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Prednisolone Induced Cushing's Syndrome : A Case Report

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Abstract

Cushing's syndrome is a hormonal disorder resulting from prolonged exposure to excessive glucocorticoids and may be caused by endogenous overproduction or exogenous steroid administration. The exogenous form is the most common and typically occurs due to long-term use of synthetic corticosteroids, especially in patients treated for chronic inflammatory or autoimmune disorders. Because of its insidious onset and nonspecific clinical manifestations, exogenous Cushing's syndrome is frequently under-recognized in routine clinical practice. We report the case of a 43-year-old woman with a history of Sjögren's syndrome, distal renal tubular acidosis, and chronic myopathy who presented with progressive bilateral lower limb edema, moon facies, facial hirsutism, and cutaneous hyperpigmentation. She had been receiving prolonged high-dose prednisolone therapy. Laboratory investigations revealed markedly suppressed serum cortisol levels along with hypokalemia, hypoalbuminemia, proteinuria, and elevated alkaline phosphatase, indicating suppression of the hypothalamic–pituitary–adrenal axis due to exogenous glucocorticoid exposure. Based on clinical features, detailed medication history, and biochemical findings, a diagnosis of prednisolone-induced exogenous Cushing's syndrome was established. Management involved gradual tapering of prednisolone to prevent adrenal insufficiency, along with supportive treatment for metabolic and electrolyte abnormalities. This case highlights the diagnostic value of suppressed endogenous cortisol in distinguishing exogenous from endogenous Cushing's syndrome and emphasizes the importance of early recognition, rational corticosteroid use and regular monitoring to reduce steroid-related morbidity and prevent long-term complications.

Keywords: Cushing's syndrome; Exogenous Cushing's syndrome; Iatrogenic endocrine disorder; Prednisolone; Steroid toxicity

Introduction

Cushing syndrome is a relatively uncommon endocrine disorder characterized by the excessive elevation of cortisol (also known as hydrocortisone) levels within the body. The most prevalent etiological factor contributing to Exogenous Cushing's syndrome (ECS) is the type of Cushing syndrome (CS) affects persons who use glucocorticoids, often known as corticosteroids or steroids.[1]

Epidemiology –

Cushing's syndrome is rare and has an estimated incidence of 0·2–5·0 per million people per year and a prevalence of 39–79 per million in various populations; 5–8 median age of onset/diagnosis was 41·4 years with a female-to-male ratio of 3:1.

Studies suggest an increased but variable prevalence in people with uncontrolled type 2 diabetes, hypertension or early-onset osteoporosis.[2]

Etiology –

Exogenous (Iatrogenic) Causes :

Prolonged therapeutic administration of synthetic glucocorticoids for various medical conditions.

Endogenous Causes :

ACTH-Dependent Cushing's Syndrome

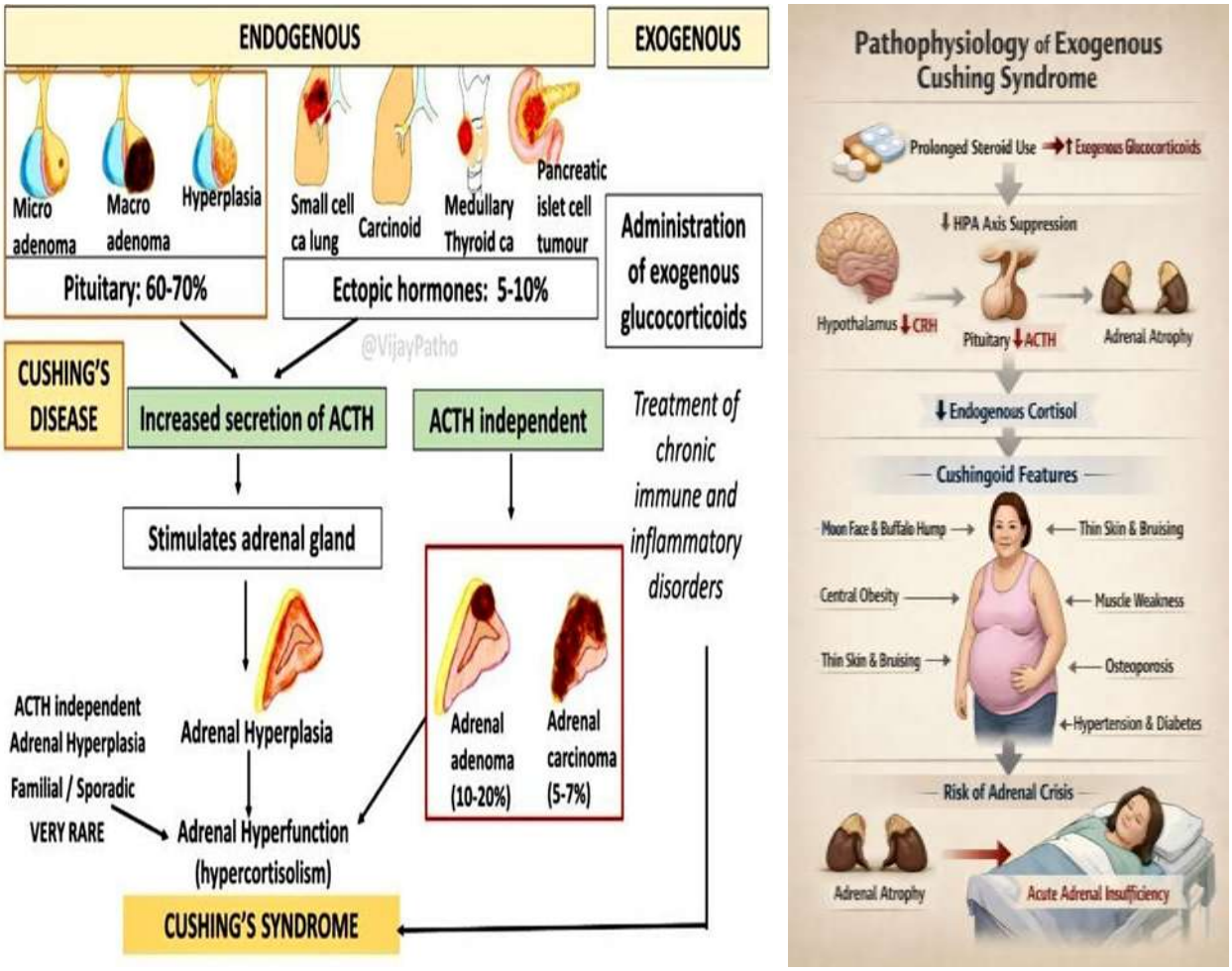
1. Pituitary corticotroph adenomas producing excess ACTH (Cushing's disease).
2. Ectopic ACTH secretion, most commonly from neuroendocrine tumors.

ACTH-Independent Cushing's Syndrome

1. Autonomous cortisol overproduction by the adrenal glands.
2. Benign solitary adrenocortical adenoma (most common cause).
3. Adrenal carcinoma and hyperplastic syndromes.[3]

Pathophysiology

Fig 01 : Pathophysiology of Cushing's syndrome[4]



Clinical manifestations –

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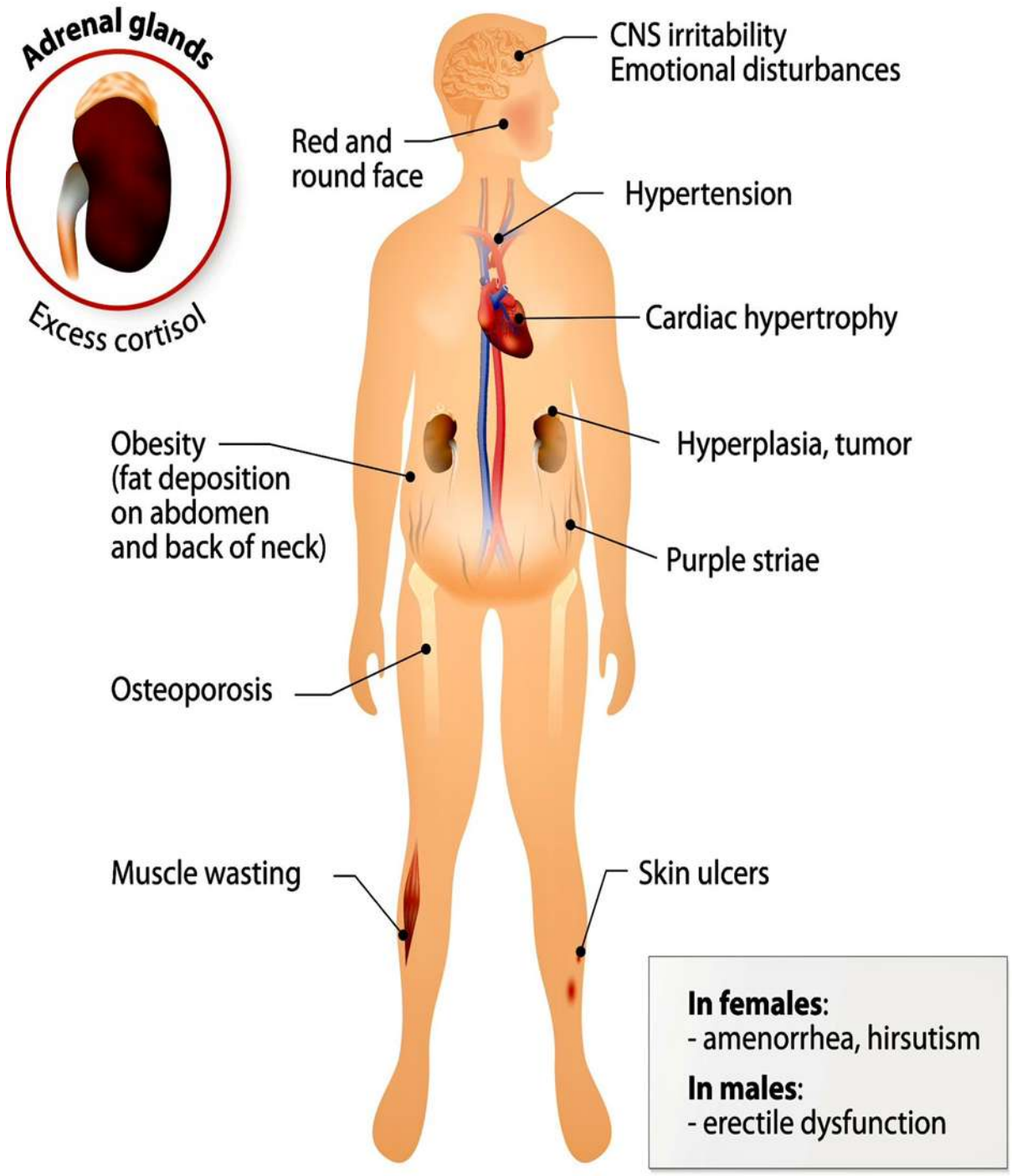
Fig 02 : Signs and symptoms of Cushing's syndrome[5]



Diagnosis –

No single clinical feature is diagnostic; presentation depends on severity and duration of hypercortisolism.

Screening Tests

1. 24-hour urinary free cortisol (UFC) : High sensitivity and specificity; unreliable in renal failure.
2. Overnight 1 mg dexamethasone suppression test : Morning cortisol <50 nmol/L excludes Cushing's syndrome.

Additional/Confirmatory Tests

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1. Midnight plasma cortisol >50 nmol/L suggests Cushing's syndrome.
2. Late-night salivary cortisol : Simple, reliable screening tool.
3. Low-dose dexamethasone + CRH / desmopressin tests : Help differentiate true Cushing's disease from pseudo-Cushing's states.[6]

Fig 03 : Cortisol fractions and specimens[7]

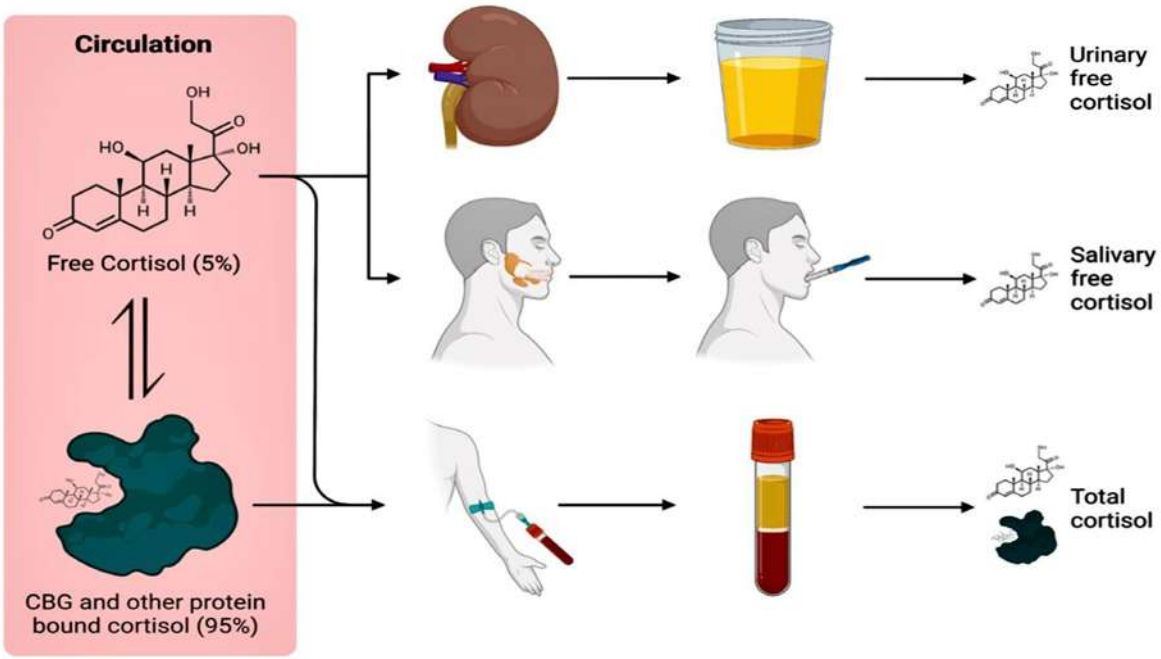

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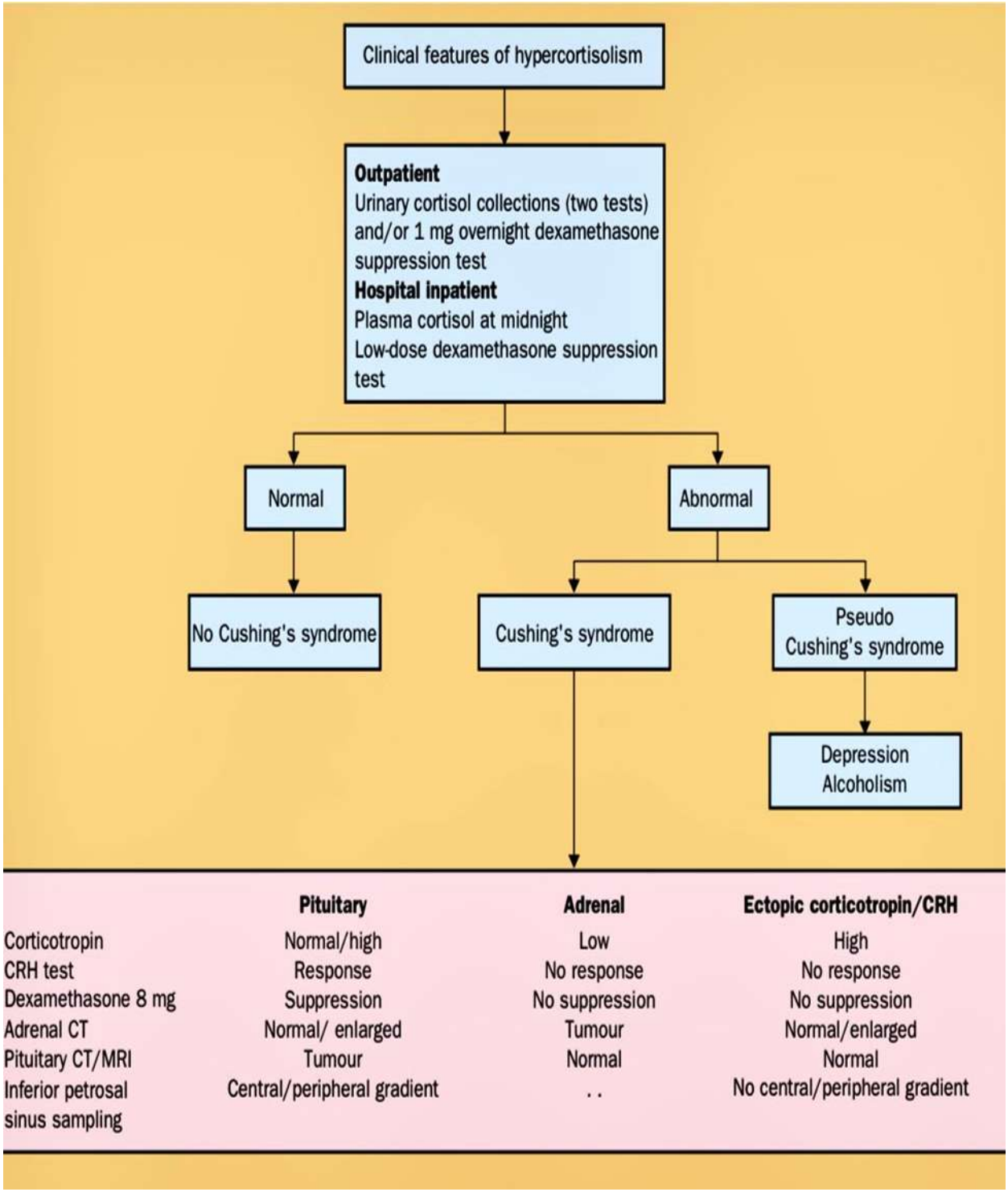
Fig 04 : Assessment of Cushing's syndrome[6]



	Pituitary	Adrenal	Ectopic corticotropin/CRH
Corticotropin	Normal/high	Low	High
CRH test	Response	No response	No response
Dexamethasone 8 mg	Suppression	No suppression	No suppression
Adrenal CT	Normal/ enlarged	Tumour	Normal/enlarged
Pituitary CT/MRI	Tumour	Normal	Normal
Inferior petrosal sinus sampling	Central/peripheral gradient	..	No central/peripheral gradient

Treatment –

Goals of therapy

1. Therapy is indicated only in confirmed Cushing's syndrome to normalize cortisol levels or action and manage related complications.
2. Treatment should not be initiated without a definite diagnosis.

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3. Patients with borderline hormonal abnormalities but no clinical features should not receive cortisol-lowering therapy, as benefit is unproven.

Table 01 : Treatment of Cushing's syndrome

CATEGORY	TREATMENT	EXPLANATION
First-line	Surgical removal of cause	Definitive and potentially curative
	Transsphenoidal surgery	Treatment of choice for pituitary adenoma
	Unilateral adrenalectomy	Adrenal adenoma/carcinoma
	Ectopic tumor resection	If tumor is localized
Second-line	Repeat pituitary surgery	Persistent or recurrent disease
Radiotherapy	Conventional / stereotactic	If surgery fails or is contraindicated
Medical therapy	Ketoconazole, metyrapone	Inhibit cortisol synthesis
	Mitotane	Refractory disease / adrenal carcinoma
	Cabergoline, pasireotide	Pituitary-directed therapy
	Mifepristone	Blocks cortisol action
	Etomidate (IV)	Severe, life-threatening cases
Definitive salvage	Bilateral adrenalectomy	Rapid control; lifelong steroids
Supportive care	Treat comorbidities	HTN, diabetes, osteoporosis
Follow-up	Long-term monitoring	Recurrence, adrenal insufficiency

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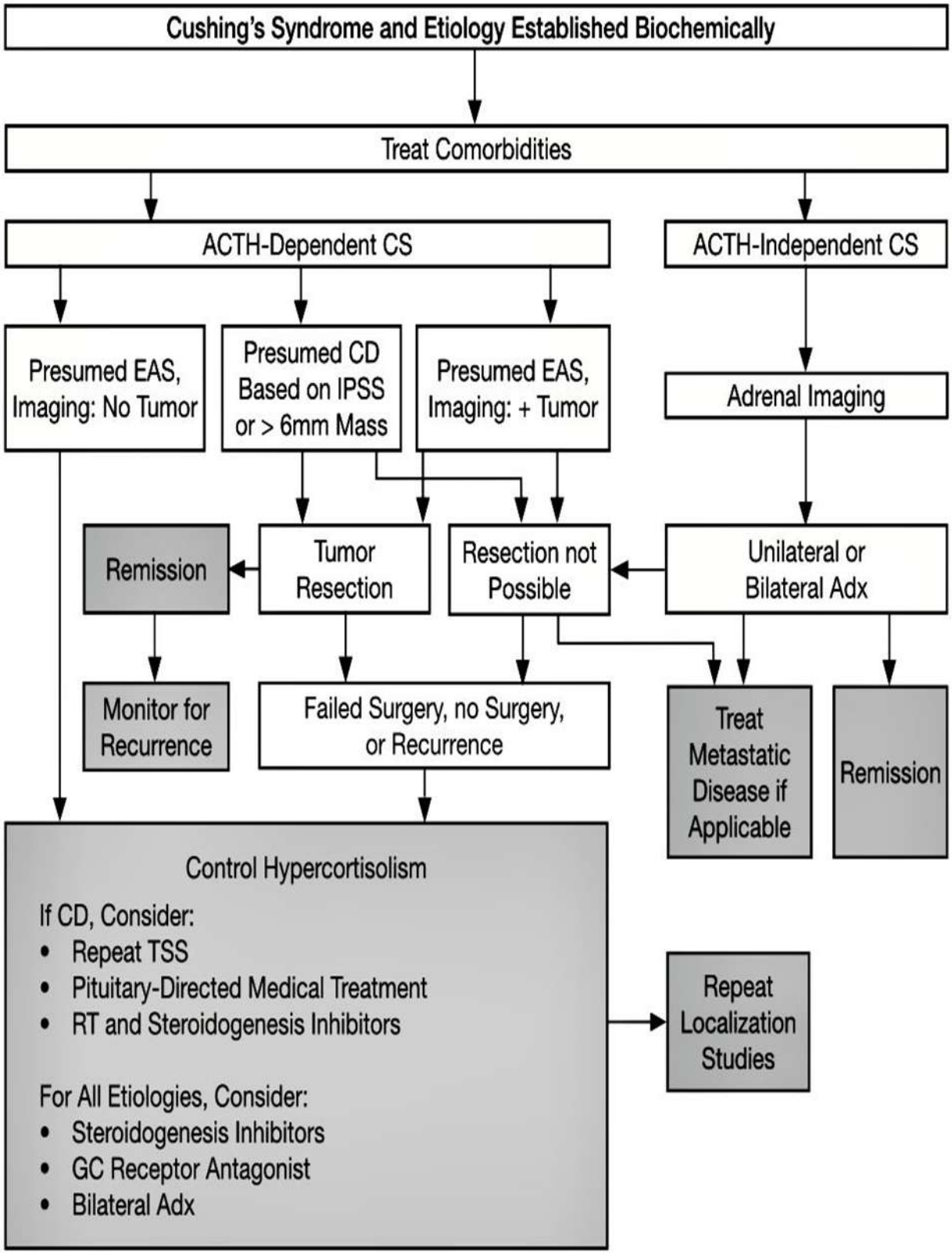
Fig 05 : An algorithm for the treatment of Cushing's syndrome[8]



Case Presentation –

A 43-year-old female patient was admitted to vijayanagara institute of medical science (VIMS), Ballari (Karnataka) with chief complaints of swelling of lower limbs since 15 days which is insidious onset and

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progressive in nature, which is till the mid-part of the leg, cough with expectoration since 4 days sputum which is non-blood tinged, the patient had moon facies and facial hyperpigmentation in prednisolone-induced Cushing syndrome (Fig-1,2)

Fig 01 - Facial Hirsutism due chronic prednisolone use.

Fig 02 - Moon facies due chronic prednisolone use.

Past History – k/c/o Distal RTA secondary to Sjogren's syndrome, Chronic myopathy on Tab.Prednisolone 40mg BD, Tab.HCQ 200mg BD, Syp.Potassium chloride 20ml TID.

Family history – Nothing significant.

On examination –

1. BP - 100/80 mmHg.
2. PR – 105 bpm.
3. SPO2 - 98% decreased at RA.

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4. GRBS - 85mg/dl.
5. Patient was conscious and oriented.
6. B/L AE +ve B/L Crepts +ve
7. S1 S2 heard.
8. P/A was soft and non-tender.

Laboratory investigations –

PARAMETERS	RESULT (03-01-26)	RESULT (04-01-26)	RESULT (05-01-26)	RESULT (06-01-26)	REFERENCE RANGE
Haemoglobin	12.7			6.4	12.5-16gm
WBC	14110			7000	4000-11000cells/cumm
RBC	4.54			3.28	4.5-5.5millions/cumm
Neutrophils	90			75	40-70%
Lymphocytes	06			15	20-40%
PCV	39.7			23.7	35-46%
MCV	87.5			72.3	60-100fl
MCH	28.2			19.5	27-34pg
MCHC	32.2			27.0	31-36%
RDW-CV	15.2			14.7	11.5-14.5%
PDW-CV	15.7			8.4	10-18%
Sodium	135		137		136-145mEq/L
Potassium	3.4		3.6		3.48-5mEq/L
Albumin	3.0				3.2-5.4g/dl
Globulin	3.3				2.5-3g/dl
ALP	343				20-140U/L
Cortisol	9.54	7.59			133-537nmol/L
Urine protein-creatinine ratio	321.1				10-150mg/g
TSH			4.88		0.27-4.2mclU/ml
FT4			0.69		0.92-1.68ng/dl

Other investigations –

Abdomen and Pelvis :- Left ovarian cyst.

Treatment chart

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SL.NO	NAME OF MEDICATIONS	DOSE	ROUTE	FREQUENCY
1.	INJ.FUROSEMIDE	20mg	IV	BD FOR 3 DAYS
2.	TAB.HCQ	200mg	PO	BD FOR 4 DAYS
3.	TAB.PREDNISOLONE	20mg	PO	OD FOR 4 DAYS
4.	INJ.CEFTRIAXONE	1gm	IV	BD FOR 4 DAYS
5.	CAP.DOXYCYCLINE	100mg	PO	BD FOR 4 DAYS
6.	SYP.AMBROXOL	5ml	PO	TID FOR 4 DAYS
7.	SYP.POTASSIUM CHLORIDE	15ml	PO	TID FOR 4 DAYS
8.	TAB.PARACETAMOL	500mg	PO	TID FOR 4 DAYS
9.	TAB.CALCIUM	500mg	PO	OD FROM DAY2 TO DAY4
10.	TAB.SODIUM BICARBONATE	500mg	PO	OD FROM DAY2 TO DAY4
11.	NEB.DOLIN			TID FROM DAY3 AND DAY 4

Discharge medication

SL.NO	NAME OF MEDICATIONS	DOSE	ROUTE	FREQUENCY
1.	TAB.AMOXYCLININE	625mg	PO	BD FOR 5 DAYS
2.	SYP.POTASSIUM CHLORIDE	15ml	PO	TID FOR 15 DAYS
3.	T.HCQ	200mg	PO	BD FOR 15 DAYS
4.	TAB.PREDNISOLONE	20mg	PO	OD FOR 15 DAYS
5.	TAB.CALCIUM	500mg	PO	OD FOR 15 DAYS
6.	TAB.PARACETAMOL	500mg	PO	TID FOR 15 DAYS
7.	TAB.TRAMADOL	100mg	PO	BD FOR 15 DAYS

Discussion

Cushing's syndrome encompasses a spectrum of clinical and biochemical abnormalities resulting from chronic exposure to excess glucocorticoids. Among its etiological subtypes, exogenous (iatrogenic) Cushing's syndrome is the most prevalent, predominantly attributable to prolonged administration of synthetic corticosteroids. The present case illustrates a typical yet clinically significant manifestation of prednisolone-induced Cushing's syndrome in a patient receiving long-term

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high-dose glucocorticoid therapy for autoimmune and renal comorbidities.

The patient had been maintained on prednisolone 40 mg twice daily for chronic myopathy associated with Sjögren's syndrome and distal renal tubular acidosis. Sustained exposure to supraphysiological glucocorticoid doses leads to suppression of the hypothalamic–pituitary–adrenal (HPA) axis through negative feedback mechanisms, resulting in reduced endogenous cortisol secretion while simultaneously inducing systemic features of glucocorticoid excess. The clinical findings observed in this patient— moon facies, facial hirsutism, peripheral edema, muscle weakness, and cutaneous hyperpigmentation — are characteristic of chronic hypercortisolism and strongly support the diagnosis of exogenous Cushing's syndrome.

Laboratory evaluation revealed low serum cortisol levels, which is a characteristic finding in exogenous Cushing's syndrome, distinguishing it from endogenous causes where cortisol levels are elevated. This paradoxical biochemical profile underscores the importance of correlating hormonal assays with medication history and clinical presentation. Furthermore, the presence of hypokalemia, hypoalbuminemia, elevated alkaline phosphatase levels, and proteinuria reflects the widespread metabolic, renal, and catabolic effects of chronic glucocorticoid exposure.

Das et al. focused on biochemical confirmation using low morning cortisol levels and supportive imaging findings such as demyelinating lesions on MRI, suggesting systemic steroid toxicity. Our article expands upon this by incorporating serial cortisol measurements, thyroid function abnormalities, electrolyte imbalance, and proteinuria, thereby providing a more comprehensive endocrine and metabolic assessment. This broader diagnostic approach aligns more closely with current guideline-based evaluation of suspected Cushing's syndrome, where no single clinical or biochemical parameter is considered diagnostic in isolation.

Management of exogenous Cushing's syndrome primarily involves gradual tapering of the offending glucocorticoid to prevent adrenal insufficiency while maintaining adequate control of the underlying disease. In this case, the prednisolone dose was appropriately reduced, and supportive therapy including potassium supplementation, calcium, diuretics, and treatment of intercurrent infection was instituted. Abrupt discontinuation of glucocorticoids was avoided, given the risk of acute adrenal crisis due to chronic HPA axis suppression.

Conclusion

This case emphasizes the critical importance of rational prescribing and regular reassessment of long-term corticosteroid therapy, particularly in patients with chronic inflammatory and autoimmune disorders. Early recognition of iatrogenic Cushing's syndrome, prompt dose adjustment, and implementation of steroid-sparing strategies where feasible are essential to minimizing morbidity and preventing irreversible complications.

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