Review Article

TEN mimics: Classification and practical approach to toxic epidermal necrolysis-like dermatoses

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Abstract

Toxic epidermal necrolysis (TEN) is an acute life-threatening dermatologic emergency. However, many dermatoses can present with a TEN-like eruption. Those "TEN-mimics" are a true diagnostic challenge and an alarming differential diagnosis to such a serious condition. Herein, we will expose and classify the landscape of TEN-mimics. Also, the key differentiating clinical and/or laboratory points will be highlighted to help an accurate diagnosis of either a TEN or a TEN-like presentation.

Key words: Toxic epidermal necrolysis, Lupus, Nikolsky's signs, SSSS, AGEP

Introduction

Toxic epidermal necrolysis (TEN) is a life-threatening T-cell mediated hypersensitivity reaction mostly to certain medications.[1] The high-risk drugs include anti-epileptics, cotrimoxazole, sulfonamides and allopurinol.[2,3] It takes between one and eight weeks to develop a TEN reaction after the 1st administration of the triggering medication and only a few hours in cases of drug re-intake.[1,4] According to the classification by Bastuji-Garin *et al.*, the features of Stevens-Johnson syndrome (SJS) can overlap with TEN.[5]

Toxic epidermal necrolysis may start with a maculopapular, purpuric or atypical targetoid rash. Eventually, cutaneous tenderness, blistering and denudation with a positive *Nikolsky's* sign supervene. Painful erosive mucositis occurs in most patients with oral involvement in up to 100% of cases. Sepsis with multi-organ failure is the most common cause of death. Survivors may develop cutaneous scarring and/or ocular complications.[1]

The hallmark histopathological finding in TEN is "keratinocyte necrosis." In the well-established lesions, full-thickness epidermal necrosis and sub-epidermal bullae can be seen. A scant perivascular lymphohistiocytic infiltrate with eosinophils is present in the superficial dermis.[6]

The standard management for TEN involves cessation of the offending agent and supportive therapy in a burn unit or an intensive care facility. Intravenous immune globulin (IVIG) with or without systemic corticosteroids has been used with conflicting results. In refractory cases; plasmapheresis could help.[7]

It may appear to some that a definite diagnosis of TEN is easily made based on the above-mentioned clinical and pathological presentation. However, many other dermatoses of an autoimmune or even infective aetiology can simulate TEN syndrome at different stages of evolution. Misdiagnosis of such conditions can potentially result in devastating outcomes.

Recently, a TEN-like presentation for different disorders or "TEN-mimics" has been increasingly reported in the literature. *Herein,* we will classify [Table 1] the landscape of TEN-mimics. Also, the key differentiating clinical and/or laboratory points [Table 2] will be highlighted to help an

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accurate diagnosis of either a TEN or another dermatosis with a TEN-like presentation.

The landscape of TEN-mimics

Proposed classification [Table 1]

Infections

- Bacterial/Bacterial-Toxin:

Staphylococcal scalded skin syndrome (SSSS)

Staphylococcal scalded skin syndrome (SSSS) is caused by an infection with exfoliative toxin-producing *Staphylococcus aureus*. Typically, SSSS is seen in infants and children however, it can occur in susceptible adults.[8]

SSSS starts with fever and irritability. Then, the skin becomes abruptly tender, erythematous with sheets of a TEN-like epidermal detachment with a characteristic "potato chip desquamation"[9] [Figure 1a] mainly at the mid-face and flexural sites associated with a positive *Nikolsky's* sign. Within 24 hours, characteristic perioral and/or periorbital crusts can be seen without mucosal involvement. With appropriate treatment, the skin is expected to heal with no scarring within two weeks.[8,10] The main histopathological finding in SSSS is a sub-corneal cleavage. Once a diagnosis of SSSS is established, anti-staphylococcal antibiotic therapy should be given immediately.[11]

Toxic shock syndrome (TSS)

Toxic shock syndrome (TSS) presents with a high fever of more than 38.9C, hypotension, the involvement of at least three organ systems as well as diffuse erythroderma and desquamation.[12] Menstrual TSS accounts for most reported cases, but non-menstrual TSS cases have increased.[13] TSS is mainly caused by *Staphylococcus aureus* that can produce toxic shock syndrome toxin 1 (TSST-1) and by toxigenic strains of Streptococcus pyogenes rarely.[14] Blood culture is usually positive in streptococcal TSS, and an identifiable source of soft tissue infection can be found. In staphylococcal TSS, the blood culture is only positive in <5% of menstrual cases and in 50% of non-menstrual cases.[15] The histopathological findings are non-specific without evidence of infectious organisms or necrotic keratinocytes.[16] Treatment of TSS involves anti-shock measures, broad-spectrum antibiotics and IVIG.[15]

Purpura fulminans (PF)

Purpura fulminans is a haematological emergency. It is frequently associated with acute disseminated intravascular coagulation (DIC) secondary to sepsis by endotoxin-producing bacteria. The main features of (PF) include tissue necrosis and small vessel thrombosis.[17] Initially (PF) presents with well-demarcated erythematous macules with irregular central areas of blue-black haemorrhagic necrosis.[18]

Epidermal necrolysis secondary to the thrombotic and haemorrhagic cutaneous infarction in (PF) might be confused with TEN, especially with a positive *Nikolsky's* sign. Moreover, both syndromes have been reported simultaneously or successively.[19] Multi-organ failure is very common.[18] Prompt initiation of broad-spectrum antibiotics is the main treatment.[17]

Table 1: Proposed aetiological classification of TEN-mimics

Etiology	TEN-mimics
1-Infections	**a-Bacterial/Bacterial-toxin:**
	Staphylococcal scalded skin syndrome (SSSS)
	Toxic shock syndrome (TSS)
	Purpura fulminans
	b-Viral:
	Coxsackievirus-induced severe mucocutaneous disease
	Chikungunya fever
	Epstein–Barr virus) EBV (positive T-cell lymphoproliferative disease
2-Immune-mediated	Lupus erythematosus
	Dermatomyositis
	Pemphigus vulgaris
	Paraneoplastic pemphigus
	Lichen Planus Pemphigoides
	Graft−versus−host−disease (GVHD)
	Inflammatory epidermolysis *bullosa* acquisita
	Inflammatory epidermolysis *bullosa*
3-Drug-induced	Acute generalised exanthematous pustulosis (AGEP)
	Generalised bullous fixed drug eruption
	Drug-induced linear IgA bullous dermatosis
	Drug reaction with eosinophilia and systemic symptoms (DRESS)
	Contact dermatitis to dendrimers
4-Metbolic	Pseudoporphyria
5-Burn	**a-Chemical:**
	Boric acid intoxication
	Bleaching agents
	b-Thermal:
	Fire or hot liquids injury

- Viral:

Coxsackievirus–induced severe mucocutaneous disease

Type B5 Coxsackievirus has been reported to induce TEN-like skin lesions. The eruption may present with generalised purpura and maculopapular/vesicular lesions. Mucosal lesions include conjunctival hyperaemia, and oral ulcers with haemorrhagic crusts on the lips and/or genitals. Skin lesions heal with hyperpigmentation and onychomadesis may supervene. The disease can result in significant corneal opacities.[20] The histopathological changes mimic those of TEN.[21,22]

Chikungunya fever

An acute viral infection caused by the chikungunya virus. The virus can be transmitted via the Aedes aegypti mosquito to humans. There are several reported outbreaks of chikungunya fever in India with a TEN-like presentation. The histopathological findings are overlapping with TEN syndrome. The extensive keratinocyte necrosis or ballooning

Table 2: Key differentiating points of TEN-mimics

TEN/ TEN-mimics	Cause	Age/Sex	Cutaneous lesions	*Nikolsky's* sign	Mucosal lesions	Systemic symptoms	Laboratory/pathology findings	Treatment
TEN	Drug-induced	Any age group, more in females	Epidermal detachment according to the classification of *Bastuji-Garin et al.*	+Ve	Yes & Painful	Fever, cardio-pulmonary, renal, and gastrointestinal dysfunction	Keratinocyte necrosis "Hallmark"	Supportive, IVIG, systemic steroids, others
Staphylococcal scalded skin syndrome (SSSS)	staphylococcus	More in infants & children	TEN-like, peri-oral & peri-orbital crusts "Potato chips" desquamation	+Ve	No	Fever	Subcorneal cleavage without keratinocyte necrosis Frozen sections & Tzanck smear for a quicker diagnosis Isolation of the organism from colonisation sites	Anti-Staph antibiotics
Toxic shock syndrome	TSST-1	More in menstrual women	TEN-like with marked septic presentation	−Ve	No	Fever, hypotension, internal organs dysfunction	Blood culture Non-specific histopathology	Anti-shock measures Antibiotics IVIG
Purpura fulminans	Endotoxin	Any age group	TEN-like, retiform purpura	+Ve	No	Multiorgan failure	DIC workup Blood culture Bacterial 16S rRNA gene sequencing Skin necrosis & embolism with microthrombi	Immediate broad-spectrum antibiotics
Coxsackievirus-induced severe mucocutaneous disease	Coxsackie-B virus	Any age group	TEN-like	−Ve	Yes	Ocular	Viral culture PCR for throat swab	Supportive
Chikungunya fever	Chikungunya virus	Any age group, endemic in some countries	TEN-like	−Ve	No or minimal	Fever	Anti-Chikungunya IgM nearly 5 days following symptoms Extensive keratinocyte necrosis or ballooning degeneration	Supportive
Epstein–Barr virus positive T-cell lymphoproliferative disease	Epstein–Barr virus	Any age group	TEN-like	−Ve	No	Fever, cough, LNs, HSM, jaundice	Skin infiltration by atypical CD8+ lymphocytes, *in situ* hybridisation, EBV serology, PCR	Supportive
Lupus erythematosus	Autoimmune	Any age group, more in females, no drug causality	TEN-like, more on sun-exposed sites	−Ve	Yes & Painless	Of SLE or SCLE, should be considered in a known case of lupus	Serology, complement levels, histopathology & DIF of lupus	Systemic steroids ± others
Dermatomyositis	Autoimmune MDA5-DM	Any age group	TEN-like	−Ve	No	Exacerbation of DM manifestations	Anti-MDA5, DM laboratory findings	Systemic steroids ± others
Pemphigus vulgaris	Autoimmune	Any age group	TEN-like	+Ve	Yes	No	Acantholysis, IF deposits of PV, anti-Dsg by ELISA	Systemic steroids ± others
Paraneoplastic pemphigus	Autoimmune	More in old adults	TEN-like	−Ve	Yes	Underlying tumour e.g., NHL	Acantholysis, anti-plakin & anti-Dsg antibodies	Systemic steroids ± others, treatment of related tumour
Lichen Planus Pemphigoides	Autoimmune	Any age group	TEN-like, both LP & BP lesions	−Ve	Yes	No	IF findings of BP	Systemic steroids ± others
Graft-versus-host-disease	Autoimmune	Any age group, after hematopoietic stem cell transplantation	TEN-like	−Ve	No	Jaundice, diarrhoea, ileus	Abnormal LFT with hyperbilirubinemia (>15mg/dL), ch.ch. GIT & liver pathology, Analysis of chimerism	Systemic steroids ± others

(Contd...)

Table 2: *(Continued)*

TEN/ TEN-mimics	Cause	Age/Sex	Cutaneous lesions	*Nikolsky's* sign	Mucosal lesions	Systemic symptoms	Laboratory/pathology findings	Treatment
Inflammatory epidermolysis *bullosa* acquisita	Autoimmune	Any age group	TEN-like, with heterogeneous, overlapping or alternating clinical lesions of EBA, DH & BP	+Ve	Yes	No	Absent keratinocytes necrosis DIF, salt-split skin test is diagnostic	Systemic steroids
Acute generalised exanthematous pustulosis (AGEP)	Drug-induced mainly	Any age group	Rapid onset of TEN-like lesions, pustules, facial oedema	+Ve	Yes in 20% of cases	No	Subcorneal /intra-epidermal pustules, marked dermal oedema, patch test	Stop the drug, Systemic steroids ± others
Generalised bullous fixed drug eruption	Drug-induced	Any age group	TEN-like, recurrent blistering, PIH	−Ve	Mild or absent	No	TEN-like histopathological changes	Stop the drug, Systemic steroids ± others
Drug-induced linear IgA bullous dermatosis	Drug-induced especially with vancomycin and phenytoin	Any age group	TEN-like, annular, or poly cyclic configuration of blisters	+Ve or −Ve	No	No	DIF from perilesional skin with linear deposition of IgA along the basement membrane zone is diagnostic	Stop the drug, dapsone, Systemic steroids ± others
Drug reaction with eosinophilia and systemic symptoms (DRESS)	Drug-induced but with ch.ch. delayed presentation after drug intake	Any age group	urticarial, maculopapular, vesicles, bullae, pustules, purpura, target lesions, facial oedema, cheilitis, erythroderma and TEN-like	−Ve	Rare	Hepatitis, pneumonitis, myocarditis, pericarditis, nephritis, and colitis	Eosinophilia, lymphocytosis with atypical lymphocytes and other internal organ dysfunctions	Stop the drug, Systemic steroids ± others
Contact dermatitis to dendrimers	Drug-induced	Adults with history of contact with dendrimers	TEN-like	+Ve	No	None	Patch test, but not advised	Supportive
Pseudoporphyria	Metabolic	Any age group, with end-stage-renal disease on haemodialysis	TEN-like	−Ve	No	Chronic renal failure	Detection of porphyrins in serum or blister fluid, festooned dermal papillae with perivascular PAS reactive material in skin biopsy	Supportive
Boric acid intoxication	Acid Intoxication	Mostly in children and infants	TEN-like, diffuse 'boiled lobster' skin	+Ve	No	Disturbed conscious level, tachycardia, abnormal breathing sounds, blue-green vomitus, body stiffness, fever	Absent keratinocyte necrosis Elevated serum boric acid level (normal <100 mcg/l)	Supportive ± haemodialysis
Bleaching agents	Topical bleaching compounds	Adults, more in females	TEN-like	−Ve	No	No	None	Supportive
Thermal	Fire or hot liquids Accidentally or intentionally in case of child abuse	Any age group	TEN-like, charred hairs in fire injuries, glove, and stocking with "donut sign" in immersion burns	−Ve	No	Fire injury: shock. carbon monoxide poisoning, stridor and respiratory failure	Carboxy-haemoglobin levels in suspected fire/ inhalation burns	Supportive, immersion burns indicate physical abuse

+Ve; Positive, -Ve; Negative, TEN; toxic epidermal necrolysis, IVIG; Intravenous immune globulin, TSST-1; toxic shock syndrome toxin-1, DIC; disseminated intravascular coagulation, PCR; polymerase chain reaction, EBV; Epstein–Barr virus, SLE; systemic lupus erythematosus, SCLE; subacute cutaneous lupus erythematosus, DIF; direct immunofluorescence, DM; dermatomyositis, MDA5; melanoma differentiation antigen-5, IF; immunofluorescence, PV; pemphigus vulgaris, ELISA; enzyme-linked immunoassay, NHL; non-Hodgkin lymphoma, Dsg; desmoglein, BP; bullous pemphigoid, LFT; liver function tests, GIT; gastrointestinal tract, EBA; epidermolysis *bullosa* acquisita, DH; dermatitis herpetiformis, PIH; post inflammatory hyperpigmentation, PAS; periodic acid Schiff.

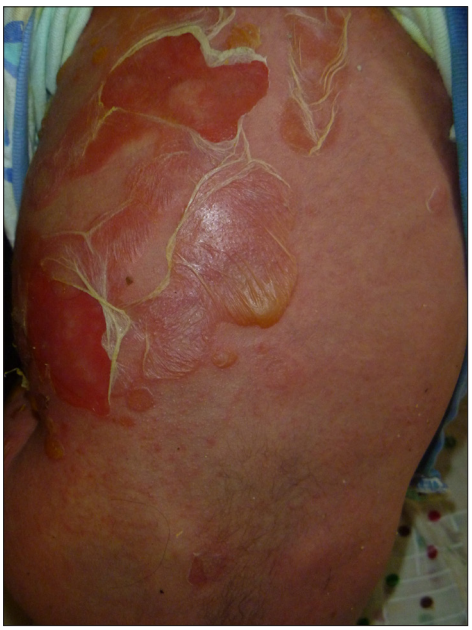

Figure 1a: Toxic epidermal necrolysis-mimics A-Staphylococcal scalded skin syndrome (SSSS) in a 6-months infant

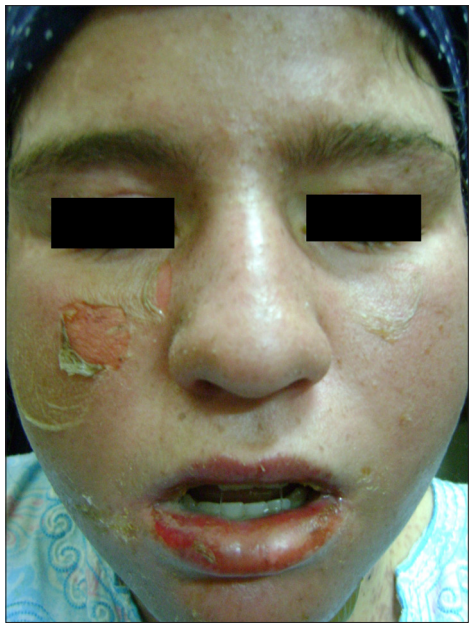

Figure 1b: Toxic epidermal necrolysis-like lupus Note the predilection to the sun-exposed sites

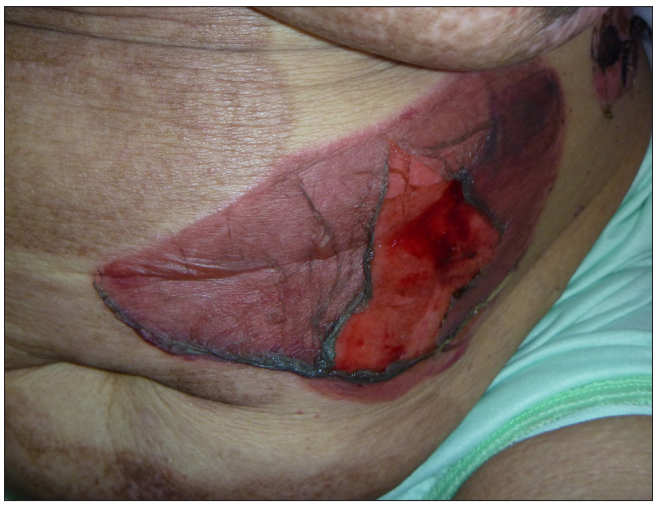

Figure 1c: Bullous fixed drug eruption Note the post-inflammatory hyperpigmentation affecting the abdominal skin from recurrent bullous eruptions

degeneration by the direct spreading of the virus into the skin, or via endothelial injury may explain the TEN-like features. The treatment of TEN-like chikungunya fever is only supportive in nature, and is associated with a favourable prognosis.[23–25]

Epstein–Barr virus) EBV (positive T-cell lymphoproliferative disease

Sharma *et al.* have described a TEN-like eruption in an infant with EBV-positive T-cell lymphoproliferative disease associated with haemophagocytic lymphohistiocytosis. Initially, the patient presented with fever, cough, maculopapular, jaundice, hepatosplenomegaly, and lymphadenopathy. Then, the rash became tender with extensive bullous eruptions and skin detachment.[26] The histopathological findings included epidermal necrosis, basal layer vacuolar changes with apoptotic cells and dermo/epidermal infiltrating lymphocytes with irregular nuclei. Many of the lymphocytes were CD8 + T cells with positive staining for *in situ* hybridisations of EBV-encoded small RNA (EBER). Serum IgM and IgG antibodies against EBV were positive. The virus was isolated using PCR from the blister fluid. Bone marrow biopsy demonstrated histiocytosis and further infiltration by T-lymphocytes positive for EBER.[26]

Earlier to this report, Kawachi *et al.*, have reported a patient demonstrating a similar clinical presentation but with classic TEN-like pathological findings associated with prominent infiltration by EBV-infected CD8 + T-lymphocytes.[27]

Immune-mediated

- Lupus erythematosus:

Acute syndrome of apoptotic pan-epidermolysis (ASAP) is a new term that was 1st proposed by *Ting et al.* to designate clinical entities characterised by acute and massive cleavage of the epidermis resulting from hyperacute epidermal basal cell apoptotic injury.[28] According to Sontheimer's classification for vesiculobullous lesions occurring in the setting of lupus erythematosus, both acute cutaneous lupus or systemic lupus erythematosus (SLE), subacute cutaneous lupus erythematosus (SCLE), and even SLE patients who are not presenting with the lupus-specific skin lesions can mimic TEN,[29] because of an extensive ASAP. Furthermore, TEN-like cutaneous lupus erythematosus is one of the clinical criteria for the diagnosis of SLE according to the Systemic Lupus International Collaborating Clinics classification system.[30]

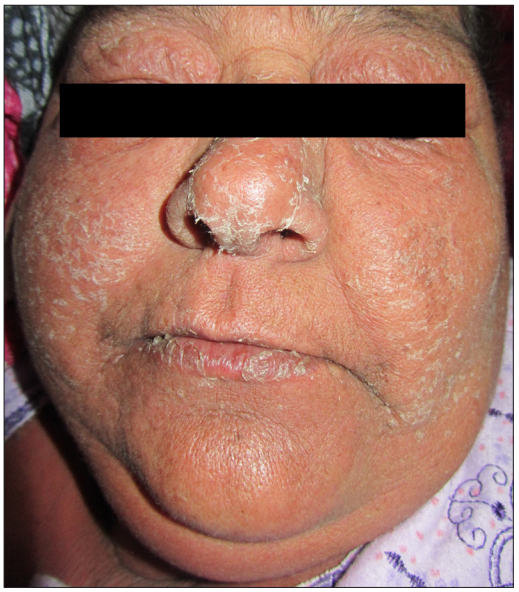

Figure 1d: Drug reaction with eosinophilia and systemic symptoms (DRESS). Note the marked facial oedema and erythroderma

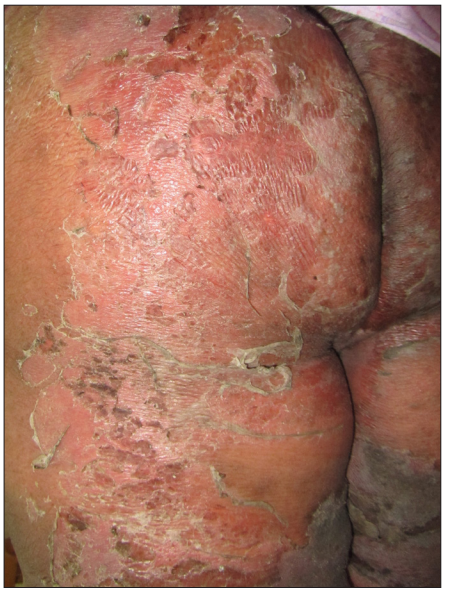

Figure 1e: Drug reaction with eosinophilia and systemic symptoms (DRESS). TEN-like skin detachment

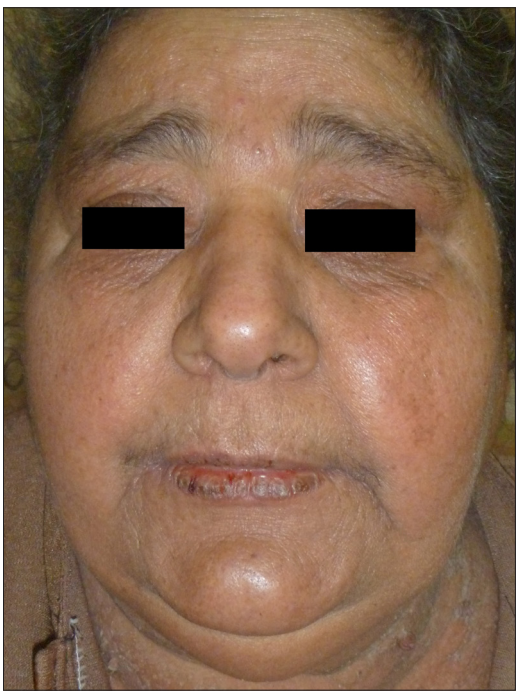

Figure 1f: Drug reaction with eosinophilia and systemic symptoms (DRESS). Resolution of facial oedema after treatment

The TEN-like rash of lupus is clinically indistinguishable from the drug-induced TEN unless there is a high index of suspicion.[31] However, the lack of a triggering medication, the subacute evolution of the TEN-like lesions over weeks, the photo-distribution [Figure 1b] of the TEN-like eruption (although it may also may occur in sun-protected sites), the presence of painless oral ulcers, associated malar or discoid rash, are all suggestive of TEN-like lupus. The serological profile including ANA is highly valuable to support the clinical suspicion of TEN-like lupus.[31] The histopathological findings include both epidermal necrosis and features of cutaneous lupus.[32] Direct immunofluorescence demonstrates positive IgG, IgM and C3 deposition in the basement membrane zone in a granular pattern.[33] The most commonly reported treatments for TEN-like lupus are systemic corticosteroids with or without hydroxychloroquine, IVIG and mycophenolate mofetil.[34]

- Dermatomyositis:

Kushnir-Grinbaum *et al.* have reported a patient with anti-melanoma differentiation antigen-5 (MDA5) dermatomyositis and a TEN-like skin eruption. The new TEN-like rash was mostly triggered by the patients' abrupt quitting of prednisone therapy. However, *Nikolsky's* sign was negative, and the mucous membranes were uninvolved. Skin biopsy revealed similar features to TEN.[35–37]

- Pemphigus vulgaris (PV):

Wakumoto-Nakashima *et al.* have reported a rare presentation of PV associated with TEN-like keratinocytes necrosis. However, the patient also demonstrated lesional intercellular IgG and C3 by direct immunofluorescence (IF) associated with high serum anti-Dsg1 and anti-Dsg3 antibodies by ELISA.[38]

- Paraneoplastic pemphigus (PNP):

There are five main clinicopathological presentations of PNP as described by Nguyen *et al.*[39] Additionally, a TEN-like presentation of PNP is a novel distinctive subtype.[40] It is critical to differentiate true TEN from

TEN-like PNP as the latter is associated with a greater mortality rate. Severe mucositis and keratinocyte necrosis with interface dermatitis are the main overlapping features between the two conditions. Also, the delayed appearance of frank acantholysis in PNP and the possibility of PNP preceding the occurrence of internal malignancy might add more confusion.[40]

In PNP, immunoblotting for envoplakin (EP) or periplakin (PP) is almost 100% sensitive, but with some false-positive results.[41] IIF on rat bladder is commonly used to confirm PNP, given a recorded specificity near 100%; however, SJS/TEN sera also can react with rat bladder.[42]

- Lichen planus pemphigoides (LPP):

Lichen planus pemphigoides (LPP) is an immunobullous disorder that is characterised by a concomitant eruption of bullous pemphigoid (BP) and lichen planus (LP) lesion.[43] Immunofluorescence studies show BP findings with linear deposition of IgG and C3 along the BMZ. Circulating autoantibodies to BP180 is another distinguishing feature.[44]

Ondhia *et al.*, have reported a patient with LPP with extensive TEN-like denuded skin and mucosal ulcers. A skin biopsy from the denuded skin revealed full-thickness epidermal necrosis while a biopsy from an intact blister showed a sub-epidermal split with a lichenoid inflammatory pattern. Direct and indirect IF studies revealed typical BP findings.[44]

- Graft-versus-host-disease (GVHD):

Four clinical stages of cutaneous acute GVHD have been defined of which stage IV is characterised by a TEN-like dermo-epidermal detachment in addition to gastrointestinal and hepatic impairment.[45,46] The differentiation of a TEN-like acute GVHD and true TEN is challenging as both may coexist. In GVHD, skin biopsies can show dyskeratotic keratinocytes, lymphocyte exocytosis, basal cell necrosis, depletion of Langerhans cells and satellite lymphocytes next to the dyskeratotic keratinocytes.[47–49]

- Inflammatory epidermolysis bullosa acquisita (EBA):

Epidermolysis bullosa acquisita (EBA) of the immunopathological type (EBA-IP) has a heterogeneous inflammatory and/or non-inflammatory phase. The inflammatory phase may mimic bullous pemphigoid (BP), mucosal pemphigoid or dermatitis herpetiformis (DH) and the non-inflammatory mechanobullous phase of EBA displays skin fragility, blistering, scarring and milia at the sites of trauma.[50]

Madan *et al.* have described a rare presentation of inflammatory EBA mimicking clinically both TEN and DH. DIF revealed linear basement membrane zone (BMZ) staining with IgG, IgA, IgM and C3. Salt-split skin test showed staining at the base of the blister, favouring the diagnosis of EBA.[51]

Drug-induced

- Acute generalised exanthematous pustulosis (AGEP):

Acute generalised exanthematous pustulosis (AGEP) is a drug-induced cutaneous adverse reaction. The onset of AGEP is rapid, often occurring hours to days after drug exposure.[52] Clinically, it is characterised by fever and the sudden eruption of numerous, tiny, non-follicular, pustules on oedematous erythematous skin. This eruption tends to be generalised with a predilection for the face and/or flexures. Mucosal involvement occurs in only 20% of cases.[53] Atypical lesions in AGEP may include marked facial oedema, purpura, atypical target lesions and skin blisters.[54] Occasionally, a coalescence of pustules occurs resulting in skin peeling resembling TEN. This effect is often referred to as a positive "pseudo" *Nikolsky's* sign.[55] The typical histopathology of AGEP shows spongiform sub-corneal and/or intra-epidermal pustules, often-marked oedema of the papillary dermis and perivascular infiltrate with neutrophils and exocytosis of some eosinophils.[54] Patch testing with the causative drug is often helpful. Generally, AGEP is a self-limiting reaction that heals within two weeks following the withdrawal of the trigger.[53]

- Generalised bullous fixed drug eruption (FDE):

Fixed drug eruption (FDE) is characterised by the recurrence of the skin eruption at the same sites whenever the same drug is re-administered. The lesions usually develop within 30 minutes to 8 hours after drug intake. This eruption appears as well-defined round or oval violaceous plaques associated with itching; however, it can develop generalised blistering or extensive skin detachment simulating TEN.[56] Lesions mostly heal with residual post-inflammatory hyperpigmentation [Figure 1c]. The pathological findings in FDE can be indistinguishable from TEN.[57,58]

- Drug-induced linear IgA bullous dermatosis (LABD):

Most cases of LABD arise spontaneously, but associations with drugs (most commonly vancomycin), infections, lymphoproliferative disorders and internal malignancies have been reported. The disease has a wide spectrum of clinical manifestations including a TEN-like presentation.[59] DIF from perilesional skin is the main diagnostic tool for detecting the linear deposition of IgA along the basement membrane zone.[60,61]

- Drug reaction with eosinophilia and systemic symptoms (DRESS):

Drug reaction with eosinophilia and systemic symptoms (DRESS) is a drug-induced delayed-type hypersensitivity reaction that includes skin eruption, haematologic abnormalities, lymphadenopathy and internal organ involvement.[62] The clinical manifestations usually appear 2–8 weeks after drug intake[63] adding to diagnostic difficulty. The most common drugs to cause this reaction can also induce TEN including anti-epileptics, allopurinol, and sulphonamides. The cutaneous

manifestations [Figure 1d-f] typically consist of an urticarial, maculopapular eruption and, in some instances, vesicles, bullae, pustules, purpura, target lesions, facial oedema, cheilitis, erythroderma and TEN-like lesions.[64,65] One important clinical finding in most patients is head and neck oedema which is often noticeable by looking at the ears.[66] Mucous membrane involvement is very rare.[62] Systemic involvement is the major cause of mortality in DRESS syndrome.[67,68] Furthermore, the clinical features of DRESS may overlap with AGEP.[69]

The histopathological features of DRESS include spongiosis, a superficial perivascular lymphocytic infiltrate and an eosinophilic infiltrate in the dermis.[69] Basal cell vacuolization and keratinocytes necrosis resembling EM may be found. Walsh *et al.* reported a correlation between those EM-like changes and severe liver dysfunction with higher mortality.[70]

Treatment consists of immediate withdrawal of all suspected medications, followed by careful monitoring and supportive care. Systemic corticosteroids are the first line of therapy, however; other immunosuppressive have been used.[71]

- Contact dermatitis to dendrimers:

Dendrimers belong to a class of polymers that are recently used for drug delivery systems in nanotechnology. In 2008, *Toyama et al.* reported a severe case of contact dermatitis to dendrimers with a TEN-like presentation in a student who was handling dendrimers during some laboratory experiments. The skin biopsy showed confluent epidermal necrosis with partial eosinophilic degeneration and mononuclear cell infiltration at perivascular and subepidermal areas.[72]

Metabolic

- Pseudoporphyria:

A TEN-like rash was reported in a patient on chronic haemodialysis who developed pseudoporphyria. Serum and vesicular fluid analysis using direct spectrofluorometry showed diagnostic findings of pseudoporphyria. Multiple skin biopsies revealed sub-epidermal blisters with an intact roof and a characteristic festooning of dermal papillae. Also, a Periodic Acid-Schiff (PAS) positive material was seen around the superficial blood vessels.[73]

Burn

- Chemical:

Boric acid intoxication

Boric acid is an inorganic acid well absorbed across the gastric *mucosa* and abraded skin surfaces. It is colourless, odourless and water-soluble. The acid is used in wound irrigation solutions, eyewashes and mouthwashes. Toxicity mainly occurs in infants and children.[74]

Patients present with disturbed conscious levels, tachycardia, abnormal breathing sounds, blue-green vomitus, body stiffness, fever, and a skin rash.[75] Within hours of ingestion, a diffuse erythematous "boiled lobster" desquamation occurs with a positive *Nikolsky's* sign. Haemorrhagic lip crusts with intra-oral sparing and conjunctival oedema can be seen.[74,76] Histopathologic examination reveals mainly an intact epidermis, but with alternating parakeratosis and orthokeratosis.[74] There is no antidote to boric acid poisoning and only supportive care is recommended.[77]

Bleaching agents

In 2015, Totri *et al.*[78] reported a severe TEN-like presentation which was induced by a self-skin bleaching recipe of "betamethasone 0.05% ointment, hydrogen peroxide 12%, and potassium persulfate" combination. Furthermore, a skin biopsy revealed TEN-like pathology.

- Thermal:

The early description of TEN syndrome was as a "burn-like" syndrome. Thermal burns can occur accidentally or intentionally as in the case of child abuse. Furthermore, thermal burn victims may develop later an attack of TEN. Clinically, patients with thermal burns present with painful, erythematous, blistering or deeply denuded skin with variable degrees of shock. Inhalation injury, carbon monoxide poisoning, upper airway swelling, stridor and respiratory failure are common in patients with fire-induced burns[79]. Furthermore, immersion burns of the buttocks and extremities are caused by dunking a child into scalding water. Immersion burns are usually sharply demarcated in a glove and stocking distribution. Sparing of the buttocks or "donut sign"[80] suggests child abuse.[81]

Conclusion

Toxic epidermal necrolysis (TEN) is a serious dermatologic emergency. Many other dermatoses can present with TEN-like manifestations which is a critical diagnostic challenge. The proposed etiological classification and key differentiating points of all TEN-mimics would help our colleagues reach the correct diagnosis. Detailed history taking, careful skin and mucosal examination in addition to the proper selection of laboratory workup are mandatory to distinguish between TEN and TEN-mimics.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflict of interest

There are no conflicts of interest.

Abbreviations

TEN; toxic epidermal necrolysis
SJS; Stevens-Johnson syndrome
IVIG; intravenous immunoglobulin
SSSS; staphylococcal scalded skin syndrome
Dsg-1; desmoglein-1
TSS; toxic shock syndrome
TSST-1; toxic shock syndrome toxin-1
DIC; disseminated intravascular coagulation
EBV; Epstein–Barr virus
EBER; Epstein–Barr virus EBV-encoded small RNA
PCR; polymerase chain reaction
ASAP; Acute Syndrome of Apoptotic Pan-Epidermolysis

SLE; systemic lupus erythematosus
SCLE; subacute cutaneous lupus erythematosus
IF; immunofluorescence
Ig; immunoglobulin
MDA5; anti-melanoma differentiation antigen-5
PV; pemphigus vulgaris
ELISA; enzyme-linked immunosorbent assay
PNP; paraneoplastic pemphigus
EP; envoplakin
PP; periplakin
IIF; indirect immunofluorescence
LPP; lichen *planus* pemphigoides
BP; bullous pemphigoid
LP; lichen planus
GVHD; graft versus host disease
EBA; epidermolysis *bullosa* acquisita
EBA-IP; epidermolysis *bullosa* acquisita-immunopathological
DH; dermatitis herpetiformis
DIF, direct immunofluorescence
BMZ; basement membrane zone
AGEP; acute generalised exanthematous pustulosis
FDE; fixed drug eruption
LABD; linear IgA bullous dermatosis
DRESS; drug reaction with eosinophilia and systemic symptoms
EM; erythema multiforme
PAS; Periodic acid–Schiff.

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