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Severe Dermatological Blistering Diseases with Maternal-Fetal Risk: Two Case Reports during Pregnancy

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Abstract

Objective: To report two cases of severe blistering dermatoses during pregnancy—a toxic epidermal necrolysis (TEN) induced by sulfadoxine-pyrimethamine and a relapsing pemphigus vulgaris (PV) complicated by transient neonatal pemphigus, and to review the literature highlighting diagnostic, therapeutic, and prognostic considerations. **Case reports:** The first case involved a 27-year-old primigravida at 28 weeks' gestation who developed TEN after intermittent preventive treatment of malaria with sulfadoxine-pyrimethamine. On admission, her SCORTEN score was 4/7, corresponding to an estimated mortality of −58%. Intensive multidisciplinary management, including emergency cesarean delivery for acute fetal distress, led to a favorable outcome with the birth of a healthy infant at term. The second case concerned a 39-year-old multigravida with mucosal PV, diagnosed based on clinical presentation and indirect immunofluorescence (IIF) in the absence of biopsy, DIF, and ELISA testing. She relapsed in the first trimester after discontinuing corticosteroids, later complicated by intrauterine growth restriction (IUGR). Both maternal remission and neonatal recovery were achieved after resumption of systemic corticosteroid therapy. **Discussion:** TEN is a rare but life-threatening hypersensitivity reaction mediated by cytotoxic T cells, associated with significant maternal and fetal morbidity. PV is an autoimmune blistering disease caused by IgG anti-desmoglein antibodies; pregnancy may exacerbate disease activity, while transplacental antibody transfer can occasionally result in neonatal pemphigus. In both conditions, therapeutic options are limited during pregnancy, necessitating individualized multidisciplinary management. In resource-limited settings, structured pharmacovigilance with simplified adverse drug reac-

tion reporting may help reduce preventable morbidity. **Conclusion:** TEN and PV during pregnancy are rare but serious conditions requiring prompt recognition, tailored therapy, and coordinated obstetric-dermatologic-neonatal care. Expanding diagnostic access and clinician awareness is critical to improving maternal-fetal outcomes.

Keywords

Pregnancy, Blistering Dermatoses, Toxic Epidermal Necrolysis, Pemphigus Vulgaris, Neonatal Pemphigus, Maternal-Fetal Prognosis

1. Introduction

Severe dermatological blistering diseases are uncommon during pregnancy but can pose significant risks to both maternal and fetal survival. **Toxic epidermal necrolysis (TEN)**, also known as Lyell's syndrome, is a severe mucocutaneous reaction most often triggered by drugs [1] [2], with reported mortality rates of 20% - 30% even under optimal care. **Pemphigus vulgaris (PV)** is a rare autoimmune blistering disorder caused by autoantibodies directed against desmoglein 3 and/or desmoglein 1, key desmosomal proteins responsible for keratinocyte cohesion [3] [4]. In Europe and North America, PV accounts for approximately 65% - 95% of pemphigus cases [5].

During pregnancy, TEN most frequently follows exposure to anti-infective or anticonvulsant drugs, while PV may present as an exacerbation of a preexisting disease or arise de novo, sometimes influenced by gestational immuno-hormonal changes. Both conditions require rapid recognition and prompt, multidisciplinary management tailored to the unique therapeutic and obstetric challenges of pregnancy.

2. Case Reports

2.1. Case 1—Toxic Epidermal Necrolysis

Patient M.T., a 27-year-old Senegalese primigravida with a history of asthma treated with inhaled salbutamol and fluticasone/salmeterol, and no known drug allergies, was referred at 28 weeks' gestation for extensive bullous skin lesions following a single prophylactic dose of sulfadoxine-pyrimethamine (Fansidar®) for intermittent preventive treatment of malaria in pregnancy (IPTp).

Within hours of drug intake, she developed ocular and vulvar pruritus, fever, and a diffuse maculopapular rash that rapidly progressed to flaccid blisters and large areas of epidermal detachment. On admission, she was febrile, with a positive Nikolsky's sign and purpuric necrotic macules involving −70% of her body surface area. Oral, ocular, and vulvar mucosae were also affected.

Laboratory tests showed moderate eosinophilia, lymphopenia, elevated C-reactive protein (117 mg/L), and mild hepatic cytolysis (alanine aminotransferase 1.7 × upper limit of normal), with preserved renal and electrolyte balance.

The suspected drug was immediately discontinued, and the patient was admitted to a specialized burn and dermatology unit. Management included aggressive rehydration, broad-spectrum antibiotics, sterile wound care, and symptomatic therapy. Obstetric monitoring revealed a viable fetus with growth parameters appropriate for gestational age.

During hospitalization, she developed threatened preterm labor at 28 weeks, successfully managed with bed rest and tocolysis. Gradual cutaneous improvement was observed, with drying of lesions and progressive re-epithelialization.

A **SCORTEN severity score** was calculated on admission: age < 40, no malignancy, tachycardia at 128/min, serum bicarbonate 18 mEq/L, serum glucose 190 mg/dL, blood urea 32 mg/dL, and epidermal detachment >10% body surface area (Table 1). The final score was 4/7, corresponding to an estimated mortality of −58%, emphasizing the severity of the prognosis and the need for intensive multidisciplinary care.

Table 1. SCORTEN score on admission for Case 1 (TEN).

SCORTEN criteria	Patient findings	Score
Age >40 years	No (28 years)	0
Malignancy	No	0
Heart rate >120/min	Yes (128/min)	1
Serum urea >10 mmol/L	No (32 mg/dL ≈ 5.3 mmol/L)	0
Serum glucose >14 mmol/L	Yes (190 mg/dL ≈ 10.5 mmol/L)	1
Serum bicarbonate <20 mmol/L	Yes (18 mmol/L)	1
Detached BSA >10%	Yes (−70%)	1

SCORTEN severity score calculated on admission for a 27-year-old pregnant patient with TEN. The total score was **4/7**, corresponding to an estimated mortality of −58%.

At 39 weeks' gestation, a cesarean delivery was performed for acute fetal distress, resulting in the birth of a healthy male infant weighing 3200 g (Apgar score 7/8) with no skin or mucosal lesions.

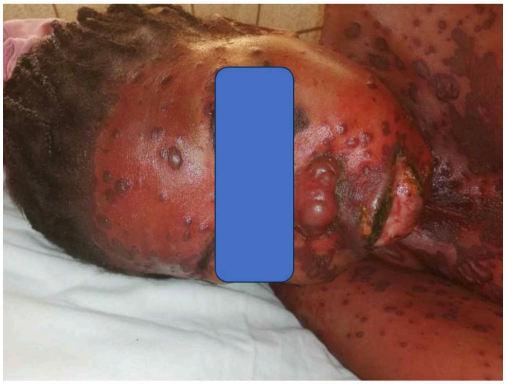

Figure 1. Patient on day 1 of hospitalization with toxic epidermal necrolysis.

Figure 1 illustrates the acute necrolytic phase with extensive epidermal detachment, while Figure 2 shows re-epithelialization and pigmentary sequelae at day 30.

Extensive erythematous and erosive lesions with multiple flaccid bullae and epidermal detachment are visible on the face, neck, and upper trunk. Periorbital edema and crusted erosions around the nostrils and lips are also noted, consistent with severe mucocutaneous involvement.

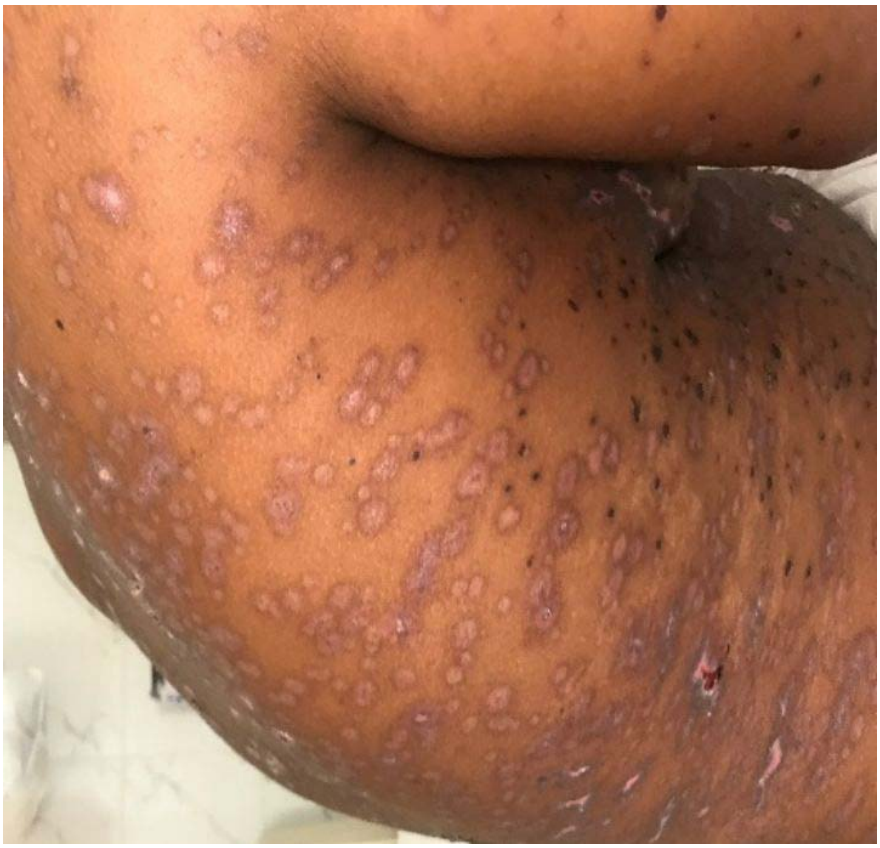

Figure 2. Day 30 of hospitalization for toxic epidermal necrolysis.

Post-inflammatory hyperpigmentation and hypopigmented patches at previous sites of epidermal detachment and bullae. No new lesions are visible. Healing erosions with crusts are noted on the lateral arm, reflecting re-epithelialization. This image illustrates the transition from the acute necrolytic phase to the reparative phase, with characteristic pigmentary sequelae.

2.2. Case 2—Maternal and Neonatal Pemphigus Vulgaris

A 39-year-old multigravida (gravida 2, para 1) had a history of recurrent oral erosions first diagnosed at age 34 as exclusive mucosal pemphigus vulgaris (PV). In Senegal, direct immunofluorescence (DIF) and ELISA testing were unavailable; the diagnosis was therefore based on clinical presentation (exclusive mucosal involvement without cutaneous lesions) and positive intercellular antibodies de-

tected by indirect immunofluorescence (IIF). This absence of confirmatory testing represented a diagnostic limitation.

The patient achieved remission for one year with oral corticosteroids. Her surgical history included a cesarean section two years before the first PV episode.

During the current pregnancy, early antenatal care was unremarkable. In the first trimester, however, she relapsed after discontinuing corticosteroids, presenting with painful erosive gingivitis and palatal lesions. Prednisone was resumed at 1 mg/kg/day (60 mg/day), tapered to 20 mg/day during the third trimester.

At 36 + 5 weeks, she was admitted for gingival bleeding after stopping corticosteroids four weeks earlier. Examination confirmed mucosal erosions with gingivitis. Laboratory evaluation showed thrombocytopenia (80,000/mm^3) without anemia and prothrombin time of 90%. Ultrasound estimated fetal weight at 1650 g, with an umbilical artery resistance index of 0.70. A dermatology consultation recommended resuming prednisone 60 mg/day.

A cesarean section was performed at 37 weeks, delivering a male infant weighing 1620 g (Apgar scores 8 and 9 at one and five minutes, respectively). The mother received two units of platelet transfusion intraoperatively.

The neonate presented with perianal blistering lesions evolving into erosions, clinically suggestive of transient neonatal pemphigus. Histology confirmed suprabasal acantholysis. The lesions resolved spontaneously within six weeks postpartum, while maternal gingival erosions healed over the same period.

Figure 3 shows the neonatal perianal erosive lesions.

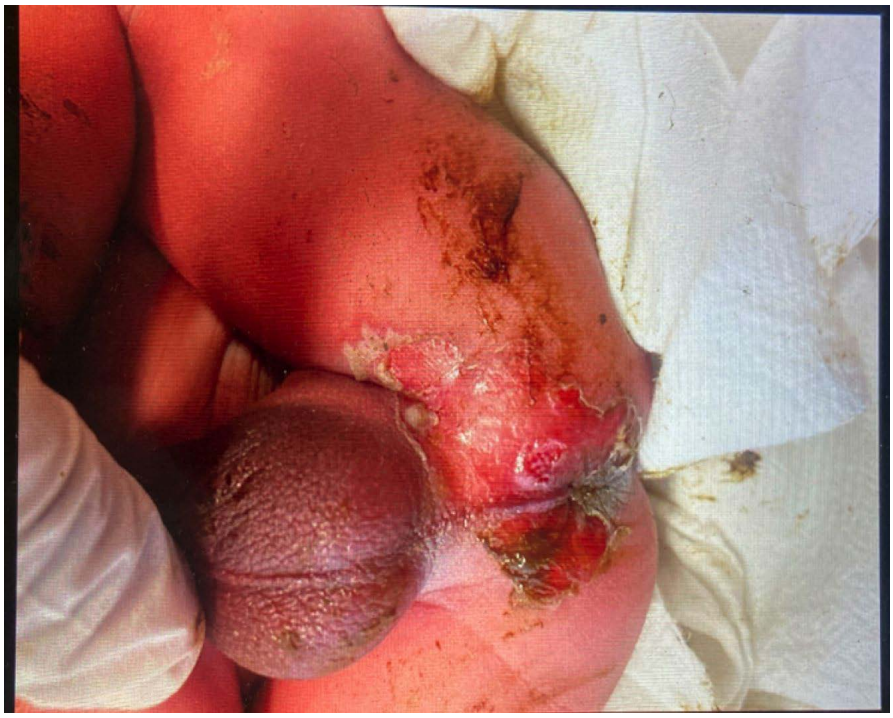

Figure 3. Transient neonatal pemphigus.

3. Discussion

3.1. Epidemiology and Severity

Toxic epidermal necrolysis (TEN) is a rare but life-threatening mucocutaneous reaction, with an annual incidence of 0.4 - 1.2 cases per million inhabitants in Western countries [1], and higher rates in regions where sulfonamides are widely prescribed [3] [4]. Mortality remains substantial (20% - 30%), even in specialized units, and increases with comorbidities, delayed diagnosis, and advanced age. Pemphigus vulgaris (PV), with an incidence of 0.1 - 0.5 cases per 100,000 per year, is the most common pemphigus variant observed during pregnancy, although its occurrence remains exceptional. Kardos *et al.* [5] identified only 47 cases of pemphigus in pregnancy reported in the literature, of which 36 were PV.

In our series, both conditions had significant maternal-fetal implications, with TEN associated with acute fetal distress (**Figure 1**) and PV complicated by intrauterine growth restriction (IUGR) and transient neonatal pemphigus (**Figure 3**). The main clinical and therapeutic differences are summarized in **Table 2**.

Table 2. Comparison of main clinical and therapeutic characteristics of TEN and PV during pregnancy.

Feature	Toxic epidermal necrolysis	Pemphigus vulgaris
Mechanism	Delayed hypersensitivity (CD8+ T cells, NK, granulysin)	Autoantibodies against desmogleins
Trigger	Drug (sulfonamides, anticonvulsants)	Idiopathic or exacerbation of preexisting PV
Lesions	>30% BSA epidermolysis + mucosal involvement	Intraepidermal blisters, mucosal ± skin
Severity	Mortality 20% - 30%	Chronic morbidity, low mortality
First-line treatment	Drug withdrawal, supportive care, ± IVIG	Systemic corticosteroids
Fetal risks	Preterm birth, stillbirth, IUGR	IUGR, preterm birth, neonatal pemphigus
Neonatal prognosis	Linked to maternal condition	Generally favorable

Comparison of underlying mechanisms, typical triggers, lesion distribution, severity, first-line treatment, and maternal-fetal outcomes between toxic epidermal necrolysis (TEN) and pemphigus vulgaris (PV) during pregnancy.

3.2. Pathophysiology

TEN is a type IVc delayed hypersensitivity reaction mediated predominantly by cytotoxic CD8+ T lymphocytes and natural killer cells. These effector cells induce keratinocyte apoptosis through perforin, granzyme B, and granulysin, the latter being a key mediator of widespread epidermal necrosis [6]. The Fas/FasL pathway also plays a role via caspase-dependent apoptosis [7], amplified by pro-inflammatory cytokines (TNF-α, IFN-γ). Prognostic assessment relies on the SCORTEN severity score, originally validated by Bastuji-Garin *et al.* [8], which helps estimate mortality risk and guide management decisions, amplified by pro-inflammatory

cytokines (TNF-α, IFN-γ). Sulfadoxine-pyrimethamine, a sulfonamide widely used for IPTp, is a recognized trigger of severe hypersensitivity reactions in endemic regions [4].

PV is an autoimmune blistering disease caused by IgG antibodies targeting desmoglein 3 (and sometimes desmoglein 1), leading to suprabasal acantholysis. Pregnancy-related immune modulation, with a shift toward a Th2 profile, may exacerbate disease activity in the first trimester, while partial remission can occur in the third trimester. The transplacental transfer of maternal IgG explains neonatal pemphigus, which is typically benign and self-limiting [9]-[12].

3.3. Diagnostic Considerations in Low-Resource Settings

The diagnosis of TEN is primarily clinical, supported by the SCORTEN severity score. In our patient, a score of 4/7 corresponded to a mortality risk of nearly 58%, justifying intensive multidisciplinary care. Histology, although confirmatory, is not essential in acute settings.

PV diagnosis normally combines clinical features, histology, and direct immunofluorescence (DIF). However, in our context, DIF and ELISA were unavailable, and the diagnosis relied on clinical presentation and positive IIF. This illustrates the constraints faced in low-resource settings and highlights the need for improved diagnostic access.

3.4. Therapeutic Strategies and Obstetric Implications

Management of TEN rests on immediate withdrawal of the culprit drug and supportive measures: fluid-electrolyte balance, infection prevention, wound care, and pain management. In our case, multidisciplinary supportive care allowed maternal recovery and the birth of a healthy neonate. Adjunctive therapies such as systemic corticosteroids, IVIG, or cyclosporine have been used with variable efficacy [9], but their use in pregnancy is constrained by safety concerns. **Figure 2** illustrates the progressive healing phase, confirming favorable maternal prognosis with supportive care alone.

In PV, systemic corticosteroids (prednisone or prednisolone) remain the gold standard during pregnancy [13] [14]. Our patient required dose escalation during relapse, with close obstetric monitoring due to the risk of IUGR. Steroid-sparing agents (azathioprine, cyclosporine, IVIG) may be considered for refractory disease [15] [16], while mycophenolate, cyclophosphamide, and methotrexate remain contraindicated. Rituximab is not recommended within 12 months before conception due to potential fetal B-cell depletion [17].

3.5. Prognosis and Literature Insights

TEN in pregnancy carries a high risk of maternal and fetal complications, including preterm labor, fetal distress, and intrauterine fetal demise [2]. In our case, prompt withdrawal of sulfadoxine-pyrimethamine and multidisciplinary support allowed survival despite a high SCORTEN score.

PV generally carries a favorable prognosis for both mother and infant when adequately controlled, although complications such as IUGR and prematurity may occur. Neonatal pemphigus, as seen in our case (**Figure 3**), is the result of passive maternal antibody transfer and usually resolves spontaneously within weeks [11]-[13]. Importantly, the severity of maternal disease does not predict neonatal involvement.

3.6. Recommendations for Low-Resource Settings

Our cases underscore several priorities:

1) systematic drug allergy assessment before prescribing IPTp with sulfadoxine-pyrimethamine;

2) clinician awareness of severe dermatological emergencies during pregnancy;

3) integration of obstetric, dermatologic, neonatal, and intensive care expertise;

4) expanding access to confirmatory diagnostics (DIF, ELISA);

5) implementing structured pharmacovigilance with simplified adverse drug reaction forms, even at the primary care level.

4. Conclusion

Toxic epidermal necrolysis and pemphigus vulgaris during pregnancy are rare but potentially life-threatening conditions with significant maternal and fetal risks. Early recognition, cautious drug prescription, and continuity of dermatological follow-up are essential. In resource-limited settings, strengthening diagnostic capacity, implementing pharmacovigilance systems, and ensuring multidisciplinary collaboration between obstetricians, dermatologists, neonatologists, and intensive care specialists are crucial to improving outcomes.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

References

[1] Roujeau, J.C., *et al.* (1995) Toxic Epidermal Necrolysis (Lyell Syndrome): Incidence and Drug Etiology in Europe. *Archives of Dermatology*, **131**, 37-42.

[2] Mockenhaupt, M., *et al.* (2008) The Risk of Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis Associated with Medications: A Review. *Dermatology*, **215**, 181-187.

[3] WHO (2015) Guidelines for the Treatment of Malaria. 3rd Edition, World Health Organization.

[4] Roll Back Malaria Partnership (2012) Intermittent Preventive Treatment in Pregnancy (IPTp).

[5] Kardos, M., *et al.* (2009) Pemphigus and Pregnancy: A Review. *Journal of the European Academy of Dermatology and Venereology*, **23**, 737-744.

[6] Chung, W., Hung, S., Yang, J., Su, S., Huang, S., Wei, C., *et al.* (2008) Granulysin Is a Key Mediator for Disseminated Keratinocyte Death in Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis. *Nature Medicine*, **14**, 1343-1350.

https://doi.org/10.1038/nm.1884

[7] Viard, I., *et al.* (1998) Involvement of Fas (APO-1/CD95) in the Pathogenesis of Toxic Epidermal Necrolysis. *Science*, **282**, 490-493.

[8] Fouchard, N., Bertocchi, M., Roujeau, J., Revuz, J., Wolkenstein, P. and Bastuji-Garin, S. (2000) SCORTEN: A Severity-of-Illness Score for Toxic Epidermal Necrolysis. *Journal of Investigative Dermatology*, **115**, 149-153. https://doi.org/10.1046/j.1523-1747.2000.00061.x

[9] Schneck, J., *et al.* (2008) Effects of Treatments on the Mortality of Stevens-Johnson Syndrome and Toxic Epidermal Necrolysis. *Archives of Dermatology*, **144**, 1520-1526.

[10] Ioannides, D., *et al.* (2011) Pemphigus Vulgaris: Epidemiology and Pathogenesis. *Clinics in Dermatology*, **29**, 393-398.

[11] Campo-Voegeli, A., *et al.* (2004) Neonatal Pemphigus from Maternal Mucosal Pemphigus Vulgaris. *British Journal of Dermatology*, **150**, 122-127.

[12] Toth, G.G., *et al.* (1994) Neonatal Pemphigus Vulgaris. *Pediatric Dermatology*, **11**, 331-334.

[13] Kaplan, I., *et al.* (2009) Pemphigus Vulgaris in Pregnancy: A Case Series and Review of the Literature. *Oral Surgery, Oral Medicine, Oral Pathology, Oral Radiology, and Endodontology*, **107**, 844-848.

[14] Chi, C.C., *et al.* (2010) Safety of Systemic Corticosteroids in Pregnancy. *Journal of the American Academy of Dermatology*, **62**, 633-639.

[15] Tincani, A., *et al.* (2006) Autoimmune Diseases and Pregnancy Outcomes: A Comprehensive Review. *Autoimmunity Reviews*, **6**, 76-82.

[16] Amagai, M., *et al.* (2014) Intravenous Immunoglobulin Therapy for Autoimmune Blistering Diseases: Current Status and Future Directions. *The Journal of Dermatology*, **41**, 757-765.

[17] Dehghanimahmoudabadi, A., Daneshpazhooh, M., Balighi, K., Mahmoudi, H., Goodarzi, A., Shahidi-Dadras, M. and Chams-Davatchi, C. (2022) Pregnancy Outcomes in Women with Pemphigus Exposed to Rituximab before or during Pregnancy. *Journal of the American Academy of Dermatology*, **87**, 123-130.