highlights PASS masquerading as COAD, a presentation not previously reported. Low-dose systemic corticosteroids demonstrated rapid efficacy and maternal-fetal safety, reinforcing their role as first-line therapy. Clinicians should consider PASS in pregnancy-related dermatoses with the abrupt onset of erythematous plaques presenting as cutaneous dirt-adherent-like lesions, even in the absence of systemic symptoms such as fever or hematologic abnormalities. Further studies are needed to explore potential therapeutic strategies in the future, as well as to evaluate long-term outcomes of emerging therapies.

Data Sharing Statement

All supporting documents have been submitted along with the case report.

Ethics Approval and Informed Consent

The patient had given written informed consent for the publication of her clinical details and accompanying images. Institutional approval is not applicable for this case report.

Consent for Publication

Written informed consent was obtained from the patient for publishing this report.

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Author Contributions

Zhenqiang Ruan and Luan Yang are co-first authors and contributed equally to this work. All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The author(s) report no conflicts of interest in this work.

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