Journal of Clinical & Experimental Dermatology Research

Sugiura and Sugiura, J Clin Exp Dermatol Res
2018, 9:2
DOI: 10.4172/2155-9554.1000441

Letter to Editor Open Access

A Case of Bullous Pemphigoid with a High Titer of Anti-BP180 NC16a IgG Antibody

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Received date: December 01, 2017; **Accepted date:** January 31, 2018; **Published date:** February 08, 2018

Letter to Editor

Bullous pemphigoid (BP) is related to various diseases, and it is often difficult to treat because of comorbidities. The coexisting of psoriasis vulgaris (PV) and BP are rare [1]. We reported a case of BP with PV in a male with diabetes mellitus (DM) and chronic renal failure (CRF) [2]. We treated him for three weeks by using the systemic administration of steroid tablets, and his cutaneous conditions were improved. After about four weeks from improvement of his BP, this case recurred with a high titer of anti-BP180 NC16a antibody (18700 U/ml [0-9 U/ml]) (Figure 1). We previously [2] reported that one risk factor for developing BP in psoriasis cases was high titers of anti-180-kDa and 230-kDa IgG antibodies. The anti-BP180 NC16a IgG antibody titer may have been associated with the psoriasis, but the particulars were unknown.

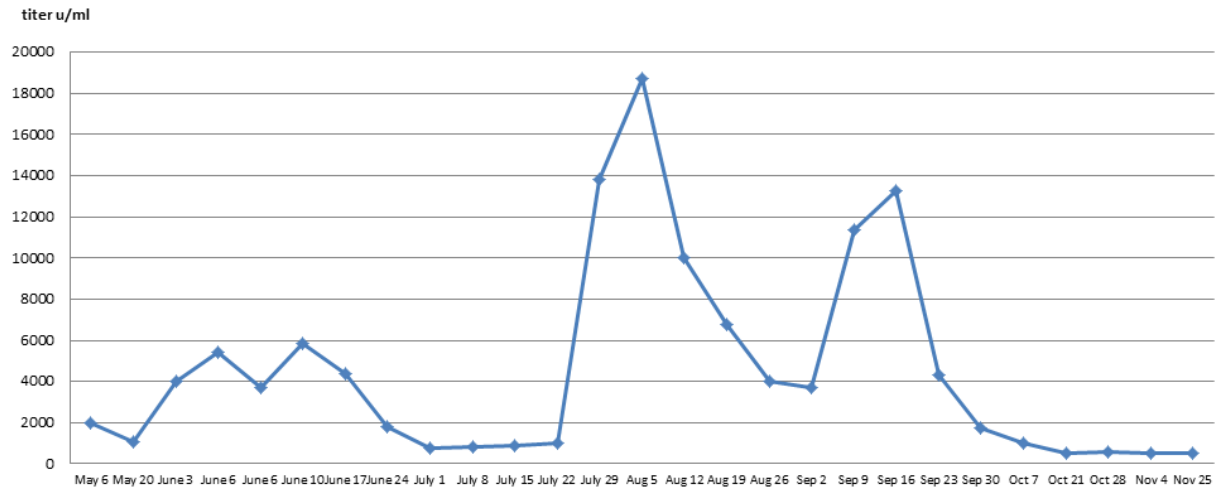

Figure 1: The titer of anti-BP 180 NC16a antibody.

In our case, Bullous Pemphigoid Disease Area Index (BPDAI) scores (Table 1) [3] at the recurrence of BP were skin activity 83, skin damage 10, scalp activity 4, scalp damage 1, and mucous membrane activity 4, for a total activity score of 91 and total damage score of 11. Because the patient's disease complications included chronic renal failure (renal dialysis patient) due to diabetes mellitus, steroid medication dose possibility was limited. We used steroid (methylprednisolone 20 mg/day), minocycline, diaminodiphenylsulfone and cyclosporine for the BP, but with little effect. We then treated the patient by plasmapheresis (1-2 times per week), and his skin condition gradually improved. We found that by decreasing his serum immunoglobulin G, the frequency of plasmapheresis could be reduced to once a week.

Skin	Activity		Damage
Anatomical Location	Erosion/Blisters or new erythema		Post-inflammatory hyperpigmentation or erythema from resolving lesion
	0 absent	Number of lesions if ≤ 3	0 absent
	1-3 lesions, up to one 2>cm in any diameter, none>6 cm		1 present

	2-3 lesions, at least two>2 cm diameter, none>6 cm		
	3>3 lesions, none>6 cm diameter		
	5>3 lesions, And/or at least one>6 cm		
	10>3 lesions, and/or at least one lesion>16 cm diameter or entire area		
Ears			
Nose			
Rest of the face			
Neck			
Chest			
Abdomen			
Back, buttocks			
Arms			
Hands			
Legs			
Feet			
Genitals			
Total skin	83/120		12/10
Scalp			
Scalp	Erosion/Blisters or new erythema	Number of lesions if ≤ 3	Post-inflammatory hyperpigmentation or erythema from resolving lesion
	0 absent		0 absent
	1 in one quadrant		1 present
	2 two quadrants		
	3 three quadrants		
	4 affects whole skull		
	10 at least one lesion>6cm		
Total Scalp (0-10)	10/4		1/1
Mucous membrane			
Anatomical Location	Erosion/Blisters	Number of lesions if ≤ 3	0 absent
	1 lesion		
	2-3 lesion		
	5>3 lesions or 2 lesions>2 cm		
	10 entire area		

Eyes		
Nose		
Buccal mucosa		
Hard palate		
Soft palate		
Upper gingiva		
Lower gingiva		
Tongue		
Floor of mouth		
Labial bucosa		
Posterior pharynx		
Anogenital		
Total Mucosa	4/120	
Total Activity Score :91		Total Damage Score:11

Table 1: BPDAI score.

After one month from the end of plasmapheresis, the patient was completely recovered, and his anti-BP180 NC16a IgG antibody titer was decreased (Figure 1) to 499 U/ml, although this was still beyond the normal range (0-9 U/ml). Despite his improved skin condition, we hypothesized two reasons why his anti-BP180 NC16a IgG antibody titer remained outside normal range: (1) some of the antibodies were non-significantly morbid antibodies related with BP; and (2) the continued production of antibodies. The first reason was most likely due to the improving skin condition. The reason that the titer of anti-BP180 NC16a antibody was not within normal range might be that antibodies that were not significant were still being measured. Such non-significant morbid antibodies were uncertain; it might be that these were not morbid and were related to PV [4,5].

This patient's PV is under good control, and the triggers of his BP are unknown, although his PV might be in a causative factor of the high titer of anti-BP180 NC16a antibody. There were some risk factors for developing BP in psoriasis cases, elderly person, comorbidities and the high titer of both 180 kDa and 230 kDa IgG antibodies [2]. Our case was an elderly patient with psoriasis and CRF due to DM, all of which are closely related to the appearance of BP.

We surmised that about 2.67% (499/18700) of the antibodies in this case were not significantly morbid, and that other patients with BP might also have about 2-3% non-significantly morbid anti-BP180 NC16a IgG antibodies.

The patient's skin condition is currently good, and he has not experienced further recurrence.

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