Case Report

ANNALS OF MEDICINE & SURGERY

OPEN

Factitious cushing's syndrome with unusual presentation: a case report and literature review

Almotazbellah M. M. Zeer, MD[a,*], Mahmoud Noman, MD[a], Zahraa M. M. Zeer, MD[a], Alaa Attawneh, PhD[b], Zinah A. Bairmani, MD[e], Khadeejeh M. A. Alfroukh, MD[c], Layth Oweina, MD[d]

Introduction: Munchausen syndrome is known as a factitious disorder imposed on the self. Factitious Cushing's syndrome (CS) is a very rare form of Munchausen syndrome, presenting with varied clinical and biochemical features, making diagnosis challenging.
Case Presentation: A 40-year-old female patient who worked as a registered nurse presented with clinical features of CS but denied any exogenous corticosteroid use. The endocrine workup revealed that the patient had a high 24 h urinary-free cortisol collection before admission. Subsequent evaluations showed low levels of morning cortisol and plasma adrenocorticotropic hormone along with a suppressed overnight low-dose dexamethasone suppression test, leading to an investigation of hypercortisolism. Unexpectedly, subsequent testing showed a normal 24 h urinary-free cortisol level. Additionally, the patient was diagnosed with panhypopituitarism, the radiological investigations showed normal pituitary and adrenal glands. Despite consistently denying the use of corticosteroids, it was finally discovered that the patient had been surreptitiously taking prednisone and receiving multiple dexamethasone injections over the past few months. The patient received treatment through a gradual prednisone tapering regimen, accompanied by comprehensive psychiatric evaluation and management.
Conclusion: This case underscores the exceptional rarity of factitious CS and emphasizes the importance of considering it as a potential differential diagnosis in hypercortisolism cases, particularly when the patient's medical history contradicts investigative findings. Furthermore, it highlights the criticality of adopting a multidisciplinary approach to investigate patients whose clinical presentation aligns with factitious CS.

Keywords: adrenal gland, case report, cushing syndrome, factitious syndrome, hypercortisolism

Introduction

Factitious Cushing's syndrome (CS) is an exceptionally rare form of Munchausen syndrome, a psychiatric disorder in which individuals intentionally produce or feign physical or psychological symptoms for the purpose of assuming the sick role[1]. Factitious CS is a rare and challenging disorder in which an individual deliberately exaggerates or fabricates symptoms of CS[2].

This type of factitious disorder is characterized by self-inflicted injuries, the use of glucocorticoid drugs[3], or the manipulation of laboratory tests to produce false results[4].

Diagnosing Factitious CS is critical as it can lead to unnecessary and potentially harmful treatments, including surgical procedures to remove adrenal glands or pituitary tumors[5]. Furthermore, this syndrome can mimic actual CS, leading to delays in diagnosing the underlying medical conditions[6].

In this case report, we present the case of a 40-year-old female who presented with features of Cushing's, including a moon face, easy bruising, and central obesity. Laboratory investigations revealed panhypopituitarism and decreased levels of cortisol and adrenocorticotropic hormone (ACTH), which initially suggested

HIGHLIGHTS

- Factitious Cushing's syndrome (CS) is a very rare form of Munchausen syndrome in which an individual deliberately exaggerates or fabricates symptoms of CS. This entity is exceptionally rare, with a few cases reported in the literature.
- We highlight the importance of considering Factitious CS in the differential diagnosis of patients with unexplained features of Cushing's syndrome.
- Early diagnosis of Factitious CS helps with saving time, money, and resources and decreases mortality and morbidity from excess steroid use.
- Laboratory findings can provide important diagnostic information and should raise suspicions of a factitious disorder if certain abnormalities are present.
- We highlight the importance of a team-based approach with interdisciplinary collaboration between endocrinology and psychiatry for the optimal management plan.

[a]Faculty of Medicine, Al-Quds University, [b]Endocrinologist at Augusta Victoria Hospital, Jerusalem, [c]Internal Medicine - Alhli Hospital, Hebron, Palestine, [d]Psychiatry at Bethlehem Psychiatric Hospital, UK and [e]Ain Shams University, Cairo, Egypt

Sponsorships or competing interests that may be relevant to content are disclosed at the end of this article.

*Corresponding author. Address: Al-Quds University, Dura, Hebron, Palestine, State Of. Tel.: +970 597 38 7212. E-mail: almotazalzeer@gmail.com (A.M.M. Zeer).

Annals of Medicine & Surgery (2023) 85:4161–4166

Received 18 May 2023; Accepted 1 July 2023

Published online 14 July 2023

http://dx.doi.org/10.1097/MS9.0000000000001050

a pituitary or adrenal disorder. However, further evaluation led to the diagnosis of Factitious CS.

This case highlights the importance of considering Factitious CS in the differential diagnosis of patients with unexplained features of CS. We discuss the clinical presentation, diagnostic workup, and management of this rare and challenging syndrome with the aim of raising awareness among clinicians and improving the overall care of affected patients.

This case was written according to the Surgical CAse REport (SCARE) Criteria 2020[7].

Case presentation

A case report regarding a 40-year-old female patient, who has been married, and lived with her family in poor socioeconomic status. She had worked as a registered nurse for 20 years.

The patient's medical history was significant for a recent diagnosis of hypertension, psychogenic seizures diagnosed after neurological evaluation 3 years ago, and migraines diagnosed 10 years ago. Additionally, the patient had a history of menstrual irregularities with menorrhagia and underwent a total hysterectomy a year ago. Furthermore, 3 months ago, she underwent a bilateral mastectomy due to ductal carcinoma in situ and granulomatous mastitis.

The patient was admitted to the hospital due to the development of a cushingoid appearance. Notably, she reported a significant weight gain of 20 kg over the past 4 months, exhibiting a central distribution pattern of body fat, facial puffiness, frequent headaches, and spontaneous bruising on her extremities. She also experienced intermittent episodes of pleuritic chest pain that increased with respiration and movement, associated dyspnea that increased upon lying down, fatigue, emotional lability, and sleep disturbance. Furthermore, the patient reported being easily agitated and had a history of self-mutilating behaviors when stressed.

An unusual erythematous skin rash that stained her clothes and tissue paper was noted during the examination. The rash was characterized by red, flat patches on the skin with a distinct pattern resembling streaks or lines. The rash was mainly distributed on her face, upper extremities, and anterior chest. The patient denied any known allergies or irritant exposure but refused to disclose any information about potential exposure to dyes or other chemicals.

The patient stated that she did not experience any symptoms of hirsutism, voice deepening, hair thinning, or temporal balding. She also confirmed that she did not smoke or drink and did not exhibit any clinical signs of depression. Furthermore, she consistently denied taking prescribed glucocorticoids or herbal supplements. The patient's medications included amlodipine, doxazosin, omeprazole, topiramate, and levetiracetam.

During the physical examination, manifestations of CS were noticeable. The patient displayed a centripetal accumulation of fat in the trunk and abdomen, which included a prominent dorsocervical fat pad and a full, flushed appearance known as a moon face. Additionally, she had enlarged fat pads that filled the supraclavicular fossae. Upon examining the skin, an erythematous rash was noted, multiple reddish striae were visible on the lower abdomen and chest, and bruises and scratches were present on the lower extremities. Furthermore, the patient had grade 2 pitting edema.

The patient's blood pressure was recorded as 157/96 mmHg, with a heart rate of 89 BPM, a weight of 95 kg, and a height of 170 cm.

The laboratory results indicated a hemoglobin level of 13.5 g/dl, a white cell count of 24.0 10e3/μl with 87% neutrophils and 8% lymphocytes in the differentiation, a creatinine level of 0.65 mg/dl, a sodium level of 134 mmol/l, a potassium level of 3.7 mmol/l, a fasting blood sugar level of 201.3 mg/dl, and a HbA1C level of 6.1.

The patient had a high 24 h urinary-free cortisol collection before admission, and an abdominopelvic computed tomography scan revealed the normal size and appearance of the adrenal glands, which are isodense to the liver with no mass or abnormal findings. Upon initial investigation, the patient was found to have low levels of morning cortisol (1.91 μg/dl) and plasma ACTH levels of less than 1.50 pg./ml, along with a suppressed overnight low-dose dexamethasone suppression test. However, subsequent testing showed a normal 24 h urinary-free cortisol level (33 mcg/24 h), which argued against endogenous CS and suggested the possibility of exogenous glucocorticoid use[8]. Additionally, the patient was found to have panhypopituitarism, with low levels of thyroid-stimulating hormone, free thyroxine, free triiodothyronine, and suppressed prolactin levels but normal gonadotropins. A brain MRI scan showed normal shape, size and signal intensity of the pituitary stalk, and anterior and posterior pituitary lobes with homogenously enhancing and no focal lesions after IV contrast injection (Fig. 1).

The patient was confronted with evidence that her high steroid levels were not endogenously produced and initially became defensive. She became angry and accused doctors of thinking she had a mental illness, insisting that her concerns were legitimate. However, she eventually confessed to taking prednisone 40 mg daily for the past 4 months and receiving multiple dexamethasone injections over the past few months.

The patient received a psychiatric evaluation where she exhibited a blunted affect with low mood, expressed reduced pleasure in socializing, and insomnia. She was diagnosed with factitious disorder, functional neurological symptom disorder (conversion disorder), and dependent personality disorder. It is worth noting that although a connection between factitious and borderline personality disorder has been established in the literature, not much can be found regarding dependent personality disorder[9].

Regarding the management plan, the patient received regular follow-up care with her endocrinologist and was initially prescribed a tapering course of prednisone. Later, she was switched to a daily dose of oral hydrocortisone 10 mg, because of the persistent suppression of the hypothalamic-pituitary-adrenal (HPA) axis[10], which resulted in symptomatic improvement and normalization of laboratory abnormalities. In addition, a psychiatric re-evaluation was performed, and treatment with a serotonin reuptake inhibitor antidepressant (sertraline 50 mg daily) was commenced for 6 months.

Discussion

Munchausen syndrome is known as a factitious disorder imposed on the self according to the DSM-5 criteria. It usually presents with an atypical presentation not supported by the clinical examination including physical examination, laboratory test, or

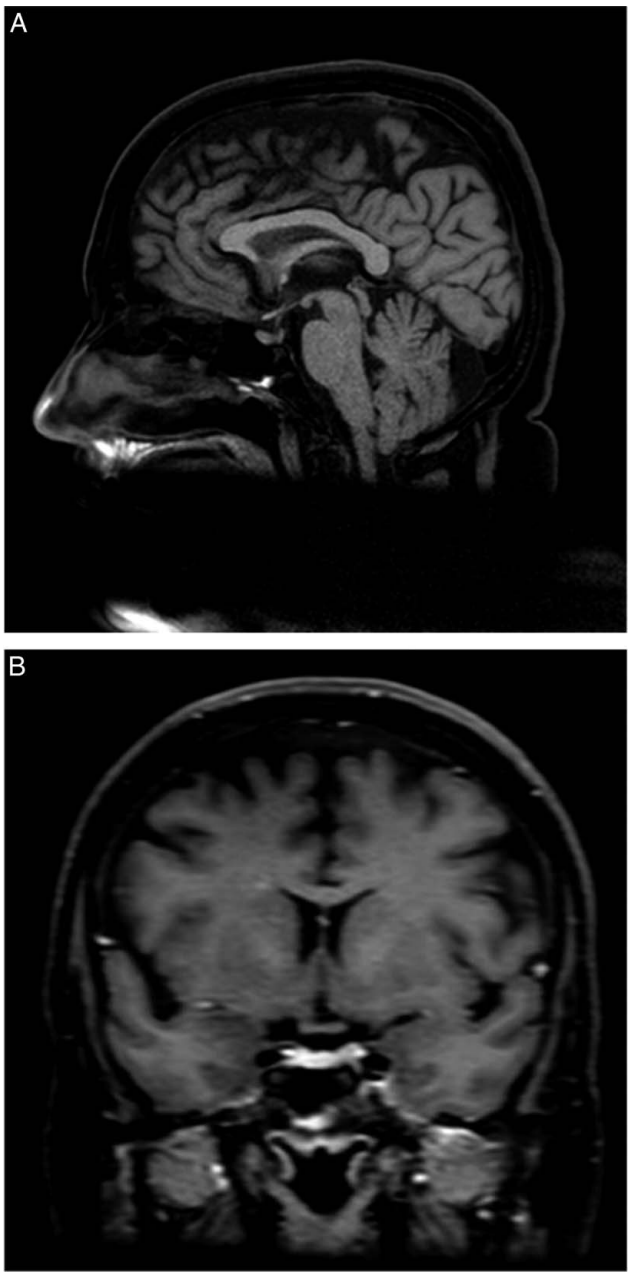

Figure 1. MRI on T1-weighted of the brain, (A) Sagitta view, (B) Coronal view; showed normal shape, size and signal intensity of the pituitary stalk, and anterior and posterior pituitary lobes.

imaging[11]. Patients with factitious illnesses tend to produce physical or psychological symptoms intentionally for internal incentives and to receive medical attention[4]. Clinical features of the factitious disorder are diverse, but most documented cases are women under the age of 30 who have a generally stable social life and work in or have access to the healthcare field, have a high rate of substance addiction, and have mood or personality disorders[12]. Medical records aid in the diagnosis of these patients because they tend to have a history of childhood illnesses and operations, an atypical clinical course of disease or healing, and unique clinical findings or laboratory results[13].

In the literature, the surreptitious administration of several hormones has been reported. Examples include catecholamine injections mimicking pheochromocytoma, thyroid hormone administration mimicking thyrotoxicosis, and insulin administration mimicking insulinoma[1]. Despite the extensive use of glucocorticoids in medicine, factitious CS cases are rare, accounting for less than 1% of all Cushing disorder cases[3].

Maria M Pineyro *et al.* conducted a literature review of 23 patients with factitious CS and found that 82% were female with a median age of 37 years old. Among them, 57% had a history of psychiatric disorders, and 43% had contact with the medical field[4]. A summary of the previously reported cases in the last 10 years is listed in Table 1.

The diagnosis of factitious CS presents a challenge as physical symptoms are largely indistinguishable between endogenous and exogenous forms of the condition, while psychiatric symptoms are of limited utility due to their high prevalence in nonfactitious cases. However, a detailed patient history may offer clues to the presence of a factitious disorder[1].

Laboratory findings can provide important diagnostic information and should raise suspicions of a factitious disorder if certain abnormalities are present. For instance, low levels of cortisol in both urine and serum in the absence of adrenal insufficiency symptoms[1], as well as discrepancies in cortisol levels between the two, are significant clues[3]. Additionally, low levels of serum corticotropin, 17-hydroxyprogesterone, progesterone, dehydroepiandrosterone sulfate, and 17-hydroxypregnenolone may also indicate a factitious disorder[15]. It should be noted that while ACTH levels are expected to be low in patients receiving steroid treatment, certain radioimmunoassay tests for ACTH may lack specificity, leading to inaccurate results[16].

The case at hand displays numerous similarities to previously reported cases, including working in the medical profession, recent surgeries, numerous outpatient subspecialist visits for a wide range of complaints, including headaches, skin changes, spontaneous bruising, and episodes of rage, as well as a history of psychogenic seizures and a concealed a history of self-mutilation, which her spouse subsequently revealed. In addition, hypopituitarism as one of the multiple heterogenous presentations of exogenous steroid administration is also reported by Cannavo and Cannavo[2] case report. However, the diagnosis of factitious Cushing's was initially overlooked due to a negative history of corticosteroid exposure and the confounding effects of synthetic steroids on cortisol measurements.

The degree of cross-reactivity between synthetic and endogenous glucocorticoids can vary depending on the timing of ingestion and the patient's endogenous cortisol secretion. Prednisone, an inactive metabolite, is rapidly converted to its active form, prednisolone, which cross-reacts with cortisol[16]. As such, liquid chromatography-tandem mass spectrometry (LC-MS/MS) is considered the most reliable diagnostic method for confirming factitious CS, as it enables the measurement of synthetic glucocorticoid levels alongside concomitant low cortisol levels and measures the level of glucocorticoid precursors like corticosterone, which tend to be high in cases of endogenous hypercortisolism[14]. Unfortunately, our initial request for a synthetic steroid test was denied due to its unavailability. However, alternative means were sought, and observations by a nurse on the ward led to the discovery of prednisone tablets in the patient's medicine cabinet. Additionally, the patient showed the disappearance of the rash after taking a shower, and a review of

Table 1

Summary of the previously reported cases in the last 10 years

	Age/ Sex/ Marital status	Close contact with medical profession (patient or family member)	Main symptoms of hypercortisolism	Psychiatric symptoms	Medical diseases or multiple surgeries unrelated to Cushing syndrome	DST	24-hour urine test for cortisol	Random Serum Cortisol/ Random urine-free cortisol / Random Late-night salivary cortisol	Plasma ACTH	Pituitary MRI/ Adrenal CT	Medication ingested
1[2]	52/F/ Married	NA	cushingoid features, bruising, headache, myalgia, fatigue	self-induced skin lesions, Münchausen syndrome	No	NA	Low*	Serum cortisol: low	Low	Brain MRI: 4 mm hypointense lesion in the anterior pituitary gland/ Adrenal CT: non	betamethasone
2[4]	26/F/single	She has a brother who works as a nurse	Cushingoid features, facial plethora, muscle weakness	dependent and histrionic personality disorder	orofacial granulomatosis	Paradoxical increase	High	Urine cortisol: Normal	Low	Pituitary MRI: Non/ Adrenal CT: Normal	prednisone and prednisolone
3[14]	32/F/NA	The patient is a nursing student	Wight gain and fatigue	NA	laparoscopic banding surgery	Paradoxical increase	NA	Serum Cortisol: high salivary cortisol: high	Normal	Normal/Normal	prednisone
4[3]	54/F/NA	No	Cushingoid features, weight gain, proximal myopathy, Hypertension, deteriorating glycemic control	No	Asthma, recent distal fibula fracture	CRH stimulation suggests ACTH-dependent Cushing syndrome	High	Serum Cortisol: normal salivary cortisol: high	Normal	Normal/Normal	prednisolone
5[5]	26/F/ Married	NA	Cushingoid features, weight gain, proximal myopathy,	Complex familial relationship, defense mechanisms of Projection, negation, somatization and reactive reactions.	Macroprolactinoma, which was treated by Transsphenoidal surgery	NA	High	Serum Cortisol: normal salivary cortisol: high	Normal	Adrenal CT: Bilateral atrophic glands Pituitary MRI: a small nodular cystic lesion	serum prednisone and prednisolone
Our case	40/F/ Married	The patient is a registered nurse	Weight gain, fatigue, easy bruising, irritability, muscle weakness	Factitious disorder, functional neurological symptom disorder (conversion disorder), and dependent personality disorder.	Bilateral mastectomy, total hysterectomy	Suppression	normal	Serum cortisol: low	Low	Normal/Normal	prednisone

*Associated: hypopituitarism; Central hypothyroidism, and hypogonadotropic hypogonadism.

CRH, Corticotropin-releasing Hormone; DST, Dexamethasone suppression test; F, Female.

her abdominal CT scan revealed fat stranding in the buttocks area consistent with multiple injections. Despite being repeatedly questioned about her medical history, the patient denied exposure to various medications. These observations provided significant clues to suggest that the patient had been falsifying symptoms, providing misleading information, and omitting details related to her condition.

The most important differential diagnosis for factitious CS is cyclic CS, an unusual variant of CS characterized by periods of high cortisol levels interspersed with periods of normal cortisol levels[17].

This rare disease is more common in elderly individuals in their fifth to sixth decade with a female-to-male ratio of 3:1. Multiple diagnostic criteria have been suggested for cyclic CS, including two peaks and one trough suggested by Krystallenia *et al.* and three peaks and two troughs suggested by Meinardi *et al.* Further studies are needed to evaluate the sensitivity and specificity of these different diagnostic criteria in large sample studies[18].

The pathophysiology of cyclic CS is not yet fully understood, but several theories have been suggested, including spontaneous episodic hemorrhage or necrosis of the tumor, the serotonin effect, central dopamine tone changes, and the properties of inflammatory cytokines as an antitumor effect[19].

Cyclic CS can be ACTH-dependent or independent. The most common cause is an ACTH-secreting pituitary adenoma, followed by ectopic ACTH secretion, most commonly associated with carcinoid syndrome. Adrenal origin is the least common, typically presenting as an adenoma or a rare disease known as primary pigmented nodular adrenocortical disease[17].

Periodic CS and factitious CS have wide fluctuations in plasma or urine cortisol levels and ACTH. The periodicity of the fluctuations is the most important factor for distinguishing between the two conditions, with patients with periodic CS exhibiting fluctuations over days to months, while those with factitious CS typically exhibit rapid fluctuations, often appearing only when under supervision[5].

Another differential diagnosis to consider is pseudo-Cushing states (PCS); this diagnosis poses a real diagnostic challenge. Patients with pseudo-Cushing have signs and symptoms similar to those with CS. Both conditions are related to prolonged exposure to corticosteroids, but pseudo-Cushing patients have nonendocrine causes that affect the HPA axis. Severe depression and prolonged alcohol exposure are important predisposing causes for pseudo-Cushing. History in these cases should not be underestimated when interpreting laboratory values[20]. PCS patients have persistently elevated cortisol levels throughout the day, but they do not lose the normal cortisol diurnal pattern as opposed to patients with CS. Based on this assumption, late-night salivary cortisol seems better than low-dose dexamethasone or urine-free cortisol in differentiating Cushing from PCS patients[21]. Moreover, PCS patients with depression do not have suppression with a low-dose dexamethasone suppression test compared to our patient[22].

The treatment for factitious CS mainly involves a non-confrontational approach, as confrontation can lead to denial and the patient seeking medical care elsewhere[4]. Our patient agreed with the statement that 'sometimes we treat inner stress by inflicting physical illness on ourselves', but she refused to acknowledge that she had any conscious or subconscious mental disorder. Currently, she is following up with a neurologist for her migraine. She was instructed to take amitriptyline 25 mg once daily before bed and gabapentin 300 mg once daily and to stop all other medications. For concurrent functional neurological disorders, physical therapy is helping under the theory of 'somatic complaint, somatic treatment'[23].

Additionally, due to long-term corticosteroid use, the HPA axis is suppressed, so sudden withdrawal of steroids should be avoided, and replacement doses are necessary to prevent secondary adrenal insufficiency[1].

Conclusion

In our case, we highlight the importance of considering factitious CS as a differential diagnosis of hypercortisolism when clues in history, laboratory findings, and environmental investigation indicate exogenous corticosteroid administration, especially when LS-MS/MS as the most important diagnostic method is not available. Additionally, we highlight the importance of the multidisciplinary approach involving the endocrinology, radiology, and psychiatry specialists in uncovering factious CS and managing the patient's condition effectively.

Ethical approval

This study is exempt from ethical approval in our intuition.

Consent

Written informed consent was obtained from the patient for the publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Sources of funding

The study did not receive any financial help.

Author contribution

A.Z. and M.N.: Data collection; A.Z., Z.M.M.Z., Z.A.B., and K.M. A.A.: study concept or design; A.Z., K.M.A.A., Z.M.M.Z., and M.N.: writing the manuscript; A.Z., Z.A.B., Z.M.M.Z.: review and editing the manuscript; L.O.: psychiatric evaluation and follow-up; A.A.: endocrinology, evaluation, and follow-up.

Conflicts of interest disclosure

The authors have no conflict of interest to declare.

Research registration unique identifying number (UIN)

1. researchregistry9038.
2. Researchregistry.com.
3. https://www.researchregistry.com/browse-the-registry#home/

Guarantor

Dr Alaa Attawneh.

Acknowledgements

The authors are thankful to the patient and his family for their great cooperation.

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