Case Report

Unregulated topical steroid use in India: From skin relief to life-threatening complications

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ABSTRACT

Unregulated and prolonged use of corticosteroids, both topical and oral, remains a significant public health concern in India, resulting in severe iatrogenic complications. We report a 35-year-old male who developed iatrogenic Cushing's syndrome, steroid-induced diabetes mellitus, and pulmonary aspergilloma within an old tuberculous cavity following prolonged unsupervised use of a high-potency topical steroid (likely clobetasol-based) for widespread eczema. This case highlights the complex interplay of endocrine disruption, metabolic derangements, and immunosuppression leading to opportunistic fungal colonization in structurally damaged lungs. Strict regulation of over-the-counter sales of potent corticosteroids, enhanced public awareness, and improved oversight of healthcare practices are urgently needed to prevent such avoidable morbidity.

Key words: Corticosteroids, Iatrogenic Cushing's syndrome, Post-tuberculous cavity, Pulmonary aspergilloma, Steroid-induced diabetes mellitus, Topical steroid misuse

Iatrogenic Cushing's syndrome secondary to exogenous glucocorticoids is well-documented, yet cases arising from unregulated self-medication or unsupervised use of potent topical and oral corticosteroids continue to occur frequently in India due to easy over-the-counter availability [1,2]. Super-potent topical agents, such as clobetasol propionate and oral preparations, such as prednisolone, when used indiscriminately and without monitoring, can cause significant systemic absorption, hypothalamic-pituitary-adrenal (HPA) axis suppression, metabolic complications, and increased susceptibility to opportunistic infections.

While most reported cases of topical steroid-induced iatrogenic Cushing's syndrome involve infants and children, adult presentations, though less common, are increasingly recognized, particularly with widespread application over large body surface areas [3]. Residual structural lung damage from prior pulmonary tuberculosis (TB), especially cavitary lesions, predisposes individuals to *Aspergillus* colonization and chronic pulmonary aspergillosis (CPA), including aspergilloma. Exogenous corticosteroids further exacerbate this risk through local and systemic immunosuppression [4].

This case illustrates how a seemingly benign dermatological condition can escalate into multisystem, life-threatening complications due to unregulated steroid use. This case is being reported because it represents a rare adult presentation of the dangerous triad of iatrogenic Cushing's syndrome, steroid-induced diabetes mellitus, and pulmonary aspergilloma complicating a post-tuberculous cavity, all stemming from unsupervised high-potency topical corticosteroid misuse. It highlights critical gaps in the regulation of over-the-counter potent steroids in India and the need for heightened awareness among practitioners and the public to prevent such avoidable, multisystem iatrogenic harm. In addition, it provides valuable insight into the diagnostic differentiation of CPA from allergic bronchopulmonary aspergillosis (ABPA) in corticosteroid-immunosuppressed patients with structural lung disease.

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CASE PRESENTATION

A 35-year-old Indian male presented to the emergency department with progressive shortness of breath, productive cough, anasarca, and chest heaviness for 7 days. There was no associated fever, hemoptysis, orthopnea, paroxysmal nocturnal dyspnea, or urinary complaints. He had completed a 6-month course of anti-tubercular therapy 10 years previously for pulmonary TB. Two years prior, he developed eczematous lesions on the abdomen and sought care from a registered medical practitioner. He received irregular, prolonged, and unsupervised applications of high-potency topical corticosteroids (presumably clobetasol-based) due to self-administration and failure to follow-up, leading to progressive generalization of lesions across the body. Approximately 1–1.5 years ago, he was diagnosed with diabetes mellitus and initiated on oral hypoglycemic agents.

On examination, he exhibited classic cushingoid features: Moon face, plethoric facies (Fig. 1a), violaceous abdominal striae (Fig. 1b), proximal muscle wasting with thin extremities (lemon-on-a-stick appearance)(Fig. 1c), and steroid-induced cutaneous atrophy with widespread eczematous dermatitis compared to previous clinical photographs (Fig 1d). He was tachypneic (respiratory rate 28 breaths/min), hypoxic (SpO_2 88% on room air), and required supplemental oxygen. Anasarca was evident, with coarse crepitations over the left lung fields.

Laboratory findings included: Hemoglobin 10.7 g/dL, total leukocyte count 9620 cells/mm^3, platelets 212,000 cells/mm^3, and normal renal and liver function tests. Serology was non-reactive for human immunodeficiency virus, hepatitis B surface antigen, and hepatitis C virus. Electrocardiogram showed normal sinus rhythm with troponin I 0.01 ng/mL. Hemoglobin A1c (HbA1c) was 9.8%, random blood glucose 312 mg/dL, urine glucose 2+, and urine ketones 2+. Morning serum cortisol was markedly suppressed at 2.23 µg/dL. Chest X-ray revealed a mass-like opacity in the left upper zone (Fig. 2a). High-resolution computed tomography (HRCT) (Fig. 2b) of the chest demonstrated a 3.5 × 3 cm fungal ball (aspergilloma) within an old tuberculous cavity in the left upper lobe, surrounded by fibrosis. Raised Serum total immunoglobulin E (IgE) was >2500 IU/mL, *Aspergillus fumigatus*-specific IgE 18.31 IU/mL (elevated), and *Aspergillus* IgG 0.46 NTU (borderline). Absolute eosinophil count was 72/mm^3 (normal). The HRCT demonstrated a classic fungal ball within the old tuberculous cavity, consistent with pulmonary aspergilloma (a form of CPA). This was distinguished from ABPA by normal eosinophil count (72/mm^3), absence of asthma/cystic fibrosis history, lack of fleeting infiltrates or proximal bronchiectasis, and failure to meet full diagnostic criteria (predisposing condition + total IgE typically >1000 IU/mL + sensitization + eosinophilia or characteristic radiology per ISHAM criteria) [5,6].

Serological findings indicated *Aspergillus* sensitization in the context of structural lung damage and immunosuppression, supporting the diagnosis of aspergilloma rather than ABPA or invasive aspergillosis. Dermatology consultation confirmed steroid-induced skin atrophy and eczematous dermatitis. Dual-energy X-ray absorptiometry scan revealed osteopenia at anteroposterior spine site, L1-L4 region, with a Z score of 1.1 (Fig. 2c).

The patient received supportive oxygen therapy, intravenous fluids, and initial broad-spectrum antibiotics. Glycemic control was optimized by transitioning to intermediate-acting insulin along with oral hypoglycemic agents. Antifungal therapy with itraconazole was commenced for aspergilloma. Dermatological management included oral terbinafine, levocetirizine, ciclopirox olamine cream, and emollients. To prevent adrenal crisis during HPA axis recovery, oral prednisolone was continued with a gradual taper starting at 40 mg/day. Close monitoring for potential

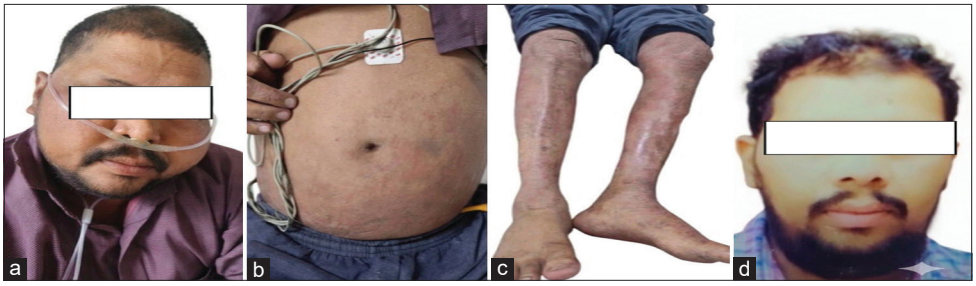

Figure 1: (a-d) The classic cushingoid features of the patient

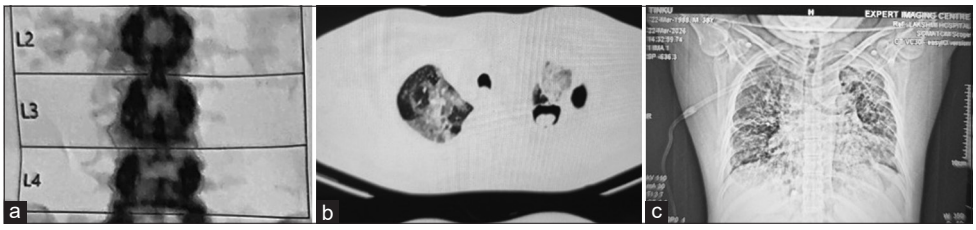

Figure 2: (a) Chest X-ray showing a mass-like opacity in the left upper zone; (b) high-resolution computed tomography of the chest; (c) dual-energy X-ray absorptiometry scan

pharmacokinetic interaction between itraconazole (a potent CYP3A4 inhibitor) and prednisolone was performed through clinical assessment, electrolytes, and glucose levels [5].

The patient showed gradual improvement with resolution of respiratory symptoms and better glycemic control. No hemoptysis occurred during hospitalization. Cushingoid features (moon face, plethora) showed early improvement with the steroid taper by discharge. Random blood glucose improved to 168 mg/dL by day 5 with resolution of ketonuria on optimized insulin and oral agents. Although repeat HbA1c was not performed during the brief admission, it was scheduled for a 3-month endocrinology follow-up to assess long-term glycemic control. Full resolution of cushingoid habitus and HPA axis recovery typically requires 4–12 weeks or longer after complete steroid withdrawal and was discussed with the patient, who was advised close monitoring and gradual taper under supervision to avoid adrenal crisis. The timeline of events is shown in Table 1.

DISCUSSION

This case exemplifies the multisystem sequelae of unregulated corticosteroid misuse in India, a growing public health concern particularly in dermatology and now increasingly recognized in internal medicine and pulmonology. Prolonged unsupervised use of potent topical agents, such as clobetasol over large body surface areas led to significant systemic absorption, resulting in full-blown iatrogenic Cushing's syndrome (moon face, violaceous striae, proximal myopathy, skin atrophy, suppressed cortisol) [1,3], new or worsened steroid-induced diabetes mellitus requiring insulin, and immunosuppression that facilitated *Aspergillus* colonization of a pre-existing post-TB cavity, culminating in pulmonary aspergilloma [4].

Prolonged high-potency topical steroid application resulted in Iatrogenic Cushing's syndrome, evidenced by moon face, violaceous striae, proximal myopathy, skin atrophy, and suppressed morning cortisol [1,3]. Steroid-induced diabetes mellitus with poor control requiring insulin; immunosuppression facilitating *Aspergillus* colonization of a pre-existing post-tuberculous cavity, culminating in aspergilloma with serological evidence of sensitization [4].

Table 1: Timeline of events

Time period	Event
10 years ago	Completed anti-tubercular therapy for pulmonary tuberculosis
2 years ago	Onset of eczematous lesions; initiated high-potency topical steroid
1–1.5 years ago	Diagnosed with diabetes mellitus; started oral antidiabetic agents
Current presentation	Dyspnea, cough, anasarca; diagnosed with iatrogenic Cushing's syndrome, poorly controlled diabetes, and pulmonary aspergilloma

Post-TB lung damage provides an ideal nidus for *Aspergillus* species. Exogenous glucocorticoids impair immune surveillance, promoting progression to CPA forms, including simple aspergilloma [7]. We have clarified the diagnosis as pulmonary aspergilloma (simple CPA) based on characteristic HRCT fungal ball in a stable cavity, positive serology (elevated specific IgE indicating sensitization, borderline IgG), in the absence of eosinophilia or other ABPA criteria (no asthma, no eosinophilia >500/µL, no fleeting infiltrates). This distinguishes it from ABPA (hypersensitivity disorder) and chronic cavitary pulmonary aspergillosis (which shows progressive cavitation/fibrosis) [4]. Similar cases of topical steroid abuse leading to systemic Cushing's syndrome and secondary infections are increasingly reported from India, though adult cases with pulmonary complications remain noteworthy and underscore the need for vigilance [2,3]. Potent agents, such as clobetasol can achieve significant systemic absorption, especially over extensive areas or with prolonged use, as seen in our patient who applied it irregularly for ~2 years [8].

Management posed challenges in balancing gradual steroid taper (to restore endogenous adrenal function) against antifungal therapy with itraconazole, given the significant CYP3A4-mediated pharmacokinetic interaction that can increase prednisolone exposure by 2–3 fold, potentially worsening iatrogenic Cushing's or risking adrenal crisis during taper [9,10]. Close monitoring of electrolytes, glucose, and clinical status was essential, as performed. Multidisciplinary involvement of internal medicine, dermatology, and endocrinology was crucial for optimal outcomes. The early improvement in cushingoid features and glycemic control during admission supports the reversibility of many complications with timely intervention, although full HPA axis recovery and resolution of skin/muscle changes may take several months. This case reinforces the importance of patient education on steroid risks, pharmacist oversight, and regulatory measures to curb over-the-counter sales of super-potent topical corticosteroids in India [6].

CONCLUSION

Unregulated patient-driven, unmonitored use of potent corticosteroids can transform a common dermatological condition into severe, multisystem complications involving endocrine, metabolic, and pulmonary systems. This case underscores the critical need for strict regulation and enforcement prohibiting over-the-counter dispensing of potent topical and oral corticosteroids; widespread public awareness campaigns targeting patients, pharmacists, and general practitioners; mandatory training and oversight for rural and unqualified medical practitioners; early recognition and cautious, monitored withdrawal of steroids in iatrogenic cases. Robust public health interventions are essential to curb this preventable epidemic in low- and middle-income settings, such as India.

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