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Case Report

Unmasking Takayasu Arteritis in a patient with steroid-induced Cushing syndrome

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Abstract

Prolonged corticosteroid use causes Cushing syndrome to emerge with characteristic features like moon face, central obesity, buffalo hump, striae and hypertension. However, the use of corticosteroids also can mask underlying autoimmune conditions, delaying diagnosis. We describe the case of a 25-year-old women who initially presented with complaints of wide-spread body pain and obesity. Further examination ensured cushingoid features due to prolonged self-medication with tablet prednisolone of dose 5mg taken orally. On gradual steroid tapering, she developed limb claudication, unequal blood pressure and pulse discrepancies. Elevated inflammatory markers strengthened the suspicion of Takayasu arteritis, a large vessel arthritis. This case emphasizes the importance of evaluating potential underlying inflammatory diseases in individuals exhibiting exogenous cushingoid features, especially when unexplained vascular signs emerge. Unsupervised corticosteroid use not only increases the risk of adverse effects but may delay the diagnosis of serious conditions, emphasizing the need for cautious prescribing and public awareness.

Keywords: Cushing syndrome, Prednisolone, Corticosteroids, Takayasu arteritis, Self-medication, Diagnostic challenge, Adverse effect

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1. Introduction

Treatment of chronic inflammation, autoimmune conditions and allergic reactions with oral corticosteroids is a common practice in the healthcare system. Positives like effective outcome and ease of administration lead some patients to self-medicate with corticosteroids, overlooking the potential for serious adverse effects; one such serious complication is exogenous Cushing's syndrome (CS).[1]

Cushing syndrome is a rare condition experienced due to prolonged exposure to high cortisol levels in the blood. It can be caused exogenously or endogenously. While exogenous CS is caused by prolonged use of corticosteroids, endogenous CS is linked to the pituitary gland's production of adrenocorticotropic hormone, which triggers and raises the level of cortisol in blood. CS occurs with an average incidence of 24 cases per million people, and it has a notable female-to-male ratio of 15:1.[2]

Takayasu arteritis (TA) is an uncommon autoimmune condition characterized by cell-mediated inflammation of blood vessels, primarily impacting large and medium-sized arteries, especially the aorta and its major branches.[3,4] Inflammatory processes can result in thickening of the arterial walls, narrowing of the lumen, formation of aneurysms, and fibrosis, which ultimately disrupts blood flow and leads to ischemic injury in organs.[5] The documented ratio of females to males affected by TA is roughly around 1.58:1.[6]

We present this case to highlight the clinical and pharmacological complexity arising from chronic, unsupervised corticosteroid use, which not only resulted in CS but also masked the diagnosis of an underlying inflammatory vasculitis. Early diagnosis is critical in order to avoid worsening of patient condition.

2. Case Presentation

A 25-year-old female is admitted to the female medicine ward with chief complaints of generalised body pain, morning stiffness of the knuckle joints, and progressive fatigue over several months. The symptoms were aggravated by exertion and minimally relieved during rest. Over the preceding

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month, she had also developed facial puffiness, central obesity, and rapid weight gain.

She had been consuming prednisolone 5 mg tablet unsupervised, 3–4 times per week for approximately two years, obtaining the medication over-the-counter for musculoskeletal pain. There was no history of medical supervision, tapering, or evaluation during this period.

Specific cushingoid features like_moon face, buffalo hump, white striae and central obesity were evidently present in the patient during physical examination. Two of her cushingoid features, namely, moon face and buffalo hump, are photographically presented in **(Figure 1)** and **(Figure 2)**, respectively.

Figure 1: Patient Presenting with Moon face characteristic

This figure shows the image of 25-year old female patient presenting with “moon face” symptom, full facial features due to fat deposition after self-medicating with oral prednisolone for the past two years. Published with patient consent.

Figure 2: Patient illustrating buffalo hump appearance

This figure shows abnormal fat accumulation in the dorsocervical region of the female patient, commonly associated with Cushing syndrome. Published with patient consent.

Elevated blood pressure of 160/130 mmHg with a pulse rate of 82 bpm was observed on Day 1. On Day 2 of hospitalization, this condition was treated by administering amlodipine 5 mg in tablet form and her BP dropped precipitously to 80/60 mmHg. During the assessment of peripheral pulses, a delay between the radial and femoral pulses as well as a weakened left radial pulse were identified. There was also a notable discrepancy in upper limb blood pressures: 140/80 mmHg in the right arm and 80/50 mmHg in the left arm. These vascular findings raised clinical suspicion for large-vessel vasculitis, most notably Takayasu arteritis.

Table 1: Laboratory investigations

Parameters	Value
WBC count	$14.6 \times 10^3/\mu L$
Neutrophils	81.9%
Lymphocytes	16.6%
Haemoglobin	12.5 g/dl
Platelets	$285 \times 10^3/\mu L$
Erythrocyte Sedimentation Rate (ESR)	21 mm/hour
C-Reactive Protein (CRP)	10.9 mg/L
Antinuclear antibody (ANA)	1+ at 1:100 dilution
Rheumatoid Factor	Negative
Antistreptolysin O Titer	Negative
Cortisol	2.3 µg/dl
Urea	21mg/dl
Creatinine	0.83 mg/dl
Aspartate Aminotransferase	48 U/L
Alanine Aminotransferase	36 U/L
Albumin	3.1 g/dl
Urine Protein to Creatinine Ratio	0.25mg/mg
Tri-Iodothyronine	32.15ng/dl
Thyroxine	8.78µg/dl
Thyroid Stimulating Hormone	1.30µIU/mL

This table represents the values of laboratory tests taken for the patient, which helped in evaluating the patient - diagnosis and ruling out certain condition.

Electrocardiogram findings revealed sinus arrhythmia, a short PR interval, and an abnormal Q wave in lead III. Abdomen and pelvis Computed Tomography helped in ruling out endogenous Cushing syndrome by showing no adrenal masses. Renal Doppler studies demonstrated low-velocity, low-resistance, and low-amplitude flow in both renal arteries, consistent with large-vessel compromise.

The patient was diagnosed with Takayasu arteritis and exogenous Cushing syndrome, attributed to chronic, unsupervised prednisolone use over two years.

On the day of admission, the patient was started on injectable dexamethasone (4 mg IV stat) due to the patient's symptomatic complaints of ongoing joint pain. Once Cushing syndrome was suspected on Day 2, steroids were temporarily withdrawn. Later the reduced cortisol level (Mentioned above in **(Table 1)** led to the cautious reintroduction of injectable hydrocortisone (100 mg IV twice daily). It was gradually tapered over the week. Tablet prednisolone, 5 mg daily was restarted on Day 8 due to inflammatory symptoms.

Supportive medications included Tablet hydroxychloroquine 200 mg once daily, Tab. Paracetamol 500 mg, Tab. Folic acid 5 mg, Tab. vitamin C 500 mg, and anti-hypertensive (Tab. amlodipine 5 mg once daily). Blood sugars and electrolytes were regularly monitored and remained within acceptable ranges.

3. Discussion

The real-life challenge and diagnostic dilemma imposed by the chronic misuse of corticosteroids is evident in this case. The signs of Takayasu arteritis were concealed by the immunosuppression and endocrine alterations resulting from long-term corticosteroid misuse.

The mechanism of action of corticosteroids explains their role in both controlling inflammation and modulating immune responses in this case. In order to generate immunosuppressive and anti-inflammatory effects, corticosteroids operate through two primary pathways namely, genomic (slower) and non-genomic (rapid). Through the genomic pathway, they enter cells, attach to cytoplasmic glucocorticoid receptors, and then proceed into the nucleus to alter gene expression and lower the synthesis of inflammatory chemicals (adhesion proteins, cytokines, and chemokines). As a result, immunological and structural cells that contribute to inflammation are less active. Through membrane or intracellular receptors, the non-genomic pathway activates in a matter of rapidly, inhibiting enzymes such as phospholipase A2, lowering the release of arachidonic acid, and affecting cell survival, including thymocyte death. Corticosteroids further diminish immunological responses by suppressing the production of B and T cells at high dosages.[7]

Either Endogenous or exogenous, Cushing syndrome results from prolonged exposure to elevated glucocorticoid levels.[8] In this case, the patient had been self-medicating with oral prednisolone (5 mg Tablet), intermittently over two years unsupervised by a medical practitioner. Her clinical presentations like moon face **(Figure 1)**, obesity, wide striae, buffalo hump **(Figure 2)**, and sustained hypertension, strongly indicated steroid-induced Cushing syndrome.[2,8] Suppressed serum cortisol level and the absence of adrenal abnormalities on imaging ruled out endogenous causes, confirming an iatrogenic etiology.

Simultaneously, observation of significant inter-arm blood pressure difference, diminished left radial pulse, radial-radial pulse discrepancy **(Table 2)** and radio-femoral delay, raised suspicion and turned the spotlight to Takayasu arteritis. These are hallmark signs of large-vessel vasculitis involving the aortic arch branches. Further, the diagnosis of TA was confirmed by The American College of Rheumatology (ACR) and European Alliance of Associations for Rheumatology (EULAR) 2022 diagnostic criteria which yield a score of 9.[9] The absolute requirements to diagnose Takayasu arteritis and its standard diagnostic score based on ACR/EULAR 2022 are elaborated in **(Table 2)** and **(Table 3)**, respectively.

Table 2: Absolute requirements of ACR/EULAR 2022 Takayasu arteritis diagnostic criteria

Absolute Requirements	Status	Justification
Age 60 years or less at the time of diagnosis	Present	The patient reported is 25 years old
Evidence of vasculitis on imaging	Present	Doppler imaging confirmed bilateral renal artery involvement.

This table ensures the presence of absolute requirements to confirm diagnosis of Takayasu arteritis as per the latest diagnostic criteria – ACR/EULAR 2022.

Table 3: Diagnostic justification for Takayasu Arteritis

Diagnostic Criteria	Score board	Justification
Female gender	+1	Patient is a female
Upper or Lower Limb Claudication	+2	Patient complaints of joint pain in upper and lower limbs which exaggerate on work and reduce during rest
Diminished pulsation of superior limb arteries	+2	Radial-radial pulse discrepancy and weaken left pulse was noted.
Minimum 20 mmHg difference between right and left arms	+1	Right BP: 140/80 mmHg Left BP : 80/50 mmHg
Two arterial territories involved	+2	Bilateral Renal artery involvement confirmed on Doppler.
Paired artery affected	+1	Bilateral renal arteries involved

This table includes justification for the diagnosis of Takayasu arteritis using ACR/EULAR 2022 diagnostic criteria along with the scoring method.

Additional supportive features included constitutional symptoms (fatigue, malaise), and elevated inflammatory markers (ESR: 21 mm/hour; CRP: 10.9 mg/L).(4) The elevated inflammatory markers and further lab investigations are tabulated above in **(Table 1), (Table 2)** and **(Table 3)**.

Management posed unique challenges due to iatrogenic suppression of the hypothalamic-pituitary-adrenal (HPA) axis. On admission, the patient received intravenous dexamethasone 4 mg immediately, administered empirically for musculoskeletal pain. Once CS was suspected and cortisol levels were found suppressed, dexamethasone was promptly discontinued. To prevent adrenal crisis, intravenous hydrocortisone 100mg twice daily was initiated and gradually tapered, later transitioning to low-dose oral prednisolone in tablet form (5 mg daily). This management strategy complies with Endocrine Society guidelines, which recommend glucocorticoid replacement and tapering in patients with suspected HPA suppression.[10]

Considering the autoimmune and inflammatory characteristics of TA, low-dose prednisolone was deemed appropriate to reduce vascular inflammation and alleviate systemic symptoms. Tablet hydroxychloroquine 200mg was added as a steroid-sparing medication, appreciated for its immunomodulatory effects and favorable safety profile, especially in cases where starting methotrexate or azathioprine was not immediately feasible.[11]

Written informed consent was obtained from the patient to ensure ethical publication of clinically relevant information and images.

4. Conclusion

This case illustrates the severe, yet preventable complication caused when self-medication results in adverse outcomes. Unsupervised use of corticosteroids not only caused exogenous Cushing syndrome but also masked and delayed the timely diagnosis of Takayasu arteritis, worsening the patient's condition. It underscores the critical need for recognising vascular signs in patients with steroid-inducing CS. This case highlights the urgent need for promoting registered steroid use, patient education on the risks of self-medication and regular clinical monitoring in the. Early multi-disciplinary involvement including endocrinology, rheumatology and vascular specialists can aid in the timely diagnosis and tailored medication management. Gradual tapering of corticosteroids while ensuring adequate immunosuppression is essential in preventing long-term complications.

5. Authors Contribution

1. **Rengaraj Thirunanamoorthy:** Supervised clinical execution at the hospital, ensured ethical compliance, provided final approval for the manuscript.
2. **Thaslim Ridhwana Barakath Ali:** Contributed to the literature search, data collection, preparing initial draft of manuscript and manuscript revision.
3. **Vignesh Vaithiyanathan:** Contributed in literature search, preparing the informed consent form, data collection and critical inputs on manuscript.
4. **Vilvarajeshwaran Balamurugan:** Helped with data collection, patient follow up and reviewed draft.
5. **Dhinesh:** Helped with literature review, manuscript editing and formatting.

6. Source of Funding

None

7. Conflict of Interest

None

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