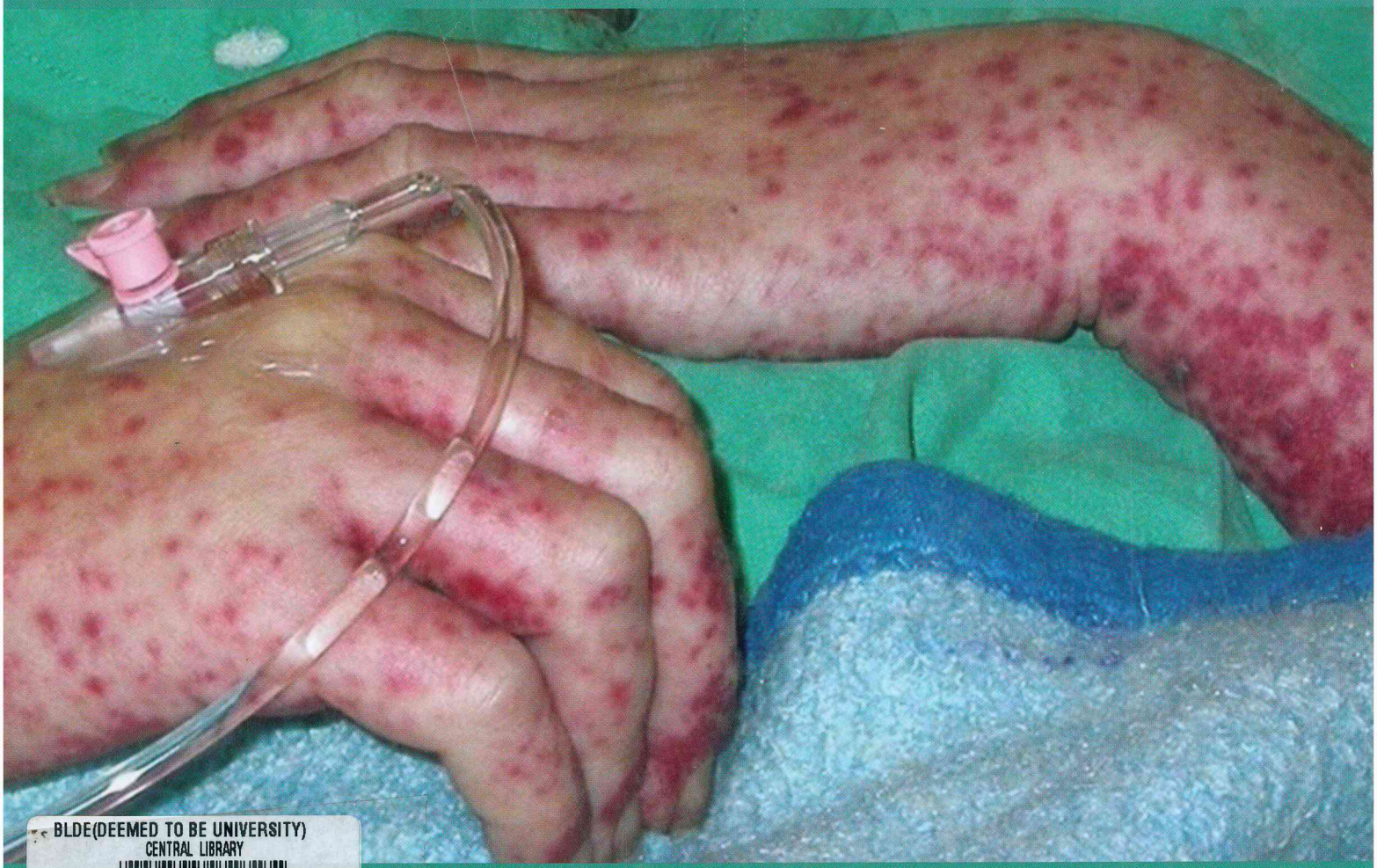
Emergency Dermatology

Second Edition



Edited by
Ronni Wolf • Lawrence Charles Parish
Jennifer L. Parish

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MEDICINE

Emergency Dermatology

Second Edition

There are many emergencies that the dermatologist needs to address and many cutaneous diseases in the emergency room that require rapid dermatologic consultation. The dermatologist is frequently the first physician to examine such patients before a hospital admission and also the first to identify a critical situation, stabilize the patient, and choose urgent and appropriate intervention. Both the practicing dermatologist and the emergency physician will benefit from the revised and updated edition of this text from top international dermatologists, enabling them to hone their diagnostic skills, expand their knowledge and understanding of pathologic events, and learn treatment options available for acute life-threatening skin diseases in this complicated and multifaceted field.

From reviews of the first edition:

"Overall, *Emergency Dermatology* contains an exceptionally up-to-date body of information, addressing the use of many newer drugs and treatment modalities such as use of biologics in cutaneous emergencies. This solid text features a broad collection of topics, an efficient writing style, and content that can easily be applied by practitioners in various medical settings... The content and structure of the chapters gives the book the feel of a handy but thorough field guide that will be frequently referenced by physicians and medical practitioners in primary care and across specialties."
—*Journal of the American Medical Association*

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Acute skin failure

Arun C. Inamadar, Shivanna Ragunatha, and Aparna Palit

INTRODUCTION

Acute skin failure (ASF) is an emergency state encountered by dermatologists, akin to other organ failures. ASF does not imply simple disruption of cutaneous integrity and functional loss, but it also is a multiorgan failure. Although many authors have attempted to define ASF in various ways, the definition given by Irvine in 1991 appears most comprehensive: "Loss of normal temperature control with inability to maintain the core body temperature, and failure to prevent percutaneous loss of fluid, electrolytes and protein, with resulting imbalance, and failure of the mechanical barrier to prevent penetration of foreign materials."[1]

A similar but better-studied condition is "burn injury." In some patients with ASF (e.g., Stevens–Johnson syndrome/toxic epidermal necrosis [SJS-TEN]), the skin involvement is similar to superficial burn. Most of the understanding of ASF to date is from the evidence drawn from burn patients.

Some authors have considered "pressure ulcer" as a form of skin failure and has categorized it as "acute," "chronic," and "end stage" based on chronicity of patients' underlying illness, a state of hypoperfusion, and presence or absence of risk factors for developing pressure ulcers.[2,3]

The discussion in this chapter will be restricted to ASF due to widespread cutaneous disorder and multiorgan involvement.

EVOLUTION OF THE CONCEPT

When Sam Shuster from Newcastle on Tyne, United Kingdom, delivered the "Parkes Weber Lecture" at the Royal College of Physicians of London, on "Systemic effects of skin disease" in 1967,[4] he became the first dermatologist to relate disturbed thermoregulation, anemia, hypoalbuminemia, and hemodynamic disturbances to erythroderma.[4] His pioneering work on systemic effects of skin disease is the basis for the concept of skin failure. Catriona Irvine in 1991 defined skin failure as a real entity comparable to any other major organ dysfunction.[1] In the same year, Terence J. Ryan of Oxford, United Kingdom, also described skin failure as one of the causes of disability in dermatology.[5]

Jean-Claude Roujeau of Paris mentioned the term "acute skin failure" in a paper to describe the systemic effects of TEN.[6] He pleaded for the management of such cases to be in specialized "dermatology intensive care units (DICU)" rather than in burn units.

CAUSES

Morphologically, we propose to categorize underlying causes of ASF as "dry disorders," i.e., disorders giving rise to erythroderma and "wet disorders," i.e., vesiculobullous disorders. Such categorization has practical implications, as hemodynamic alterations, extent of fluid, electrolyte, and nutrient-loss and management aspects may differ in these two broad types. Various underlying dermatologic disorders giving rise to ASF are presented in Table 6.1.[7] The list is comprehensive but not exhaustive.

ALTERED PHYSIOLOGY IN PATIENTS WITH ASF

Hemodynamic Changes

In patients with ASF, there are vasodilatation and enhanced blood flow to the chronically inflamed skin (normal 0.5–1 L/min versus 5 L/min).[8] Clinically, it is evident as bright erythema and edema of the skin (Figure 6.1). This leads to increased cardiac output (normal 5 L/min versus >10 L/min) and venous return.[8] There are tachycardia and decreased perfusion to the vital organs. In case of preexisting functional compromise, there is risk of sudden cardiac or renal failure.

High levels of vascular permeability factor (VPF) and vascular endothelial growth factor (VEGF) have been detected in patients with erythroderma of long duration. These make capillary leakage, causing expansion of extracellular space and resultant dependent edema.[9] Various causes of peripheral edema are presented in Box 6.1.

Though patients with ASF are in a "high-cardiac-output" state, hypovolemic shock may occur due to dehydration and septicemia.

Altered Temperature Regulation

Temperature regulation of the body is an energy-consuming process partially achieved at the cost of calorie expenditure. Cutaneous blood circulation and evaporation of eccrine sweat are the two principal mechanisms for thermoregulation in the human body.[8] About 200–300 mL of eccrine sweat is produced per day, and it evaporates from the skin surface.[8] This, along with transepidermal water loss (TEWL), consumes 600 Kcal/L of fluid loss.[8] Cutaneous vasculature is a complex network, specially designed in several layers to regulate body temperature. About 15%–20% of cardiac output flows through cutaneous vessels[8] and maintains the surface temperature balanced between environmental and core body temperatures.

In the state of ASF, both the mechanisms are jeopardized. There is increased cutaneous blood flow, and sweat glands are either occluded or damaged in patients with long-standing erythroderma.[10] Increased cutaneous blood flow leads to enhanced heat loss from the body surface leading to hypothermia. To maintain core body temperature, the patient starts shivering at the cost of a raised basal metabolic rate (BMR). When there is failure to compensate, core body temperature becomes equal to that of environment (poikilothermia).[10]

Table 6.1 Causes of Acute Skin Failure[7]

1. Erythroderma (dry disorders):
- Congenital causes
  - Congenital ichthyosis (syndromic and nonsyndromic) (Figure 6.2)
  - Leiner disease
- Acquired causes
  - Psoriatic erythroderma (Figure 6.3)
  - Acute generalized pustular psoriasis of Von Zumbush (Figure 6.4)
  - Pityriasis rubra pilaris
  - Atopic and other dermatitides
  - Crusted scabies
  - Drug-hypersensitivity syndrome (DHS) (Figure 6.5)
  - Pemphigus foliaceous (Figure 6.6)
  - Collagen vascular disorders (acute cutaneous lupus erythematosus, dermatomyositis)
  - Paraneoplastic (lymphoma, leukemia, solid organ tumors)
  - Langerhans cell histiocytosis
  - Erythrodermic sarcoidosis
  - Graft versus host disease
  - Idiopathic

2. Vesiculobullous disorders (wet disorders):
- Genetic disorders
  - Epidermolysis bullosa
  - Bullous congenital ichthyosiform erythroderma
- Acquired disorders
  - Immunobullous disorders
  - Hailey-Hailey disease (generalized form)
  - Drug-induced; SJS (Figure 6.7), TEN (Figure 6.8)
  - Staphylococcal scalded skin syndrome (SSSS)
  - Bullous mastocytosis

Box 6.1 Various Causes of Peripheral Edema in Patients with ASF

- Peripheral vasodilatation
- Capillary leakage
- Edema associated with the original inflammatory condition (e.g., SJS-TEN, psoriatic erythroderma)
- Hypoalbuminemia
- Associated cardiac failure
- Associated renal failure
- Fluid overload during resuscitation

Figure 6.1 Generalized erythema in a patient with acute skin failure. Also note mild ectropion.

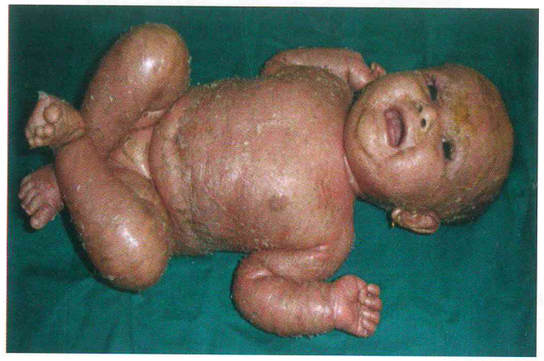

Figure 6.2 Congenital ichthyosiform erythroderma.

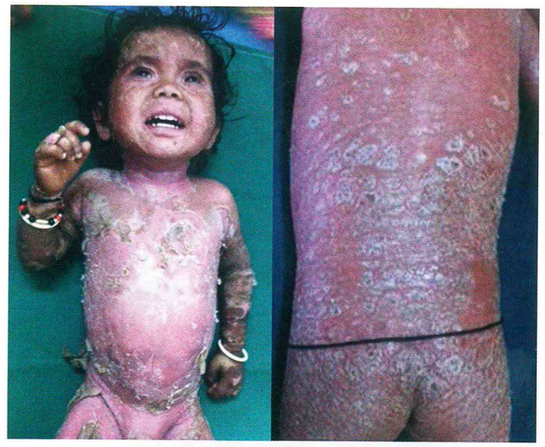

Figure 6.3 Psoriatic erythroderma in a child.

Rarely, extensive skin loss and release of interleukin-1 from necrotic keratinocytes may induce hyperthermia in these patients. Mechanical factors, such as occlusion of sweat ducts in patients with long-term erythroderma, may also give rise to hypohidrosis and hyperthermia.[10]

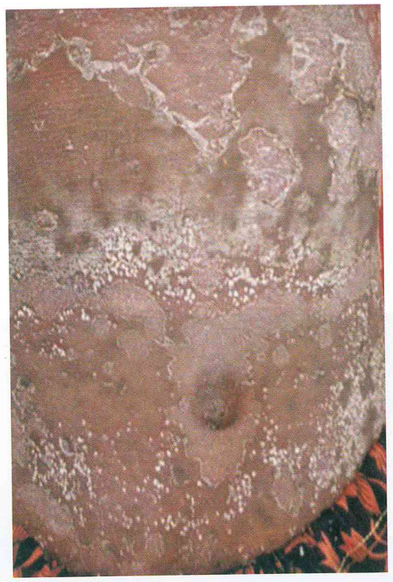

Figure 6.4 Acute generalized pustular psoriasis with lakes of pus.

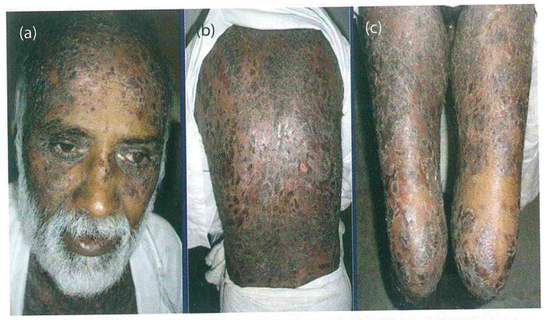

Figure 6.6 (a–c) Erythroderma due to pemphigus foliaceous.

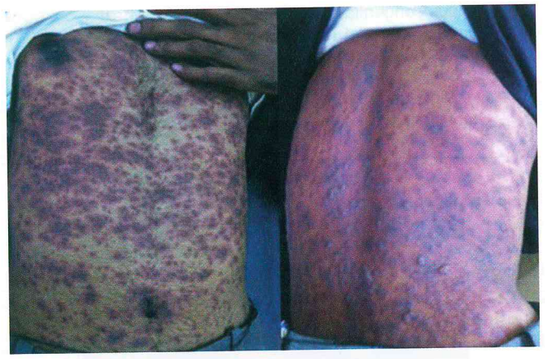

Figure 6.7 Stevens–Johnson syndrome.

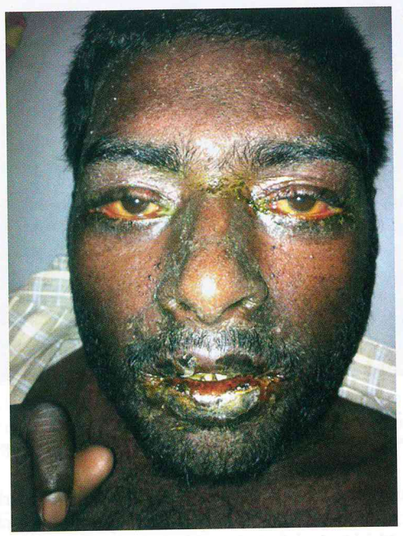

Figure 6.5 Drug hypersensitivity syndrome.

Figure 6.8 Toxic epidermal necrolysis.

Altered Fluid and Electrolyte Balance

Composition and Distribution of Body Fluids in Health

Water is the major constituent of the human body comprising approximately 50% (women) to 60% (men) of body weight. Total body water is distributed into two major compartments, intracellular fluid (ICF) and extracellular fluid (ECF). The former constitutes 55%–75%, and the latter 25%–45% of the total body water.[11] The total volume of ECF in an adult is 270 mL/kg and is distributed in plasma and lymph (55 mL/kg), muscle and organs (85 mL/kg), and skin and connective tissue (130 mL/kg).[12] Skin constitutes a major reservoir of ECF in the form of interstitial fluid.

Osmotic equilibrium between ECF and ICF is important for normal functioning of cells. To maintain the equilibrium, osmolality of both ECF and ICF should be equal. It is achieved by movement of water across the cell membrane. The osmolality of the fluid is determined by the solute or particle concentration. The major ECF particles are Na^+, Cl^- and HCO_3^-, whereas K^+, adenosine triphosphate (ATP), creatinine phosphate, and phospholipids are major ICF particles. The number of intracellular particles remains constant as these are required for normal cell function. Therefore, the changes in ICF osmolality are due to changes in ICF water content.[11] To maintain homeostasis, the intake of water should be equal to its excretion. Normally, a measurable amount of water is lost through the kidneys and gastrointestinal tract; however, additional water is lost through evaporation from the skin and respiratory tract. Typically, the latter two are not measured and are termed as insensible water loss (IWL). TEWL contributes 70% of IWL.[13] The normal composition and distribution of body fluids and TEWL vary depending on age groups and climatic conditions like humidity and temperature. These factors should also be considered along with underlying pathological changes during fluid resuscitation.

Fluid and Electrolyte Balance in Children

The percentage of total body water in fetal life gradually reduces with gestational age, from around 86% at 26 weeks to 80% at 32 weeks and to about 78% at full term.[14] Neonates have high body surface area (BSA) to weight ratio and functionally immature kidneys. As compared to adults, infants and younger children have a larger daily turnover of water, relative to the total body water and large ECF space. All of these factors predispose children to a greater risk of fluid and electrolyte imbalance than adults.[15]

Fluid and Electrolyte Balance in Elderly

The percentage of water content of the body depends on lean body mass. With age, the lean body mass decreases resulting in decreased body water content; hence, fluid balance in the elderly requires special considerations. The fluid balance in the elderly is further compromised by decreased intake of food and water due to loss of appetite and thirst, physical dependence, and biochemical changes of aging. All of these factors result in increased susceptibility of the elderly to fluid deprivation. It has been reported that mortality associated with disturbances in water balance in the elderly may be as high as 40%–70%.[16]

Nature of Fluid and Electrolyte Imbalance in ASF

The composition and amount of fluid loss differ depending on type and severity of diseases causing acute skin failure. In diseases presenting as erythroderma (dry disorders), the TEWL leads to hypernatremic dehydration, whereas in diseases presenting with extensive erosions (wet disorders) both water and electrolytes are lost. In addition, fever, sepsis, and comorbid conditions involving the central nervous system and kidneys put the patient at risk of fluid and electrolyte imbalance.

Fluid Loss in TEN

In adult patients with TEN involving >50% total body surface area (TBSA), the daily fluid loss exceeds 3–4 liters.[17] The blister fluid of autoimmune bullous disorders and TEN contains approximately 120–150 mmol/L of Na^+, 100 mmol/L of Cl^-, and 5–10 mmol/L of K^+.[18] The composition of blister fluid of pediatric patients with TEN was found to be similar to that of burns, except that the latter has a threefold higher albumin and protein. The lactate dehydrogenase, calcium, and magnesium were significantly high in both blister fluid specimens when compared to serum levels.[19]

Fluid Loss in Erythroderma

The TEWL in a child with ichthyosis ranges from 746 ± 468 mL/day (mean basal TEWL of 39.6 ± 20.6 mL/m²/hour compared to upper limit of normal 8.7 mL/m²/hour) to 209 mL/day seen in age-matched children with competent skin barrier.[20] An estimation of TEWL from normal and abnormal skin of a collodion baby using evaporimeter at day 4 demonstrated a loss of 18 ± 2 g/m²/hour and 112 ± 2 g/m²/hour of water, respectively, at room temperature of 27°C and relative humidity of 25%.[21] The same reduced significantly, in parallel with the clinical improvement of the skin at day 30, to 5.5 ± 2 g/m²/hour and 16 ± 2 g/m²/hour, respectively, at room temperature of 23°C and relative humidity of 37%.[21]

Metabolic Alterations

There is a raised BMR in patients with ASF, as explained above. About 50% of the patients demonstrate hyperglycemia, which may be multifactorial: stress, low insulin level due to pancreatitis, and relative insulin resistance.[8,10] Hypophosphatemia is common in patients with wet disorders and augments insulin resistance.[10] Protein depletion may occur to maintain the level of BMR, later manifested as muscle wasting. Overall, a catabolic state prevails.[10]

Alteration in Barrier Function

Both physical and immunologic barriers are at stake in patients with ASF. Loss of physical barrier, i.e., disruption of the stratum corneum, is the primary event, whereas loss of immunologic functions occurs secondarily. There is little evidence in the literature of local immunosuppression in patients with ASF in particular, but, such conclusion has been drawn from similar effects in patients with burn.[8] There may be systemic immunosuppression in patients with TEN in the form of neutropenia (30%), total lymphopenia (90%), and selective CD4+ T lymphocytopenia.[10] Granulocytic functions, e.g., chemotaxis and phagocytosis, are impaired.[10] Altered immune functions make these patients prone to acquire infections.

Nutritional Loss

Loss of nutrients occurs through the damaged skin along with fluid and electrolytes. Normally, 500–1000 mg of material is lost per day through exfoliation, and this amount may increase

inefold in patients with ASF. Diffuse scaling leads to protein oss of approximately 20–30 g/m² BSA/day.[22] The blister fluid f autoimmune bullous disorders and TEN contains approxiately 40 g/L of protein. In addition, protein is lost through ozing and hypercatabolism, accounting for total protein loss f 150–200 g/day.[18] The associated hypercatabolism, fever, and epsis increase protein metabolism and energy expenditure by 0%.[23] In pediatric patients with ichthyosis, the total calorie loss rom daily TEWL ranges from 84–1015 kcal (21 ± 9.8 kcal/kg/ lay with a mean of 433 ± 272 kcal/day) to 41–132 kcal/day seen n age-matched children.[20]

Dermatologic enteropathy leads to chronic diarrhea in patients with long-term erythroderma. Malabsorption of iron, vitamin B_{12}, and micronutrients occurs. Diarrhea also depletes he intestinal microflora resulting in deficiency of vitamin B complex. High cellular turnover in erythrodermic patients eads to folate deficiency.

ESTABLISHING THE UNDERLYING CAUSE

Establishing the underlying cause of ASF has two implications: first, obviously, making a complete diagnosis of an inpatient is obligatory, and second, it helps the clinician to administer definitive treatment during the recovery period of the patient; however, establishing the underlying illness giving rise to ASF in an admitted patient is a difficult task indeed! This is because most of the times at this point the patient is unable to give a correct history and that obtained from the relatives may be incomplete and misleading. The responsibility falls upon the first clinician, who attends the patient in the emergency department, to elicit a plausible history, and to identify the clinical clues to the underlying cause. If few definite skin lesions (e.g., individual papule/vesicle) are identified, it is appropriate to perform simple bedside tests (e.g., Tzanck test, KOH mount from scales, etc.) and skin biopsy at this stage; otherwise, such investigations are postponed until the crisis period is over. Table 6.2 presents diagnostic clues for some of the underlying conditions in patients with ASF.

COMPLICATIONS

Complications of ASF are manyfold. During the acute stage, there may be life-threatening complications arising mainly from hemodynamic alterations.[10] Effects of this massive cutaneous injury leave cicatricial changes on skin, mucosa, and appendages; these are considered as "late complications."[10] Various complications of ASF have been presented in Table 6.3.

MANAGEMENT

Hospitalization

All patients with ASF must be hospitalized, preferably in an intensive care unit (ICU). In some institutions, a facility for DICU is available, which meet the special needs of patients with ASF;

Table 6.2 Diagnostic Clues for Underlying Disorders in Patients with ASF

History/clinical features/investigations	Probable diagnostic interpretation
Scaly plaques, long-term scaling of scalp, nail changes	Psoriasis
Showers of pustular lesions with febrile episodes, lakes of pus	Pustular psoriasis
Spiny, follicular lesions, islands of normal skin between orange red erythema, palmoplantar keratoderma (PRP sandal)	Pityriasis rubra pilaris
Scaling since birth or soon after, H/O collodion membrane at birth	Congenital ichthyosis
H/O suggestive drug intake, icterus, hepatosplenomegaly, eosinophilia, elevated liver enzymes	Drug hypersensitivity syndrome
H/O drug intake, target lesions, mucosal involvement, hemorrhagic crusts on lip, positive Nikolsky sign	SJS-TEN
Poor hygiene, pruritus, mite demonstrable on light microscopy	Crusted scabies
Lymphadenopathy ± hepatosplenomegaly, bleeding episodes, abnormal peripheral blood smear	Lymphomas, leukemias
Greasy, scaly papular lesions in a neonate, hepatosplenomegaly, thrombocytopenia	Langerhans cell histiocytosis
Presence of bullae since/soon after birth, healing with scarring, milia formation, nail changes	Epidermolysis bullosa
Flaccid bullae, positive Nikolsky sign, positive Tzanck smear	Pemphigus

Table 6.3 Complications of ASF

Acute complications		Long-term complications	
Infection	Septicemia	**Ocular**	Ectropion, entropion, exposure keratitis, dry eyes (Figure 6.9), corneal ulcer, symblepharon (Figure 6.10)
Pulmonary	Acute respiratory distress syndrome (ARDS) Aspiration pneumonia Pulmonary embolism Pulmonary edema due to fluid overload	**Esophageal**	Stricture and dysphagia
Cardiovascular	High-output cardiac failure Congestive heart failure due to fluid overload Hypovolemic shock	**Genital**	Urethral meatal stricture, phimosis or vaginal stenosis (Figure 6.11)
Renal	Prerenal uremia Acute renal tubular necrosis	**Cutaneous**	Hypo- and hyperpigmentation, scarring
Gastrointestinal	Stress ulcers and gastric hemorrhage	**Hair**	Scarring alopecia
Neurological	Mental confusion and stupor resulting from electrolyte imbalance Meningitis spreading from septicemia	**Nails**	Beau lines, nail dystrophy, total shedding of nails (Figure 6.12)

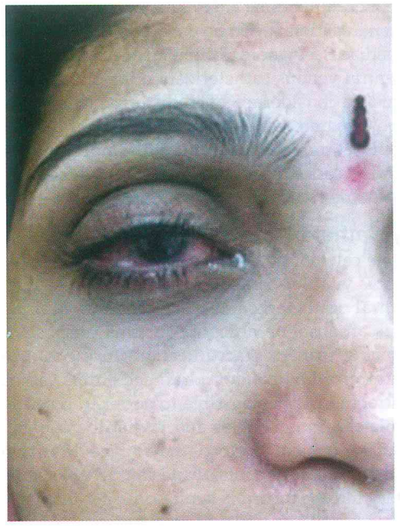

Figure 6.9 Keratoconjunctivitis sicca following SJS.

Figure 6.10 Symblepharon in a patient following SJS.

however, in the absence of such a facility, such patients may be admitted to a general ICU or even in a burn unit.

A multispecialty approach is required for appropriate care of patients with ASF. The basic team includes an internist or pediatrician, ophthalmologist, dietician, and dedicated nurses in addition to a dermatologist. Other specialists may be called upon as and when necessary.

Initial Clinical Evaluation

The first dermatologist attending a patient with ASF has the responsibility of assessing the hemodynamic status of the patient, to decide the immediate management protocol, and to initiate the treatment. An in-and-out history taking (recent and past) makes the job easier. The points and clinical parameters to be recorded at this stage are presented in Table 6.4.

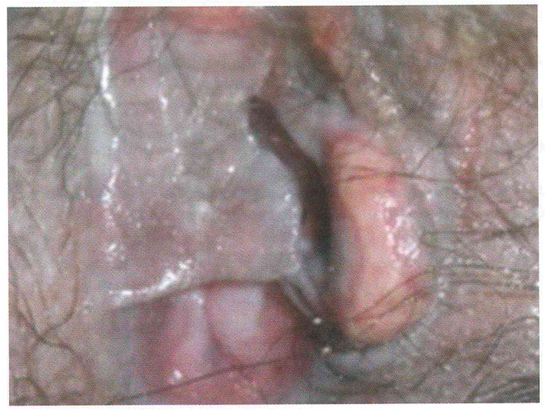

Figure 6.11 Adhesion of labia minora causing vaginal stenosis as a sequelae of SJS.

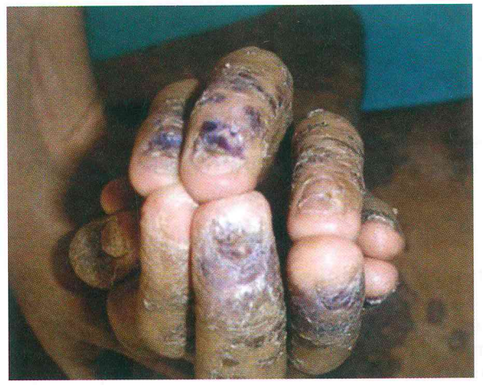

Figure 6.12 Shedding of nails during convalescence period of SJS-TEN.

TBSA involvement should be calculated.[10,15] There are different methods of estimating BSA involved, used in patients with burn; *viz*, rule of nine, palm area of the patient, and Lund-Browder chart. The rule of nine is used for immediate assessment and it gives approximate values. This method should not be used in children less than 15 years of age.[25] With this technique, there is a tendency for overestimation of BSA involved, resulting in fluid overload, tissue edema, and subsequent pulmonary complications.[26] Similarly, use of palmar area is not accurate in patients with high body mass index (BMI). In these patients, it accounts for 0.64%, instead of 1% of BSA. A more accurate method is drawing the BSA involved on the Lund-Browder chart.[27]

If a definitive diagnosis is possible, disease-specific indices are utilized to assess degree of involvement and prognosis (e.g., "psoriasis area severity index" [PASI] or "eczema area severity index" [EASI], etc.). In patients with TEN, a

ble 6.4 Initial Examination of a Patient with ASF[24]

:ord vital parameters	Pulse, blood pressure, respiratory rate, body temperature
:sess hydration	Hydration of tongue Skin turgidity
:vel of consciousness	Conscious/unconscious If conscious, check orientation
uick look all over skin, mucosa, hair, and nails ny abnormal smell eneral and systemic examination	Search for clue to underlying disease Look for foci of infection May indicate secondary infection Dependant edema, Pallor, Lymphadenopathy, Hepatosplenomegaly, Pulmonary congestion, Muscle weakness
ssessing risk factors	Old age Infants and children Pregnant women Patients with convulsive disorders Alcoholism, smoking habits
ıssessing for comorbid conditions	Hypertension, diabetes mellitus, ischemic heart disease, chronic obstructive pulmonary disease Cirrhosis of liver, chronic kidney disease Tuberculosis, HIV infection

pecific disease severity score, "SCORTEN," must be assessed n admission and thereafter while monitoring.[24]

Baseline Investigations

udicious baseline investigations must be undertaken in all patients with ASF. A list of these investigations is presented in Box 6.2.

Setting Intravenous Channel

Setting an intravenous (IV) channel is the immediate action following admission to ICU. If the patient is critically ill, insertion of more than one canula may be necessary for hemodynamic monitoring and interventions. In all cases, the piercing should be done through relatively uninvolved skin as far as practicable to reduce the chances of introducing infection. If such an area of skin is not identifiable, setting a central venous line should be considered. The advantage of establishing vascular access immediately, even though the patient is not in critical condition, is manyfold; it enables the attending clinician to draw blood samples for multiple investigations, administer injectable drugs, infuse IV fluids as and when indicated, and institute total parenteral nutrition (TPN).[28]

Often in patients with severe SJS-TEN or extensive immunobullous diseases, where peripheral venous access in routine way or central venous catheterization is not possible, venesection should be considered.[28] The distal saphenous vein serves this purpose in an excellent way as it is easier to access due to consistent location, noncontiguity with important arteries and nerves, and distance from other resuscitative gadgets.[28]

Box 6.2 Baseline Investigations in Patients with ASF

- Complete hemogram
- Blood glucose level
- Blood urea and creatinine
- Serum electrolytes (Na^+, K^+, Cl^-, Ca^{++})
- Serum HCO_3
- Liver function test
- Urinalysis
- Chest x-ray
- Electrocardiography (ECG)
- Arterial blood gas analysis

Urinary Catheterization

An accurate calculation and maintenance of the intake/output (I/O) chart is vital for patients with ASF. In bedridden patients transurethral catheterization or condom drainage (male patients) is an absolute necessity for this purpose.[28] The risk of introducing urinary tract infection during catheterization can be minimized by condom drainage (Texas catheter); however, this method is not possible to adopt in cases with damaged penile skin. In neonates and pediatric patients, neonatal and pediatric "urine collectors" may be used for collection of urine.[28] In ambulant patients, urine can be collected in graduated bottles and the record is kept.

Nasogastric Intubation

Any patient with ASF due to any cause should be encouraged to take fluids and food orally as far as possible; however, this may be difficult for patients with SJS-TEN and pemphigus due to painful oropharyngeal and esophageal erosions. Insertion of a nasogastric tube is helpful in such cases. All acutely ill patients with ASF unable to take orally should have a nasogastric tube inserted. Various advantages of nasogastric intubation in patients with ASF are presented in Box 6.3.

Following insertion, the correct location of the tube must be double-checked to prevent accidental aspiration.[28] The clinician must undertake due precaution to insert a nasogastric tube in conditions with fragile mucosa to avoid the risk of traumatic hemorrhage and esophageal perforation.[28]

A schedule for Ryle tube feeding must be prepared by the clinician, dietician, and on-duty nursing staff. The total amount is divided into small feeds (one fourth of the desired amount) and administered at regular intervals as overfeeding may lead to regurgitation and aspiration.[24]

While recovering, nasogastric feeding is gradually substituted by oral intake (liquid or semisolid) and as the condition permits, full oral diet is instituted.[24]

Box 6.3 Various Usage of Nasogastric Tube in Patients with ASF[28]

- Feeding
- Administration of oral medications
- Periodic aspiration of gastric contents
- Detection of stress-induced gastrointestinal bleeding
- Management of gastric immobility and paralytic ileus in acutely ill patients

Use of Air-Fluidized Bed

The purpose of using these devices in patients with ASF is prevention of pressure sores. Water beds are made up of soft polyvinyl chloride (PVC) or similar material and are provided with single or interconnected flow chambers to fill it with water.[29] Air-fluidized beds have interconnected air and fluid chambers. The water may be warmed with a thermostatic device connected to the bed.[29]

During use, an air-fluidized bed is shaped around the patient's body and thus reduces contact time and pressure, especially over bony prominences.[29] It may be set at body temperature or according to the patient's comfort level[29]; however, a water bed with a temperature regulation system may result in dysregulation of body temperature and dehydration in patients with ASF.[29] A floating sensation may make some patients feel uncomfortable.[29] The caregivers should be alert to accidental leakage from the bed.[29]

Maintenance of Body Temperature

Patients with ASF are at risk for developing hypothermia. At environmental temperature, it is invariably associated with shivering and loss of extra calories. This precipitates a hypercatabolic state and negative energy balance. An optimum room temperature (30–32°C)[10] has to be maintained for these patients to avoid such consequences, and this can be achieved by air-conditioning. At presentation, hyperthermia in some patients as discussed before should be taken into consideration before a final decision is made regarding regulating environmental temperature.[10]

Maintenance of Fluid and Electrolyte Balance

In a state of ASF, depending on the underlying cause and severity, significant amounts of fluid and electrolytes are lost through the skin resulting in serious hemodynamic consequences. The use of drugs in a hemodynamically unstable patient of ASF to the patient's benefit has been inconsistent; hence, the successful management of these patients mainly depends on maintenance of fluid and electrolyte balance in addition to other supportive therapies. Adequate fluid resuscitation is based on the knowledge of

- Fluid and electrolyte balance
- Accurate estimation of BSA involved
- Volume and nature of fluid lost
- Comorbid conditions and clinical scenarios that may worsen the existing condition and/or act as risk factors for development of complications related to fluid resuscitation

There is a dearth of studies on estimation of fluid and electrolyte loss and their management in ASF; therefore, extrapolation of the guidelines used for management of fluid and electrolytes in burn patients and other critically ill patients is recommended for patients with TEN and other patients with ASF.

Assessment of BSA Involved

Accurate assessment of BSA involved in patients with ASF is essential for calculation of adequate resuscitation fluid and better patient outcome.

Estimation of Replacement of Deficit Fluid

Replacement of fluid that is already lost should be provided during the first 24 hours after injury. It is essential for maintenance of tissue perfusion, blood pressure, and adequate urine output.

Estimation of Replacement Fluid in TEN

The Parkland formula[25] is used to calculate the replacement fluid, because the injury that occurs in TEN is comparable to burns. The Parkland formula is as follows:

$$\text{Resuscitation volume} = 4\ \text{mL} \times \text{Total body weight} \times \%\ \text{of epidermolytic BSA involved}$$

In TEN, the epidermal denudation occurs at the level of dermoepidermal junction like superficial burns.[12,23] In addition, the absence of the thermal effect, moderate papillary edema, and normal reticular dermis make patients with TEN less susceptible to fluid and electrolyte loss when compared to burns covering the same BSA.[30,31] Even in burns with involvement of large BSA and in patients with high BMI, the Parkland formula overestimates the volume of replacement fluid required during the first 24 hours[27]; hence, aggressive fluid resuscitation is not required in patients with TEN.[32] One half to three-quarters of the resuscitation volume calculated by the Parkland formula is recommended during the first 24 hours after injury.[18,33]

Estimation of Water Deficit in Erythroderma

Unlike TEN, in patients with erythroderma the TEWL is solute free. The water deficit is calculated here based on plasma Na^+ levels, as follows:

$$\text{Water deficit} = \frac{\text{Plasma } Na^+ \text{concentration} - 140}{140} \times \text{Total body water}$$

In hypernatremia total body water is 50% and 40% of lean body weight in men and women, respectively.[11]

Estimation of Maintenance Fluid and Ongoing Evaporative Loss

After administration of replacement fluid during the first 24 hours, the maintenance fluid and additional fluid to compensate for ongoing loss (through the damaged skin) should be provided. Maintenance fluid is designed to supply free water and electrolyte requirements in a fasting patient. The requirement of free water in a healthy child is equated with that of energy expenditure; hence, the maintenance fluid is calculated as follows:

> 100 mL/kg/day for first 10 kg (1000 mL), 50 mL/kg/day for next 10 kg (1000 mL + 500 mL), and 20 mL/kg/day above 20 kg (1500 mL + 20 mL/kg).[34]

If urine output is low and is not improving, then the volume of maintenance fluid should be reduced to 50%.[12]

> The evaporative loss of fluid is calculated by using the following formulas:
>
> - Insensible water loss = (125 + % TBSA involved) × TBSA (m^2)[35]
> - Evaporative water loss (through eroded skin in children) = 25 + % TBSA involved × TBSA (m^2)[23]

Choice of Fluid for Replacement Therapy

Various factors like age, severity of disease, and underlying metabolic changes determine the choice and rate of administration of IV fluid during replacement therapy. Generally, oral administration of fluids is preferred over IV fluids. IV fluids are recommended if TBSA involvement is >10% in children and >15% in adults. If the patient is not in severe shock, oral fluids can be started within 24 hours providing one quarter of the daily fluid requirement. As the condition of the patient improves slowly, IV fluid is replaced with oral fluids.[25] Table 6.5 presents the composition of various resuscitation fluids and advantages and disadvantages of their usage.

Choice of Fluid in TEN

The replacement fluid calculated by the Parkland formula is based on crystalloids. Usually isotonic solutions like Ringer lactate (RL) and 0.9% normal saline (NS) are recommended for replacement therapy. The choice of fluid for immediate restoration of intravascular volume is RL. Half of the calculated volume is administered in the first 8 hours and the remaining half in the subsequent 16 hours[25]; however, depending on the severity of volume depletion, the replacement fluid can be administered in 8–12 hours.[36] NS can be used especially in infants and young children who are predisposed to lactic acidosis and hypernatremia.[15]

If TBSA involved is >40%, IV colloidal resuscitation with 5% human albumin in NS is recommended due to the hypercatabolic state and movement of protein into the extravascular space, which results in a significant amount of protein loss[23,25]; however, it is not recommended during the first 24 hours due to the presence of a significant capillary leak. The dosage of albumin is calculated as 0.3–0.4 mL/kg × % of BSA involved, to be administered over 6–8 hours.[37]

Choice of Fluid in Erythroderma

In hypernatremic dehydration, restoration of intravascular volume is achieved by infusion of 10–20 mL/kg/hour of RL or NS in 1–2 hours before replacement of water deficit. In premature and small infants, NS is preferred.[34] Once the intravascular volume is restored, half of the calculated water deficit should be replaced during the first 12–24 hours and the remaining over 48–72 hours by using isotonic NS.[38]

Choice of Fluid for Maintenance Therapy

In the hypovolemic state, nonosmotic secretion of antidiuretic hormone (ADH) results in avid water resorption by kidneys and reduced urine volume. In such a situation, administration of hypotonic solution, which has been followed traditionally, may put the patient at risk of hyponatremia; hence, an isotonic solution like NS is recommended for maintenance fluid therapy.[39] The evaporative loss is replaced preferably with free water; however, 5% dextrose in 0.2% saline is used to prevent water intoxication and electrolyte imbalance.[23]

Monitoring of Fluid and Electrolyte Therapy

The formulas used for estimation of volume in the treatment of patients with ASF give only rough guidelines for fluid therapy due to various reasons described above. The cornerstone of successful fluid resuscitation is careful and continuous monitoring of clinical and laboratory parameters. Though mean

Table 6.5 Composition of Various Resuscitation Fluids and Their Usage[24]

Type of fluid	Comments	Type of fluid	Comments
Colloids	More effective in expanding intravascular volume, by maintaining colloid oncotic pressure. Volume sparing effect is an added advantage (Colloid: crystalloid = 1:3).	**Crystalloids**	Inexpensive, widely available, established role as first-line resuscitation fluid. Risk of significant interstitial edema.
A. Natural			
1. Human albumin (4%–5%) in saline	Reference colloid solution Ideal IV fluid in early sepsis Limiting factors: high cost, limited availability in resource-poor setup	1. Normal saline (0.9%) (NS)	Most commonly used crystalloid solution. Large volume transfusion results in hyperchloremic metabolic acidosis, acute renal injury.
B. Semisynthetic colloids	Shorter duration of action than albumin, but actively metabolized, and excreted	2. Hartman/ Ringer lactate	Contains K^+(5.4 mmol/L), Ca^{++} (2 mmol/L) and lactate (29 mmol/L); advantageous over NS when supplementation required.
1. Hydroxyethyl starch	Colloid source is potato or maize starch. Recommended maximum daily dose 33–50 mL/kg/day. Contains K^+, Ca^{++}, Mg^{++}, lactate, and malate in addition to Na^+ and Cl^-, depending on the brand. Adverse events: pruritus, altered coagulation, acute kidney injury, and increased death rate	3. Balanced salt solution	Considered as initial resuscitation fluid. Chemical composition approximates ECF. Relatively hypotonic due to lower sodium concentration. Nonbicarbonate anions, like lactate, gluconate, acetate, and malate, are used. Excess use: risk of hyperlactatemia, metabolic alkalosis, hypotonicity, and cardiotoxicity. Some brands contain calcium and have the risk of microthrombi formation with citrate containing red cell transfusion.
2. Dextran	Infrequently used.		
3. Succinylated modified fluid gelatin (4%) and urea-linked gelatin (3.5%) (Hemaccel)	Colloid source is bovine gelatin. Hemaccel contains K+ (5.1 mmol/L) and Ca^{++} (6.25 mmol/L)		

Box 6.4 Monitoring Fluid Transfusion Based on Urine Output

- Urine output equal to or less than one third of predicted value over 2 consecutive hours: Rate of IV fluid infusion increased by 20%/hour.
- Urine output more than predicted value: Rate of IV fluid infusion decreased by 20%/hour.[35]

Other recommendation[25]:

- Urine output <1 mL/kg/hour: Increase IV fluid infusion by 50%.
- Urine output >2mL/kg/hour: Decrease the rate of infusion.

arterial pressure, central venous pressure, and central venous oxygen saturation are the accurate indicators of hemodynamic response to fluid therapy, in patients with ASF invasive monitoring techniques are not recommended to avoid the risk of sepsis.[15,39]

The main goal of fluid resuscitation is maintenance of normal urine output (0.5–1.0 mL/kg/hour), blood pressure, heart rate, and serum Na^+. In children, capillary filling time is measured to assess the response to fluid resuscitation. Based on urine output, monitoring of fluid infusion is presented in Box 6.4.

The clinical signs and symptoms suggestive of dehydration and electrolyte imbalance (Table 6.6) should be monitored frequently. A pulse rate of ≥120 in a patient with ASF may be indicative of negative fluid balance, even in the presence of other factors like fever and septicemia; the recording of blood pressure in these patients may be misleading as it tends to remain normal in the initial stage.[10]

During treatment of hyponatremia, rapid correction of plasma Na^+ by 1–2 mEq/L/hour should be limited to the initial phase of management. Later, to prevent osmotic demyelination, the correction of plasma Na^+ levels should not exceed 8–12 mEq/L in 24 hours.

Patients with erythroderma are more prone to develop severe hypernatremic dehydration due to solute free water loss. In such a situation, water enters the cells during rehydration leading to intracellular edema; hence, the fluids should be administered very slowly to prevent brain cell injury. The serum sodium should fall by 0.4–0.8 mEq/L/hour or 10–12 mEq/L/day, and the maximum rate of fall in plasma Na^+ should not exceed 2 mEq/L/hour.[38]

Nutritional Supplementation in ASF

The main goal of nutritional therapy is to supplement the catabolized proteins, provide proteins required for healing of skin lesions, and ensure growth in case of children.[15] Adults with >15% and children with >10% of BSA involved have an increased nutritional requirement;[25] therefore, aggressive nutritional supplementation should be started preferably through the enteral route.

Enteral feeding has the advantage of preserving gastrointestinal integrity and decreasing the incidence of bacterial transmigration across the gut, but complications pertaining to insertion of the nasogastric tube have already been discussed. TPN is also associated with complications related to IV line insertion, sepsis, and metabolic risk.[40] Delivery of nutrition without using nasogastric tube or central venous line has been proposed. It is done in two phases, initially low osmolarity TPN is administered through peripheral veins followed by oral supplementation of nutrition as soon as the clinical condition permits.[41]

If the patient is not toxic and gastrointestinal functions are intact, the feeding can be started within 6 hours of injury.[25] In children with burns involving <20% of BSA, the gastrointestinal motility returns to normal in 72 hours, and in extensive involvement, paralytic ileus may persist for 5–6 days.[37] In such patients with impaired gastric emptying, enteral feeding is initially started with one quarter of the desired volume and is gradually increased at a rate of 5 mL/hour. The residual gastric volume should be checked by periodic aspiration and feeding is stopped if it is more than 50 mL.[15]

As in fluid and electrolyte therapy, various formulas used for calculation of protein and energy requirements in burns can be extrapolated for TEN. The calorie requirement of pediatric patients with TEN was found to be 22% less per day than those with burns. A formula has been developed as follows[42]:

Calorie requirement = Baseline body weight (Kg) × 24.6 + Wound size (%TBSA) × 4.1 + 940

Table 6.6 Signs and Symptoms Suggestive of Dehydration and Electrolyte Imbalance

Age Group	Dehydration	Electrolyte Imbalance
Children and infants[34]	Mild: 3%–5% loss of body weight, reduced urine volume, minimal clinical sign Moderate: 6%–10% loss of body weight, tenting of skin, lethargy and sunken eyes Severe: 11%–15% loss of body weight, hypotension, tachypnea, tachycardia, oliguria, and altered sensorium Note: In hyponatremia, degree of dehydration is less and in hypernatremia degree of dehydration is more than clinical features suggest.[36]	• **Hyponatremia** Muscle weakness, dizziness, hypotension, and tachycardia[39] (Symptoms uncommon if plasma Na^+ is >120–125 mmol/L) Rapid fall in plasma Na^+ to <125 mmol/L results in appearance of symptoms, and to <110 mmol/L causes seizures and coma **Risk factors for osmotic demyelination:**[38] Alcoholics, malnourished, hypokalemia, elderly women on thiazide diuretics, and patients with plasma Na^+ <105 mEq/L • **Hypernatremia** Restlessness, irritability, lethargy, confusion, and somnolence
Adults	Dry tongue and mucosae Loss of skin turgidity Intense thirst Confusion and somnolence Hypovolemic shock	

Protein requirement is calculated by Davies formula, as ollows:[25]

Children : 3 g / kg + 1 g / % BSA involved
Adults : 1 g / kg + 3 g / % BSA involved

Isodense formulas that give 100 kcal/100 mL are pre-erred and are well tolerated. Based on the caloric value of ood articles like milk (100 mL = 60 kcal), sugar (1 tsp = 20 kcal), nd cereals (1½ tsp = 20 kcal), various isodense kitchen-based nteral feeds are prepared, as enumerated below:

1. High energy milk	100 mL milk + 1 tsp sugar + 1/2 tsp oil
2. Cereal milk	100 mL milk + 1 tsp sugar + 1½ tsp cereal flour
3. Fruit juice	1 orange + 2 tsp sugar + water up to 100 mL
4. Egg flip	1 egg + 2 tsp sugar + 150 mL milk

Commercially available enteral preparations can also be ised.[43] In addition to protein and energy, daily supplements of itamins, minerals, and trace elements are provided.[23]

Immune modulating parenteral nutrition containing glutamine, arginine, and omega-3-fatty acids has been demon-trated to benefit the patients with TEN. The immune modulating pproach has been used successfully in burn and other critically ll patients. Glutamine acts as oxidative fuel for lymphocytes nd mucosal cells; arginine promotes lymphocyte maturation nd activation; and omega-3-fatty acids lead to decreased pro-duction of proinflammatory mediators; thus, these substrates enefit the patients with critical illness by reducing infections, requirement for ventilator, and length of stay.[44]

Prevention of Infection

Superadded infections are common in patients with ASF. These worsen the existing skin disease and enhance chance of septicemia. During hospitalization of a patient with ASF, the attending dermatologist must look for focus of skin infections, like folliculitis, abscess, infected fissures, chronic suppurative otitis media, tooth abscess, etc. History of fever in the past few days may indicate underlying systemic infection. The presence of tachypnea may indicate underlying pneumonia.[10]

Patients with ASF are always at risk of septicemia and septicemic shock. Even in frank septicemia, patients may remain afebrile or even hypothermic causing dilemma in diagnosis. Two subtle signs of septicemic shock are reduction in urine volume and altered sensorium, which should be detected at the earliest possible time.[10]

Even in the absence of frank infection on admission, a culture sensitivity test from skin should be sent for all patients of ASF routinely. This will help in the selection of antimicrobials, if required in the future. If initial skin culture is negative, a repeated skin culture sensitivity test should be sent weekly during the patient's hospital stay, for early detection of nosocomial infection. In a febrile patient with ASF, blood culture and urine culture are part of the diagnostic panels.

Barrier nursing must be practiced stringently. Daily sponging and cleaning of body orifices, like oral cavity, nostrils, external auditory canal, genitalia, as well as teeth and gums, eyes, perineum and perianal area must be undertaken. Any pus collection must be drained.

As soon as the patient's condition improves, a gentle bath in lukewarm water with antibacterial soap should be started. During the cleaning process, all debris, dead skin, discharge, and crusts are removed, preferably following lubrication with liquid paraffin.

Antimicrobials should be started in the presence of cutaneous and systemic infection, initially empirically and thereafter based on culture sensitivity report. Caution should be taken in patients with DHS or drug-induced SJS-TEN to avoid accidental use of the same group of drug.

Skin Care

In patients with ASF due to "wet disorders," the skin is tender and bleeds easily. Gentle handling is recommended for them to avoid further damage to the skin. This is especially true for neonates with epidermolysis bullosa, where trauma may precipitate new lesions. Patients should be laid on a non-adherent McIntosh sheet, spread over the bed. Loose clothing without any elastic bands is preferred. Adhesive tape is avoided and resuscitative gadgets should be fixed with roller bandage instead. An air-fluidized bed or frequent posture change is recommended to avoid the formation of pressure ulcers.

Intact bullae should be incised to drain the fluid and the roof is allowed to rest on the floor so that it acts as a biological covering. Raw areas of skin are covered with sheets of lubricated (white soft or liquid paraffin) gauze, changing every day. Various biological dressings are available, which can be used depending on availability.

The skin of patients with erythroderma (dry disorders) often develops deep, painful fissures. These can be managed with lubricants and topical antimicrobials.

Ocular Care

Sight-threatening ocular involvement can occur in SJS-TEN and long-term erythroderma. In patients with SJS-TEN, conjunctival adhesions are separated with a clean glass rod several times a day to prevent synechiae formation. Ectropion and exposure keratitis are the sequelae of long-term erythroderma. Ectropion is managed with instillation of artificial tears at periodic intervals and eye-pads at bedtime. In the presence of exposure keratitis, gas-permeable scleral contact lenses may be used to reduce photophobia and discomfort.[10]

Genital Care

In "wet disorders" with ASF, there is risk of adhesions on opposing denuded surfaces of male and female genitalia. The sequelae are phimosis in men and vaginal stenosis in women. Genitalia of these patients must be inspected and cleaned daily, and adhesions must be broken gently with a glass rod at the earliest. Placing a sterile wet gauze piece between prepuce and glans penis in men and a wet swab between the walls of the vagina in women prevents adhesions.

Therapeutic Intervention

Therapeutic interventions may be "supportive" or "disease specific." Medications must be used very judiciously in patients with ASF, especially when the underlying cause is drug-induced reactions; however, often it is crucial to administer drugs as supportive therapy, but the indications must be genuine and close supervision is mandatory. Some drugs are administered prophylactically, whereas others only when indicated. Various indications of supportive therapy in these patients are presented in Table 6.7.

The relevance of the use of systemic antimicrobials in patients with ASF is controversial and should be used only when indicated, as presented in Box 6.5.

Disease-specific therapy can be started initially when a tentative underlying cause has already been identified or subsequently when the diagnosis is established. A list of these drugs is presented in Table 6.8.

Table 6.7 Indications of Supportive Therapy in Patients with ASF

Indications		
Prophylactic administration	Administration on indication	Drugs administered
Prevention of stress ulcer		H2-blockers/proton-pump inhibitors
	Evidence of frank infection (cutaneous/systemic)	Antimicrobials based on culture sensitivity report
Prevention of stress	Anxious patient	Short-acting benzodiazepine (Alprazolam) at bedtime
	Disturbing pruritus	Antihistamines
	SJS-TEN	Short course of systemic steroid to reduce inflammation
	Hyperglycemia	Intensive insulin therapy[8]
Prevention of nutritional deficiency		Supplementation of the following: • Vitamin B complex • Vitamin D • Vitamin C • Iron • Commercially available protein powder mixed with milk

Box 6.5 Indications of Antibiotic Use in Patients with ASF

1. Isolation of single-strain bacteria (high colony count) from skin specimen/catheter sample of urine
2. Sudden hypothermia in a relatively stable patient (septicemia)
3. Sudden confusion/delirium (meningitis)
4. Pneumonia
5. Urinary tract infection

Table 6.8 Disease-Specific Therapy in Patients with ASF

Conditions	Treatment
Psoriasis	Methotrexate, cyclosporine, phototherapy
Dermatitis	Systemic corticosteroids, cyclosporine, azathioprine
Ichthyosis	Systemic retinoids
Immunobullous disorders	Systemic corticosteroids, adjuvant immunosuppressive drugs
Dermatophyte infection	Systemic antifungals; terbinafine, itraconazole
Crusted scabies	Repeated topical application of permethrin (5%) + oral ivermectin
SSSS	Antistaphylococcal antibiotics; cloxacillin, linezolid
SJS-TEN	Systemic steroid, intravenous immunoglobulin G
DHS	High-dose systemic steroid tapered slowly over a period of time

PROGNOSTIC FACTORS IN ASF

Poor prognostic factors in a patient with ASF on admission are extremes of age, high TBSA involvement, the presence of cytopenias (neutropenia, thrombocytopenia), chronic kidney disease, and other comorbid conditions (hypertension, diabetes, obesity/malnutrition, tuberculosis, HIV infection). In drug-induced cases, if the precipitating drug has a long half-life, it confers a poorer prognosis.[10]

While under treatment in the ICU, development of any of the acute complications as described in Table 6.3 carries poor prognosis. These are associated with risk of mortality in patients with ASF.

CONCLUSIONS

Irvine's concept of "acute skin failure" has proven to be a real entity. Awareness and discussion on this subject in dermatologic meetings have gained momentum in recent years; however, proper research work involving patients with ASF is still sparse in the dermatology literature, and a "burn model" is being used for pointing out therapeutic strategies for these patients. DICU is available in limited institutions, and the concept of ASF, among other medical and paramedical fraternities, is still vague. Dermatologists will have to work still harder to establish the concept of ASF for the benefit of these patients.

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