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A Case Study On Iatrogenic Cushing's Syndrome: Medical Error Of Frequent Parenteral Steroid Injections

[1]S. Pushpa Sree, [2]P. Ruchitha, [3]S.U. Salma, [4]Dr. N. Lokeswara Reddy, [5]Dr. K.Thirumala Naik

[1]Pharm D Intern, Sri Balaji Medical College Hospital & Research Institute, Krishna Teja Pharmacy College, Tirupati, Andhra Pradesh, India, [2]Pharm D Intern, Sri Balaji Medical College Hospital & Research Institute, Krishna Teja Pharmacy College, Tirupati, Andhra Pradesh, India,[3]Pharm D Intern, Sri Balaji Medical College Hospital & Research Institute, Krishna Teja Pharmacy College, Tirupati, Andhra Pradesh, India.

[4]Dr. N. Lokeswara Reddy, Assistant Professor Dept of Pulmonology,Sri Balaji Medical College Hospital & Research Instuite, Tirupati, Andhra Pradesh,India.

[5]Dr. K.Thirumala Naik, Associate professor & Clinical Preceptor, Krishna Teja Pharmacy College, Tirupati, Andhra Pradesh, India.

Abstract:

Background

Unintentional and irrational usage of oral/parenteral steroids is the root cause of chronic iatrogenic cushing's syndrome.

Case presentation

Here, we present a iatrogenic cushing's syndrome case which is caused by longterm exposue to both parenteral and oral intake of steroids.

Conclusion

An evidence of constant use of over-the-counter medications is required to identify the source of exogenous glucocorticoids in iatrogenic Cushing's syndrome.

Index Terms - cushing's syndrome, steroids, cortisol**.**

I. INTRODUCTION

Cushing's syndrome is an endocrine disease precipitated while the adrenal glands produce excess cortisol. It is also known as hypercortisolism. Cushing's syndrome can arise for diverse motives. One of the most common causes is long-term exposure to corticosteroid medications like prednisone. Cushing's syndrome that is resulting from steroids is referred to as iatrogenic (or exogenous) Cushing's syndrome[1].

The most common cause of iatrogenic Cushing's syndrome (ICS) is long-term, high-dose oral or parenteral steroid usage.

Cortisol is a substance that is evidently produced by means of the body, especially during the time of stress. Cortisol has several actions, which includes regulating inflammation and controlling the metabolism of carbohydrates, fats, and proteins in our body.

The adrenal glands cortex produces corticosteroids, which are hormone mediators that are further divided into three categories: androgenic sex hormones, mineralocorticoids (aldosterone is the body's primary mineralocorticoid), glucocorticoids (cortisol is the major glucocorticoid produced by the body). Medical

professionals imply their actions to treat the various conditions. The list of glucocorticoid indications is incredibly long.[2]

Glucocorticoids helps in metabolism and have immunosuppressive, anti-inflammatory, and vasoconstrictive impacts. Whereas mineralocorticoids regulate water and electrolytes balance by influencing particle transport within the epithelial cells of the renal tubules.[5]

However, prolonged exposure to cortisol, whether from the body's own production or from using corticosteroid medications can lead to weight gain, insulin resistance, and elevated glucose levels[3].

Cushing syndrome patients may experience purple striae, moon facies, buffalo hump, facial plethora, truncal obesity, and supraclavicular fat pads. Proximal muscular weakness, bruising easily, weight gain, hirsutism, and development retardation in youngsters are common symptoms. There is also a chance of osteopenia, diabetes, hypertension, and weakened immune system.[5]

EPIDEMIOLOGY:

Glucocorticoid administration at supraphysiologic dosages is the most frequent cause of exogenous or iatrogenic Cushing's syndrome.[4]Endogenous Cushing's syndrome is extremely uncommon, incidence of this is 0.7–2.4 times per million people annually.[4]Glucocorticoids are administered pharmacologically to over 10 million Americans annually.[6]

CASE REPORT:

A 24-year-old lady was referred to our medical centre (Sri Balaji Medical College Hospital & Research Institute) with possible cushingoid-like disease and Tinea incognito. She was presented with complaints of severe rashes, erythematous lesions, and itching all over her arms and legs &abdomen. She had central obesity and a history of 8kg gain in weight over 6 months duration.

On examination, she had facial hair and slightly ulcerated acne on her forehead and cheeks. History of Photosensitivity and itching for 1 year which is progressive, and Arthritis (Bilateral knee joint pain, Swelling, and stiffness of bilateral knee joint, ankle, and elbow) for more than 12 weeks duration, Decreased urine output for two weeks, with intermittent moderate to high-grade fever spikes.
Her family members also noticed a few psychological symptoms like agitation, restlessness, mood swings, and insomnia. She was on symptomatic therapy for allergy and benzodiazepines for mood swings and insomnia by local physician .

On her arrival, she showed numerous Cushingoid features which included moon facies, enlarged dorsocervical fat pads, truncal obesity, easy bruisability. On presentation she was febrile 100.6 F with HR 131 bpm, BP 110/90mm of Hg, SpO2 98%, Her preliminary lab presentation Shows urine analysis shows nil sugar , Trace albumin, and pus cells around 4-6 cells/HPF.

Later she was admitted to the Internal medicine department. Underwent basic investigations including blood counts , serum cortisol levels (mid night, 8am and 4 pm), chest imaging and 24 hours urine for cortisol. 8am cortisol test 53mcg/dl (5-25mcg/dl) hemoglobin 11.2 g/dL with, sodium of 128.8 mmol/L (115–145mmol/L) and potassium of 2.89 mmol/L(3.6-5.0mmol/L); HbA1C was 6.1(<5.7%); , epithelial cells : 2-4cells/HPF(15-20cells/HPF) .CRP value is 32mg/L (upto 6mg/L).

Later she was hospitalized and evaluated . She was initially started on symptomatic therapy for fever spikes and fatigue . Later dermatologist opinion was obtained for skin lesions.

The dermatologist evaluated for Eczema and skin lesions. multiple erythematous lesions with irregular borders and scales. She was diagnosed with Tinea Corporis. She was started on Itraconazole 200mg/day and later combined with Terbinafine 500mg /day. Treatment was continued for 4 weeks and then doses were tapered after reassessment and continued for another 2 weeks .

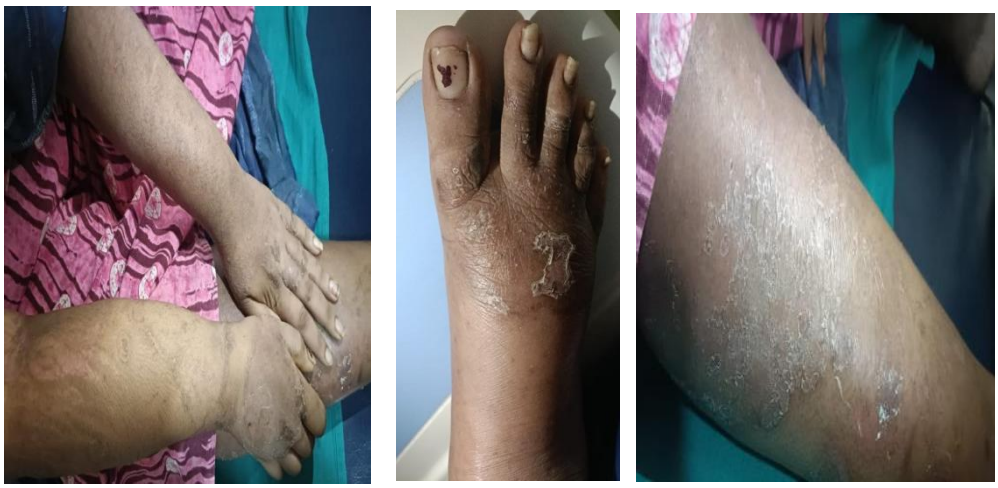


Fig:-1 Images of Tinea corporis

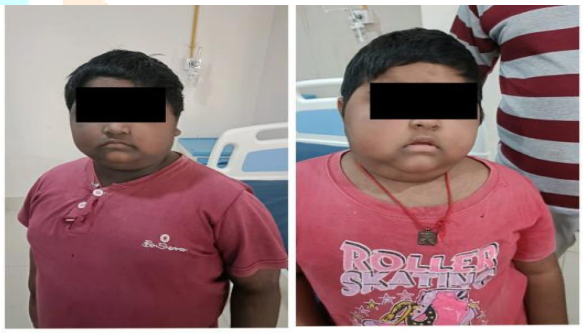
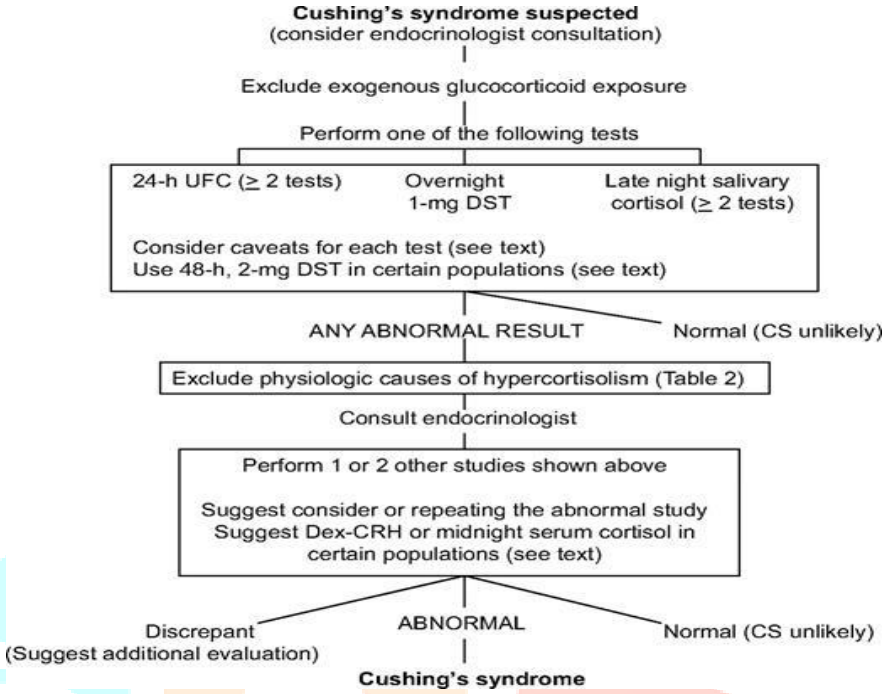


Fig:-2 Her children with similar cushingoid features

DIAGNOSTIC EVALUATION:

No pituitary abnormality / adenoma / cystic lesions noted in Magnetic resonance imaging (MRI) . Chest imaging showed normal bronchovascular markings and no parenchymal or bony abnormality found.



DISCUSSION :

In this case, it was concluded that the patient's regular use of the steroid supplement was most likely the cause of their Cushingoid symptoms. No other discernible source of exogenous glucocorticoids, such as topical, intranasal, or inhaled formulations, was present.

In this case, rather than starting from a known prior dose, the patient was started on physiologic prednisolone with the intention of attempting tapering from there, as her previous steroid dose was unknown. In this case, along with the patient her family members (2 sons and her husband) also diagnosed with iatrogenic Cushing syndrome with the same etiology. They were also received treatment in the same hospital. In few cases, female patient also had gynaecological issues like dysmenorrhea, ammenorrhea, PCOS. But in our cases there is no such gynaecological complaints.[9]

Diagnosing Cushing's illness might be difficult. Cushingoid syndrome is usually misdiagnosed since it overlaps with several prevalent medical conditions, delaying treatment. In this instance, the female patient, 24yrs, had been unwell for at least 1.5 months prior to receiving a diagnosis. She was presented with round full moonface, , accumulation of fat in trunk region with excessive weight gain, infection of skin, purple striae on the skin , abdomen, and thighs, easy bruising, backache, severe muscle weakness, fat deposit between the shoulders and above the collar bone. We have encountered other patients who have long-term denials of a Cushing's diagnosis. Certain patients have very little clinical symptoms, although they test positive for Cushing's disease. Some people are severely sick by the time they receive a diagnosis, despite exhibiting many of the clinical signs of Cushing's disease. Recovery from hypercortisolism is frequently difficult and unpleasant due to the harm it causes to the body, particularly the muscles, joints, and bones.

In this situation, it became determined that the affected person's Cushingoid capabilities had been maximum possibly because of usage of parenteral steroids. No further biochemical checking out for hypercortisolism was pursued on the basis of her compelling physical features and signs and symptoms of Cushing's syndrome with a known source of exogenous glucocorticoids.

Her two sons, ages 8 and 10 were seen with growth retardation got admitted in pediatric ward in the same hospital and get treated by a pediatrician.

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