J. Pharm Pract Comm Med, 2026; 12(3):180-183.
www.jppcm.org

Case Report

# Exogenous Cushing's Syndrome with Steroid-Induced Adrenal Insufficiency Complicated by Sepsis and a Non-healing Diabetic Foot Ulcer: A Case Report

**Hardik Prajapat*, Meharunisha Siddiqui, Daizy Tak, Narendra Bheemraj Parihar, Jishi Joshi Joseph, Mahendra Singh Rathore**

Department of Pharmacy Practice, Geetanjali Institute of Pharmacy, Geetanjali University, Udaipur, Rajasthan, INDIA.

**ABSTRACT**

This case involves a 52-year-old man with subsequent development of Exogenous Cushing's syndrome and steroid induced adrenal insufficiency after 1 year of unmonitored betamethasone therapy for Arthritis. The patient reported a 4 months old ulcer of the left foot, accompanied by newly detected type 2 diabetes mellitus. Investigations showed exogenous hypercortisolism complicated by sepsis, hypoalbuminemia, raised transaminases and leucocytosis. Radiographic assessment identified absence of the left kidney. The patient was managed in the ICU with a multidisciplinary approach, including intravenous antibiotics, stress dose intravenous hydrocortisone, strict glycemic control, and later transition to a tapering oral corticosteroid regimen. This case illustrates consequences of chronic exogenous corticosteroid exposure and emphasizes the importance of judicious steroid use, vigilant monitoring for Hypothalamic-Pituitary Axis (HPA) axis suppression, and prompt management of secondary infections.

**Keywords:** Betamethasone, Exogenous Cushing's syndrome, Hypoalbuminemia, Steroid induced adrenal insufficiency, Type 2 diabetes mellitus.

**Correspondence:**
**Hardik Prajapat**
Research Scholar, Department of Pharmacy Practice, Geetanjali Institute of Pharmacy, Geetanjali University, Udaipur, Rajasthan, INDIA.
Email: kumarhardi789@gmail.com

## INTRODUCTION

Cushing's disease is an uncommon and severe endocrine disease manifested by over secretion of Adrenocorticotropic Hormone (ACTH) by a corticotroph pituitary tumor therefore causing an over secretion of cortisol by adrenal glands. Cushing's disease indicates the most recurrent cause of endogenous type of (Figure 1) Cushing's Syndrome (CS), constituting approximately 70% of cases (Lacroix *et al*., 2015; Pivonello *et al*., 2016).

CS is a complicated endocrine disease with possibility for serious repercussions if not treated properly (Lacroix *et al*., 2015). The incidence of CS differs across studies, from 0.7-2.4 per million population per year (Lacroix *et al*., 2015; Nieman *et al*., 2015; Pivonello *et al*., 2016). A recent analysis demonstrated a prevalence of 75 per million and incidence of 1.8 per million per year for CS (Pivonello *et al*., 2015). About 80% cases of intrinsic hypercortisolism and ACTH dependent, ACTH origin being pituitary in about 70-80% and ectopic in 10-15% (Feelders *et al*., 2019; Invitti *et al*., 1991; Lamberts *et al*., 1989; Patel, 1999).

**DOI:** 10.5530/jppcm.20260023



**Publishing Partner :** Manuscript Technomedia. [www.mstechnomedia.com]

CS is prevalent in specific high-risk population, example- 2.5% in patients with type 2 diabetes mellitus with impaired glucose regulation (Hofland *et al*., 2005) and 0.5-1% in hypertensive patients (Bruns *et al*., 2002).

Adrenal insufficiency is a serious endocrine disorder that may occur because of intrinsic adrenal damage or suppression of the HPA (Arlt and Allolio, 2003; Bornstein, 2009; Reddy *et al*., 2011). Compared to primary adrenal failures, secondary adrenal insufficiency is more prevalent (Arlt and Allolio, 2003). It affects approximately 150-280 individuals per million, shows a female predominance (Bates, 1996; Laureti *et al*., 1999; Nilsson *et al*., 2000; Regal *et al*., 2001; Tomlinson *et al*., 2001) and is most commonly diagnosed in patients in their 50s and 60s.

## CASE HISTORY

A 52-year-old male patient reported a 4-month history of a persistent discharging ulcer over the left ankle. The ulcer arose spontaneously without any history of injury and showed no improvement with routine local treatment. The patient's history revealed prolonged daily intake of betamethasone without medical supervision for 1 year for presumed arthritis, accompanied by a recent diagnosis of type 2 diabetes mellitus. On examination, the patient had elevated blood pressure (140/100 mL of mercury) and a raised heart rate (111/min). The wound demonstrated signs of infection with purulent discharge and associated

cellulitis. Investigations showed a critically low morning cortisol level of 3.03 µg/dL, confirming adrenal insufficiency. Systemic inflammatory markers were significantly raised (C-reactive protein 195 mg/L, procalcitonin 0.615 ng/mL), along with leucocytosis, low serum albumin, and deranged liver enzymes. Abdominal ultrasound incidentally detected absence of the left kidney since birth. A definitive diagnosis of exogenous Cushing's syndrome with Secondary Adrenal insufficiency, complicated by Diabetic Foot Ulcer and Sepsis, was made. Management included admission to the ICU for intravenous antibiotics, stress-dose hydrocortisone, insulin therapy, and wound care, after which the patient was shifted to oral steroids and antihyperglycemic medications, leading to stabilization and discharge.

The relevant laboratory findings are summarized below in Table 1.

## DISCUSSION

This case is an example of the severe treatment-related complications resulting from unmonitored, long-term glucocorticoid use. The patient's prolonged betamethasone intake over 1 year led to HPA axis suppression, producing a dual state of exogenous Cushing's syndrome and Secondary Adrenal inadequacy. Profoundly reduced stress related cortisol levels confirm the suppression of HPA axis, representing a potential life-threatening state requiring

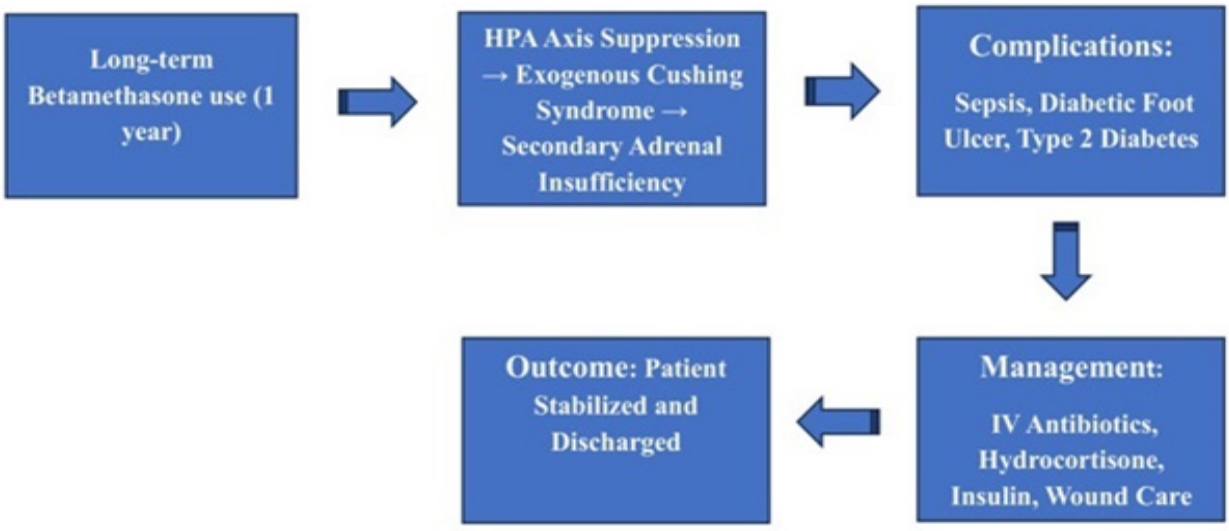


**Figure 1:** Representation of Bethamethasone-induced Cushing's syndrome.

**Table 1: Laboratory Findings.**

| Parameter | Observed value | | | Reference range | Interpretation |
|---|---|---|---|---|---|
| | Day 1 | Day 3 | Day 6 | | |
| Morning serum cortisol | 3.03 µg/dL | 16.7 µg/dL | 9.6 µg/dL | 5-25 µg/dL | ↓ at admission, improved due to steroid therapy. |
| CRP | 195 mg/100 ml | 215 mg/100 ml | 147 mg/100 ml | 0.00-10.00 mg/100 ml | ↑ at admission with gradual decline by Day 6, indicating improvement in systemic inflammatory status. |
| Procalcitonin | 0.615 ng/ml | 0.32 ng/ml | 0.09 ng/ml | <0.5 ng/ml | ↑ initially with progressive decline, indicating resolving sepsis. |
| TLC | 16.69 1,000/cumm | 22.55 1,000/cumm | 15.82 1,000/cumm | 4-11 1,000/cumm | Leucocytosis with gradual improvement. |
| Serum Albumin | 2.40 g/100 ml | 1.98 g/100 ml | 3.1 g/100 ml | 3.5-5.2 g/100 ml | Hypoalbuminemia with partial recovery. |

**Abbreviations: CRP:** C-Reactive Protein; **TLC:** Total Leucocyte Count.

urgent glucocorticoid replacement during acute settings. Simultaneously, glucocorticoid-induced immunosuppressive effects, compounded by poorly controlled and newly diagnosed Diabetes, facilitated a favorable environment for a localized infection to progress into a chronic ulcer and subsequent Sepsis. In addition, the catabolic state resulted by steroids lead to marked hypoalbuminemia, impairing tissue repair and oncotic pressure.

The management required a nuanced, concurrent approach to opposing problems: eradicating the infection while preventing Addisonian crisis, and glycemic control while correcting malnutrition. This underscores the importance of a multidisciplinary strategy comprising endocrinology, critical care, and surgical services. The incidental identification of a lone kidney aggravated pharmacotherapy, emphasizing meticulous renal-adjusted dosing.

Eventually, this case strengthens the essential principle of the steroid-stewardship. It emphasizes to prescribe systemic glucocorticoids at the lowest effective dose and shortest possible duration, along with mandatory patient education about risks of self-medication and withdrawal. It also emphasizes the need for regular monitoring for suppression of HPA axis, signs of infection, and glycemic dysregulations in any patient receiving long-term steroid therapy. This case report serves as a cautionary example of how a commonly prescribed anti-inflammatory medication can cause a multisystem crisis when used in the absence of appropriate supervision.

## CONCLUSION

Prolonged unsupervised glucocorticoid use may result in serious and potentially fatal complications, including adrenal insufficiency, severe infections, and metabolic abnormalities. This case brings forward the significance of steroid stewardship and routine monitoring in patients receiving long-term glucocorticoid therapy; the importance of maintaining a high index of suspicion for adrenal insufficiency in any patient receiving glucocorticoids presenting with acute illness; the pivotal role of a multidisciplinary approach in the management of complex cases involving endocrine, infectious, and surgical complications; the importance of patient education in preventing treatment related complications.

## ACKNOWLEDGEMENT

We would like to express our sincere gratitude to all faculty members for their support and guidance in the preparation and publication of this case report.

## ABBREVIATIONS

**ACTH:** Adrenocorticotropic Hormone; **CRP:** C-Reactive Protein; **CS:** Cushing's Syndrome; **DC:** *Dichrostachys cinerea* (L.) Wight and Arn.; **HPA:** Hypothalamic-Pituitary-Adrenal (axis); **ICU:** Intensive Care Unit; **IV:** Intravenous; **MetS:** Metabolic Syndrome; **TLC:** Total Leucocyte Count.

## CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

## PATIENT CONSENT

A written informed consent was obtained from the patient for collecting data and publication of this case report.

## REFERENCES

Arlt, W., and Allolio, B. (2003). Adrenal insufficiency. Lancet, 361(9372), 1881-1893. doi: 10.1016/S0140-6736(03)13492-7, PubMed: 12788587

Bates, A. S., Van't Hoff, W., Jones, P. J., and Clayton, R. N. (1996). The effect of hypopituitarism on life expectancy. Journal of Clinical Endocrinology and Metabolism, 81(3), 1169-1172. doi: 10.1210/jcem.81.3.8772595, PubMed: 8772595

Bornstein, S. R. (2009). Predisposing factors for adrenal insufficiency. New England Journal of Medicine, 360(22), 2328-2339. doi: 10.1056/NEJMra0804635, PubMed: 19474430

Bruns, C., Lewis, I., Briner, U., Meno-Tetang, G., and Weckbecker, G. (2002). SOM230: A novel somatostatin peptidomimetic with broad somatotropin release inhibiting factor (SRIF) receptor binding and a unique antisecretory profile. European Journal of Endocrinology, 146(5), 707-716. doi: 10.1530/eje.0.1460707, PubMed: 11980628

Feelders, R. A., Newell-Price, J., Pivonello, R., Nieman, L. K., Hofland, L. J., and Lacroix, A. (2019). Advances in the medical treatment of Cushing's syndrome. Lancet. Diabetes and Endocrinology, 7(4), 300-312. doi: 10.1016/S2213-8587(18)30155-4, PubMed: 30033041

Gatto, F., Arvigo, M., Amarù, J., Campana, C., Cocchiara, F., Graziani, G., Bruzzone, E., Giusti, M., Boschetti, M., and Ferone, D. (2019). Cell specific interaction of pasireotide: review of preclinical studies in somatotroph and corticotroph pituitary cells. Pituitary, 22(1), 89-99. https://doi.org/10.1007/s11102-018-0926-y

Hofland, L. J., van der Hoek, J., Feelders, R., van Aken, M. O., van Koetsveld, P. M., Waaijers, M., Lamberts, S. W. J. (2005). The multi-ligand somatostatin analogue SOM230 inhibits ACTH secretion by cultured human corticotroph adenomas via somatostatin receptor type 5. European Journal of Endocrinology, 152(4), 645-654. doi: 10.1530/eje.1.01876, PubMed: 15817922

Invitti, C., Giraldi, P., Dubini, F., Piolini, A., and Cavagnini, M. (1991). Effect of Sandostatin on CRF-stimulated secretion of ACTH, beta-lipotropin and beta-endorphin, β-lipotropin and α-endorphin. Hormone and Metabolic Research, 23(5), 233-235. doi: 10.1055/s-2007-1003660, PubMed: 1651895

Lacroix, A., Feelders, R. A., Stratakis, C. A., and Nieman, L. K. (2015). Cushing's syndrome. Lancet, 386(9996), 913-927. doi: 10.1016/S0140-6736(14)61375-1, PubMed: 26004339

Lamberts, S. W., Uitterlinden, P., and Klijn, J. M. (1989). The effect of the long-acting somatostatin analogue SMS 201-995 on ACTH secretion in Nelson's syndrome and Cushing's disease. Acta Endocrinologica, 120(6), 760-766. doi: 10.1530/acta.0.1200760, PubMed: 2543178

Laureti, S., Vecchi, L., Santeusanio, F., and Falorni, A. (1999). Is the prevalence of Addison's disease underestimated? Journal of Clinical Endocrinology and Metabolism, 84(5), 1762. doi: 10.1210/jcem.84.5.5677-7, PubMed: 10323417

Nieman, L. K., Biller, B. M. K., Findling, J. W., Murad, M. H., Newell-Price, J., Savage, M. O., Endocrine Society (2015). Treatment of Cushing's syndrome: An Endocrine Society clinical practice guideline. Journal of Clinical Endocrinology and Metabolism, 100(8), 2807-2831. doi: 10.1210/jc.2015-1818, PubMed: 26222757

Nilsson, B., Gustavasson-Kadaka, E., Bengtsson, B. A., and Jonsson, B. (2000). Pituitary adenomas in Sweden between 1958 and 1991: Incidence, survival, and mortality. Journal of Clinical Endocrinology and Metabolism, 85(4), 1420-1425. doi: 10.1210/jcem.85.4.6498, PubMed: 10770176

Patel, Y. C. (1999). Somatostatin and its receptor family. Frontiers in Neuroendocrinology, 20(3), 157-198. doi: 10.1006/frne.1999.0183, PubMed: 10433861

Pivonello, R., De Leo, M., Cozzolino, A., and Colao, A. (2015). The treatment of Cushing's disease. Endocrine Reviews, 36(4), 385-486. doi: 10.1210/er.2013-1048, PubMed: 26067718

Pivonello, R., Isidori, A. M., De Martino, M. C., Newell-Price, J., Biller, B. M. K., and Colao, A. (2016). Complications of Cushing's syndrome: State of the art. Lancet. Diabetes and Endocrinology, 4(7), 611-629. doi: 10.1016/S2213-8587(16)00086-3, PubMed: 27177728

Reddy, S. V. B., Prabhudesai, S., and Gnanasekaran, B. (2011). Origin of primary adrenal lymphoma and predisposing factors for primary adrenal insufficiency in primary adrenal lymphoma. Indian Journal of Endocrinology and Metabolism, 15(4), 350-351. doi: 10.4103/2230-8210.85604, PubMed: 22029014

Regal, M., Páramo, C., Sierra, S. M., and Garcia-Mayor, R. V. (2001). Prevalence and incidence of hypopituitarism in an adult Caucasian population in northwestern Spain. Clinical Endocrinology, 55(6), 735-740. doi: 10.1046/j.1365-2265.2001.01406.x, PubMed: 11895214

Tomlinson, J. W., Holden, N., Hills, R. K., Wheatley, K., Clayton, R. N., Bates, A. S., Stewart, P. M. (2001). Association between premature mortality and hypopituitarism. West Midlands Prospective Hypopituitary Study Group. Lancet, 357(9254), 425-431. doi: 10.1016/s0140-6736(00)04006-x, PubMed: 11273062

**Cite this article:** Prajapat H, Siddiqui M, Tak D, Parihar NB, Joseph JJ, Rathore MS. Exogenous Cushing's Syndrome with Steroid-Induced Adrenal Insufficiency Complicated by Sepsis and a Non-healing Diabetic Foot Ulcer: A Case Report. J Pharm Pract Comm Med. 2026;12(3):180-3.