RAPID PROGRESSIVE GLOMERULONEPHRITIS IN DRUG-INDUCED BULLOUS PEMPHIGOID

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ABSTRACT

Accepted: Month Year,

Rapid progressive glomerulonephritis (RPGN) is characterized by rapid loss of kidney function. This is a severe form of kidney inflammation in some patients with renal disease associated with autoimmune conditions like drug-induced bullous pemphigoid (DIBP). DIBP-induced RPGN involves a complex of mechanisms, pinvolving immune system responses and the formation of crescent structures within glomeruli.

Keywords: DABP, kidney, RPGN, skin

Received: Month Year,

INTRODUCTION

Rapid progressive glomerulonephritis is a serious kidney condition characterized by a swift decline in kidney function, often within days or weeks, and is marked by nephritic features like proteinuria. ¹

Drug-induced bullous pemphigoid (DIBP) is a form of bullous pemphigoid (BP), an autoimmune disease characterized subepidermal blisters, that can be triggered by certain medications. DIBP typically presents with itchy, tense blisters. Medications can induce BP, and medicationinduced BP can occur at any age. New medication exposures should be evaluated when examining patients with an acute onset BP. DIBP-induced **RPGN** multifaceted process where the immune antibodies system, through and inflammatory cells. ³

CASE

A 62-year-old man with a history of diabetes mellitus came to emergency ward with a rash that had been present for approximately 2 days ago. The rash consisted of pink urticarial plaques and tense bullae with areas of erosion and hemorrhagic crust

the trunk and all 4 extremities. predominantly on both the forearms and dorsal aspect of the hands (Fig 1). Of note, this rash developed in the patient 2 days after starting acarbose for his diabetes mellitus. Aside from acarbose, he take other medications regularly, which is premix insulin.. He had no prior significant dermatologic history or history autoimmune disease.

Published: Month Year

During hospitalization, kidney function tests were performed, and the patient experienced rapid kidney function in a matter of days (Table 1). Then the patient was given high-dose systemic corticosteroids 500 mg per day for 3 days, and the suspected drug that triggered DIBP was stopped. After 7 days of therapy, kidney function improved.



Figure 1. clinical feature

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Table 1. Laboratory examination

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Laboratory examination	Day 1	Day 5	Day 7	
hematology difference count ureum/creatinin	11.8/33.8/10.620/330.000 0.5/12.9/70.4/9.5/6.7 62.8/3.4 (20)	92/3.7(16.5)	118/4.5 (13)	
urinalysis IgE total	Microalbuminuria 1549.1 11137 IU/ml			

between the new medication in this patient, it is likely that this represents a case of DIBP. Rapidly progressive glomerulonephritis is a condition characterized by the presence of glomerular crescents.

DISCUSSION

Drug-induced bullous pemphigoid (DIBP) has been reported with several medications and medication classes. The mechanisms by which these drugs induce BP are not yet fully understood, but several mechanisms have been proposed. Possible mechanisms of DIBP include autoimmune damage from altered antigenicity of the structure within the lamina lucida, negative action on immune suppressor cells, or direct splitting of the skin without development of antibody formation.⁴ The new theory suggests that these drugs can modify the structure of proteins in the skin's basement membrane, thereby inducing an immune response against novel antigens. A similar but distinct mechanism has suggested that certain medications can structurally modify proteins and thereby create novel epitopes that stimulate the immune system.

DIBP exhibits the same antibody-antigen pattern as classic BP and presents similarly, consisting of a prodromal phase followed by a bullous phase. However, the presentation often exhibits greater heterogeneity than that of classic BP. The temporal relationship between starting the offending drug and the appearance of blisters can be highly variable, ranging from 2 weeks to months. Interestingly, this patient was most prominently affected on the dorsal aspect of the hands and forearms rather than a generalized distribution.

This patient presented with a rash consistent with BP both clinically and histologically.⁵ The case had a score of 5 on the Naranjo Adverse Drug Reaction Probability Scale, indicating a probable adverse drug reaction based on the temporal association of drug administration with the development of the rash.⁶ Given the temporal relationship

DIBP is defined by C3 and IgG deposition at the basement membrane. C3 glomerulopathy features C3-dominant deposits within renal glomeruli, stemming from defects in the alternative complement pathway.

CONCLUSION

These case reports theorize that there could be cross-reactivity of the autoimmune mechanism between DIBP and RPGN. ⁷

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Vol. x No.x Year DOI:

ISSN: on proses

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Vol. x No.x Year DOI: