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Case Report

Cushing Syndrome Induced by Methylprednisolone: A Case Study

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ABSTRACT

Corticosteroids, naturally produced by the adrenal cortex or synthetically formulated, play a vital role in various medical treatments, including asthma management. They are categorized into mineralocorticoids and glucocorticoids, each serving distinct physiological functions. Glucocorticoids, such as methylprednisolone and hydrocortisone, possess potent anti-inflammatory, immunosuppressive, anti-proliferative and vasoconstrictive properties, making them essential in controlling inflammatory diseases. However, prolonged corticosteroid use can lead to Iatrogenic Cushing's Syndrome (ICS), characterized by central obesity, moon facies, osteoporosis, hypertension, hyperglycaemia and increased infection susceptibility. This case report discusses a 34-year-old female with a history of bronchial asthma managed with long-term methylprednisolone use, presenting with acute respiratory distress, lower limb swelling, facial swelling and abdominal pain. Clinical evaluation revealed elevated cortisol levels, leucocytosis and electrolyte imbalances. Her treatment included intravenous furosemide, nebulized bronchodilators (Levo salbutamol and ipratropium bromide), corticosteroids (budesonide and hydrocortisone), antibiotics (cefoperazone-sulbactam, gentamicin, metronidazole) and supportive therapy with oxygen and anticoagulants. The synergistic effect of bronchodilators and corticosteroids in reducing airway inflammation and bronchospasms significantly improved her respiratory function. This case underscores the importance of carefully monitoring corticosteroid therapy to prevent adverse effects like ICS while effectively managing asthma exacerbations. A balanced approach in steroid administration is crucial to minimizing complications while maximizing therapeutic benefits.

Keywords: Crushing syndrome, Glucocorticoid Side Effects, Hyper cortisol, Methylprednisolone.

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INTRODUCTION

The corticosteroids are naturally made by the adrenal cortex that act as steroids and can also be synthesized synthetically analogues. There are primarily two forms of these corticosteroids: mineralocorticoids and glucocorticoids. These corticosteroids come in a variety of formulations, such as topical solutions, parenteral, inhalations and oral forms and they are frequently prescribed in medical procedures. In the adrenal cortex, cholesterol is converted into corticosteroids. Aldosterone synthase (for aldosterone) or 11β-hydroxylase (for corticosterone) mediates the final step of their biosynthesis pathway, which starts with aldosterone and corticosterone. Glucocorticoids have anti-inflammatory, immunosuppressive, anti-proliferative and vasoconstrictive properties. They also influence the metabolism of proteins, fats and carbohydrates. Blocking the activity of inflammatory mediators produces anti-inflammatory effects, while immunosuppressive effects are Direct action on T-lymphocytes suppresses delayed hypersensitivity reactions, inhibits DNA synthesis and epidermal cell turnover to have anti-proliferative effects and inhibits the action of inflammatory mediators like histidine to produce vasoconstrictive effects.[1] Excess glucocorticoid receptor activation results in Cushing syndrome. The most common type of Cushing syndrome is iatrogenic (exogenous), which is brought on by long-term use of synthetic glucocorticoids like prednisolone. The rare endogenous Cushing syndrome is brought on by the excessive pituitary tumour production of ACTH, excessive cortisol synthesis by the adrenal glands due to an adrenal tumour, or ectopic ACTH production by other tumors.[2] Weight increase, typically central obesity, truncal fat redistribution, the development of dorsocervical and supraclavicular fat pads (buffalo hump) and the iconic moon face are the hallmarks of Iatrogenic Cushing Syndrome (ICS).

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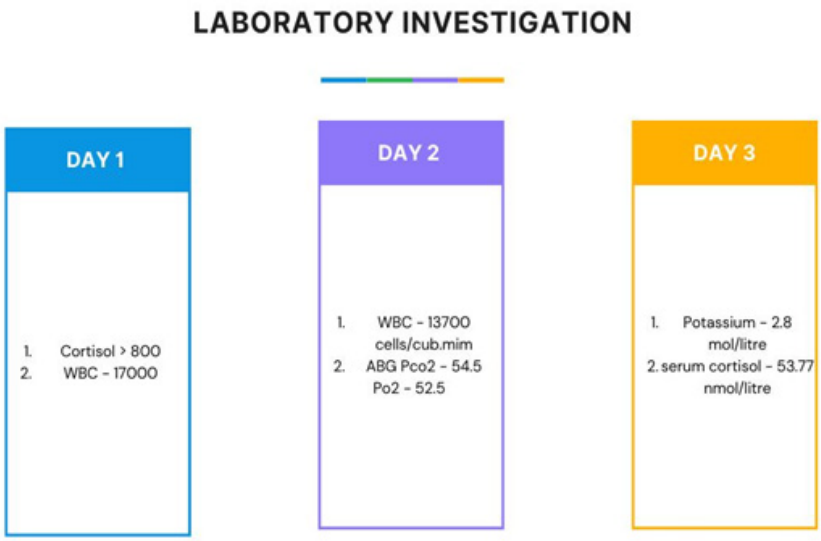
There are other signs of myopathy, muscle weakness, thin skin, easy bruising, osteoporosis and abdominal striae. Psychosis and sadness are two of the negative psychological outcomes. Additionally, patients are vulnerable to inadequate wound healing, which raises the risk of atherosclerotic disease and infections.[3] ATH, which is secreted by the pituitary gland in healthy people, causes the adrenal glands to release cortisol. This hypothalamus pituitary adrenal axis is suppressed when steroids are given.[3] Inducing gluconeogenesis and preventing the absorption of glucose by the cells, causing hypertension and hyperglycaemia. The catabolic effects cause loss of collagen and reabsorption resulting in the development of osteoporosis and increased susceptibility to fractures. Because glucocorticoids weaken the immune system, patients with Cushing syndrome are susceptible to a variety of infections.[4] Certain features such as an increase in intraocular pressure, cataracts, benign intracranial hypertension, osteoporosis and pancreatitis are more common in the ICS than in the exogenous Cushing syndrome. Cushing's syndrome has two primary causes: endogenous hypercortisolism and exogenous hypercortisolism. The most frequent cause of Cushing's syndrome, exogenous hypercortisolism, is primarily iatrogenic and arises from long-term glucocorticoid use. Endogenous Cushing's syndrome, which can be both ACTH-dependent and ACTH-independent, is caused by the adrenal glands producing too much cortisol. Secretion of ACTH pituitary adenomas (Cushing's disease) and ectopic ACTH release by neoplasms are responsible for ACTH-dependent Cushing's syndrome; while adrenal hyperplasia, adenoma and carcinoma are significant causes of ACTH-independent Cushing's syndrome.[5]

PATIENT INFORMATION

A 34-year-old female patient presented with a three-year history of breathing difficulties, accompanied by palpitations, decreased urine output, bilateral swelling of the lower limbs, abdominal swelling, facial swelling and abdominal pain over the past two days. She denied experiencing nausea, dizziness, vomiting, or symptoms associated with a cold or cough. Her past medical history was significant for acute exacerbations of bronchial asthma, for which she had been receiving regular treatment for the past three years. Additionally, she had a history of taking methylprednisolone 40 mg once daily. On physical examination, the patient was found to be conscious, oriented and afebrile. Her vital signs revealed a normal body temperature, elevated blood pressure of 140/80 mmHg, an increased pulse rate of 130 beats per minute, a respiratory rate of 26 breaths per minute and a reduced oxygen saturation level (SpO_2) of 82%. Laboratory investigations showed elevated cortisol levels. Renal function tests indicated reduced creatinine and uric acid levels, while potassium levels were noted to have decreased by the third day of admission. On complete blood count shows increased WBC. Liver function tests revealed elevated Serum Glutamic-Oxaloacetic Transaminase (SGOT) levels.

Upon admission, the patient was initially managed with an intravenous bolus dose of furosemide (10 mg, stat) to facilitate fluid management. In addition, she received nebulized ipratropium bromide and Levo salbutamol (stat) to relieve bronchospasms, nebulized budesonide (stat) to reduce airway inflammation and one Liter of oxygen supplementation to improve her oxygen saturation and respiratory function. Further therapeutic management included the administration of intravenous cefoperazone and sulbactam (1.5 g, twice daily) for infection control, along with intravenous hydrocortisone (100 mg, three times daily) to manage inflammation and potential adrenal insufficiency. Respiratory support was continued with nebulized ipratropium bromide and Levo salbutamol four times a day via the nasal route, as well as nebulized budesonide twice daily. Additionally, the patient was started on subcutaneous low molecular weight heparin (0.4 cc, twice daily) as prophylaxis



for thromboembolic complications. Her diuretic therapy was adjusted with intravenous furosemide, administered in a dosage regimen of 40 mg in the morning, 20 mg in the afternoon and no dose at night (40 mg-20 mg-0, IV). Inj. Gentamicin 80 mg, IV-BD, Inj. Metronidazole 500 mg, IV-TDS, Inj. Pantoprazole 40 mg, IV- BD.

CASE DESCRIPTION

Recurrent exacerbations of bronchial asthma were part of the 34-year-old's medical history and she has been receiving regular medication for the past three years. She also previously took 40 mg of methylprednisolone once daily. An intravenous bolus dosage of furosemide was administered as a stat after the patient was diagnosed with an acute exacerbation of bronchial asthma. The patient had past medication history methylprednisolone 40 mg once daily this drug was taken before 2 days.

Naranjo Adverse Drug Reaction Probability Scale (with modifications): The following scale is used to assess the likelihood a particular adverse reaction is related to a medication. Answer each of the 10 questions, calculate the total score and determine if an adverse drug reaction is Definitely, Probably, Possibly, or unlikely related to the drug in question. (Interpretation of the probability classification can be found in Table 1 on the next page).

Furosemide is used to treat oedema and belongs to the class of loop diuretics. To treat bronchospasms, she was also given Levo salbutamol (stat) and nebulized ipratropium bromide. The nebulizer ipratropium bromide under the class of anticholinergic

Table 1: Naranjo Adverse Drug Reaction Probability Scale.

Question	Yes	No	Do Not Know	Score
Are there previous CONCLUSIVE reports on this reaction? Answer yes if 2 or more well-described case reports can be found in the literature.	+1	0	0	0
Did the adverse reaction appear after the suspected drug was administered? Answer yes if a reaction occurs in close temporal relation (e.g., within 1-2 days) after drug administration.	+2	-1	0	2
Did the adverse reaction improve when the drug was discontinued or a specific antagonist given? Answer yes if the reaction lessens or disappears after the suspect drug stops or a pharmacologic antagonist given.	+1	0	0	1
Did the adverse reaction reappear when the drug was readministered? Answer yes if reaction disappears after drug discontinuation but reappear when the drug was restarted.	+2	-1	0	0
Are there alternative causes (other than the suspect drug) that could have caused the reaction? Answer yes if the reaction can be explained by causes or medications other than the suspect drug.	-1	+2	0	2
Did the reaction reappear when a placebo was given? Answer yes if the reaction reappears after administration a placebo.	-1	+1	0	0
Was the drug detected in blood or other fluids in concentrations known to be toxic? Answer yes if drug concentration is in the toxic or supratherapeutic range.	+1	0	0	1
Was the reaction more severe when dose was increased or less severe when dose was decreased? Answer yes if the intensity of the reaction is stronger with higher dose or weaker with lower dose.	+1	0	0	1
Did the patient have a similar reaction to the same or similar drugs in any previous exposure? Answer yes if patient has a similar documented reaction when exposed to the suspect drug or related medication in the past.	+1	0	0	0
Was the adverse reaction confirmed by any objective evidence? Answer yes if the reaction can be confirmed by abnormal lab values, imaging, or physical examination.	+1	0	0	1
	Total Score		8	

(M3 antagonist, Levo salbutamol and ipratropium bromide are commonly used in combination to treat asthma because they both efficiently induce bronchodilation. By inhibiting Muscarinic (M3) receptors in the smooth muscles of the airways, ipratropium bromide, a Short-Acting Muscarinic Antagonist (SAMA), stops acetylcholine from causing bronchoconstriction. The reduction of mucus development, airway resistance and airway relaxation therefore improves airflow. By targeting β2 receptors in bronchial smooth muscles, Levo salbutamol, a Selective β2-Adrenergic Agonist (SABA), activates adenylate Cyclase, which raises cyclic AMP (cAMP) levels. This process encourages smooth muscle relaxation, which leads to bronchodilation and lessens airway inflammation. In particular, these medications cooperate to enhance bronchodilation, nebulized budesonide (stat) to lessen airway inflammation and she required one Liter of oxygen supplementation to improve her oxygen saturation and respiratory function because of her low saturation. By intravenous as preventative antibiotics, cefoperazone and sulbactam (1.5 g) are administered intravenously. These drugs block the penicillin-binding protein, which prevents the cross-linking transpeptidation process and stops the growth of bacteria. H. Cort was a member of the corticosteroid class. Corticosteroid injections are used to treat inflammation because they have strong anti-inflammatory effects.

Examples of inflammatory cytokines that promote hyperresponsiveness in the bronchi and are reduced by binding to glucocorticoid receptors. Budesonide is classified as a corticosteroid (glucocorticoid) and, rather than acting as a direct bronchodilator, it acts as an anti-inflammatory. Injectable gentamicin 80 mg belongs to the class of aminoglycosides that block the 50s ribosome, which prevents the synthesis of proteins and results in cell death. Injectable metronidazole, a member of the nitroimidazole class, inhibits DNA synthesis and results in cell death. Proton pump inhibitors of the inj. pantoprazole class, which reduce stomach acid secretion as a result of multiple medication ingestion (40 mg).

DISCUSSION

This case highlights the potential development of iatrogenic Cushing's syndrome due to chronic methylprednisolone (40 mg/day) use over three years. Long-term administration of exogenous corticosteroids leads to Hypothalamic-Pituitary-Adrenal (HPA) axis suppression, resulting in adrenal insufficiency upon abrupt withdrawal. This patient presented with classic features of Cushing's syndrome, including hypertension, tachycardia, hypokalaemia, generalized oedema (facial and limb swelling) and metabolic abnormalities (elevated cortisol levels, leucocytosis and reduced renal function markers).

CONCLUSION

This case highlights the complex interplay between prolonged corticosteroid use and the development of iatrogenic Cushing's syndrome in an asthma patient. Despite being essential for reducing airway inflammation, glucocorticoids must be carefully monitored during long-term use to avoid negative immunosuppressive and metabolic consequences. The combination of bronchodilators, corticosteroids, diuretics, antibiotics and supportive care was effective in treating this patient's acute exacerbation. This highlights the necessity of customized treatment plans to balance the advantages of corticosteroid therapy with risk reduction. It is imperative for clinicians to stay alert for early indicators of problems caused by corticosteroids and implement suitable treatment adjustments to maximize patient outcomes.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

ABBREVIATIONS

BP: Blood Pressure; **PR:** Pulse Rate; **RR:** Respiratory Rate; **SpO_2:** Oxygen Saturation; **RFT:** Renal Function Test; **Cr:** Creatinine; **UA:** Uric Acid; **K^+:** Potassium; **CBC:** Complete Blood Count; **WBC:** White Blood Cell; **LFT:** Liver Function Test; **GOT:** Serum Glutamic-Oxaloacetic Transaminase; **IV Inj.:** Intravenous Injection; **Neb.:** Nebulization.

PATIENT CONSENT

Informed consent was obtained from the patient for the publication of this case report, ensuring the protection of her privacy and confidentiality.

SUMMARY

This case report discusses a 34-year-old female patient with a history of recurrent bronchial asthma, who developed Iatrogenic Cushing's Syndrome (ICS) due to prolonged methylprednisolone use (40 mg/day for three years). She presented with breathing

difficulties, palpitations, edema, hypertension and metabolic abnormalities, including elevated cortisol levels and hypokalemia. Her symptoms were managed with a combination of diuretics, bronchodilators, nebulized corticosteroids, antibiotics and supportive care. This case underscores the importance of monitoring long-term corticosteroid therapy to prevent adverse effects such as HPA axis suppression and metabolic complications.

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