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Beyond Biologics: Evaluating Efficacy of Dexamethsone Cyclophosphamide Pulse Therapy and Conventional Corticosteroid Therapy in Pemphigus Vulgaris in Resource Limited Settings.

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Abstract:

Pemphigus vulgaris is a chronic autoimmune blistering disorder of skin and mucosal surfaces. Treatment consists mainly of corticosteroids given either in moderate to high oral doses or in the form of intravenous pulse therapy along with adjuvant immunosuppressant. Recently rituximab, an anti CD20 monoclonal antibody, has been approved as first line treatment. With recent advancements and targeted molecular therapies, treatment of pemphigus has been revolutionized. But looking at their high cost, pulse therapy with fewer side effects and with high and rapid remission rates, remains a treatment option in pemphigus in resource limited settings.

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Introduction

Pemphigus is an epidemiologically heterogeneous group of autoimmune chronic bullous disease of skin and mucous membrane, comprising of pemphigus vulgaris (PV), pemphigus foliaceus (PF), pemphigus vegetans, paraneoplastic pemphigus, IgA pemphigus and pemphigus herpetiformis.[(1)] Histologically, there are intraepidermal blisters and immunopathologically, in vivo bound circulating antibodies directed against adhesion molecules (desmogleins and desmocollins) on keratinocyte surface.[(2,3)] Recently association between PV and autoimmune, cardiovascular, endocrine, hematological and neuropsychiatric diseases have been found.

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According to Langan et al, recently, incidence of PV are increasing due to undefined factors and is associated with high death risk.[5] Risk factors include an older age of onset,[6,7] concomitant involvement of skin and mucosal surface at early disease presentation,[8] delayed initiation of corticosteroid therapy,[9] and absence of corticosteroid sparing drugs. The levels of anti-dsg autoantibodies is also a candidate for prediction of overall survival rates.[10]

Current studies show that B cells play an important role in autoimmunity and disease expression along with T lymphocytes. B cells are critical to pathogenesis of pemphigus by producing pathogenic antibodies (anti-desmoglein 1and 3 IgG). Autoreactive B cells are activated by T-cells, differentiating into plasma cells that drive blister formation, making them the primary target of therapeutic strategies like Rituximab.

As with other autoimmune diseases, treatment consists of corticosteroids either in the form of conventional oral or pulse therapy alone or in combination with steroid sparing immunosuppressants. In the present scenario, beneficial effects of rituximab in terms of fewer side effects and rapid induction of remission has made it first line therapy in pemphigus. Many clinical trials now focus on targeted therapy in pemphigus. More recently, advances in our understanding of pathogenesis have led to a paradigm shift in treatment protocol from blanket immunosuppression towards targeted restricted therapies. Research advancements have led to emerging therapies that include anti-CD20 antibodies. BAFF inhibitors, BTK inhibitors, and CART-T therapy, along with FcRn antagonist. Though rituximab has been approved as first-line therapy, cost restraints remain a major limiting factor in India.

Objective: The primary objective was to conduct comparative analysis of therapeutic response of dexamethasone cyclophosphamide pulse therapy (DCP) and conventional corticosteroid regime (CCR) and to evaluate the side effects of both forms of therapies.

Secondary objectives were (1) To compare the relapse rate and time for induction of remission (2) To assess the impact on the patient's quality of life using DLQ1 (3) To determine the cumulative steroid burden in both groups.

Material and Method: The study was conducted in the department of dermatology venereology and leprosy, Mahatma Gandhi Medical College, Jaipur for 1 year. A total of 50 patients of pemphigus vulgaris were included who were subjected to random selection for either DCP or CCR. Each subgroup had 25 patients.

Apart from clinical diagnosis, inclusion criteria were aged between 20 to 60 years, Tzanck smear showing acantholytic cells and hazy nucleoli and histopathological examination and direct immuno fluorescence (DIF) consistent with pemphigus vulgaris.

Exclusion criteria were age less than 20 and more than 60 years, bullous eruption associated with drugs, malignancy or pregnancy and eruption showing seasonal or environmental variation. Patients having HIV, tuberculosis, ischemic heart disease, psychiatric issues, epilepsy, systemic fungal infection, herpetic keratitis, myelosuppression, septicemia, major electrolyte imbalance, hepatic or renal dysfunction and hypersensitivity to injected drugs were also excluded.

Patients were subjected to detailed clinical and laboratory evaluation before, during and after treatment. Monitoring parameters included (1) PDAI (2) DLQI score (3) Lab investigations (4) Adverse drug events.

Key end points considered were (1) Time to initiate response for (a) Skin (b) Mucosa (2) Time for complete remission (3) Relapse rates both during treatment and after treatment (4) Adverse Events (5) Steroid exposure & hospital stay.

Dexamethasone Cyclophosphamide Pulse therapy: Patients were subjected to detailed clinical and laboratory evaluation before, during and after pulse. Intravenous infusion of dexamethasone 100 milligrams in 500 ml of 5% dextrose over 2 hour was given on 3 consecutive days with 500 milligram of intravenous cyclophosphamide in 500 ml of 5% dextrose added on second day. This schedule was repeated at 4 Weeks with close watch on vital parameters during pulse therapy. The structured 4 phase approach (initially described by Dr. JS Pasricha) was followed. All relevant data were recorded on specialized performa.

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Conventional corticosteroid therapy: Before initiation of treatment, thorough clinical and investigative evaluation of the patients was undertaken. Thereafter, according to disease severity 40-120 mg at dose of 0.5-1 mg/kg body weight of prednisolone equivalent doses of steroids, along with adjuvant (cyclophosphamide or dapsone) was started. When the disease was under control the doses were reduced to minimum effective levels (15 to 60 mg daily or on alternative days). Once the disease was stable, attempts were made to withdraw steroids and maintain only on adjuvants.

In both groups, supportive therapy like antibiotics, antifungals, acid blockers, iron and potassium supplements were added according to the patient's requirement. During flare-up, the dose of steroid was increased during with subsequent taper.

Complete remission was defined as absence of new lesions and healing of all existing lesions after withdrawal of intermittent steroids.

Relapses were defined as the appearance of 3 or more new lesions that did not heal spontaneously within a week or by extension of established lesions in patients who had achieved control full stop. Failure of therapy was defined as inability to control disease activity with a full therapeutic dose of systemic steroids.

Observation: It was observed that induction of remission of both cutaneous and oral lesions was faster in patients with DCP (76% cutaneous and 45% mucosal) compared to CCR (cutaneous 46% and mucosal 25%) during initial 1-4 months. Complete remission was achieved in a significantly higher number of patients with DCP (84%) compared to CCR (40%) beyond 12 months of therapy.
About 17 of 25 cases in Group A (68%) and 6 of 25 cases in Group B (24%) evidenced clinical remission within one to 6 months of institution of therapy although cumulative dose of prednisolone equivalent was higher in DCP (1000-20000mg) as compared to CCR group (1000-10000 mg).

Table 1: Induction of Remission

Duration in months	DCP			CCR		
	Mucosal lesions	Cutaneous lesions	Completed remission	Mucosal lesions	Cutaneous lesions	Complete remission
1-4	11	19	10	8	12	6
5-8	8	2	9	4	1	3
9-12	2	0	1	2	1	1
Beyond 12 months	1	1	1	-	-	-
No remission	3	3	3	11	11	15

4 patients did not respond to DCP even after 9 months, while 11 patients in CCR continued to have active disease despite a cumulative dose of 10,000 milligram of prednisolone equivalent given for an equal period. Complete remission was seen in 21(84%) patients of DCP group and in only 1(4%) case in CCR Group at the end of 12 months of therapy.

Table 2: Comparison of PDAI outcome between DCP and CCR (n=25 per group)

Time Point	DCP Mean PDAI ± SD	CCR Mean PDAI ± SD	Mean change from baseline DCP	Mean change from baseline CCR	Complete Remission
Baseline	45±10.2	45.3±9.8	-	-	-
6 Month	10.2±6.1	18.4±8.0	34.8±9.3	26.9±10	DCP=40% CCR=20%
12 Month	3.1 ± 4	8.2±6.2	41.9±10	37.1±11.2	DCP=64% CCR=20%

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DCP patients showed faster and more sustained reduction in PDAI compared to CCR. This faster control, allows early tapering of steroids and mitigating its side effects. It also enables sepsis prevention and fluid and electrolyte stability by earlier re-epithilization of erosions. Faster visible improvement reduces depression, anxiety and social withdrawal often associated with the disfiguring nature of pemphigus lesions. While not universal, a rapid decline in PDAI is often associated with more durable long term clinical responses compared to slow, recalcitrant cases.

Table 3: Comparison of side effects of DCP and CCR among pemphigus patients.

S. No.	Side effects	Group A (DCP) n1=25		Group B (CCR) n2= 25	
		No.	%	No.	%
1	Pulse rate				
	Increased	4	16	3	12
	Decreased	1	4	1	5
2	Increase BP	16	64	11	44
3	Increased weight	15	60	15	60
4	Dermatological side effects				
	Cushingoid faces	13	52	11	44
5	Pigmentation	1	4	2	8
	Purpura	2	8	0	0
	Telangiectasia	4	16	1	4
	Secondary infections				
	Viral	1	4	1	4
	Bacterial	2	8	12	48
	Mycobacterial	1	4	2	8
	Candidal	4	16	9	32
	Dermatophytosis	1	4	9	32
6	Psychosis	3	12	5	20
7	Hyperglycemia	3	12	3	12
8	Muscle weakness	6	24	5	20
9	ECG changes	5	20	0	0
10	GIT upset				
	Gastritis	3	12	8	32
	Hiccup	3	12	0	0
	Constipation	2	8	0	0
11	Edema	8	32	7	24
12	Menstrual dysregulation	1	4	2	8
13	Osteoporosis	1	4	7	24

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Weight gain, hypertension and cushingoid facies were common in both groups. Telangiectasia, hypertension and hiccups were more frequent in the DCP group while bacterial, fungal and candidal infections along with osteoporosis were greater in the CCR group.

Table 4: Comparison of cumulative steroid dose, adjuvant and supportive drugs and adherence to treatment:

S. No.	Parameters	DCP	CCR
1	Mean cumulative dose of Prednisolone equivalent	22533 mg	4366 mg
2	Mean treatment duration	15.6 week	10.8 week
3	Adjuvant cyclophosphamide	19	16
4	Adjuvant dapsone	6	9
5	Antibiotics	14	20
6	Antifungal	15	14
7	Hematinics	25	25
8	Antacid	6	12
9	Potassium supplements	6	5
10	**Treatment adherence**		
	Regular	15	7
	Irregular	10	18
11	Off steroids	13	1
12	Lost follow up	5	9

Patient adherence was more in DCP and the therapeutic response was satisfactory in 84%. cases of DCP. It shows the mean cumulative dose of steroids was 5 times in DCP. Patients on DCP were regular and remission rates without steroids were high in them.

Table 5: DLQ1 outcome in pemphigus patients T/T with DCP vs CCR (n=25 per group)

DLQ parameter	DCP	CCR	P value	Statistical Test
Baseline DLQ1	18.4±4.2	19.1±3.9	0.542	Independent t test
DLQ1 week 12	4.2±2.8	8.6±3.5	<0.001	Independent t test
DLQ1 (change from baseline)	-14.2±3.9	-10.5±4.1	0.003	ANCOVA
DLQ1 week 24	3.1±2.1	6.9±3.2	<0.001	Mann-Whitney U test
Patients achieved DLQ1 (0/1) week 24 (n%)	14 (56%)	6 (24%)	0.018	Chi-Square test
Patients with DLQ1 ≥5%	23 (92%)	18 (72%)	0.067	Fisher's exact

This shows DCP therapy significantly reduced DLQI at both 12 and 24 weeks compared to CCR. More than half of the DCP treated patients achieved DLQI of 0 or 1 by week 24 compared to 24% in the CCR group.

Table 6: Concise Comparison of patients receiving DCP and CCR

				DCP	CCR
1	Therapeutic response	a) Time for Induction of Remission (months)	Oral	5.4	3.6
			Cutaneous	2.0 9	3.66
			Complete	5	4.5
			Remission	21	10

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		b) Response	Disease activity present	4	15
2	Side effects	General	-	17	16
		Dermatological	-	14	13
		Systemic	-	14	17
		Metabolic	-	3	3
		Infections	-	11	8
3	Course and prognosis	Off steroids	-	13	1
		Continuing Treatment	-	7	15
		Lost to follow up	-	5	91

Induction of remission was faster and DCP group. Persistent active lesions were seen in the majority of patients of CCR (60%) compared to the DCP group (16%). Steroids could be withdrawn in 13 patients in the DCP group and only in one case in the CCR group.

Thus, it can be summarized that Pemphigus usually affects middle aged people in the 3rd and 5th decade. The period for achieving remission for oral and cutaneous lesions is shorter in DCP compared to CCR. Further, remissions are maintained for longer periods without frequent relapses in the DCP group. Persistent active lesions were seen in the majority of patients in the CCR (60%) group compared to DCP (16%). Steroid withdrawal and suppression of disease activity was higher in the DCP group though there was a great difference in the mean cumulative dose of steroid. There was a difference in incidence of side effects in 2 groups. This concludes, that DCP is superior to CCR for induction of remission for both oral and cutaneous lesions and disease activity suppression For longer time without steroids with no evidence of increased incidence of side effects.

Discussion: The general opinion about all autoimmune disorders is that there is no permanent cure but reasonable remission. Therapeutic management of pemphigus remains challenging. Conventional oral corticosteroids have remained the cornerstone of treatment, but they were not able to induce long term clinical remission and were associated with adverse effects. Corticosteroids sparing adjuvant drugs decreased cumulative dose of steroids but did not have morbidostatic effect. Although life saving, these strategies were not specific to pemphigus pathology and had considerable side effects. With oral corticosteroids, there are many differences in initial doses, tapering schedule and management of relapses between different groups.[14]

Immunosuppressive agents such as azathioprine, mycophenolate mofetil are widely used but adjuvant application and dose regime of different recommendations are not standardized. The difference is attributed to the clinical scoring adopted, the standards for disease severity evaluation, the publication year of each guideline and local and regional healthcare differences. [14]

To decrease the side effects of long term steroids, for better efficacy and quicker results the concept of pulse therapy was introduced by Pasricha and Gupta.[20] The basic difference between DCP and CCR is the dose and duration of therapy. Pasricha et al used intermittent high doses in 79 pemphigus patients and reported rapid induction of remission.[22] It is speculated by various authors the DCP causes clonal suppression of specific B cell producing auto antibodies in PV.[21]

Corticosteroids effects on T- cell include redistribution, immune-suppression by decrease cytokine secretion, favoring the lineage development, induction of apoptosis and attenuating T-cell receptor signaling.[23] The combined immunosuppressive and anti-inflammatory effects of both immunosuppressive drugs and supra pharmacological doses of corticosteroid has been utilized in pulse therapy to induce immune tolerance. The rapid elimination of intravenously administered drugs allows lesser side effects.

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In the current study also, moderate to severe skin and mucosal lesions of pemphigus vulgaris responded better with DCP. It is difficult to maintain regular follow up of pemphigus patients receiving DCP and achieving quick remission beyond the second phase of DCP.[20] Hence comparative analysis of DCP and CCR therapy beyond the active stage is difficult. Roy et al reported remission with DCP in 40% of 20 regular follow up patients out of 87 registered patient.[24]

Werth et al, also stated DCP to be more effective compared to patients of oral steroids group who had protracted course and no long-term remission.[30] Therefore superiority of DCP during active stage of disease is justified (remission rate 76% in DCP and 56% in CCR). Kanwar et al, mentioned 4 remission patterns (a) quick initial response within one to 2 months of DCP (b) moderate response with partial remission (c) poor response requiring daily oral steroids (d) poor response with rapid relapses requiring in between mini pulse[25]. Therapeutic response may not be homogeneous in all pemphigus patient.

The variable response of DCP may probably be due to genetic variation, physiological variable response of GC receptors and individual metabolic status. Curative effect has been reclaimed by Pasricha et al in 100 patients followed for 10 years with mild relapses controlled by additional DCP and few death.[26] Many researchers conclude admixture of DCP and CCR regime for better management of pemphigus.[27] A lot of modifications and variations have been described[28] with complete cure for long periods possible only with DCP in pemphigus.[29] DCP has immediate suppression of serological and clinical activity of pemphigus with minimal HPA suppression while CCR is used to avoid acute life threatening complications of CS like cardiac arrhythmias electrolyte imbalance and seizure.

The quality of life of individuals with Immunobullous disorders is significantly impacted negatively in terms of their mental health.[48] It is important to note that quicker remission and reduced relapse rates translate into meaningful improvements in patient quality of life. Mucosal lesions, nutritional compromise, pain, and visible skin erosions have profound effects on daily functioning, social interactions, and mental health. Chronic diseases, like pemphigus vulgaris show great impairment of DQOI of patients.

The symptom and also the side effects of treatment contribute to decreased life quality.

In our study, the DCP group showed significantly reduced DLQI at both 12 and 24 weeks compared to CCR suggesting a greater impact on mental, social and physical well being of the patient. Not only DQLI, pemphigus vulgaris significantly affects the Family Dermatology Life Quality Index. Education and counseling of family caregivers by various support groups such as Pemphigus Family Associations could be effective in improving the quality of life of the caregivers[48].Chronic diseases can affect many aspects of the quality of life of both patients and their families including their social, mental and physical health, living expenses, time spent with the patient and issues related to job or studies.

The important side effects encountered with supra pharmacological doses are electrolyte shift, cardiac dysrhythmia, ECG changes, seizure along with hyperglycemia and hyperlipidemia. Pulmonary infection and reactivation of underlying tuberculosis and increase in bacterial viral and fungal infections is common with DCP. Other transient effects include hiccups[31], facial flushing[32], diarrhea, weakness, joint and muscle pain, generalized swelling and weight gain.

Modifications of DCP include Dexamethasone-azathioprine pulse (DAP) in which cyclophosphamide is replaced by daily oral azathioprine. No bolus dose of azathioprine is given during the pulse. DAP is recommended for unmarried patients who have not completed their family. In Dexamethasone-methotrexate pulse (DMP), cyclophosphamide is replaced by 7.5 mg of oral weekly methotrexate (three doses of 2.5 mg at 12 h apart), during the three phases of pulse therapy.
DMP is recommended for patients not responding to DCP/DAP after 12 pulses in Phase 1.

Intravenous immunoglobulin (IVIG) and immunoadsorption are also a therapeutic option in severe or refractive pemphigus[43]. Since the advent of target therapies, the management of pemphigus has gradually changed. Among conventional adjuvant immunosuppressants both EADV and BAD guidelines suggest azathioprine and mycophenolate mofetil as first line. However different variables including patient comorbidities, single institution experience and costs have to be taken into account and other drugs like methotrexate and cyclophosphamide also

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demonstrate efficacy. They do not lead to improvement in achieving remission but reduce the risk of relapse.

In recent years, much advancement has been made in treatment of pemphigus. Specific targeted therapies to the pathogenic immune pathway have been introduced like monoclonal CD 20 antibodies (rituximab), BAFF and BTK inhibitors, CARR-T therapy[34] FcRn antagonist and TNF-Alpha inhibitors.

Other biological agents like calcineurin inhibitor and immunoadsorption appear promising.[35] Other advancements, include blocking antigen specific T cell via CD154 monoclonal antibody.[36,37] Other novel approach in research is altered peptide ligands that may be engineered to prevent interaction between auto reactive T cells and autoantigen peptides that present on disease associated HLA class molecules.[37,38]

In a study on pathogenic molecular pathways, which highlighted mechanism based therapeutic pathways targets in PV 25 molecules (uPA, snc, p35, MAPK, caspase 3 etc.) were recognized, inhibition of which, resulted in reduction in acantholysis.[41]·Out of these ,Rituximab a highly effective, anti-CD20 monoclonal antibody therapy for moderate-to-severe pemphigus vulgaris (PV), designed to deplete B-cells and reduce pathogenic autoantibodies, has been extensibly used.

A recent multicentric study supports using rituximab as first line adjuvant showing superior efficacy compared to corticosteroid alone.[15,16] Rituximab exerts a deep modulation of both humoral and acquired immune function explaining disease amelioration lasting longer than B cell reappearance[17] without affecting plasmablast[18] and memory B cell compartment resident in lymphoid tissue.[19] Patients in complete remission display increase number of IL-10 producing regulatory B cells and absence of Dsg 3 IgG+ B cells[17] ·Rituximab, targeting CD 20 positive B cell is approved as first line therapy in moderate to severe pemphigus.[14]. CD 20 is believed to function as calcium channel and play role in maturation and activation of B cells.[42] Patients treated with rituximab showed B cell depletion with reduction in serum autoantibodies and Dsg 3 specific CD 4+Th1

Combination of rituximab with immunoadsorption

induces rapid clinical remission and long term control of pemphigus.[40] Rituximab is a viable treatment in older patients, albeit with diligent monitoring and vigilance.[45]

However, the optimal doses and timing of maintenance therapy of rituximab are uncertain and prognostic factors are unknown. Several relapses occurred during the course of chronic disease. It does not lead to permanent remission due to B cell repopulation within 1 to 2 years after receiving rituximab. Two limiting factors are (1) infusion reaction[44,46] and (2) occurrence of anti-rituximab antibodies.

Despite remote serological or clinical relapses, the majority of pemphigus patients could achieve normal life without steroids in the DCP group and that is the first and last aim of a treatment in pemphigus. Complete remission of about 10 years has been reported by Pasricha in pemphigus patients on DCP. They claimed, if substantiated by further follow-up, this treatment schedule may prove curative in this potentially fatal disease[50]. Comparable clinical efficacy between DCP and rituximab was observed in some studies,[51] Rituximab and DCP are both popular treatments for pemphigus but they differ in cost efficacy and side effects. Though rituximab offers advantage of early and prolonged remission, lesser side effects, faster improvement in PDAI and DLQ1[33] and fewer infusions, it has not been able to completely replace DCP in India due to its higher cost (one-time downpayment). Rituximab represents a higher upfront investment. Pulse therapy is considered a vital option for managing pemphigus efficiently, especially in developing areas, but it is not completely without risks, requiring careful patient management.[49] Even with Rituximab incidence of relapse was at least 50%.[52] Rituximab is an expensive drug and it demands a one-time payment to procure the drug, The remitting and relapsing nature of the disease, imposes financial burden of treatment and the adverse effects associated with the treatment modalities (systemic immunosuppressants) even when the disease is under control. In resource limited settings, rituximab may be used for the treatment of PV patients who are nonresponsive to or who develop serious side effects to conventional therapy.

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Conclusion: DCP therapy was found to have greater efficacy in controlling the symptoms with longer remission periods. Quick healing of lesions reduced the hospital stay compared to oral steroids. DCP is superior in terms of safety, efficacy and longer remission. Though Rituximab is a promising first line therapy, cost remains a major barrier in its widespread use in India with the majority of patients in the middle income group attending primary health centers where accessibility to biologics may be limited. Accessibility and affordability remains a primary concern in the peripheral rural areas. Thus, DCP is still a feasible treatment option even if patients need long term & multidisciplinary follow up.

Reference's:

1. Kridin K, Schmidt E. Epidemiology of pemphigus. *JID Innov.* 2021;1(1):100004.
2. Eichkorn RA, Schmidt MF, Walter E, Hertl M, Baron JM, Waschke J, Yazdi AS. Innate immune activation as cofactor in pemphigus disease manifestation. *Front Immunol.* 2022;13:898819.
3. Davis G, Hathway R, Shipley D, et al. The management of pemphigus vulgaris and mucous membrane pemphigoid in a joint oral medicine and dermatology clinic: a five-year narrative review. *Br Dent J.* 2024;236:311–316.
4. Ingold CJ, Sathe NC, Khan MAB. Pemphigus vulgaris. In: *StatPearls*. Treasure Island (FL): StatPearls Publishing; 2024.
5. Langan SM, Smeeth L, Hubbard R, Fleming KM, Smith CJ, West J. Bullous pemphigoid and pemphigus vulgaris: incidence and mortality in the UK. *BMJ.* 2008;337:a180.
6. Shamsadini S, Fekri AR, Esfandiarpoor I, Saryazdi S, Rahnama Z, Zandi S, et al. Determination of survival and hazard functions for pemphigus patients in Kerman. *Int J Dermatol.* 2006;45:668–671.
7. Huang YH, Kuo CF, Chen YH, Yang YW. Incidence, mortality, and causes of death of patients with pemphigus in Taiwan. *J Invest Dermatol.*2012; 132:92
8. Chams Davatchi C, Valikhani M, Daneshpazhooh M, Esmaili N, Balighi K, Hallaji Z, et al. Pemphigus: analysis of 1209 cases. *Int J Dermatol.* 2005;44:470–476.
9. Seidenbaum M, David M, Sandbank M. The course and prognosis of pemphigus. *Int J Dermatol.* 1988;27:580–584.
10. Baican A, Chiorean R, Leucuta DC, Baican C, Danescu S, Ciuce D, Sitaru C. Prediction of survival for patients with pemphigus vulgaris and foliaceus. *Orphanet J Rare Dis.* 2015;10:48.
11. Porro AM, Seque CA, Ferreira MCC, Enokihara MMSES. Pemphigus vulgaris. *An Bras Dermatol.* 2019;94(3):264–278.
12. Abraham A, Roga G, Job AM. Pulse therapy in pemphigus: ready reckoner. *Indian J Dermatol.* 2016;61(3):314–317.
13. Kountz SL, Cohn R. Initial treatment of renal allografts with intrarenal immunosuppressive drugs. *Lancet.* 1969;1:338–340.
14. Zhao W, Wang J, Zhu H, et al. Comparison of guidelines for management of pemphigus. *Clin Rev Allergy Immunol.* 2021;61:351–362.
15. Joly P, Horvath B, Patsatsi A, Uzun S, Bech R, Beissert S, et al. Updated S2K guidelines on pemphigus. *J Eur Acad Dermatol Venereol.* 2020;34(9):1900–1913.
16. Werth VP, Joly P, Mimouni D, Maverakis E, Caux F, Lehane P, et al. Rituximab versus mycophenolate mofetil in pemphigus vulgaris. *N Engl J Med.* 2021;384:2295–2305.
17. Colliou N, Picard D, Caillot F, Calbo S, Le Corre S, Lim A, et al. Long-term remission after rituximab therapy. *Sci Transl Med.* 2013;5:175ra30.
18. Mouquet H, Musette P, Gougeon ML, Jacquot S, Lemercier B, Lim A, et al. B-cell depletion immunotherapy in pemphigus. *J Invest Dermatol.* 2008;128:285–296.
19. Cho A, Bradley B, Kauffman R, Priyamvada L, Kovalenkov Y, Feldman R, et al. Memory responses after rituximab therapy. JCI insight. 2017;2:e93222.

20. Pasricha JS, Gupta R. Pulse therapy with dexamethasone cyclophosphamide in pemphigus. Indian J Dermatol Venereol Leprol.1984;50:199-203.
21. Pasricha JS. Pulse therapy in pemphigus and other diseases. 2nd ed. New Delhi: Pulse therapy foundation;2000.
22. Pasricha JS, Thanzama J, Khan UK. Intermittent high-dose dexamethasone cyclophosphamide therapy. Br J Dermatol. 1973;88:73-77.
23. Williams LC, Nesbitt LT Jr. Syytemic glucocorticosteroids in dermatology. Dermatol. Clin.2001;19(1):63-77.
24. Roy R, Kalla G, Dexamethasone-cyclophosphamide pulse therapy in pemphigus. Indian J Dermatol Venereol Leprol. 1997;63:354-356.
25. Kan AJ, Kaur S, Thami GP. Long-term efficacy of dexamethasone-cyclophosphamide pulse therapy. *Dermatology.* 2002;204:228–231.
26. Pasricha JS, Seetharam KA, Das U. Further studies on pemphigus patients treated with dexamethasone. *Indian J Dermatol Venereol Leprol.* 1989;55:98–104.
27. Ramam M. Dexamethasone pulse therapy in dermatology. *Indian J Dermatol Venereol Leprol.* 2003;69:319–322.
28. Rao PN, Lakshmi TS. Pulse therapy and its modifications in pemphigus. *Indian J Dermatol Venereol Leprol.* 2003;69:329–333.
29. Pasricha JS. Current regimen of pulse therapy for pemphigus. *Indian J Dermatol Venereol Leprol.* 2008;74:217–222.
30. Werth VP. Treatment with high-dose intravenous glucocorticoids. *Arch Dermatol.* 1996;132:1435–1439.
31. Kanwar AJ, Kaur S, Dhar S, Ghosh S. Hiccup as a side effect of pulse therapy. *Dermatology.* 1993;187:27.
32. Dhar S, Kanwar AJ. Facial flushing as side effect of pulse therapy. *Dermatology.* 1994;188:332.
33. Das S, Agarwal K, Singh S, Halder D, Sinha S, De A. Rituximab versus DCP therapy in pemphigus vulgaris. *Indian J Dermatol.* 2021;66(2):223.
34. Abulikemu K, Hu F, Liang J, Kang X. Targeting therapy in pemphigus. Heliyon. 2023;9:e16679.
35. Jessop S, Khumalo NP. Pemphigus. Am J Clin Dermatol. 2008;9:147-154.
36. Kridin K. Emerging treatment options for pemphigus vulgaris. Ther Clin Risk Manag. 2018;14:757-778.
37. Aoki-Ota M, Kinoshita M, Ota T, Tsunoda K, Iwasaki T, Tanaka S, et al. Tolerance induction in pemphigus model. *J Invest Dermatol.* 2006;126:105–113.
38. Anhalt G, Werth V, Strober B, et al. Safety of PI-0824 in pemphigus vulgaris. *J Invest Dermatol.* 2005;125.
39. Eming R, Nagel A, Wolff-Franke S, Podstawa E, Debus D, Hertl M. Rituximab dual effect in pemphigus vulgaris. *J Invest Dermatol.* 2008;128:2850–2858.
40. Behzad M, Möbs C, Kneisel A, Möller M, Hoyer J, Hertl M, Eming R. Immunoadsorption and rituximab therapy. *Br J Dermatol.* 2012;166:844–852.
41. Bavleen K, Jenna K, Jia K, Manreet K, Stefanos K, Nicola C. Mechanism-based therapeutic targets. *Exp Dermatol.* 2022;31:154–171.
42. Pierpont TM, Limper CB, Richards KL. Rituximab therapy review. *Front Oncol.* 2018;8:163.
43. Didona D, Paolino G, Di Zenzo G, Didona B, Pampena R, Di Nicola MR, Mercuri SR. Therapeutic strategies in pemphigus vulgaris. *Dermatol Pract Concept.* 2022;12:e2022037.
44. Kasi PM, Tawbi HA, Oddis CV, Kulkarni HS. Adverse events of rituximab. *Crit Care.* 2012;16:231.
45. Mielnik P, Sexton J, Lie E, Bakland G, Loli LP, Kristianslund EK, et al. Rituximab safety in older age. *Drugs Aging.* 2020;37:617–626.
46. Bhandari PR, Pai VV. Novel applications of rituximab. *Indian Dermatol Online J.* 2014;5:250–256.
47. Penha MA, Farat JG, Miot HA, Barraviera SR. Quality of life in bullous dermatosis. *An Bras Dermatol.* 2015;90:190–194.
48. Sajedianfard S, Handjani F, Saki N, Heiran A. Family dermatology life quality index in pemphigus. *Indian J Dermatol Venereol Leprol.*

2021;87:375–378.

49. S P, N S, J, Y V. Managing challenging cases of pemphigus vulgaris. *Int J Res Dermatol.* 2019;5:434–436.
50. Pasricha JS, Khaitan BK, Raman RS, Chandra M. DCP therapy for pemphigus. *Int J Dermatol.* 1995;34:875–882.
51. Khandpur S, Sharma P, Sharma VK, Das D, Sharma A, Bhari N, Sreenivas V. Rituximab vs DCP therapy comparative study. *Indian Dermatol Online J.* 2024;15(3):464–47.
52. Ahmed AR, Shetty S. Treatment outcomes with rituximab in pemphigus vulgaris. *Autoimmun Rev.* 2015;14:323–331.