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Dexamethasone Pulse Therapy in Pemphigus Vulgaris Associated Myocardial Infarction in Resource-Limited Settings

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Abstract

Pemphigus vulgaris is an autoimmune blistering disorder where biologics are the cornerstone of therapy. In resource-limited settings, a dermatologist should rely on systemic steroids. Dexamethasone pulse therapy is a cheap and established treatment of pemphigus where a supra-pharmacological dose of dexamethasone is used. High dose of dexamethasone is supposed to have atherogenic potential which could promote a hypercoagulable state. Here, we report a case of a 30-year-old woman in the resource-limited setting of Nepal, who was under monthly dexamethasone pulse therapy for pemphigus vulgaris, who had an acute episode of ST-segment elevated myocardial infarction. In such setting, although steroids are the cornerstone of therapy in various disorders, physicians should be equally cautious in using a high dose of steroids which might lead to fatal complications. This could probably be the first case report of steroid-induced acute myocardial infarction during treatment of pemphigus worldwide.

Key words: Dexamethasone; Myocardial Infarction; Pemphigus vulgaris; Pulse therapy.

Dear Editor,

Pemphigus vulgaris (PV) is an immune-bullous disorder where autoantibodies are directed against cell adhesion molecules, desmogleins. Rituximab is considered as the first line option in the management of PV.¹ Recently, these drugs are being sparingly available in Nepal but have limited availability at many centers and are not affordable because of its cost effectiveness. Thus, many dermatologists should depend on systemic steroids in treatment of PV. Intravenous dexamethasone pulse (DP) therapy is one of the better modalities for its remission in the long run but is associated with various risks and adverse outcomes.² We report a case of myocardial infarction associated with DP used in the management of PV which could be the first case report from Nepal.

A 30-year-old woman with severe muco-cutaneous pemphigus vulgaris of three months duration visited to our clinic for a third cycle of dexamethasone azathioprine pulse which consisted of an infusion of dexamethasone i.e. 100 mg in 5% dextrose over three consecutive days and daily dose of azathioprine 100mg in a month. She had no known comorbidities. The basic investigations like complete blood count, blood sugar, liver and renal function test, and lipid profile was normal. The baseline ECG was normal. The histopathologic findings comprised of intra-epidermal bulla, acantholytic cells, and suprabasal splitting. (Figure 1)

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During the third monthly pulse of dexamethasone, on the second day, after completion of the infusion, she had a sudden onset of chest pain, shortness of breath, and sweating. ECG showed features of inferior wall myocardial infarction, (Figure: 2) which was supported by positive troponin I, high creatinine phosphokinase MB. Patient was managed conservatively by aspirin (300mg), clopidogrel (300mg), Inj. morphine (5mg), and heparin drip (5000U bolus) in addition to atorvastatin (10mg), who later was handed over to cardiology team.

Because of the unavailability of Rituximab in many settings, steroids are a cornerstone of therapy. However, steroids are associated with adverse outcomes like change in vasculopathy and atherogenesis, which can lead to ischaemic heart disease. It has been found that dexamethasone is a promoter of an atherogenic potential as it increases Von Willebrand Factor and P-selectin.³ The cardiac ischemia due to steroids could be explained by a decrease in nitrate, nitrite, and nitric oxide due to calcium mobilization and downregulation of cyclooxygenases leading to increase thromboxane and decrease prostacyclin.⁴ Many researchers found that glucocorticoids for longer duration and high doses are associated have greater risk.⁵ As Rituximab is first line treatment of PV, its widespread availability would help in effective management of PV and many potential fatal side effects of systemic corticosteroids could be minimized. Thus, during use of systemic corticosteroids, physicians should be aware of its possible severe outcomes.

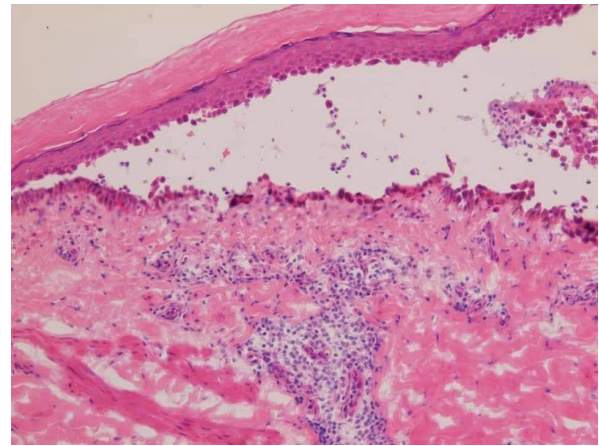


Figure 1: Histopathological section showing acantholytic cells and intraepidermal bullae with suprabasal split. (Magnification 10X, with Hematoxylin and Eosin staining)

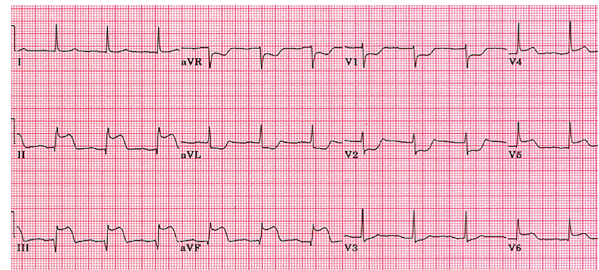


Figure 2: The ECG showing ST elevation in the inferior leads i.e. II, aVf and III and reciprocal ST depression in V1 and V2

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