International Journal of Research in Medical Sciences
Gopi M et al. Int J Res Med Sci. 2023 Jan;11(1):xxx-xxx
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20223306

Case Report

Topical steroid induced Cushing's syndrome-a rare entity

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Received: 20 November 2022
Accepted: 09 December 2022

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ABSTRACT

The topical steroid therapy is the commonest mode of treatment for Psoriasis. When the therapy is overused, it can cause unusual side effects like Cushing's syndrome which is very rarely reported and we are reporting such a case. Treatment with maintenance dose of steroids is necessary based on basal cortisol levels to avert hypocortisolemic crisis in Cushing's syndrome, however in some select asymptomatic cases maintenance dose of steroids can be withdrawn and only stress dose be given in spite of low basal serum cortisol. We managed such a case and it is reported because of its rarity in clinical practice. This can alert the clinicians to avoid prescribing steroids as maintenance dose for a long time in asymptomatic Cushing's syndrome with low morning cortisol and thereby reducing the side effects of long-term steroids on them.

Keywords: Topical steroid, Psoriasis, Cushings syndrome

INTRODUCTION

Psoriasis is one of chronic inflammatory diseases affecting 1-3% of total grand population.[1] Introduction of topical steroid has revolutionized treatment of psoriasis. However topical corticosteroid is double-edged sword and unfortunately it is available over the counter and tends to be overused by patients who find benefit. When used for prolonged periods of time in large quantities in extensive skin disease systemic adverse effects including iatrogenic Cushing's and suppression of hypothalamic pituitary adrenal axis can occur though rare. Systemic side effects of topical corticosteroids are more common in children than in adults. We report case of adult male who had extensive psoriasis who presented with iatrogenic Cushing's due to prolonged use of large quantities of topical clobetasol and prolonged suppression of HPA axis after withdrawal of topical steroid.

CASE REPORT

A 48-year-old male has come with fever, tender swelling of left lower limb and painful inguinal adenitis. Clinical examination showed features of streptococcal cellulitis of left lower limb with tender left inguinal lymphadenoapathy. He reported recurrent similar episodes of streptococcal cellulites since one year. Impaired glucose tolerance (IGT) was detected 3 years ago and he was managed with only life style measures. He had psoriatic lesions all over the body (Figure 1) since 20 years. He had been advised topical clobetasol ointment for the lesions 20 years ago when he had only a few lesions. However, he developed extensive skin lesions and started using this preparation on his own for the lesions distributed all over his body. He was using one 20gm tube of clobetasol in 4 days for the last several years.

He developed striking Cushingoid features with purple striae over the abdomen (Figure 2) and in the axilla since 4 months and gained a lot of weight. The onset of his impaired fasting glucose was after he started using large quantities of clobetasol cream. He had a mild hypertension (BP 150/100). Fundus examination revealed a posterior polar cataract consistent with the steroid induced cataract.

His cellulitis was managed with parenteral Antibiotics and it resolved promptly. His 8 AM serum cortisol was 0.03 ug/dl (Normal 5-14 ug/dl) indicating pronounced suppression of his hypothalamic pituitary adrenal (HPA) axis. Patient denied history of oral or injectable corticosteroid use. The only corticosteroid he was getting was the topical steroid cream. The topical clobetasol was stopped and in order to prevent hypoadrenal crisis, he was started on replacement doses of oral hydrocortisone (5-2.5-2.5 mg/day) with a view to tapering it off. His hypertension was controlled with telmisartan.

For his extensive psoriasis he was started on methotrexate orally once in a week and folic acid. He was advised oral penicillin long-term as prophylaxis against recurrent streptococcal infection.

On follow up after 6 months hydrocortisone was discontinued gradually and even though his 8 AM cortisol was low, he did not have any symptoms of hypocortisolemia. He was therefore advised only hydrocortisone cover for acute severe illness. He developed 1 episode of diarrhoeal illness a year later for which he needed IV hydrocortisone for 48 hours. After withdrawal of steroids his weight decreased from 97 kg to 85 kg over the next 2 years. His blood sugars remained normal. He did not have any recurrence of the cellulitis. The psoriatic lesions have mostly disappeared and the striae and other Cushingoid features have regressed.

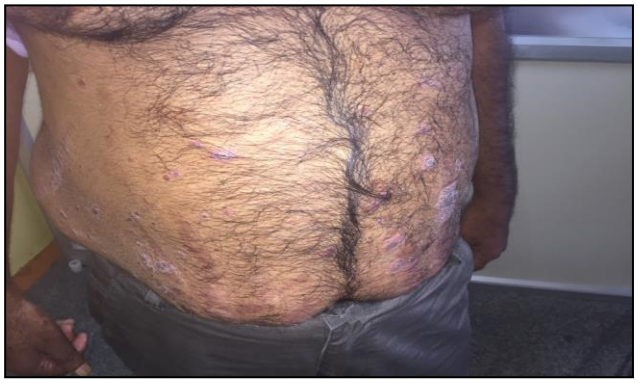

Figure 1: Psoriatic lesions over the abdomen.

Figure 2: Thick purplish striae over the abdomen suggestive of Cushing's syndrome.

DISCUSSION

Cushing's syndrome refers to groups of signs and symptoms developed as a result of either endogenous cortisol overproduction or exogenous steroids supplementation.[2] These includes hypertension, weight gain, buffalo hump, moon face, proximal muscle weakness, wide purple striae, recurrent infections, thromboembolic disease and cardiovascular disease.[2]

The most common form of Cushing's syndrome in clinical practice is iatrogenic Cushing's syndrome.[3] The severity of iatrogenic Cushing's syndrome depends on the dose, potency, duration of the corticosteroid used.[4]

Most commonly iatrogenic Cushing's is due to oral or parenteral corticosteroid therapy over prolonged periods of time in asthma arthritis or skin problems. Topical corticosteroids when used in large doses for prolonged periods of time can also cause iatrogenic Cushing's as in our patient. Topical steroids tend to get absorbed more readily if they are applied on denuded skin or applied over the groins or armpits or if they are used with an occlusive dressing.[5] Iatrogenic Cushing's due to topical steroids is more common in children than in adults, as children have a thinner dermis layer and the ratio of body surface to volume is greater in children.[5]

Treatment with maintenance doses of glucocorticoids is necessary in iatrogenic Cushing's syndrome in order to avert a hypocortisolemic crisis. Periodically the oral drug should be discontinued for about 48 hours and 8 AM cortisol measured.[6] If the 8 AM cortisol is >10 ug/dl, it implies that the HPA axis has recovered.[6] However a value <3 ug/dl indicates the need for continuing oral steroid replacement.[6] Values between 3 and 10 ug/dl indicate the need for a Synacthen stimulation test to assess recovery. If after IV synacthen the cortisol after 1 hour is >20 ug/dl it implies that the HPA axis has recovered. However, Synacthen is not freely available in India and so this approach is difficult and one has to go by the clinical symptomatology and the basal cortisol levels. In our patient after withdrawal of hydrocortisone, there were no symptoms to suggest a hypocortisolemic state and he continued to require a high dose of telmisartan for controlling his hypetenison even though his 8 AM cortisol was 0.34 ug/dl. Therefore, we opted to use only hydrocortisone cover for stressful situations. Even after 2 years of steroid withdrawal he continues to have an 8 AM cortisol of 1.7 ug/dl which is higher than his initial values but lower than the prescribed cut-off for oral steroid replacement. However, he has not experienced frequent hypocortisolemic crisis. These findings suggest that in select asymptomatic subjects with low 8 AM cortisol values after steroid withdrawal, hydrocortisone cover for stress may suffice and daily glucocorticoid replacement with its attendant complications such as (exacerbation of diabetes and hypertension) may not be warranted.

CONCLUSION

Large doses of topical steroids applied over extensive areas of skin can cause iatrogenic Cushing's syndrome. Patients should be strictly advised to use the topical steroid in the prescribed strength and for the prescribed duration and should not buy topical steroids over the counter and use them indiscriminately. Tight regulation of topical steroid preparations (only on prescription) is mandatory. After withdrawal lifelong follow-up is mandatory. Patients should be educated about the potential for hypocortisolemic crisis and should be advised to seek early hospitalization for IV hydrocortisone in the event of an acute illness. Patients should also carry a card or bracelet indicating this requirement so that there is no undue delay in initiation of parenteral glucocorticoid when required in a crisis situation. In select patients such as ours the decision to use maintenance doses of glucocorticoids should be individualized and in the absence of symptoms it may be justified to offer only hydrocortisone cover in situations of stress. Lifelong follow-up of patients with iatrogenic Cushing's is mandatory.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Gopi M, Seshadri MS. Topical steroid induced Cushing's syndrome-a rare entity. Int J Res Med Sci 2023;11:xxx-xx.