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NAVIGATING STAPHYLOCOCCAL SCALDED SKIN NDROME FROM ETIOPATHOGENESIS TO MULTIDISCIPLINARY MANAGEMENT

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ABSTRACT:
Background: Staphylococcus aureus produces epidermolytic toxins leading to Staphylococcal Scalded Skin Syndrome (SSSS), characterized by blistering, erythematous cellulitis, and skin peeling. It primarily affects adults and some pediatric patients.
Methods: This review outlines the clinical foundation, etiopathogenesis, complications, treatments, and anticipated future developments of SSSS.
Results: SSSS is a toxin-mediated infection requiring wound care, antibiotic therapy, and supportive care to reduce mortality. A multidisciplinary approach involving dermatologists, infectious disease specialists, and surgeons is crucial due to its differential diagnoses, including toxic epidermal necrolysis, adverse drug reactions, and Steven Johnson syndrome.
Conclusion: Understanding SSSS's clinical aspects and management strategies is essential. Further research is needed to enhance treatment efficacy and patient outcomes.

KEYWORDS: infection with Staphylococcus aureus, staphylococcal scalded skin syndrome, and staphylococcal toxic shock syndrome.

INTRODUCTION:

It is associated with an acute dermatosis that manifests clinically as fever, erythema, and widespread epidermal detachments in Pakistan. It is brought on by the epidermolytic toxins A and B of Staphylococcus aureus. Only a few more than 32 species and subspecies that comprise the Staphylococcus genus are harmful. Despite the introduction of antibiotics, Staphylococcus aureus

remains the most pathogenic species in the genus and is still a common cause of illness and death. It exhibits opportunistic behavior and can cause septic metastases and abscesses, producing invasive and toxic infections, such as S (Fellows et al., 2021).
This microorganism colonizes between 30 and 55 percent of healthy individuals, either temporarily or permanently. This percentage is even higher in insulin-dependent diabetic patients who have HIV infection, dialysis patients who are scheduled for parenteral drug addiction, and people who have chronic skin diseases. The axillae, perineum, vagina, nasal passageways, and oropharynx are the sites of colonization that occur most frequently.

Pathophysiology Reference	Summary
Al-Niaimi et al., 2020	Methicillin-resistant strains of S. aureus, producing exfoliative toxins A and B, contribute to the pathogenesis of St. These toxins cleave desmoglein complex 1, disrupting keratinocyte adhesion and leading to epidermal detachment.

Epidemiology Reference	Summary
Pereira et al., 2022	Staphylococcus aureus colonizes between 30-55% of healthy individuals, with higher rates in certain populations like insulin-dependent diabetics, HIV-infected individuals, and those with chronic skin diseases. Risk factors for SSSS include immunosuppression and chronic renal failure.
De Seta et al., 2021	SSSS carries a substantial risk of morbidity and mortality, particularly in immunosuppressed individuals. Mortality rates in adults can reach 65%, while children typically have lower mortality rates around 6%.

Historical Background Reference	Summary
Fellows et al., 2021	Staphylococcal scalded skin syndrome (SSSS) was initially reported by German physicians in 1878. It is associated with fever, erythema, and widespread epidermal detachments. It is caused by epidermolytic toxins A and B produced by Staphylococcus aureus.

These locations are more common in people who have already been colonized because they serve as reservoirs for future infections. With a gender ratio of 3:1, a prevalence of 0.08 to 0.55 persons per million people is reported, with a higher frequency in newborns (acquired through the birth canal) and those under the age of 5.5. The age range for a presentation is three to four years old at most (Pereira et al., 2022).
Immunosuppression, immunoglobulin deficiency, chronic renal failure, and renal immaturity are risk factors for SSSS. However, SSSS has a substantial risk of morbidity and death if it is not promptly and well treated. Adult mortality rates as high as 65% have been documented; these deaths are most likely due to immunosuppressive conditions or major underlying illnesses. Children usually die at a rate of no more than 6% (De Seta et al., 2021).

Pathophysiology Methicillin-resistant strains of S. aureus are also colonizing more frequently, leading to an increasing number of problems, including S. Despite this, exfoliative toxins A (ETA) and B (ETB) are only produced by 6% of S. aureus isolates from people. ETB is considered the more aggressive of the two atypical serum proteins comparable to trypsin and specific glutamic acid that builds up in the skin. A desmosomal cadherin implicated in keratinocyte cell adhesion, desmoglein complex 1, is cleaved due to ETA and ETB building up in the skin. Exfoliation is finally brought on by the breakdown of the desmosomes that hold the granular layers in place. Since the toxin can travel through the bloodstream to locations far from the site of the primary infection, although typically originating in the head and neck area (conjunctivitis, nasopharyngitis, otitis media), the staph that initially causes the disease is frequently not found in biopsies or cultures (Al-Niaimi et al., 2020).

Factors at risk

It is a pathology that can strike at any age. Still, as previously noted, children under the age of six are more likely to experience it than adults, with no discernible sex difference. There have also been

reports of an increase in this incidence in youngsters in the summer and fall. Several explanations have been proposed to explain the higher occurrence in youngsters, two of which are the immaturity of the kidneys for the excretion of exfoliative toxins and the absence of development of protective antibodies against them. Adults with underlying diseases are primarily at risk due to their higher mortality rates (61% and higher), immunosuppression linked to renal failure, diabetes mellitus, malignant neoplasms, chemotherapy, intravenous drug use, or human immunodeficiency virus infection (Paraiso et al., 2020).

The inability of the toxin to be excreted and the inability of the body to produce antibodies against it would be the pathogenesis linked to these risk factors. However, because of the presence of contaminated vascular accesses, the incapacity of renal excretion, and the ensuing immunological deficiency, patients undergoing hemodialysis are more susceptible to infection. A gene encoding the more virulent exotoxin ETB is present in the S. aureus strain linked to cases of immunocompetent persons with the illness (Paraiso et al., 2020; Tikka et al., 2020).

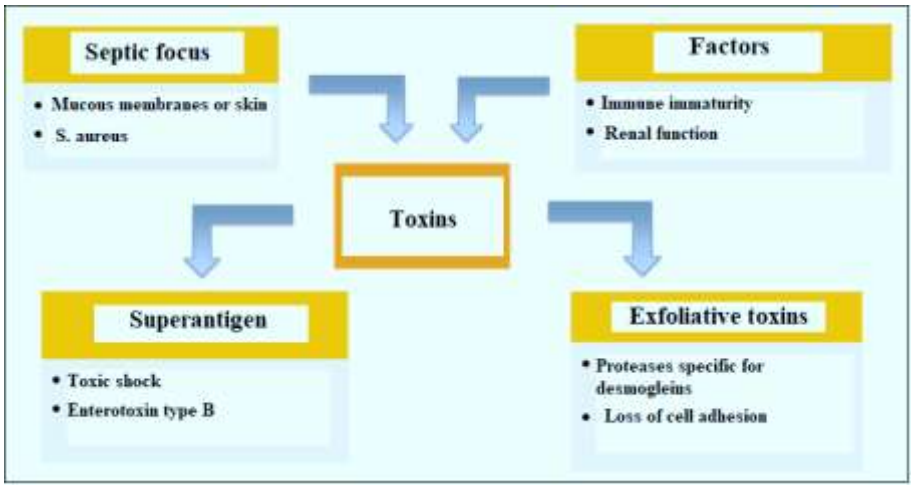

Figure 1: S pathophysiological processes.

Clinical image

From the time of infection to the ultimate expression of an S, the incubation period ranges from 1 to 11 days. Traditionally, the clinical picture starts with a prodrome marked by fever and general malaise, followed by an abruptly developing global erythematous rash. The patient presents with a scarlet rash on the second day, along with cutaneous hyperesthesia, bullous lesions, and a positive Nikolski sign (easy blister rupture under slight tangential pressure) on the trunk, folds, periorificial region, and lesions' spread sites. They develop into generalized skin exfoliation between the third and fifth day, eventually leading to well-defined erythematous patches that can spread to areas more significant than the original lesions. These are typically accompanied by fever and altered consciousness. Skin stripped of its outer layer is susceptible to serous fluid leaking, which can lead to secondary infections (Keating et al., 2024).

After ten days or so, babies typically go through a second peeling period. In 16 days, the lesions completely healed with no skin aftereffects. Adults usually experience a more severe course of symptoms, but their clinical presentation is similar to that of children. There is just one documented instance of congenital S, and it usually manifests in infants between the third and seventh day of life. Unlike toxic epidermal necrolysis or Lyell's syndrome, it does not impact the mucosa. Bacteria are never discovered in the lesions, and the illness is self-limiting and lasts 5 to 9 days. Bullous impetigo may also be linked to these lesions (Dimitrov et al., 2021).

Identification

Most of the diagnosis is clinical. The manifestation of vesiculobullous lesions, a positive Nikolsky sign, and the look of burned skin raise suspicions. At first, I may be mistaken for other conditions, such as toxic epidermal necrolysis, immunological drug reactions, or infections. These conditions

can be clinically distinguished from SSSS by the lack of mucosal involvement, the superficial peeling of SSSS, and the lack of a prescription history. Although the yield is low, blood cultures can be used where the organism is likely to be isolated. Cultures of lesions are not advised. Since our services do not offer the exfoliative toxin gene, PCR is not required for diagnosis beyond academic purposes (Klim et al., 2020).

Difficulties

Although they are uncommon, sepsis, toxic shock syndrome, pneumonia, and dehydration are among the complications of SSSS. Fluid management and laboratory monitoring are essential because electrolyte abnormalities can result from dehydration. Prompt diagnosis and intervention halt more skin peeling and avert morbidity and death (Liu et al., 2023).

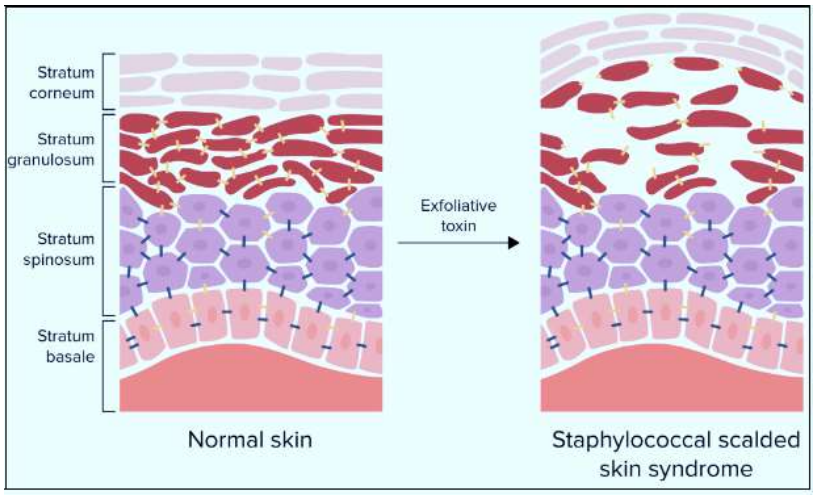

Figure 2: Histopathological depiction and biopsy of S

Handling

It necessitates a multimodal approach because histological testing can rapidly distinguish S from other comparable disorders, for which a biopsy is advised but not strictly required for diagnosis (Kamle et al., 2021).

Histopathology

The degree of excision in S is significantly more superficial than in other illnesses, such as toxic epidermal necrolysis, which is crucial for histological distinction. A ward for critically ill or burned patients is the ideal location for hospitalization. It comprises three main pillars: analgesics, antibacterial therapy, and support (King et al., 2021).

Medium:

Patients need to be treated with wound care and fluid replenishment, just like they would for thermal burns. Electrolytes must be routinely checked, fluid loss must be made up for, hypovolemia must be avoided, and hyponatremia brought on by hypervolemization requires special attention. Covering exposed skin will help to prevent subsequent infections and promote recovery. After applying saline-soaked gauze, the denuded area should be covered with a soft silicone primary dressing. Warm air blankets should keep adults and children at rest to maintain a core temperature of 37°C. In addition, physical therapy is used to preserve joint mobility, which lowers morbidity and speeds up healing (Riyal et al., 2023).

Pain Relief:

Opioids like fentanyl (1-4 µg/kg/h) and paracetamol can be given as needed. NSAIDs should be avoided since they raise the risk of bleeding and have renal excretion. Midazolam (50–100 µg/kg/h)

sedation might be helpful for younger individuals. Medication can be taken as needed to relieve itching (Khalsa et al., 2020).

Antimicrobial Treatment:

It is crucial to start antibiotic therapy as soon as possible, even if burned skin syndrome won't progress for 24 to 48 hours after it begins until the exotoxins are neutralized by antibodies or eliminated by the liver. The course of treatment involves injecting antistaphylococcal antibiotics, such as cloxacillin, intravenously for a minimum of seven days to eradicate the initial infection. It is currently thought that every staphylococcus strain has penicinylases and is penicillin-resistant. Synthetic penicillins, such as flucloxacillin, should be given immediately, 50–150 mg/kg/day for children and 500–1000 mg/day for adults, divided into four doses. Vancomycin (45 mg/kg/day in three daily doses) should be administered to methicillin-resistant S. aureus where the bacteria is expected or methicillin-resistant strains are the source of infection. It is known that clindamycin can counteract exotoxin release in staphylococcal infections. If the patient has a cloxacillin allergy, cefuroxime or ciprofloxacin may be used instead (Sánchez, 2020).

ALTERNATIVE TREATMENTS:

Although no randomized clinical trials have been carried out, the use of exotoxin-neutralizing medicines, such as fresh frozen plasma and immunoglobulin, has been investigated in the case of patients who have not improved with antibiotic treatment. Here are a few of the therapy options:

- Corticosteroids: Since they have been linked to worsening the condition, corticosteroid use should be avoided.
- Immunoglobulins: Although intravenous immunoglobulins have been suggested as a treatment for S, subsequent case studies have linked these individuals to more extended hospital stays.
- Fresh frozen plasma: children in poor condition may get a dose of fresh frozen plasma (10 mg/kg) to neutralize antibodies against exfoliative toxin. 92% of adults over 41 have antibodies against exfoliative toxin.
- Laxatives: It has been proposed that administering substances like lactulose may aid in the excretion of toxins, particularly in highly young patients whose kidneys are not fully matured.

PROJECTED

Following proper therapy, most cases resolve in 2 to 3.5 weeks without any aftereffects. The mortality rate among pediatric patients is around 5%, and it is linked to refractory sepsis, severe skin involvement, and imbalances in fluid and electrolytes. There are reports of adult fatality rates exceeding 60%, which can be attributed to underlying factors that increase an individual's susceptibility to the disease (You et al., 2023).

Differential diagnosis

Toxic shock syndrome caused by staphylococci relates to an uncommon side effect of an infection with S. aureus. Clinical symptoms include fever, rash on the skin, and shock. These can develop into multiorgan involvement, including respiratory distress, DIC, liver failure, and renal failure. It is created by the superantigen-like toxin TSST-1, which intensifies the immune response by releasing a lot of cytokines. Depending on the extent of multiorgan involvement, it might manifest as changes in laboratory tests with leukocytosis with neutrophilia, thrombocytopenia, increased prothrombin time, leukopenia, and varying degrees of biochemical profile alteration. A related investigation relates to TSST-1 gene PCR (Haion et al., 2024).

Three main components support the treatment: immunomodulators, antibiotics, and resuscitation. The first resuscitation step involves replacing crystalloids and using a central venous catheter for monitoring. Vasopressor medications (dobutamine and norepinephrine) are continued when therapy fails. After blood culture, microbiological care entails treating with antibiotics and removing any potential infection triggers. A broad-spectrum antibiotic regimen should be used, which includes clindamycin with cloxacillin or flucoxacillin to inhibit the synthesis of TSST-1 toxins. As an

alternate treatment for resistant agents, studies have demonstrated the inhibitory effects of tigecycline and linezolid on synthesizing toxins. It has been shown that it is beneficial to employ immunoglobulins as immunomodulators to prevent the TSST-1 toxin's superantigenic activity (Omar & Mohammed, 2021).

Stevens-Johnson syndrome – toxic epidermal necrolysis Both toxic epidermal necrolysis (TEN) and Stevens-Johnson syndrome (SJS) are related diseases that include a cutaneous hypersensitivity reaction that is typically brought on by medication. The percentage of impaired skin surface (less than 10% in SJS and more than 31% in TEN) indicates the difference between these two conditions. Although the exact pathophysiology is unknown, it is thought to be brought on by a compromised ability to excrete drug intermediate metabolites, which could then create antigenic complexes and incite an immunological response in the afflicted person. There have been several genetic susceptibilities identified. Although symptoms from Mycoplasma pneumonia, herpes, HIV, and hepatitis viruses have also been reported, medicines account for the majority of cases. The three related medications that are most commonly discussed are lamotrigine, carbamazepine, and allopurinol (Ghassemi et al., 2020).

An annual incidence of 0.4 to 1.3 persons per million is reported. Any age group can experience it. However, women, those living with HIV, recipients of bone marrow transplants, the elderly, and those suffering from systemic lupus erythematosus are more likely to experience it. Prodrome symptoms, including fever, myalgias, arthralgias, and poor general conditions, typically accompany the illness's initial stages. Erythematous skin lesions, also known as targets, appear a few days later. It is noteworthy that 96% of patients with SJS/TEN have mucosal involvement, which is more common than in burned skin syndrome. Eliminating the causing factor and supportive therapy are the cornerstones of treatment. Since most of these symptoms are brought on by medications, as was previously noted, the substance thought to cause these symptoms should be stopped as soon as they manifest (Schlievert et al., 2023).

If the source is bacterial, the appropriate antibiotic therapy must be implemented. Admission to intensive care or burn unit, wound superinfection detection and treatment, electrolyte balance, and nutritional support are desirable components of supportive management. Although several immunomodulatory therapies have been reported, there is debate regarding their efficacy. We can recall corticosteroids, immunoglobulins, and cyclosporine, among others. Drug response accompanied by systemic symptoms and eosinophilia. Drug reaction with eosinophilia and systemic symptoms (DRESS syndrome), an uncommon illness affecting the skin and internal organs, is another alternative diagnosis of SSSS. It is thought to occur once every 1,000–10,000 drug exposures. Among the medications linked to this illness are allopurinol, antibiotics, and anticonvulsants like carbamazepine (Mclauchlin et al., 2000).

Not all of the pathophysiology is known. It has been proposed that there is an unusual way for drug metabolites to be detoxified by enzymes. Herpes family virus reactivation has also been linked to it. *DRESS* typically appears two to six weeks following medication use. The prodrome lasts for a few days before the skin symptoms and starts with itching and fever. Although they differ, the morbilliform rash is the most typical. The traditional distribution initially affects the face, trunk, and upper limbs before moving on to the lower limbs. It could be connected to purpura, bullae, target lesions, and vesicles. As the rash worsens, exfoliative dermatitis may develop. Mucosal involvement and facial edema are frequent. This condition affects the systems (Mehrotra et al., 2000).

The most common types are hematological (leukocytosis, eosinophilia), hepatic (elevated transaminases, alkaline phosphatase), lymphatic (adenopathies), and cardiac (respiratory, pulmonary, and cardiac). The Bouquet criterion comprises skin rash, eosinophilia, and involvement of several internal organs identified for this illness, one of three diagnostic criteria presented. The most crucial therapy action is to determine which drug is causing the problem and stop giving it. Consideration should be given to symptomatic treatment, which includes topical antipyretics and corticosteroids to reduce skin complaints. Nutritional assistance, hydroelectrolyte management, and antibiotic therapy in case of bacterial superinfection should be carried out if it develops into

exfoliative dermatitis. For most patients, early systemic corticosteroid medication initiation is advised (Parsonnet et al., 2005).

CONCLUSION:

Acute dermatosis, known as S syndrome, is brought on by the toxins produced by S. aureus. This microbe can temporarily or permanently colonize a non-negligible percentage of the population. Considering the preceding, one of its salient features is that it is regarded as an opportunistic illness; on the other hand, S carries a high risk of morbidity and mortality if left untreated. It has symptoms that have been reported in several publications. Still, a high level of clinical suspicion and eliminating other illnesses with comparable symptoms and signs are necessary. In addition to the previously mentioned, even though the etiopathogenic mechanisms involved have been defined, the majority of the evidence for management, given its low occurrence, comes from case reports and expert judgments.

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