INTRODUCTION

Stevens-Johnson syndrome (SJS) is a potentially rare, deadly skin disease that usually results from a drug reaction. Another form of the disease is called Toxic Epidermal Necrolysis (TEN), and again this usually results from a drug-related reaction. Both forms of the disease can be deadly as well as very painful and distressing. There are many drugs that have been linked to SJS, and these include NSAIDs, Allopurinol, Phenytoin, Carbamazepine, Barbiturates, and Sulfa Antibiotics. Often, Stevens-Johnson syndrome begins with flu-like symptoms, followed by a painful red or purplish rash that spreads and blisters, eventually causing the top layer of your skin to die and shed. The condition can sometimes although not very often be attributed to a bacterial infection, and in some cases there are unknown causes for the onset of SJS or TEN. However, the most common cause is through drug related reaction.[1]

Case Description

A 36 year old female with complained of flu like symptoms and pink colour rashes all over the body was admitted in the medical unit of a secondary care hospital. She was diagnosed for seizure disorder, a month ago and was prescribed Tab. Phenytoin150 mg, Tab. Ranitidine150 mg and Tab. B Complex. She had no family history of seizure disorder or other chronic diseases. On admission her Serum Creatinine (SCr) and Random Blood Sugar (RBS) were found to be normal but her blood urea was slightly elevated. Her vitals were found to be normal but her RBC count, Hb and HCT values were less than normal. It was diagnosed that flu like symptoms and the pink colour rashes were indications of SJ syndrome induced by Phenytoin. Hence the physician ordered to stop Phenytoin and recommended for plenty of fluid intake to avoid dehydration. The follow up of the patient showed worsened symptoms of
SJS and developed to TEN. The pink colour rashes had spread, blisters had formed and peeling of skin was observed. While the clinical pharmacist critically reviewed the medication order it was observed that she was also prescribed with Tab. Ceftriaxone 250mg, Tab. Dexamethasone, Tab. Chlorpheniramine Maleate and Tab2mg. Alprazolam 1 mg. But as per the standard treatment guidelines for SJS,[2-5] no antibiotics should be initiated. It was also reported that use of corticosteroids like dexamethasone in SJS syndrome will aggravate the symptoms. Based on these evidences, the clinical pharmacists intervened the use of antibiotics and corticosteroids in this patient and the same was accepted by the treating physician. On day 4, both ceftriaxone and dexamethasone were stopped. However, the outcome of this decision could not be documented as the patient was referred to the tertiary care center on her personal request.

DISCUSSION

Stevens–Johnson syndrome was initially described in 1922. Alan Lyell provided an early description of TEN in 1956. Even today, disagreement exists in the literature and many researchers refer to the entity as SJS/TEN.[6-8] SJS presents a medical emergency that usually requires hospitalization. Stevens-Johnson syndrome is a minor form of toxic epidermal necrolysis, with less than 10% body surface area (BSA) detachment. It can affect all age groups, both sexes and all races. It is more common in HIV infection as increased use of medications by HIV patients. More than 200 medications have been reported in association with SJS/TEN. It is more often seen with drugs with long half-lives compared to even a chemically similar related drug with a short half-life. The medications are usually systemic but TEN has been reported in topical use as well. Infections are generally associated with less severe disease than when drugs are the cause.

At the beginning, the patient experienced flu like symptoms (fever, cough, sore-throat, runny nose and general aches and pains) followed by an abrupt onset of a tender/painful red colour skin rashes starting on the trunk and extending rapidly over hours to days onto the face and limbs. The blisters then merged to form sheets of skin detachment, exposing red, oozing dermis. In this patient, detachment >30% of body surface area (BSA) was noticed. In this patient, anemia was also seen as it is a major sign in SJS/TEN.[9] This syndrome has a significant public health impact because of high mortality and morbidity.[10] Although minimal drug therapy is required in the treatment of Stevens-Johnson syndrome, early aggressive management is necessary. It is essential that nutrition be maintained and that
treatment of infections is appropriate to the identified cultures. Antacids, H₂ receptor antagonists, or both have proven beneficial in the prevention or reduction of gastrointestinal ulcers. Most important, however, is psychological support to the patient.

The etiology of SJS in this patient was believed to be associated only with chronic use of Phenytoin as this is one of the direct and well known indications for SJS. But the co-administration of antibiotic and corticosteroid was not realized initially as causative agents for the condition of the patient. Otherwise, the administration of such drugs would have been abstained. Such a decision would have not allowed the worsening of SJS to TEN. Hence, it is the professional responsibility of the clinical pharmacists to review individual prescriptions with available evidences for their risks and benefits.

CONCLUSION
In summary this patient developed both SJS and TEN due to intake of phenytoin for a period of one month for her seizures. Her hemoglobin and hematocrit values were lesser than normal thus proved the patient was anemic, a significant marker for SJS/TEN. Though the use of antibiotics and corticosteroids are not the primary factors for Steven Johnson Syndrome, their use complicated the condition caused by Phenytoin in this case. Hence, evidence based treatment options should be adopted by the clinicians and clinical pharmacists especially in the rare and unique disease conditions like SJS and TEN.

REFERENCES