EMERGENT DIAGNOSIS OF THE UNKNOWN RASH

AN ALGORITHMIC APPROACH

Clinicians must rapidly determine whether a rash is dangerous—or even life-threatening—and act quickly to reduce the risk for mortality.

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Consider this clinical vignette: An obtunded man with limited ability to provide a pertinent history presents to the ED at 2 AM. The nurse states that the man "may have been burned." Vital signs at triage included a heart rate of 120 beats/min; respiratory rate, 24 breaths/min; and temperature, 37.7°C. In addition, a blood pressure of 110/60 mm Hg and pulse oxygenation of 98% were recorded. Your assessment finds the patient is a lethargic and toxic-appearing man about 45 years of age, with diffusely erythematous skin that is sloughing off in large sheets (Figure 1). He appears very dehydrated, with oral mucous membrane involvement. What is your differential diagnosis at this point?

Although thermal burn is in the differential diagnosis, this patient has toxic epidermal necrolysis (TEN), which is characterized by erythematous skin and sloughing and has a mortality of 30% to 35% with optimal treatment.[1] Early recognition is critical.

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INTRODUCTION

According to 2006 data, rash is among the top 20 reasons for ED visits in the United States.[2] This article presents a set of novel and easily interpreted algorithms designed to guide the emergency physician in identifying the most common—and potentially lethal—rash syndromes. While an exhaustive review of all pediatric exanthems, endemic fungal etiologies, and parasitic exanthems is beyond the scope of this article, the algorithms presented here will equip

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the emergency physician to quickly recognize the most common and critical rashes. Table 1 depicts common terms associated with the diagnosis of a rash. Table 2 categorizes rash syndromes according to patient characteristics and presenting symptoms. Following the presentation of the algorithms, we review the salient points of the most clinically important diseases.

HISTORY

When taking a history from a patient with a rash, the clinician should elicit information about the following:

Onset and Progression

The distribution and progression of the rash are essential features. The vasculidities generally begin peripherally and spread centrally. Rocky Mountain spotted fever (RMSF) classically starts on the wrists/ankles and spreads centripetally. Viral rashes usually begin centrally and progress peripherally.[3] Rashes of the palms and soles can be drug induced or infectious (eg, target lesions of erythema multiforme [EM], secondary syphilis, RMSF). Localized lesions may represent a contact dermatitis or infectious process.

Most deadly rashes progress rapidly. Urticaria with anaphylaxis can spread within minutes. The petechiae associated with meningococcemia appear after toxicity has begun and progress within hours. Drug reactions may develop over days.

Travel History

It is important to determine if the patient has recently traveled. Lyme disease is common in the mid-Atlantic, central, western, and northeastern parts of the United States. Toxic patients who have recently traveled to the Caribbean may have Dengue fever. And, of course, patients reporting recent camping and travel through wooded areas are candidates for RMSF.

Medical or Occupational History

Those with diabetes, HIV, a history of intravenous drug abuse, and patients undergoing chemotherapy are at risk for diseases of high morbidity and mortality: meningococcemia, thrombotic thrombocytopenic purpura (TTP), necrotizing fasciitis, disseminated zoster, EM, Stevens-Johnson Syndrome (SJS), TEN, and sepsis. Persons with valvular heart disease are at increased risk for endocarditis. College students, military personnel, and employees of day care facilities are more likely to contract meningococcemia. Hunters and campers are at risk for tick-borne illnesses.

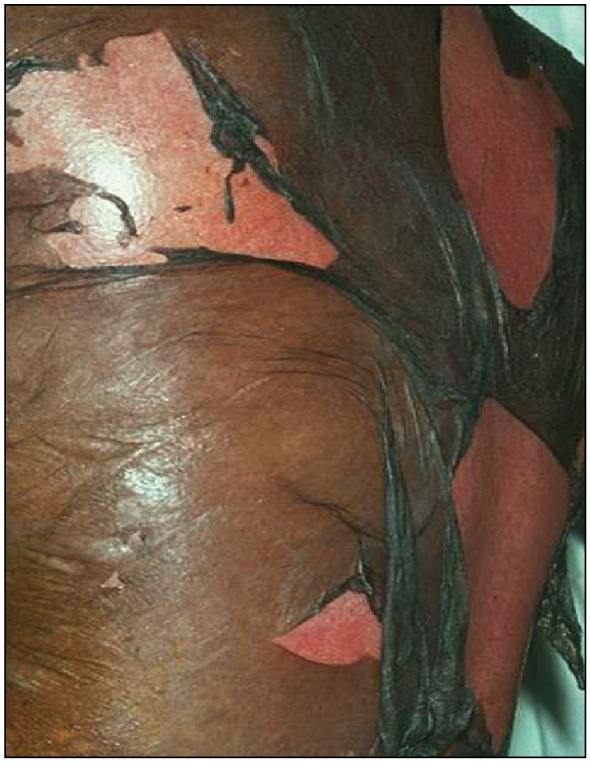


FIGURE 1. Toxic epidermal necrolysis.

Medication Regimens

Determine which medications the patient is taking. Potentially lethal drug reactions such as SJS, TEN, anaphylaxis, and angioedema mandate specific and emergent interventions. Additionally, many patients self-treat rashes prior to presentation. Steroid creams, in particular, may significantly alter rash morphology.[3]

PHYSICAL EXAMINATION POINTERS

To begin, it is essential to evaluate vital signs. Fever and hypotension, in particular, are ominous findings that mandate expedited and intensive care. Additional physical exam findings of concern include new-onset heart murmur or nuchal rigidity. Generalized lymphadenopathy is present in many illnesses, including mononucleosis and other infections, serum sickness, and drug reactions.

TABLE 1. Common Terms Used in Rash Diagnosis

Lesion	Single small, diseased area	**Rash**	An eruption on the skin; more extensive than a single lesion
Macule	Circumscribed area of change without elevation	**Pustule**	Circumscribed area containing pus
Papule	Solid raised lesion ≤1 cm	**Vesicle**	Circumscribed fluid-filled area ≤1 cm
Nodule	Solid raised lesion ≥1 cm	**Bulla**	Circumscribed fluid-filled area ≥1 cm
Plaque	Circumscribed elevated confluence of papules ≥1 cm	**Petechia**	Small red/brown macule ≤1 cm that does not blanche

TABLE 2. Clues Toward a Definitive Diagnosis

Clues to Diagnosis	Rash
Patient Age	
0 to 5 years	Meningococcemia, Kawasaki disease, viral exanthem
>65 years	Pemphigus vulgaris, sepsis, meningococcemia, TEN, SJS, TSS
Rash Characteristics	
Diffuse erythema	Staphylococcal SSS, staphylococcal or streptococcal TSS, necrotizing fasciitis
Mucosal lesions	EM major, TEN, SJS, pemphigus vulgaris
Petechiae/purpura	Meningococcemia, necrotizing fasciitis, vasculitis, DIC, RMSF
Symptom	
Hypotension	Meningococcemia, TSS, RMSF, TEN, SJS

TEN = toxic epidermal necrolysis; SJS = Stevens-Johnson syndrome; TSS = toxic shock syndrome; SSS = scalded skin syndrome; EM = erythema multiforme; DIC = disseminated intravascular coagulopathy; RMSF = Rocky Mountain spotted fever.

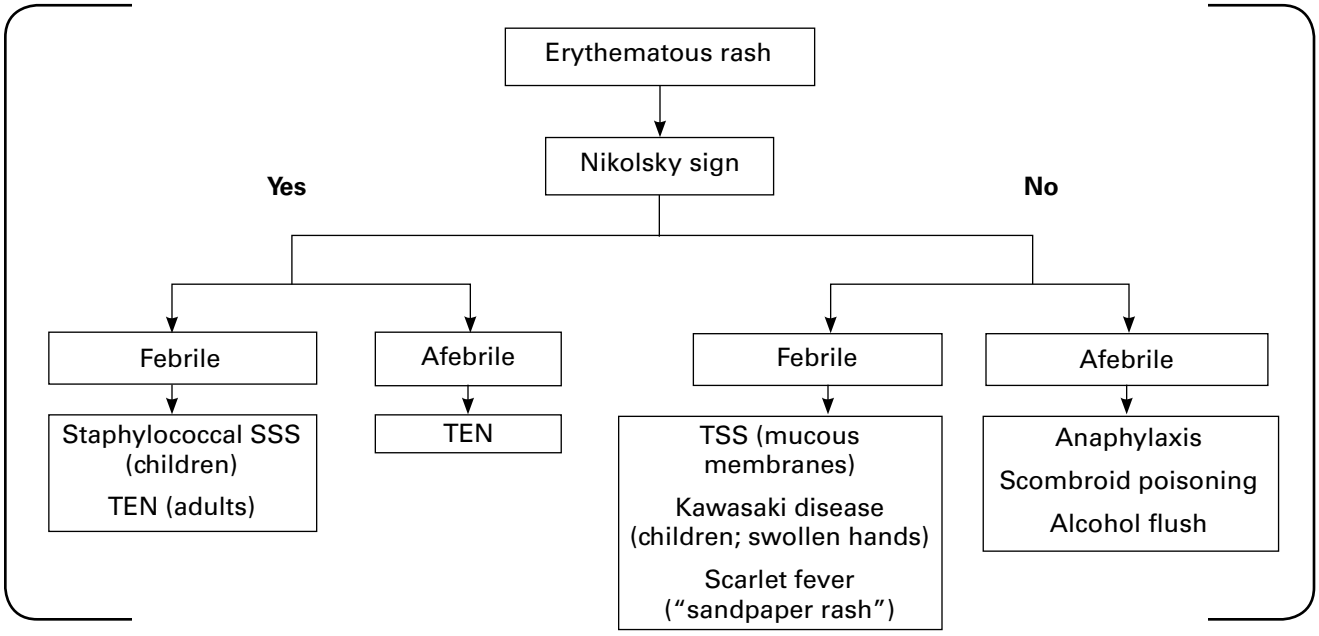
Thorough Examination

To ensure that lesions on the back, buttocks, or perineum are identified, patients should be completely undressed for the examination. Often, patients are unaware of lesions that are present in these locations. The patient's shoes and socks should also be removed (especially in diabetic persons), as significant bacterial and fungal infections may be present on the feet. Toenails should be closely inspected for signs of systemic disease or fungal infection. Additionally, the soles and palms should be examined.

Mucous Membrane Involvement

Dysphagia, eye, or genital irritation may represent mucosal involvement and possibly indicate a life-threatening condition such as TEN, SJS, or pemphigus vulgaris. Conjunctival injection is often also associated with viral syndromes, as well as with Kawasaki disease.

FIGURE 2. Diagnosis of the Erythematous Rash



SSS = scalded skin syndrome; TEN = toxic epidermal necrolysis; TSS = toxic shock syndrome.

Specific Signs

The Nikolsky sign is a sloughing of full-thickness skin with lateral pressure. The Asboe-Hansen sign is a blister that spreads into clinically normal skin with light lateral pressure. Both are seen in TEN, as well as in other conditions. It is also important to look for blanching in the diagnosis of petechial rashes.[3]

THE ALGORITHMIC APPROACH

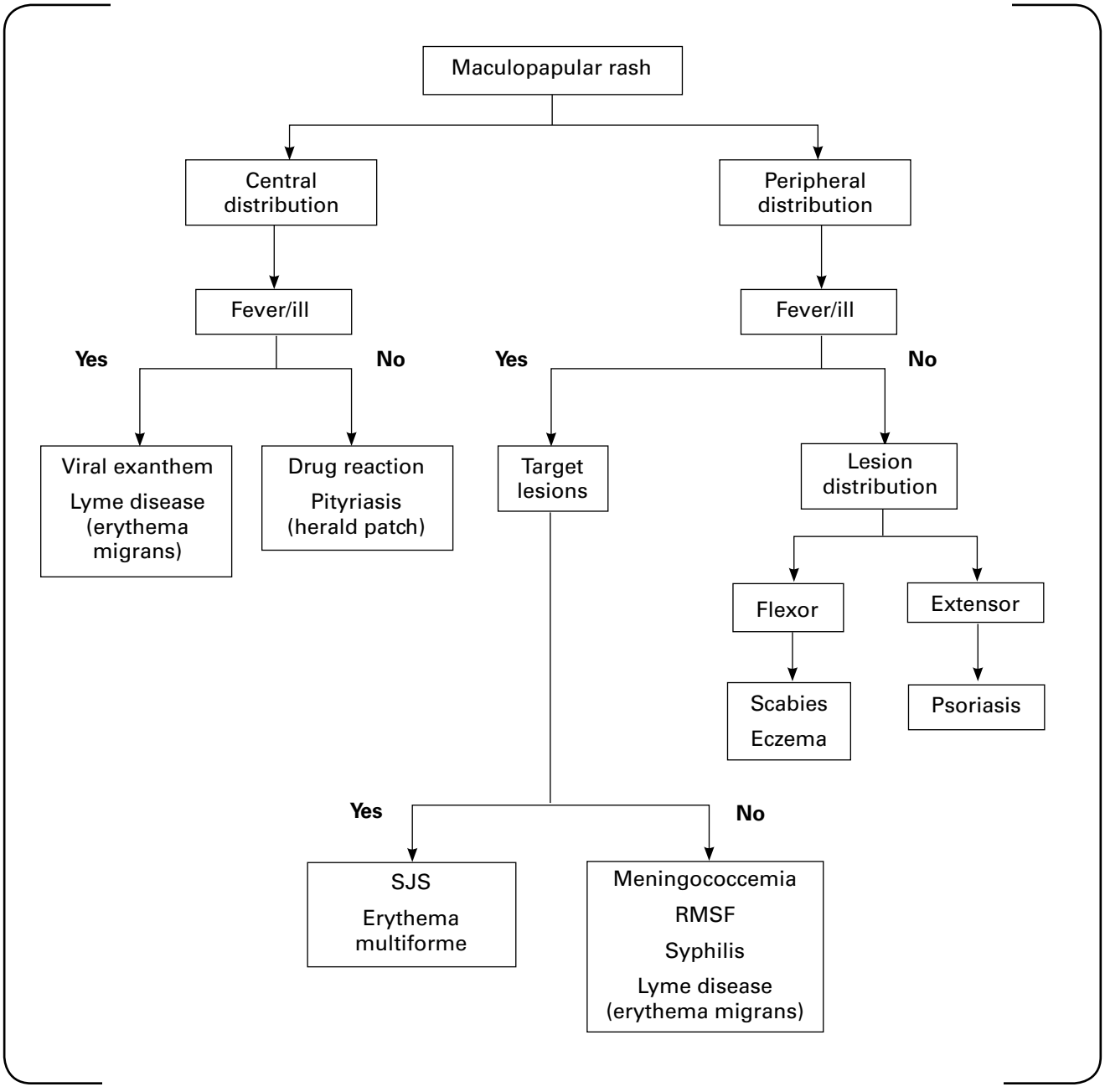
Erythematous Rashes

Characterized by diffuse redness of the skin due to capillary congestion, erythematous rashes (Figure 2, page 9) are differentiated from other rashes based on the presence or absence of fever and the Nikolsky sign. If these factors are present, the diagnosis is narrowed substantially, usually to TEN in adults and staphylococcal scalded skin syndrome (SSS) in infants and young children. If fever is present without the Nikolsky sign, the differential diagnosis includes Kawasaki disease, scarlet fever, and toxic shock syndrome (TSS). Those patients with an erythematous rash but without a fever or Nikolsky sign may be having an anaphylactic reaction or an exposure reaction to scombroid or alcohol.

Staphylococcal Scalded Skin Syndrome: Known as *Ritter disease* or *dermatitis exfoliativa neonatorum*,[4] staphylococcal SSS presents as a scarlatiniform, erythematous rash that blisters and sloughs (positive Nikolsky sign). Children younger than 5 years are at highest risk. Patients initially have an abrupt fever, erythema of the neck, axillae, and groin, and extreme skin tenderness. Diagnostic clues include a lack of mucous membrane involvement and a skin cleavage plane that is more shallow than that associated with TEN. Treatment of this infection includes antistaphylococcal antibiotics, fluid and electrolyte management, and local wound care. Young, well-appearing patients with minimal skin sloughing may be managed as outpatients. In young children, this disease has a mortality of less than 5%; in contrast, this disease is very rare in adults, but when it occurs, mortality can be as high as 60%.[4,5]

Toxic Epidermal Necrolysis: TEN, also known as *Lyell disease*, is the most serious cutaneous drug reaction and most commonly associated with sulfa drugs. Other important triggers include anticonvulsants, antivirals, NSAIDs, and allopurinol. TEN presents as sudden-onset diffuse erythema with tender skin and sloughing. Symptoms occur first on the face and around the eyes, spread caudally to the shoulders and upper extremities,

FIGURE 3. Diagnosis of the Maculopapular Rash



SJS = Stevens-Johnson syndrome; RMSF = Rocky Mountain spotted fever.

and then progress to the whole body. The skin cleavage is full thickness (positive Nikolsky and Asboe-Hansen signs), with massive skin sloughing in large sheets. Patients with TEN are toxic, with myalgias and substantial mucous membrane involvement. The mortality of TEN is considerable—30% to 35% with optimal care.[1]

At-risk populations include those with head injuries, brain tumors, systemic lupus erythematosus (SLE), and immunocompromise.[6] Importantly, HIV patients have a TEN risk that is 1,000 times greater than that in patients without HIV.[6]

Treatment consists of discontinuation of the offending agent, wound care, eye care, and fluid and

electrolyte resuscitation. Intravenous immune globulin (IVIG) may be helpful, although it is not yet FDA approved for this indication. Most physicians recommend against steroid use. Sulfadiazine should not be used on the wounds, as sulfa is the most common offending agent.[7] Patients with TEN usually require ICU admission and should be managed in a burn unit if skin sloughing is extensive.

Toxic Shock Syndrome: This toxin-mediated staphylococcal or streptococcal infection is historically associated with tampon use, although any staphylococcal or streptococcal source can precipitate TSS. Up to 45% of cases are unrelated to menses; TSS can be associated with abscesses, nasal packing, surgical wounds, and postpartum conditions.[3] Patients are overtly toxic, in shock, and febrile, with a diffuse erythematous rash that eventually leads to desquamation of the hands and feet. Treatment includes removal of the infective material, administration of IV antibiotics, fluid resuscitation and, possibly, IVIG. Patients with TSS require ICU admission.

Kawasaki Disease: This childhood illness is also known as *mucocutaneous lymph node syndrome* or *infantile polyarteritis.* Kawasaki disease is a vasculitis of unknown cause, although infective and autoimmune theories abound. It affects many systems, including the skin, mucous membranes, lymphatics, and blood vessels. Diagnostic criteria include high fever for at least 5 days, diffuse erythroderma, strawberry tongue, significant cervical lymphadenopathy, conjunctival injection, peeling of the fingers and toes, and edema of the extremities.[8] By far the most serious complication is vasculitis of the coronary arteries, leading to coronary vessel aneurysms and myocardial infarction, even in young children. Treatment comprises high-dose aspirin (given immediately), hospitalization with supportive care, and (very importantly) IVIG. Kawasaki disease does not respond to antibiotics.[8,9]

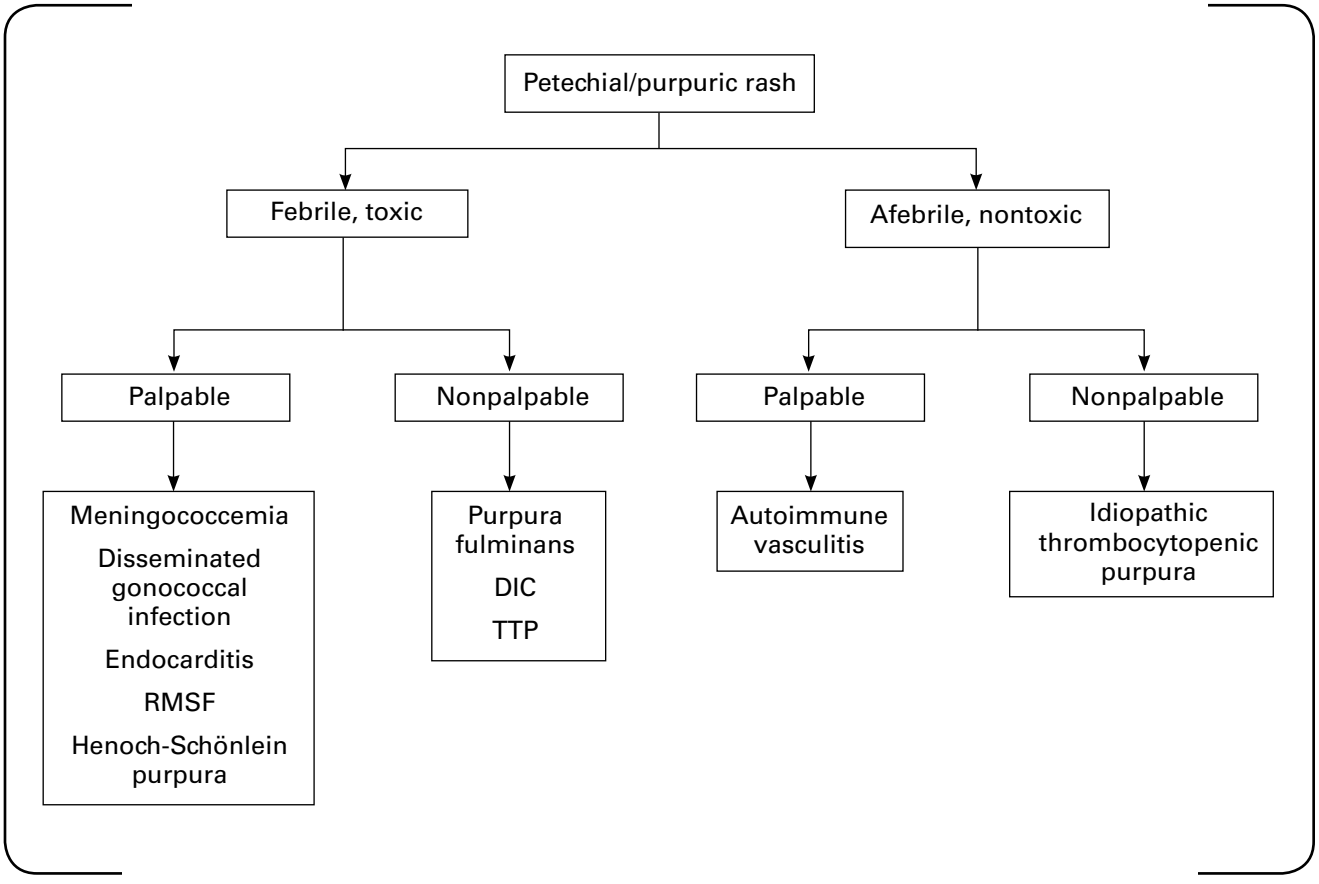
Maculopapular Rashes

The term *maculopapule* is a portmanteau, a combination of *macule* and *papule* (Table 1, page 8). Maculopapular rashes are differentiated based on the distribution of the rash and systemic toxicity (Figure 3, page 10). Patients with centrally distributed rashes who appear toxic and febrile have a wide differential diagnosis; however, it is paramount that patients living in endemic areas be assessed for Lyme disease. Those with centrally distributed rashes but without signs of toxicity usually present with either a drug reaction or pityriasis rosea. Patients with peripherally distributed rashes have a broader differential diagnosis, which is dependent upon systemic toxicity, presence or absence of target lesions, and whether the rash is located on the flexor or extensor surfaces. Target lesions are pathognomonic for SJS or EM. Patients with peripheral lesions and systemic toxicity but without target lesions require emergent evaluation for meningococcemia, RMSF, and syphilis. Nontoxic patients with a peripherally distributed rash but without target lesions require further assessment for flexor involvement (scabies or eczema) or extensor involvement (psoriasis).

Lyme Disease: This tick-borne illness is caused by *Borrelia burgdorferi*. The patient generally presents with erythema migrans (a large annular lesion with dark red border and central clearing) at the site of the tick bite. This rash begins with tick inoculation and therefore may be central or peripheral.[3] As the rash spreads hematogenously over days to weeks, the patient may experience a variety of systemic symptoms, including a secondary skin rash (annular lesions), fever, meningitis, atrioventricular nodal block, migratory arthralgias, and myalgias. Neuritis occurs as well, often manifesting as Bell's palsy (which may be bilateral); however, any nerve can be affected. The diagnosis is made clinically, although a biopsy of the of the site of the tick bite is often diagnostic. Serologic tests are positive after several weeks but do not differentiate active from inactive infection. Doxycycline is the first-line treatment in nonpregnant adult patients. Children may be treated with amoxicillin.

Stevens-Johnson Syndrome: This condition often occurs as a drug reaction, although infections and malignancies have been implicated. Previously, SJS was thought to be linked with EM, but recently it has been reclassified with TEN.[6] Patients present with diffusely distributed target lesions that include the palms and soles, as well as mucous membrane involvement. These patients are toxic, with many constitutional symptoms. Treatment involves discontinuation of the offending agent and optimizing fluid and electrolyte levels. Use of steroids is considered controver-

FIGURE 4. Diagnosis of the Petechial/Purpuric Rash



RMSF = Rocky Mountain spotted fever; DIC = disseminated intravascular coagulopathy; TTP = thrombotic thrombocytopenic purpura.

sial. Both SJS and TEN have greater mortality (10% and 30%, respectively) than do EM major (mortality less than 5%) and minor (negligible morbidity). Patients with SJS require ICU admission.[1,5-7,10]

Erythema Multiforme: This condition may present as a mild, self-limited rash (EM minor) or a severe, life-threatening disease with significant mucous membrane involvement (EM major).[6] The etiology cannot be identified in up to 50% of affected patients, but EM is thought to be autoimmune in nature and usually follows infection (herpes simplex, *Mycoplasma*, fungal diseases) or drug exposure (sulfa drugs, anticonvulsants, antibiotics). The mild form presents as pruritic, symmetrically arranged lesions on the extremities that develop into classic target lesions; these resolve in 1 to 2 weeks. This rash does not involve the mucous membranes. EM major presents with target lesions and significant mucous membrane involvement. Prodromal symptoms (eg, mild, nonspecific upper respiratory tract infection, moderate fever, general discomfort, cough, sore throat, vomiting, chest pain, diarrhea) occur 1 to 2 weeks prior to the onset of rapidly progressive lesions that spread centripetally. The rash is maculopapular and nonpruritic; it evolves into target lesions on the palms, soles, dorsa of the hands, face, and extensor surfaces. Eye involvement occurs in 10% of cases, often as a bilateral purulent conjunctivitis. Diagnosis is confirmed by biopsy. Mild cases (EM minor) require only symptomatic support (analgesics, cold compresses, topical steroids), treatment of cause (if identified), and outpatient dermatologic follow-up. Erythema major requires more aggressive care, with discontinuation of the offending agent, fluid and electrolyte balance, analgesics, wound care similar to that of thermal burns (silver sulfadiazine should be avoided), and soothing solutions for oral le-

sions.[3] The use of systemic steroids is of unproven benefit and may increase complications. Dermatologic consults are required for admitted patients, as are ophthalmologic consults for those with ocular involvement.[3]

Meningococcemia: Infection with *Neisseria meningitidis* has a predilection for adolescents and children younger than 4 years. Without proper treatment, meningococcemia is invariably fatal; mortality remains at 10% to 20%, even with immediate therapy.[3] Patients are ill appearing, febrile, and in shock, with mental status changes and a rash that develops within 24 hours of toxicity. The rash is initially erythematous and maculopapular (beginning on wrists and ankles); it then spreads and becomes petechial. Early in the illness, meningococcemia can be mistaken for RMSF. Treatment for both is mandatory when there is any diagnostic uncertainty. Diagnosis is confirmed by Gram stain and blood and/or cerebrospinal fluid (CSF) cultures. Gram staining of a specimen from a meningococcal skin lesion is more sensitive than a CSF Gram stain (72% vs 22%, respectively).[3] Ceftriaxone is first-line therapy. Vancomycin should be added in cases of diagnostic uncertainty to cover resistant streptococcal meningitis. Dexamethasone has been shown to reduce neurologic sequelae if administered early (prior to antibiotics, if possible). Rifampin prophylaxis for persons in close contact with the patient is recommended; alternatives include single-dose ciprofloxacin and IM ceftriaxone.[3]

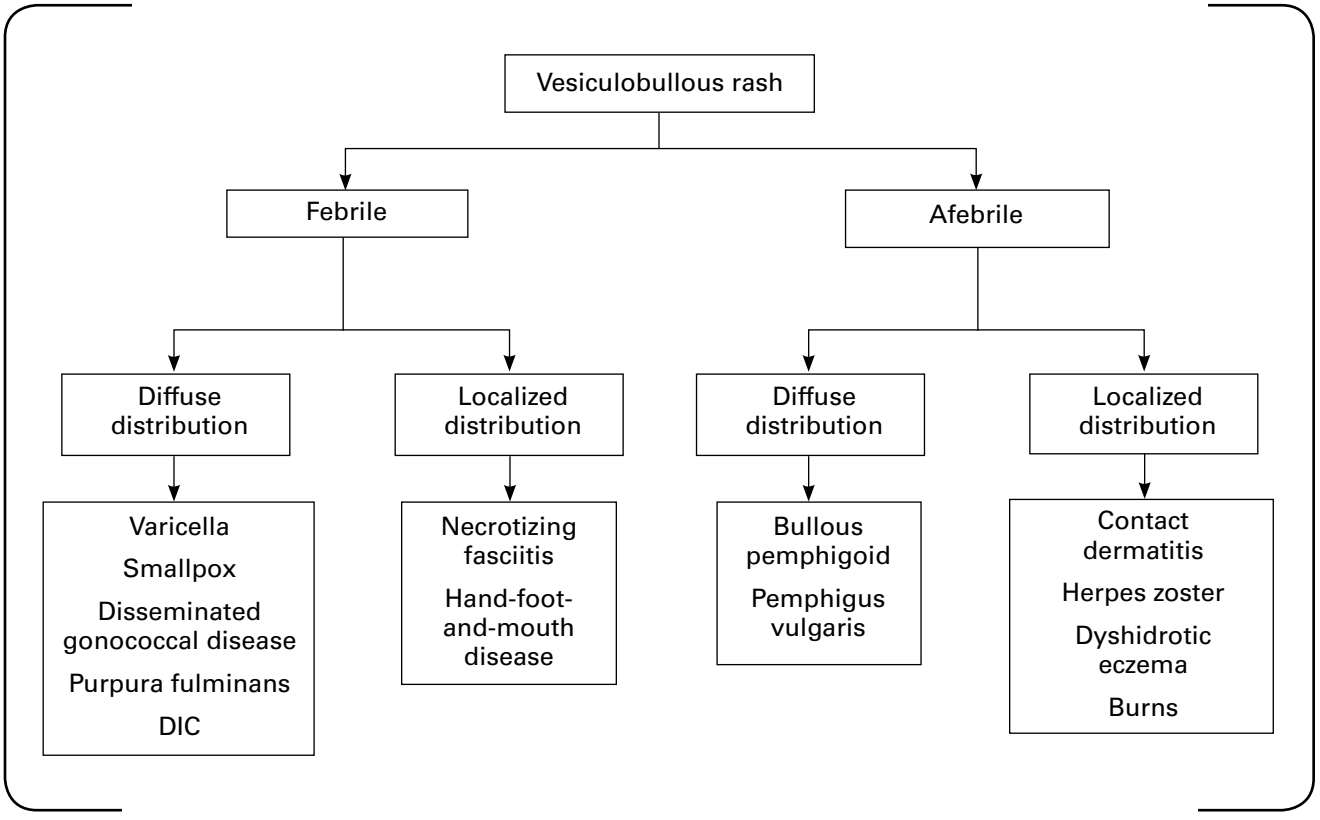
Rocky Mountain Spotted Fever: Another tick-borne illness (*Rickettsia rickettsii*), RMSF occurs primarily in the south-Atlantic region and also occurs in other regions of the United States. Only 50% of affected patients can recall being bitten by a tick.[3] The erythematous maculopapular rash begins on the wrists and ankles and spreads over the body. In its early stage, the rash presents as reddish macules that blanch, only to become petechial and purpuric later. In up to 20% of patients, the rash is absent (spotless fever).[3] Regardless of rash presence or absence, the patient will be highly febrile and toxic. For RMSF, the diagnosis is made clinically. Physicians should not wait for confirmatory antibody test results before beginning treatment, as these will be negative in the acute period. If untreated, RMSF has a mortality greater than 30%; this decreases to 5% with prompt antibiotic therapy. Permanent neurologic deficits persist in 15% of cases.[11-15] Doxycycline is the drug of choice in all nonpregnant patients, even children. Pregnant patients may be treated with chloramphenicol.[3,11-15]

Petechial/Purpuric Rashes

These rashes can be especially challenging and are associated with devastating differential diagnoses; however, an algorithmic approach can help the physician narrow the diagnosis with confidence (Figure 4, page 12). Additionally, remembering the etiology of palpable versus nonpalpable lesions is paramount. Palpable (raised) purpura occurs in vasculitic diseases secondary to inflammation or infection. Nonpalpable purpura presents in thrombocytopenic conditions (flat, subcutaneous hemorrhages). Patients with petechiae/purpura with fever or toxicity require emergent evaluation. If the lesions are palpable, the differential diagnosis includes meningococcemia, disseminated gonococcal disease, endocarditis, RMSF, and Henoch-Schönlein purpura. Those with petechiae/purpura with fever/toxicity but with nonpalpable lesions may have purpura fulminans, disseminated intravascular coagulopathy (DIC), or TTP. If the patient is afebrile with a petechial or purpuric rash, the diagnosis may be far simpler and less ominous. Nontoxic patients with palpable lesions may have a vasculitis, such as autoimmune vasculitis; those with nonpalpable lesions may have idiopathic thrombocytopenic purpura. While all patients with petechiae require complete assessment, those with nonpalpable petechiae are more likely to have thrombocytopenia.

Henoch-Schönlein Purpura: Also known as *allergic purpura* or *anaphylactoid purpura*, this disease is found mainly in children. It is an autoimmune systemic vasculitis affecting primarily the skin (usually on the legs, buttocks, and arms) and kidneys. It is often preceded by an infection or drug exposure. The classic triad associated with this disease comprises purpura, abdominal pain, and arthritis (particularly in the knees, ankles, and elbows). The purpura is palpable and pruritic. The abdominal pain may be associated with nausea, vomiting, intussusception, diarrhea, or constipation. Hematuria is classic and occurs in 10% to 20% of cases; however, less than

FIGURE 5. Diagnosis of the Vesiculobullous Rash



DIC = disseminated intravascular coagulopathy.

1% of children go on to develop end-stage renal disease.[16] Most patients require only supportive care, although relapses occur. Some patients require hospitalization for pain control, kidney biopsy, and/or administration of immunosuppressant agents or, occasionally, IVIG. The use of steroids is controversial. The prognosis is good, with full recovery in more than 80% to 90% of patients; permanent kidney damage is rare.

Purpura Fulminans: This acutely life-threatening disorder is associated with previous infection (most commonly meningococcal or gram-negative organisms), pregnancy, massive trauma, end-stage malignant disease, hepatic failure, snakebites, transfusion reactions, and anything else that may precipitate DIC. It is characterized by fever, shock, rapid subcutaneous hemorrhage (ecchymotic purpura/hemorrhagic bullae), tissue necrosis, widespread petechiae, bleeding from multiple sites, widespread organ failure, and DIC. Laboratory findings include thrombocytopenia, schistocytes, prolonged prothrombin time (PT) and partial thromboplastin time (PTT), increased fibrin degradation products, increased D-dimer levels, and decreased fibrinogen levels. Emergent hematology/oncology consult and ICU admission are mandatory. First-line therapy is treatment of the underlying cause. Folate, vitamin K, fresh frozen plasma (FFP), cryoprecipitate, platelets, and red blood cell transfusions are given as needed; heparin is used for associated thrombi. It may be necessary to initiate these treatments in the ED if the patient cannot be admitted immediately to the ICU. However, treating this disease is a tricky balancing act that is best done in consultation with a hematologist.[17,18]

Thrombotic Thrombocytopenic Purpura: Patients with this disease present with a diffuse, nonpalpable petechial/purpuric rash. The classic pentad of symptoms includes fever, thrombocytopenia, hemolytic anemia, neurologic deficits, and renal failure; however, it is more common to observe the clinical triad

of thrombocytopenia, hemolytic anemia, and elevated lactate dehydrogenase. These findings alone are sufficient for the diagnosis and initiation of treatment. There are numerous associated conditions, including HIV, SLE, pregnancy, malignancy, and transplantation. Unlike those with DIC, patients with TTP often have normal PT/PTT and fibrin values. Schistocytes and helmet cells, indicating hemolysis, are seen on blood smears. Treatment includes emergent hematology/oncology consultation, plasmapheresis, FFP, and treatment of the underlying cause. Importantly, FFP can be used as a temporizing measure, but patients with TTP should be transferred to a facility with plasmapheresis capabilities. Mortality is less than 10% when plasmapheresis is used; it is more than 90% without such treatment.[19,20] Platelets should not be administered, as they will precipitate additional thrombus formation.[19]

Vesiculobullous Rashes

Vesiculobullous rashes provoke significant angst in many physicians (Figure 5, page 14). However, the differential diagnosis can be greatly simplified by categorizing patients with these rashes as febrile or afebrile and noting whether the rash distribution is diffuse or localized. Patients with a diffuse vesiculobullous rash and a fever may have varicella or a more devastating illness, such as smallpox, disseminated gonococcal disease, purpura fulminans, or DIC. Necrotizing fasciitis and hand-foot-and-mouth disease present with localized lesions and fever. In afebrile patients with a diffuse vesiculobullous rash, the differential diagnosis includes bullous pemphigus (BP) and pemphigus vulgaris. These entities are regularly confused, and it is essential to differentiate urgently. However, the differential diagnosis is simpler and less emergent in a patient who is afebrile with a localized vesiculobullous rash; contact dermatitis, herpes zoster, dyshidrotic eczema, and burns (chemical or thermal) are included.

Pemphigus Vulgaris: Occurring primarily in persons ages 50 to 60 years, this disease is a generalized mucocutaneous autoimmune blistering with a poor prognosis. Most patients (60%) have mucosal surface involvement initially, which progresses to nonpruritic skin blisters.[5] The bullae coalesce and may produce skin sloughing similar to that found in TEN or SSS, with positive Nikolsky and Asboe-Hansen signs.

FIGURE 6. Bullous pemphigus.

Pemphigus is associated with other autoimmune disorders, especially myasthenia gravis and thymoma. Triggers include penicillamine, captopril (or other thiol-containing compounds), rifampin, and emotional stress. The diagnosis is made clinically with confirmation by biopsy. Patients with large areas of involvement require admission for fluid and electrolyte balance, pain control, monitoring for secondary infection, and treatment with immunosuppressant drugs.[5,21] Although pemphigus had a mortality rate of 50% to 90% before the advent of steroids, steroid therapy has reduced the mortality to 10% to 20%.[3]

Bullous Pemphigus: This condition is a chronic, autoimmune cutaneous blistering that classically occurs in the elderly (average age, 65 years). Curiously, the incidence of infant BP appears to be rising. It has a better prognosis and notably less oral involvement than does pemphigus vulgaris; only 10% to 25% of patients with BP have oral involvement.[5] However, the disease may persist for months or years (waxing and waning) and may be fatal in frail patients. This disease has many triggers, including lichen planus, psoriasis, ultraviolet radiation, x-ray therapy, and exposure to drugs such as furosemide, ibuprofen, captopril, penicillamine, and certain antibiotics and childhood vaccines. The rash is generalized and bullous (Figure 6). The diagnosis is established by histopathologic analysis of specimens taken from the blistering edge of the wounds. Treatment options include oral and topical steroids, tetracycline, immunosuppressive agents, dapsone, and supportive care. Dermatologic consultation is required, as is otolaryngologic consultation for patients with mucosal in-

volvement. In addition, ophthalmologic consultation is warranted for patients with ocular involvement or for those who are taking a prolonged course of high-dose steroids.[21]

CONCLUSION

After a comprehensive history has been taken and a thorough physical examination has been performed, an algorithmic approach can be used to narrow the differential diagnoses of the four most common categories of rashes. While the unknown rash can be perplexing and anxiety provoking to the emergency physician, attention to simple differentiating characteristics (fever, toxicity, distribution, specific signs, target lesions, and palpability) can guide the physician to the correct diagnosis. □

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