Case Report

Drug-induced Steven Johnson syndrome

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ABSTRACT

Adverse drug reactions (ADR) are a serious health issue. Drug hypersensitivity reactions can occur in a variety of illnesses, some of which are extremely serious. The skin is the site of the most frequent allergic reactions. Drugs (antimicrobials, such as penicillin and cephalosporins such as cefixime and non-steroidal anti-inflammatory drugs), infections, and other risk factors that have not yet been identified are the main causes of Stevens-Johnson syndrome (SJS). The skin is the site of the most frequent allergic reactions. Iatrogenic morbidity and mortality are largely caused by these reactions, which can range from minor itchy eruptions to potentially fatal situations. To improve the patient's condition, the cause must be determined, the trigger must be removed, and supportive care must be provided. Mortality is high and rises with disease severity, patient age, and underlying medical conditions despite all therapeutic efforts. Survivors may experience chronic symptoms such as mucous membrane strictures and serious eye issues. This case reports that the patient was suffering from fever and conjunctivitis for the last 1 week with lesions on both lips, and ulcerations of the surface of the lips, tongue, and palate. The ulcers were hemorrhagic and tender on palpation. Hemorrhagic crusts and erosions were seen on both lips. The provisional diagnosis revealed that it is an acute febrile illness (AFI) with skin lesions with decreased evaluation further revealed it is an AFI with SJS secondary to drug reaction.

Key words: Cefixime, Rare, Skin lesions, Stevens-Johnson syndrome

Stevens-Johnson syndrome (SJS) is a rare, severe, and potentially fatal drug-induced hypersensitivity reaction. SJS is characterized by extensive inflammation of the epidermis that eventually leads to necrosis and tissue sloughing [1]. This rare condition affects one to two people out of every million annually. This syndrome is linked to an uncommon yet dangerous condition that affects the skin, mucous membranes, genitalia, and eyes as a result of an infection or medication reaction. It starts with flu-like and feverish symptoms, then blisters emerge and a painful red or purplish ash spreads [1]. Affected skin dies, sheds, and then repairs its outermost layers. SJS is an idiopathic condition that can be caused by infections. Reactions to drugs are the most frequent cause of SJS. Early childhood is more likely for 1st-time exposure to antibiotics (such as sulphonamides, penicillin, and cephalosporins like cefixime) or certain viral infections, but adult exposure is more prevalent than that of children and females [1]. The mortality rate from SJS is 10–40%, making it potentially lethal. Survivors often experience long-term problems, such as eye injuries. When this syndrome occurs, serious eye conditions such as acute conjunctivitis, iritis, corneal blisters, erosions, and corneal holes can arise. These conditions can be fatal and cause severe vision loss [1]. Early diagnosis of the illness, stopping the responsible medication, and providing extensive supportive care are all necessary for treatment to be successful. Hydration, electrolyte replacement, body temperature regulation, and supportive and symptomatic interventions should all be part of the main care [2]. Special attention should be given to the airway that is respiratory tract, early oral diagnosis and parental nourishment should be done, pain management and vein access maintenance should be monitored depending upon the affected areas for the prevention of subsequent infections. Skin lesions are managed in accordance with dermatological recommendations [2].

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CASE REPORT

A 33-year-old male patient was brought to causality with chief complaints of fever for the last 1 week. The patient was apparently asymptomatic 1 week ago and then developed fever with chills, cold and cough, mucosal ulcers, peeling of the skin and lips, lesions over the neck, and mucosal peeling bilateral to eye redness. The past medical history revealed that the patient had skin allergy but there was no similar history in the past. No significant family history was presented.

On detailed examination, the patient was conscious/coherent/febrile with a temperature of 102° Fahrenheit, normal blood

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pressure of 120/80 mmHg, saturation was 98%, and pulse rate of 92 beats/min. The cardiovascular system sound 1 and sound 2 were positive and the abdomen was normal. Intraoral examination revealed ulcerations of the surface of lips tongue and palate. The ulcers were hemorrhagic and tender on palpation. Hemorrhagic crusts and erosions were seen on both lips (Fig. 1).

The patient biochemical parameters were as follows: Hemoglobin was-12.2 (normal values-13.8–17 g/dL), white blood cells-14.07 (4–11 k cells/μL), red blood cells-4.59) (4.7–6.1 million cells/L), hematocrit-37.9 (slightly decreased but normal) (38–48%), mean corpuscular volume-82.6 (80–100 fL), sodium-73 (decreased) (135–145 meq/L), leukocyte-13.6 (4–11 kcells/μL), eosinophils-1.5, monocytes-11.5 (4–8%), and platelets-398 k (150–450 k).

The provisional diagnosis revealed that it is an acute febrile illness (AFI) with skin lesions (mucosal peeling) eruption with decreased evaluation upon further reveal it proved that it is an AFI with SJS secondary to drug reaction.

The patient was treated with dexamethasone, azithromycin, 0.05% betamethasone dipropionate ointment, 2% mupirocin, TESS gel, candid mouthwash, eye drops, and syrup ambroxyl. The patient improved symptomatically after the altered treatment.

DISCUSSION

One of the most important clinical issues is drug hypersensitivity. Among the several forms of medication hypersensitivity, SJS is one of the most dangerous and potentially fatal side effects. There is still much to learn about the pathophysiological mechanism of SJS. A small percentage of people are genetically predisposed to these conditions. The people who are most at danger are slow acetylators, particularly those with weakened immune systems and whose livers are unable to fully detoxify reactive drug metabolites [2].

SJS is characterized by the widespread occurrence of papules and macules, which ultimately result in sloughing, skin necrosis, and idiopathic illness. When the liver and the organ that stores Vitamin A are harmed by a drug metabolite, free retinoid molecules leak into the bloodstream, resulting in acute systemic Vitamin A toxicity. The most common molecule in SJS blisters is granulysin, a cytotoxic protein that is produced in large amounts by natural killer cells and CD8+ T-lymphocytes. The keratinocyte apoptosis observed in SJS is caused by granulysin, which functions as a cytokine for broken retinoid molecules [3]. The gastrointestinal tract and the mouth's mucous membranes are usually irritated. Dehydration, sepsis, pneumonia, and multi-organ failure are among the complications. According to reports, the fatality rate for SJS is 1±13, and deaths continue to happen in significant numbers even after hospital discharge [1].

Figure 1: Conjunctivitis, skin lesions, and peeling of skin over lips

Histopathological features and clinical symptoms are used to make the diagnosis. Typical clinical symptoms include blister formation and patches of erythematous, livid skin macules, histopathological analysis reveals a widespread necrotic epidermis affecting all layers [2]. The agent of the underlying suspected infection can be identified through blood, urine, and skin cultures. According to Naranjo's ADR scale and the World Health Organization-Uppsala Monitoring Centre criteria, the causality assessment of ADRs was classified as likely caused by cefixime [3]. The management involves stopping the responsible medication right away. The maintenance of a strict aseptic environment, appropriate fluid-electrolyte balance, and body temperature are all essential. Paraffin guage covering is done were the skin has been stripped, which helps to reduce wound infection and inflammation. In medical management, immunomodulators and steroids are used. Among the surgical management techniques are debridement and non-adherent cutaneous dressings [4].

CONCLUSION

Early removal of the offending agent is essential for increasing patient survival, according to earlier research on SJS. Managing patients with SJS necessitates prompt diagnosis, severity-of-illness score for toxic epidermal necrolysis prognostic evaluation, prompt identification and discontinuation of the offending medication, and specialized supportive care due to the high risk of death. To prescribe the appropriate medication to the appropriate patient through the appropriate route at the appropriate time in the appropriate dosage, clinicians should consider safety, cost, necessity, and effectiveness. An in-depth knowledge of current therapy patterns and the underlying factors is required to enhance the effectiveness and quality of drug therapy.

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