1. Introduction

Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are two rare but potentially fatal medical conditions. Both of these conditions cause severe skin damage and can affect the whole body. In addition, there are several precipitating factors that can trigger the development of SJS and TEN, and one of the factors that can play a key role in these cases is diabetes mellitus (DM) or diabetes. SJS is a milder, though still very serious, form of the SJS-TEN spectrum. Symptoms include painful and potentially lethal skin eruptions involving mucous membranes such as the eyes and mouth. On the other hand, TEN is a more severe form of this condition, with more extensive skin damage, and can even involve almost the entire surface of the body. Both of these conditions can develop quickly and become life-threatening if not treated properly. Diabetes mellitus, commonly known as diabetes, is a chronic metabolic disorder that affects how the body manages glucose (sugar) in the blood. Diabetes can affect multiple systems in the body, including the immune system, and has the potential to increase the risk of developing SJS and TEN in individuals who have these diseases.\(^1\)\(^4\)

Diabetes mellitus (DM) can affect multiple systems in the body, including the immune system, and has the potential to increase the risk of developing Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). This occurs because DM can
interfere with the body's immune response to infection and inflammation, which are factors that can trigger the development of SJS and TEN. Most patients with DM experience changes in their immune system, which can include impaired white blood cell function, impaired inflammation, and decreased ability to fight infection. This weakened immune system can make individuals more susceptible to infections and respond to them in abnormal ways. This can increase the risk of developing a drug reaction or infection, which in turn can lead to the development of SJS and TEN. It is important to understand that other factors can also play a role in the development of SJS and TEN, such as the use of certain medications, infections, or genetic factors. However, DM can be a predisposing factor that increases individual susceptibility to this condition. Therefore, careful management of patients with DM is essential to reduce the risk of developing SJS and TEN, including monitoring their medications and maintenance of good blood sugar control. This study aimed to present cases of TEN with type 2 diabetes mellitus.

2. Case Presentation

A 50-year-old man is a patient referred to the emergency room (ER) at Jayapura General Hospital with initial complaints of a rash, itching, and pain, and extensive blisters with mucosal involvement with an estimated area of around 40%. Previously the patient had a history of taking drugs paracetamol and tetracycline given by the nurse without consulting the doctor beforehand. One day after consuming these drugs appears red hot rash on the face and chest, then the patient goes to the pharmacy to buy dexamethasone and chlorpheniramine maleate (CTM) on the patient's own initiative, then 1 day later, after consuming these drugs, blisters, and crusts appear in the face, lips, eyes, chest, back, and genital areas, accompanied by complaints of shortness of breath and swollen eyes that are difficult to open. Then, the patient was taken to the emergency room of the nearest hospital for 1 day. The patient was treated with a diagnosis of Steven-Johnson syndrome (SJS) and given therapy oxygen, IVFD ringer lactate 20 drops per minute, Dexamethasone 3x1 intravenously, Diphenhydramine 1 ampoule (extra intravenous), installation of a nasogastric tube then the patient was referred to Jayapura General Hospital with the reason that referral did not have a skin and gender specialist doctor.

On arrival at the Jayapura General Hospital, the patient arrived with skin blisters that looked like burns almost all over the body, felt severe pain, accompanied by high fever, blisters filled with black body fluids, and tended to be somnolent. Both swollen eyes are difficult to open, and there is dirt. The swollen mouth has crusts and pain. Patients denied a history of allergies to drugs and a history of other diseases such as diabetes mellitus type 2, Hypertension, and heart disease was denied by the patient.
Figure 1. A when the patient was in the initial hospital before being referred. B. Thick crusts appear on the face, and epidermolysis is seen in the mouth. C and D. Extensive epidermolysis is seen on the patient’s back and chest. E. Epidermolysis of the patient’s genitals is seen.

On physical examination of the patient upon arrival at the emergency room, the general condition of the patient looked seriously ill, conscious somnolence GCS E4V4M6, vital signs blood pressure: 110/80 mmHg; pulse: 100 beats/minute; respiratory rate: 20 breaths/minute; temperature: 38 degrees; oxygen saturation: 99% on room air. On examination of the dermatological status, it was found that, in general, there were lesions such as burns with an area of approximately 40% the size of the lenticular to the circumscrip plaque, there were multiple erythematous violase plaques of nummular size to confluent circumscrip plaques, in the facial region there were crusts in the mouth and nose, in the facial region, neck, chest, waist, buttocks, and genitals showed epidermolysis. Because our hospital does not have a special room, such as a burn center, for these patients, the patient’s family gave informed consent that we would treat these patients in the usual treatment room. The patient was diagnosed with toxic epidermal necrolysis et causa tetracycline and hyperglycemia et causa DMT2 and TEN conjunctivitis.

The therapy given when the patient arrives at the emergency from dermatologist is IVFD RL 20 drops/minute, dexamethasone 2 amp – 0 – 2 amp (iv), ranitidine 50 mg/12 hours (iv), compress NaCl 0.9% 2 times a day, fusidic acid cream 2 times a day, check blood glucose, urea and creatinine, SGOT and SGPT, family education and consult of Internal Medicine and Ophthalmology.

Table 1. The results of blood tests on the first day of treatment.

<table>
<thead>
<tr>
<th>Blood test</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin</td>
<td>14.7 g/dl</td>
</tr>
<tr>
<td>Leukocyte</td>
<td>3.320</td>
</tr>
<tr>
<td>Platelets</td>
<td>139,000</td>
</tr>
<tr>
<td>Basophils</td>
<td>0.3%</td>
</tr>
<tr>
<td>Eosinophils</td>
<td>0.0%</td>
</tr>
<tr>
<td>Neutrophils</td>
<td>75.6%</td>
</tr>
<tr>
<td>Monocytes</td>
<td>5.1%</td>
</tr>
<tr>
<td>Temporary blood sugar</td>
<td>260 mg/dl</td>
</tr>
<tr>
<td>BUN/Creatinine</td>
<td>29.7 / 1.2</td>
</tr>
<tr>
<td>Sodium/potassium</td>
<td>1214.3</td>
</tr>
</tbody>
</table>
Results of consultation with internal medicine were IVFD NaCl 500cc/12 hours, IVFD D5% 500cc/12 hours, paracetamol 3x1gr (iv), ranitidine 2x50mg (iv), sliding insulin scale with novorapid before meals according to blood glucose results, other therapy according to Dermatologist department. The results of consultation with the eye department were P-Pred eye drop 6 x ODS and cleaned the secret in the eye.

Treatment, general condition on the sixth day, looks seriously ill awareness: GCS E4V5M6 blood pressure: 100/90 mmHg; Pulse: 100 beats/minute; respiratory rate: 20 breaths/minute; temperature: 36.7 degrees; oxygen saturation: 99% on room air. GDS: 382 based on dermatological status. No new bulls were found. There were wounds almost all over the body in the facial region, neck, chest, stomach, and back, showing epidermolysis. On both lower and upper limbs, crusts appear, and both soles of the feet have been bullectomy. Treatment Seventh-day General condition: looks seriously ill consciousness: GCS E2V2M3 (delirium) blood pressure: 100/70 mmHg; Pulse: 103 beats/minute; respiratory rate: 18 breaths/minute; temperature: 36.5 degrees; oxygen saturation: 99% on room air. GDS: 400. From the dermatological status of the facial area, erosion, excoriations, and crusts appear. Epidermolysis was seen in the chest, back, palms, stomach, and genital regions. On both lower limbs, visible bullae have dried up. Before the patient was transferred to the ICU, the patient’s condition worsened, so resuscitation was carried out by the doctor on duty, but the patient was declared dead.

3. Discussion

SJS and TEN are often closely related to the use of certain drugs. Several drug classes that have been identified as potential triggers include antibiotics (such as sulfonamides and penicillins), anticonvulsants (such as phenytoin and carbamazepine), and NSAIDs (non-steroidal anti-inflammatories). This is a non-exhaustive list, and there are many other medicines that can trigger this reaction. When a person takes certain drugs, the body can respond by stimulating the immune system. In some individuals, the immune response to these drugs can be abnormal or excessive. This is the basis of the drug reactions that cause SJS and TEN. This reaction involves a T-cell response and cytokine production, which then leads to inflammation. T cells are an important component of the immune system responsible for recognizing and fighting foreign substances, including drugs. In some individuals, the reaction of T cells to certain drugs can be abnormal or exaggerated. This can occur due to genetic predisposition or environmental factors that are not fully understood.10-13

This abnormal T-cell reaction may be one of the early stages of the process that leads to SJS and TEN. Activated T cells respond to drugs by producing a variety of cytokines, which are signaling molecules that play a role in inflammation. Several cytokines, such as tumor necrosis factor-alpha (TNF-α) and interferon-gamma (IFN-γ), have been found in increased amounts in cases of SJS and TEN. An increase in these cytokines can trigger inflammation that damages epidermal cells and mucous membranes. Inflammation is the body’s response to injury or irritation, and in the context of SJS and TEN, inflammation occurs in the skin and mucous membranes. Overproduction of cytokines and activation of the immune system can produce inflammation that damages epidermal cells. This inflammation causes symptoms such as severe skin eruptions, blisters, and detachment of the epidermis, which are characteristic features of these two conditions. The combination of T-cell overreaction, increased cytokine production, and inflammation occurring at the skin and mucous membrane level are key elements in the pathophysiology of SJS and TEN. Extensive inflammation and significant epidermal cell
damage can be very dangerous for the patient, as it can affect various body systems and cause serious complications, including secondary infections and electrolyte disturbances. It’s important to note that not everyone who takes drugs that have the potential to trigger SJS or TEN will develop these conditions. There is an element of individuality in response to drugs, which includes genetic and immunological factors that may make a person more susceptible to adverse drug reactions.14-16

A large number of epidemiological studies have shown a strong association between the use of certain drugs and the risk of developing SJS and TEN. Medications most commonly associated with this condition include antibiotics (such as sulfonamides, penicillins, and cephalosporins), anticonvulsants (such as phenytoin and carbamazepine), and NSAIDs (non-steroidal anti-inflammatories). These studies have shown a significantly increased risk for individuals using these drugs. SJS and TEN often begin with the use of certain medications. This suggests that these drugs can trigger a reaction in the body that leads to the development of this condition. These reactions often involve an immune system response to drugs, which in turn can produce inflammation and damage to epidermal cells (the outermost layer of skin). Studies have also shown that the risk of SJS and TEN can vary depending on the dose and duration of use of the drugs. Some individuals may be more susceptible to the side effects of drugs if they take them in high doses or for a long period of time. Several studies have shown that there are predisposing factors that may make individuals more susceptible to drug reactions that cause SJS and TEN. These factors may involve genetics or pre-existing medical conditions. In some cases, stopping the use of drugs suspected of triggering SJS or TEN and then re-administering these drugs (re-challenge) can result in a return of similar symptoms. This provides additional evidence of the role of drugs in the development of this condition. It’s important to remember that medications vary in terms of risk for SJS and TEN, and not everyone who takes medications known to have the potential to cause these reactions will develop these conditions.17-20

4. Conclusion

Toxic epidermal necrolysis (TEN) is a serious disease that must be treated in burn centers, where experience in the management of the complications of extensive skin loss ensures the best results. The pathophysiology of the condition, fluid loss, risk of multiple organ dysfunction, and risk of sepsis are common in patients suffering from extensive burns.

5. References

6. Finkelstein Y, Soon GS, Acuna P. Recurrence and outcomes of Stevens-Johnson syndrome


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