Keywords: Autoimmune disease; Bullous Pemphigoid; Diabetes Mellitus Type 2; Management

Introduction

e most frequent blistering condition is bullous pemphigoid (BP)1. BP is a dermatological autoimmune blistering disorder marked bullae that are tense can appear on either Skin that is normal or erythematous [1,2, 3]. It consists of circulating and tissue-bound autoantibodies directed against a speci c antigen bullous pemphigoid antigen1 or bullous pemphigoid antigen or both. Diabetes mellitus without preceding corticosteroid use has been identi ed with BP and, as observed in a case control report, can predispose to DM, however there is no a connection to a particular type 2 Increased skin fragility due by Hyperglycaemia and the production of autoantibodies produced by glycosylation of dermal proteins are the mechanisms at work. It is possible to argue that Type 1 diabetes is a hereditary disorder. Type 2 diabetes is more likely in this patient. e use of DPP IV inhibitors like as vildagliptin and sitagliptin has been related to an increased risk of BP group[4-6].

Background

e most prevalent kind of sub epidermal autoimmune bullous illness is bullous pemphigoid. e condition a ects the elderly, particularly those over the age of 70. In diverse populations around the world, the annual incidence of BP has been estimated to range between 2.4 and 21.7 new cases per million populations. In people over the age of 80, it rises exponentially to 190 to 312 cases per million [7,8]. With an annual frequency of 0.2 to three cases per 100,000 people, BP is the most prevalent of the blistering illnesses. e disease has no gender preference, but it is more common in people over the age of 75. Young adults and children are also susceptible, although only infrequently. In people under the age of 50, BP is uncommon, with a reported prevalence of less than 0.5 cases per million population [9,10].

Patient information: A case of 65-year-old female admitted to AVBRH hospital in dermatology ward on date 23/06/2021 with the Fluid- lled Lesion arm, trunk, back, face for 6 months with mild to moderate itching, and discomfort, burning sensation on arm, trunk, back, face for 8 months. Now she came AVBRH for f mild to Medical, family and psychosocial history: She was admitted 2 month before in private hospital for same and treatment was taken in the form of oral medication and cream (Cap. Ce xime BD, Cap. Phagolac BD Tab. Ipinan SR BD, Tab. Bypride TDS, L/A Fusee B Cream TDS , Tab. Omnacortil 10mg BD For 5 Day ,Cycloxan 100 Mg

place, and people. She was in agony, worry, anxiety, and was terribly ill-looking, pale, cyanosed, dehydrated, and afebrile, with all vital parameters normal and a slim body built. Her body mass index (BMI) was 20.5kg/m2, and she weighed 43kg and stood 1.45m tall. Her neurological, chest, and abdominal examinations revealed no abnormalities. Multiple uid- lled vesicles and bullae on erythmatous base, multiple row region over B/L, UL, B/L, LL, Trunk and Back, Face with extreme itching Bullae had a tight demeanour. BSA is around 30%, the bulla spread sign is positive, and the nikolsky sign is negative.

Timeline: Eight months ago, she appeared to be ne. When a uid- lled pea-sized sore appeared on her arm .it was insidious in onset and progresses gradually .it was mild to moderated itching and intermittent burning sensation. Later the lesion started increasing in size and spreadand abdosizl9e2rm .it L4gesD1.2 ton her 30%, Trunk abd B.n a 7 Wh

wias

RoutibgBMIururialbuexa

both the patients and their family.