Original Research Paper

Volume - 16 | Issue - 06 | June - 2026 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Paediatrics

DIAGNOSTIC CHALLENGE IN AN ADOLESCENT WITH CHRONIC FEBRILE ARTHRITIS, STEROID-INDUCED CUSHINGOID FEATURES AND SECONDARY STAPHYLOCOCCAL SEPSIS

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ABSTRACT Cushing's syndrome, a rare endocrine disorder may result from exogenous administration of steroids. Long-term corticosteroid uses causes symptoms such as moon face, buffalo hump, pink stretch marks, and weight gain. Chronic febrile illness with arthritis in adolescents presents a diagnostic challenge due to overlap between autoimmune disorders, infections, and drug-induced complications. Prolonged corticosteroid exposure further complicates diagnosis by masking inflammation and predisposing to opportunistic infections. A 14-year-old male presented with cushingoid feature due to continuous prednisolone use. He had chronic unilateral knee joint pain from last 6 months and is currently taking steroids. He has been experiencing symptoms such as prolonged fever, mucosal ulcers, facial swelling, and greenish loose stool. Examination revealed Cushingoid features like moon face, edema, muscle weakness and diffuse hyperpigmented ichthyosis-like skin changes. Laboratory evaluation showed leucocytosis, anaemia, hypoalbuminemia, and inflammatory stool findings. Blood culture revealed methicillin-sensitive coagulase-negative Staphylococcus (MSCONS). Imaging supported inflammatory rather than septic joint disease. The patient required escalation of antibiotics and reintroduction with gradual tapering of corticosteroids. Steroid tapering is essential in managing complex paediatric cases with overlapping inflammatory and infectious aetiologies.

KEYWORDS : Prednisolone, Cushing's Syndrome (Cushingoid Features), Moon Face, Steroids, Coagulase-negative Staphylococcus

INTRODUCTION

Cushing's syndrome is a rare, multisystemic endocrine disease due to excessive glucocorticoid hormone production that result from either exogenous or endogenous aetiologies (1). Exogenous causes occur due to prolonged use of high dose of medications mostly prednisolone in the management of non-endocrine disorders (1-2). This is the most common cause in the paediatric population (2-3). Glucocorticoids are significant pharmacological agents that function as immune system stimulants and inflammation reducers to treat a variety of illnesses. On the other hand, if glucocorticoids are used inappropriately, they can cause a number of adverse effects, such as Cushing's syndrome and the suppression of the hypothalamic pituitary axis (4). Cushing's syndrome has two primary aetiologies: endogenous hypercortisolism and exogenous hypercortisolism. The most frequent cause of Cushing's syndrome, known as exogenous hypercortisolism, is primarily iatrogenic and arises from long-term glucocorticoid use (5). Understanding the pharmacokinetic characteristics, daily dosage, frequency, and variations in each person's steroid metabolism is essential to avoiding side effects associated with glucocorticoid overuse (6).

The present case involves a 14-year-old adolescent with chronic monoarthritic and prolonged fever, creating a diagnostic dilemma between autoimmune conditions such as Juvenile Idiopathic Arthritis, gastrointestinal pathology like Inflammatory Bowel Disease, and infection. Prominent features including joint involvement, morning stiffness, greenish stool, anaemia, and hypoalbuminemia suggested an inflammatory process; however, blood culture revealing coagulase-negative Staphylococcus introduced the possibility of secondary infection, complicating the clinical picture.

The history of prolonged unsupervised corticosteroid uses with features of Cushing Syndrome further masked the underlying disease while increasing susceptibility to infection. Additionally, the risk of adrenal insufficiency necessitated cautious steroid tapering. This case highlights the complexity of distinguishing primary inflammatory disease from superadded infection in a steroid-modified clinical state.

Case Presentation

A 14-year-old adolescent male, presented with chronic unilateral knee pain for 6–8 months, characterized by morning stiffness and slight limitation of movement. He also had a history of intermittent fever for the past 6 months. From last ten days before admission, he developed oral ulcers with crackling of lips associated with difficulty in opening the mouth, along with facial swelling that appeared to be temporally related to prior drug intake. He also develops complain of swelling and peeling of skin around eyelids and he is unable to open his eyes properly. In the week preceding admission, he developed loose stools. The colour of stools is greenish in colour with frequency of five to six episodes a day. On the day of admission, he was drowsy, febrile with mild respiratory distress. His heart rate was 122 beats per minute, respiratory rate was 28 breaths per minute, blood pressure was 134/80mmHg and spo2 was 92% on room air. He was unable to take anything orally due to mucosal ulcers and lips crackling. His urine output was 0.9ml/kg/day. He also having mild crepitations on both sides of lung. Patient was admitted in PICU for management. On examination, the patient exhibited Cushingoid facies with round red full moon face suggestive of prior corticosteroid exposure. The skin showed diffuse hyperpigmentation with ichthyosis-like scaling, along with abdominal striae. Oral examination revealed multiple ulcers with restricted mouth opening. Additionally, bilateral gynecomastia was noted.

Investigations on admission revealed mild anaemia (8.1gm/dl), sepsis TLC (14140/mm^3) with neutrophils predominance, hypoalbuminemia (albumin 2.0g/dl), hyponatremia (Na 134mEq/L) also elevated CRP (40mg/L) suggestive of inflammation and infection in the body. Serum cortisol level was 8 µg/dl suggestive of suppression of endogenous cortisol production. Viral marker was normal. On stool microscopy 8-10 pus cells/HPF detected. Stool and urine culture shows no growth. Blood culture shows Methicillin- sensitive coagulase- negative staphylococcus (MSCONS) heavy growth. In imaging, USG knee/gluteal region shows mild myositis, minimal effusion with no septic arthritis. Initially the patient was started on oxygen therapy with empirical antibiotics (ceftriaxone, metronidazole,) and iv fluids for mild respiratory distress, fever and gastroenteritis. For supportive care ORS, zinc, ondansetron and pantop were given. For oral and skin care betadine gargle, mild topical steroids, vitamin A were given. Nebulization with normal saline was also done for crepitations in both lungs. Based on the culture and sensitivity report, antibiotic therapy was escalated to the sensitivity pattern to piperacillin -tazobactam, vancomycin and levofloxacin. Targeted therapy guided by culture (MSCONS sensitivity profile). For steroid management reintroduction followed by gradual tapering was done. For adjunctive therapy PRBC transfusion for anaemia, albumin transfusion for hypoalbuminemia, antihistamines (bilastine) and topical steroids for dermatological symptoms were given. Initial empiric antibiotic treatment yielded a delayed and suboptimal therapeutic response. Upon receipt of the culture and sensitivity data, the antimicrobial regimen was adjusted to targeted therapy. Following this adjustment, the patient demonstrated a dramatic and rapid resolution of symptoms, leading to a full recovery.

DISCUSSION

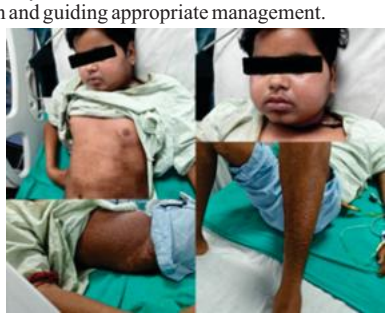
This case illustrates a complex interplay of steroid-induced Cushing Syndrome, underlying inflammatory pathology, and secondary infection. Prolonged unsupervised corticosteroid use led to

Cushingoid features and masked the primary disease, while predisposing the patient to bloodstream infection with coagulase-negative Staphylococcus. Pituitary tumours and ectopic production of the hormone ACTH are examples of endogenous causes of Cushing syndrome, while exogenous causes include external delivery of corticosteroids. Exogenous Cushing's syndrome is a disorder that arises from extended exposure to the therapeutic use of corticosteroids. Another name for them is steroid-induced. Iatrogenic Cushing syndrome, also known as Cushing's syndrome (4). Cushing's disease patients typically exhibit one or more symptoms that are a result of the presence of too much ACTH or cortisol (7). Unless the patient is taking a corticosteroid, serum cortisol levels are low in exogenous Cushing's syndrome. A round, red, full moon face, growth retardation in children, skin infections, purple marks (striae) on the skin of the breast, abdomen, and thighs, thin skin that is easily bruised, back pain during routine activities, fat deposit between the shoulders and above the collar bone, hip and shoulder muscle weakness, and fractures of the ribs and spine due to thinning of the bones are the most common symptoms of Cushing syndrome patients. Common laboratory findings associated with Cushing syndrome include low ACTH, low ACTH stimulation, elevated fasting blood sugar, decreased serum potassium, elevated blood cholesterol, and decreased bone density. The corticosteroid dose is tapered off as part of the treatment, which could take a year.

In clinical practice, glucocorticoids are frequently used to treat autoimmune, inflammatory, and allergic diseases. The most frequent use of glucocorticoids is irrational, especially when used in long-term treatments. This can have a variety of negative effects, including suppression of the hypothalamic-pituitary-adrenal axis, Cushing's syndrome, an increased risk of infections, and mental abnormalities. The factors affecting the therapeutic and side effects of glucocorticoids are the pharmacokinetic characteristics of the drug, individual differences in steroid metabolism, daily dosage, and length of treatment (8). Before initiating steroid therapy, patients should be well informed about the possible side effects of steroids. Otherwise, it may lead to severe systemic side effects including Cushing's syndrome, hypertension, dyslipidaemia, suppression of hypothalamic pituitary-adrenal axis, striae, glaucoma, skin atrophy, cataract and predisposition to life threatening infections(9).

CONCLUSION

This case highlights a steroid-modified clinical presentation in which prolonged unsupervised corticosteroid use resulted in Cushingoid features, masking the underlying disease and predisposing to secondary infection. Many clinicians prescribe these drugs, and they are used to treat a wide range of diseases. As a result, when patients are prescribed corticosteroids, all clinicians must educate them about the potential side effects, as well as the importance of close monitoring and dose tapering. Gradual reintroduction and tapering of steroids were essential to prevent adrenal insufficiency. A multidisciplinary approach and dynamic reassessment were crucial in achieving clinical stabilization and guiding appropriate management.



During Illness, At Presentation

During Recovery Phase

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