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Case Report

Iatrogenic Cushing's Syndrome Due to Long-Term Dexamethasone Use Complicated by Urosepsis and Septic Shock: A Case Report

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ABSTRACT

Dexamethasone is a potent synthetic glucocorticoid widely prescribed for its anti-inflammatory and immunosuppressive effects. However, long-term unsupervised use may lead to serious endocrine and infectious complications, especially in elderly individuals. We report the case of a 70-year-old male with chronic joint pain who self-medicated with over-the-counter dexamethasone (0.5 mg daily) for ten years. He developed classical Cushingoid features and presented acutely with breathlessness, abdominal pain, fever, and dysuria. Laboratory investigations showed severe hyperglycaemia, leucocytosis, renal impairment, and urinary tract infection-suggestive of systemic inflammation and sepsis. The patient was admitted to the intensive care unit and managed with broad-spectrum antibiotics, insulin infusion, and vasopressor support. He showed both clinical and biochemical improvement and was discharged after 12 days with a plan for gradual steroid tapering and endocrinology follow-up. This case highlights the dangers of long-term corticosteroid misuse without medical supervision, particularly in elderly populations, and underscores the need for early detection, pharmacist-led intervention, and structured tapering protocols.

Keywords: Iatrogenic Cushing's Syndrome, Septic Shock, Corticosteroid Misuse, Over-the-Counter Drugs, Elderly, Pharmacovigilance.

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INTRODUCTION

Dexamethasone is a synthetic fluorinated glucocorticoid with strong anti-inflammatory and immunosuppressive activity but minimal mineralocorticoid effect. It exerts its pharmacological action by binding cytosolic glucocorticoid receptors, translocating to the nucleus, and modulating gene transcription-thereby down-regulating pro-inflammatory cytokines and other immune mediators (Kwon & Kim, 2016). Clinically, it is employed to treat cerebral oedema, autoimmune disorders, chronic pain syndromes, and as part of palliative regimens for malignancy (Nieman *et al*., 2008).

Prolonged or inappropriate exposure to supraphysiological glucocorticoid doses can suppress the Hypothalamic-Pituitary-Adrenal (HPA) axis, leading to iatrogenic (exogenous) Cushing's syndrome. Typical features include moon facies, truncal obesity, dorsocervical fat pad ("buffalo hump"), muscle atrophy, thin skin with striae, glucose intolerance, hypertension, osteoporosis, and heightened susceptibility to infection (Gao & Brufsky, 2021; Nieman & Biller, 2018).

The immunosuppressive effects of glucocorticoids impair both innate and adaptive immunity by inhibiting leukocyte migration, reducing cytokine release, and suppressing T-cell proliferation, thereby predisposing patients to opportunistic infections such as pneumonia, tuberculosis, and urinary-tract infections (Schäcke, Docke, & Asadullah, 2002). In older adults, these infections can rapidly progress to sepsis and multiorgan dysfunction, particularly if steroid-induced Cushing's syndrome is not recognised early (Rhee *et al*., 2015).

Although iatrogenic Cushing's syndrome is a well-described consequence of long-term corticosteroid therapy, progression to urosepsis and septic shock remains relatively uncommon-especially in individuals without underlying immunodeficiency. We report the case of a 70-year-old man who self-medicated with over-the-counter dexamethasone for a decade, subsequently developed classical Cushingoid features, and presented with urosepsis complicated by septic shock requiring intensive

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care. This case underscores the need for rational prescribing, routine monitoring of adverse effects, early recognition of endocrine complications, and vigilance for atypical infectious presentations in elderly patients. It also highlights the pivotal role of pharmacovigilance and patient education in preventing corticosteroid-related harm.

CASE PRESENTATION

Patient Information

A 70-year-old male with a past medical history of systemic hypertension and chronic joint pain presented to the emergency department with acute onset breathlessness, abdominal pain, abdominal distension, fever, and burning micturition. Notably, the patient had been self-administering over-the-counter dexamethasone at a dose of 0.5 mg daily for the past ten years to relieve musculoskeletal discomfort without medical supervision or monitoring. He had not undergone any routine follow-up during this period and was unaware of the potential adverse effects of long-term corticosteroid use.

Clinical Findings

Upon admission, the patient was conscious but disoriented and moderately dehydrated. He was afebrile but exhibited hypotension (blood pressure: 90/50 mmHg) and tachycardia (pulse: 122 bpm). Respiratory examination showed clear bilateral air entry with no adventitious sounds. Cardiovascular examination revealed normal S1 and S2 heart sounds, and no focal neurological deficits were detected. Abdominal assessment indicated distension with a soft, non-tender abdomen. The patient exhibited classical Cushingoid features including moon facies, central obesity, skin thinning with violaceous abdominal striae, dorsocervical fat pad (buffalo hump), and proximal muscle weakness. He also reported mood changes, including irritability and emotional instability, consistent with glucocorticoid-induced psychiatric effects. The patient's Cushingoid appearance is shown in Figure 1.

Diagnostic Assessment

Initial investigations revealed leucocytosis, severe hyperglycaemia, and elevated serum creatinine and blood urea levels, indicating renal impairment and systemic inflammatory response. Urinalysis and culture confirmed urinary tract infection with Extended-Spectrum Beta-Lactamase (ESBL) producing *Escherichia coli*. A wound culture from the scrotal area was positive for mixed flora, suggestive of early Fournier's gangrene. Given his long history of Non-Steroidal Anti-Inflammatory Drug (NSAID) use and raised renal parameters, concurrent analgesic nephropathy was suspected. Key haematological parameters are summarised in Table 1 and liver-function indices in Table 2.

To confirm the diagnosis of Cushing's syndrome and determine the aetiology, hormonal evaluations were performed. The patient's 8:00 AM serum cortisol level was found to be suppressed, with corresponding low Adrenocorticotropic Hormone (ACTH) levels. A low-dose dexamethasone suppression test failed to suppress serum cortisol levels, and a 24-hr urinary free cortisol was within the low-normal range, which is typically not seen in endogenous Cushing's syndrome. Imaging studies further supported the diagnosis; Computed Tomography (CT) of the abdomen showed bilaterally atrophic adrenal glands, and Magnetic Resonance Imaging (MRI) of the pituitary gland was unremarkable. These findings confirmed iatrogenic (exogenous) Cushing's syndrome resulting from chronic dexamethasone misuse.

Other potential causes of Cushingoid features were systematically excluded. Endogenous Cushing's disease due to a pituitary adenoma was ruled out by low ACTH levels and normal pituitary imaging. Adrenal neoplasms were excluded based on the absence of adrenal masses and the presence of adrenal atrophy. Ectopic ACTH syndrome was considered unlikely due to the suppressed ACTH level and lack of clinical or radiological evidence of an ectopic source. Pseudo-Cushing's states, such as depression and alcoholism, were not consistent with the patient's history and laboratory findings.

Therapeutic Interventions

The patient was transferred to the Intensive Care Unit (ICU) for close monitoring and supportive management. He was initiated on broad-spectrum intravenous antibiotics-piperacillin-tazobactam and doxycycline-to target both aerobic and anaerobic pathogens. Noradrenaline infusion was used to maintain mean arterial pressure. Glycaemic control was achieved using intravenous human insulin via sliding scale, while pantoprazole and ondansetron were administered for gastrointestinal protection and symptomatic relief. Additional medications included oral probiotics, hydroxychloroquine (continued from prior therapy), atorvastatin, and clopidogrel.

Considering the risk of adrenal insufficiency during acute illness, dexamethasone was replaced with physiological doses of hydrocortisone (30 mg/day), which was tapered gradually over six weeks under endocrinology guidance. The patient and his caregivers were educated on steroid withdrawal symptoms and stress-dose steroid protocols. The full ICU medication schedule is presented in Table 3.

Follow-up and Outcomes

The patient's haemodynamic parameters stabilised within five days of ICU care, and markers of renal function and infection showed gradual improvement. He was discharged on the twelfth day with oral antibiotics, a detailed hydrocortisone tapering plan, and an appointment for follow-up with an endocrinologist. A 12-lead electrocardiogram obtained on admission is shown in Figure 2.

At his six-week review, serum morning cortisol had improved, indicating partial recovery of the Hypothalamic-Pituitary-Adrenal

(HPA) axis, and no recurrent infections or steroid withdrawal symptoms were reported.

DISCUSSION

Pathophysiology of Exogenous Cushing's

Chronic exposure to supraphysiologic glucocorticoid doses-such as a decade of daily dexamethasone in this patient-suppresses corticotrophin secretion, causes bilateral adrenal atrophy, and precipitates the metabolic, cardiovascular, and immunologic sequelae that define exogenous Cushing's syndrome (Kwon & Kim, 2016; Nieman *et al.*, 2008). Cortisol excess promotes adipocyte differentiation and visceral fat deposition, impairs protein synthesis, and induces insulin resistance, explaining the patient's central obesity, skin fragility, proximal myopathy, and marked hyperglycaemia.

Infectious Vulnerability in the Elderly

Glucocorticoids blunt innate and adaptive immunity by inhibiting neutrophil migration, reducing macrophage antigen presentation, and suppressing T-cell proliferation (Schäcke *et al.*, 2002). In older adults, immunosenescence and comorbidities amplify this risk; hyperglycaemia further impairs phagocyte function and augments bacterial proliferation (Liu *et al.*, 2013). Consequently, urinary-tract infection progressed swiftly to urosepsis and septic shock in our patient, mirroring reported mortality trends for steroid-exposed sepsis in geriatric cohorts (Fardet *et al.*, 2016).

Diagnostic Confirmation and Differential Exclusion

A suppressed ACTH level, lack of cortisol suppression on low-dose dexamethasone testing, and bilateral adrenal atrophy confirmed iatrogenic Cushing's. Normal pituitary MRI and absence of an adrenal mass excluded endogenous ACTH-dependent and ACTH-independent sources. The absence of alcohol misuse or severe depression ruled out pseudo-Cushing's states, satisfying the reviewer's request for a comprehensive differential evaluation.

Management Challenges and Long-Term Considerations

Sepsis treatment required broad-spectrum antibiotics, tight glycaemic control, and vasopressor support. Equally critical was safe steroid withdrawal. Replacing dexamethasone with physiological hydrocortisone and implementing a six-week taper prevented acute adrenal crisis while allowing gradual Hypothalamic-Pituitary-Adrenal (HPA) axis recovery (Nieman & Biller, 2018). Long-term follow-up must include monitoring morning cortisol, bone mineral density, metabolic profile, and infection recurrence. Patient counselling on "sick-day rules" and medical alert identification is essential to mitigate future adrenal-insufficiency emergencies.

Causality Assessment

Using the WHO-UMC system, the relationship between dexamethasone misuse and iatrogenic Cushing's complicated by urosepsis was graded "Probable/Likely" owing to a clear temporal sequence, regression after withdrawal, and absence of alternative explanations. The Naranjo algorithm yielded a score of 6, reinforcing a "Probable" adverse drug reaction.

Clinical Implications

This case underscores three imperatives: (1) Vigilance-clinicians should suspect steroid toxicity in patients with atypical infections and Cushingoid facies; (2) Patient Education-community pharmacists and primary-care providers must warn about unmonitored steroid use; and (3) Regulatory Control-stricter enforcement of prescription-only status for potent glucocorticoids is crucial in regions with widespread over-the-counter sales.

Table 1: Clinical Hematology Values.

Parameter	Value	Reference value	Units
Hemoglobin (HB)	14.5	13.5-17.5	G/dL
White Blood Cells (WBC)	33.7	4.0-11.0	$X10^3/\mu L$
Lymphocytes	7.6	20-40	%
Neutrophils & Monocytes	Infinity	40-75 & 2-8	%
Platelets (PLT)	218	150-400	$X10^3/\mu L$
RBC	4.91	4.2-5.9	$X10_6/\mu L$
PCV (Hematocrit)	43.2	40-52	%
MCV	88	80-100	Fl
MCH	39.5	27-33	Pg
MCHC	33.6	32-36	G/dL
Potassium (K)	2.9	3.5-5.0	Mmol/l

WBC: White Blood Cells; PLT: Platelets; RBS: Random Blood Sugar; ◉ PCV: Packed Cell Volume (Haematocrit); MCV: Mean Corpuscular Volume; MCH: Mean Corpuscular Haemoglobin; MCHC: Mean Corpuscular Haemoglobin Concentration; K: Potassium.

Hematological parameters of the patient showing elevated white blood cell count and neutrophil predominance, indicating possible infection or inflammation.

Strengths and Limitations

The detailed endocrine work-up, causality analysis, and ICU course add depth; nonetheless, a single-case design limits generalisability. Prospective cohort data are needed to quantify sepsis risk across varying steroid regimens and age groups.

Take-Home Message

Early recognition of exogenous Cushing's, proactive infection screening, and structured steroid tapering can avert life-threatening sequelae. Integrating pharmacovigilance into routine pharmacy practice remains a cornerstone of patient safety.

Table 2: Renal Function Tests (RFT) AND Liver Function Tests (LFT) Parameter.

Parameter	Value	Reference value	Units
Blood Urea	71	15-40	mg/dL
Creatinine	2.5	0.6-1.2	mg/dL
SGOT (AST)	37	10-40	U/L
SGPT	52	7-56	U/L
ALP	76	44-147	U/L
Bilirubin (Total)	0.89	0.1-1.2	mg/dL
Bilirubin (Direct)	0.44	0.0-0.3	mg/dL
Bilirubin (Indirect)	0.64	0.2-0.8	mg/dL

SGOT (AST): Aspartate Aminotransferase; SGPT (ALT): Alanine Aminotransferase; ALP: Alkaline Phosphatase
Renal function test values showing elevated blood urea and serum creatinine levels, suggestive of impaired renal function. Liver function tests with mild elevation in SGPT and normal bilirubin levels, suggesting mild hepatic involvement.

Table 3: Drug Treatment Timeline from Admission to Discharge.

Drug/Infusion	Dose & Frequency	Phase of Treatment
Piperacillin-Tazobactam	2.5 mg TDS	Day 1- Discharge
Noradrenaline infusion	5 mL/hr.	Day 1- Discharge
Pantoprazole (IV)	40 mg BD	Day 1- Discharge
Ondansetron (IV)	4 mg BD	Day 1- Discharge
Doxycycline	100 mg BD	Day 1- Discharge
Probiotic (Lactic acid bacillus)	TDS	Day 1- Discharge
Hydroxychloroquine (HCQ)	200 mg BD	Day 1- Discharge
Atorvastatin	10 mg HS	Day 1- Discharge
Clopidogrel	75 mg OD	Day 1- Discharge
Human Insulin Infusion	As Per Sliding Scale	Day 1- Discharge
Amlodipine	5 mg BD	Added post stabilization-Discharge
Zinc Cream	Local Application	Added post stabilization-Discharge

This multifaceted approach ensured aggressive infection control, stabilization of endocrine and renal functions, and prevention of further systemic complications.

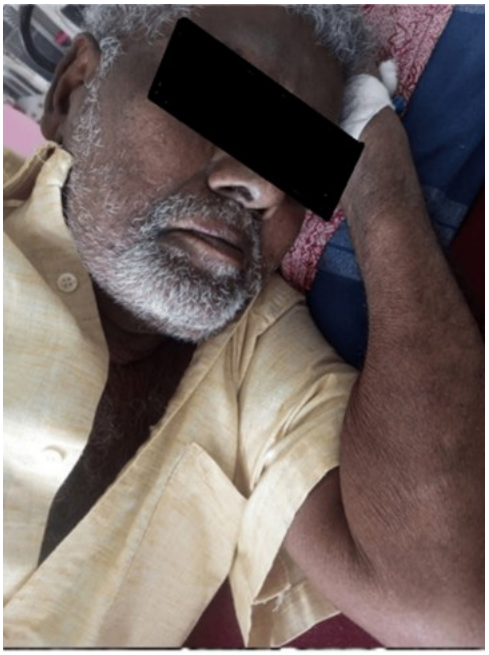

Figure 1: Clinical photograph of the patient with Cushing's syndrome. Cushing's syndrome including moon face, central obesity, buffalo hump, skin thinning/striae, hypertension, and muscle weakness.

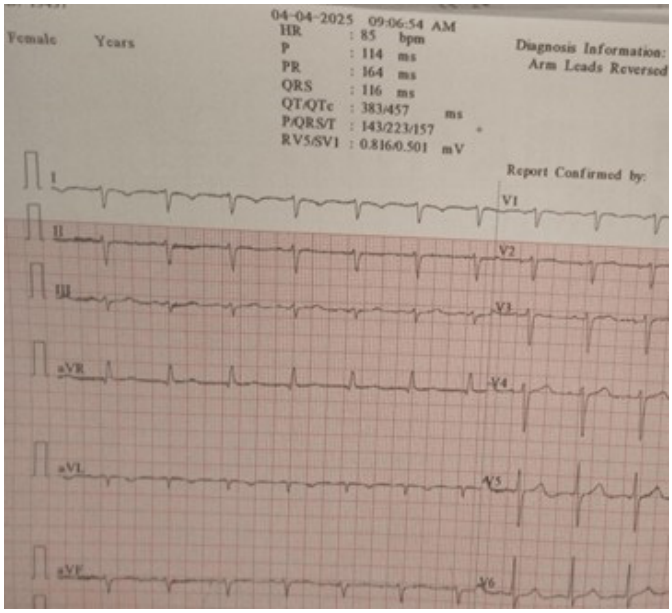

Figure 2: Electrocardiogram (ECG) report of the patient showing heart rate and rhythm analysis.The ECG helps assess cardiovascular status, which may be affected in Cushing's syndrome.

CONCLUSION

Prolonged, unsupervised dexamethasone therapy can culminate in iatrogenic Cushing's syndrome, steroid-induced hyperglycaemia, and life-threatening infections such as urosepsis and septic shock. Early recognition of corticosteroid-related adverse effects-together with prompt withdrawal, physiological steroid replacement, and multidisciplinary support-markedly improves prognosis (Nieman & Biller, 2018; Singer *et al.*, 2016). Judicious prescribing, routine metabolic and infectious surveillance, and patient education are therefore essential, particularly in older adults who often purchase glucocorticoids over the counter. Strengthening pharmacovigilance and enforcing prescription-only access remain key public-health strategies for preventing similar iatrogenic harm.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

ABBREVIATIONS

ACTH: Adrenocorticotropic Hormone; **HPA:** Hypothalamic-Pituitary-Adrenal; **ICU:** Intensive Care Unit; **OTC:** Over-the-Counter; **RBS:** Random Blood Sugar; **ESBL:** Extended-Spectrum Beta-Lactamase; **CT:** Computed Tomography; **MRI:** Magnetic Resonance Imaging; **MAP:** Mean Arterial Pressure; **WHO-UMC:** World Health Organization - Uppsala Monitoring Centre; **BD:** Bis in Die (Twice Daily); **OD:** Once Daily; **TDS:** Ter Die Sumendum (Thrice Daily); **HS:** Hora Somni (At Bedtime).

PATIENT INFORMED CONSENT FORM

Written informed consent was obtained from the patient for publication of clinical information and images. Institutional Permission: Approval to publish this case report was granted by the Medical Superintendent of Government Medical College and Hospital, Orathur, Nagapattinam. All identifying patient information has been anonymised.

PATIENT PERSPECTIVE

"I did not realise my daily tablets could be dangerous. After learning about the side-effects, I now understand why I must reduce steroids slowly and see the doctor regularly."

SUMMARY

This case describes a 70-year-old man who self-medicated with over-the-counter dexamethasone for ten years and developed exogenous Cushing's syndrome. He presented with urosepsis and septic shock, complicated by severe hyperglycaemia and renal impairment. Hormonal testing (low ACTH, failed suppression on dexamethasone test) and imaging (bilateral adrenal atrophy) confirmed the exogenous aetiology. Intensive-care management with broad-spectrum antibiotics, vasopressors, insulin infusion, and a structured hydrocortisone taper achieved clinical recovery. The report highlights the dangers of unsupervised steroid use and emphasises the roles of pharmacovigilance, patient counselling, and regulated dispensing in safeguarding patient safety.

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