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Severe Exogenous Cushing's Syndrome with Femoral Avascular Necrosis and Pulmonary Embolism Following Use of an Unregulated "Herbal" Weight-Gain Powder: A Case Report

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Case Report

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Abstract

Exogenous Cushing's syndrome (ECS) can result not only from prescribed glucocorticoids but also from unregulated "herbal" products adulterated with potent steroids. Such hidden exposures may lead to severe metabolic, musculoskeletal, and thromboembolic complications. We report a 27-year-old man who developed rapid weight gain, violaceous striae, neuropsychiatric changes, and progressive hip pain after ingesting an unlabeled weight-gain powder purchased from a local herbalist. Imaging revealed multifragmentary femoral head destruction consistent with avascular necrosis, and CT angiography confirmed acute pulmonary embolism. Osteoporosis on bone density testing further supported chronic glucocorticoid exposure. Due to resource limitations, chemical analysis of the powder could not be performed. This case highlights the serious risks of unregulated "herbal" supplements and underscores the importance of considering hidden glucocorticoid exposure in patients with unexplained Cushingoid features. Greater regulatory oversight and public awareness are essential to prevent similar cases.

Introduction

Exogenous Cushing's syndrome (ECS) is the most frequent cause of hypercortisolism and typically results from long-term exposure to glucocorticoids given by oral, topical, inhaled, or injectable routes (1–3). In recent years, an important novel source has been unregulated over-the-counter products marketed as "herbal" or "natural," which may contain undisclosed corticosteroids potent enough to produce clinically significant adrenal suppression (4). Patients affected by unrecognized steroid ingestion may develop classic Cushingoid features, rapid weight gain, violaceous striae, muscle weakness, and psychiatric changes, as well as major complications such as osteoporosis, atraumatic fractures, avascular necrosis, and thromboembolic events (5). Because these products are often supposed as safe, diagnosis can be delayed (6). These cases highlight the role of social determinants of health, including reliance on traditional remedies, limited access to regulated healthcare, and widespread availability of unmonitored supplements. The present case involves a young man who developed severe ECS with femoral avascular necrosis and pulmonary embolism after consuming an unlabeled weight-gain powder bought from a local herbalist, highlighting the notable risks caused by such unregulated products. This manuscript conforms to the CARE guidelines for the reporting of case reports (7).

Case Presentation

A 27-year-old male with no prior medical history presented with progressive right femoral pain, generalized weakness, and significant behavioral changes. Eight months before admission, he had begun consuming herbal preparations bought from a local herbalist with the goal of gaining weight. Following regular ingestion of these products, he experienced an unexplained 35-kg weight gain. Over the subsequent months, he developed increasing fatigue, emotional instability, and cognitive disturbances, eventually exhibiting disorganized thoughts and episodes of agitation. Upon admission to the internal medicine ward, he appeared weak and obese, with marked abdominal striae suggestive of hypercortisolism. Figure 1 is a clinical photograph (with patient consent) or an illustration of the striae.

Shortly after hospitalization, his mental status worthened further; he became increasingly irritable and aggressive, which required psychiatric assessment and patient transfer. His psychiatric profile was consistent with Paranoid disorder, OCD, cluster B personality features, and body integrity identity disorder, in the context of a documented opium use disorder. During his hospital stay, he repeatedly complained of dyspnea. Initially, oxygen saturation was within normal limits, but it subsequently declined to below 90%. Spiral CT angiography of the chest revealed central filling defects in the lobar and segmental branches of the left lower lobe, consistent with acute pulmonary thromboembolism, accompanied by pleural effusion, basal consolidations, and patchy ground-glass opacities in both lungs. Anticoagulation therapy was promptly initiated. In addition to his respiratory symptoms, the patient reported severe right hip pain. Three-dimensional pelvic CT with reconstruction showed a multi-fragmentary fracture and dislocation of the femoral head, findings that were not consistent with any reported trauma. Given the suspicion for steroid-induced bone disease, bone mineral density testing confirmed the presence of osteoporosis. The combination of rapid weight gain, prominent striae, neuropsychiatric changes, and decreased bone density raised strong possibility of Cushing syndrome (CS), most likely due to exogenous corticosteroid contamination in the herbal supplements he had consumed. A detailed hormonal evaluation was undertaken. Morning plasma cortisol was markedly suppressed (< 0.05 µg/dL), and adrenocorticotropic hormone (ACTH) levels were also abnormally low (< 5 pg/mL; reference range 10–56 pg/mL). In addition, 24-hour urine-free cortisol concentrations measured 1.1 µg/24 h, significantly below normal limits. These findings confirmed suppression of the hypothalamic–pituitary–adrenal (HPA) axis, most consistent with prolonged exogenous corticosteroid exposure from the herbal preparations. All hormonal assays were repeated on multiple occasions during the course of hospitalization, yielding consistent results. Ultrasound evaluation of the hepatobiliary system revealed a liver of normal size but with increased echogenicity, compatible with grade I hepatic steatosis, while the gallbladder and biliary ducts appeared normal. Because of lower-limb swelling, a vascular surgery consultation was obtained. Assessments revealed no evidence of deep vein thrombosis, and no surgical intervention was required. The patient's acute issues, including pulmonary embolism and psychiatric instability, were stabilized medically. Plans were made for elective orthopedic management of the right femoral fracture-dislocation on an outpatient basis. Further endocrine evaluation was recommended to confirm the diagnosis of hypercortisolism and identify potential exogenous sources in the herbal products he had been using. During follow-up over the subsequent three months, the patient showed partial clinical improvement after discontinuation of the suspected herbal powder and initiation of supportive care. His psychiatric symptoms, including agitation and disorganized thought patterns, gradually stabilized with antipsychotic therapy and structured outpatient follow-up. However, right hip pain persisted, and repeat imaging confirmed progressive collapse of the femoral head, for which he was scheduled for elective orthopedic intervention. Although his cushingoid features began to lessen, morning cortisol levels remained suppressed, suggesting ongoing hypothalamic–pituitary–adrenal axis impairment. The patient was advised to continue endocrine follow-up, avoid all non-prescription supplements, and undergo rehabilitation for functional mobility.

Discussion

This case highlights a severe presentation of exogenous CS associated with the ingestion of an unlabeled “herbal” weight-gain powder obtained from a local herbalist. Similar to numerous reports documenting corticosteroid contamination in unverified natural treatments our patient’s clinical course, strongly suggests chronic exposure to a potent glucocorticoid. Multiple studies support the plausibility of this mechanism. For example, Patel et al. (8) described two patients who developed ECS after consuming the contaminated Over The Counter (OTC) supplement *Artri King*, later confirmed to contain dexamethasone. Their findings align closely with our patient's multisystem complications, reinforcing the likelihood of similar hidden steroid content in the powder he consumed. Similarly, Franke et al. (9) reported Cushingoid features resulting from a “herbal” ointment that subsequent laboratory testing revealed to contain glucocorticoids, demonstrating how undisclosed corticosteroid contamination in non-approved formulations can lead to significant systemic toxicity. These studies provide strong external support for our suspicion of exogenous hypercortisolism originating from the patient’s powder. More directly similar to our case, several authors have reported musculoskeletal and systemic complications mirroring those observed in our patient. A recent systematic review examining adverse events linked to *Artri King* documented not only adrenal suppression but also osteonecrosis and fragility fractures, findings nearly identical to the multifragmentary femoral head collapse in our patient (10). These similarities support the hypothesis of chronic steroid exposure as the underlying pathology.

However, other reports highlight detailes that differ from our case. For instance, Insalaco et al. (11) described iatrogenic CS in a pediatric patient using long-term intranasal corticosteroids, where the steroid source was fully known and dosage quantifiable. In contrast, our case involves an unknown formulation, which introduces diagnostic uncertainty and underscores the challenge of confirming exposure when products are unlabeled. Similarly, cases involving topical herbal creams adulterated with clobetasol (2) and intranasal betamethasone drops (1), demonstrate that systemic absorption can occur even with formulations typically considered low-risk. In contrast, our patient ingested the substance orally, which may result in greater and more rapid systemic bioavailability, highlighting the particularly severe risk associated with oral intake of unregulated powders.

The report by Wei et al. (12), examining the broader impact of OTC steroid-containing preparations on the development of ECS, also reinforces the clinical relevance of our findings. Their work highlights the diversity of routes and formulations capable of inducing hypercortisolism, consistent with our observation that even products marketed for weight gain can carry significant systemic toxicity. At the same time, they describe cases in which laboratory confirmation of the offending product was feasible, an advantage we did not have.

Indeed, one of the key limitations in our case is the inability to perform chemical analysis of the powder. Due to resource and laboratory constraints, we were unable to submit the product for mass spectrometry or chromatographic analysis. Several articles, such as the report by Saad-Omer et al. (13), who identified dexamethasone contamination through laboratory testing, demonstrate how analytical confirmation strengthens diagnostic certainty. Although our clinical diagnosis remains strongly supported by the patient’s phenotype and trajectory, the absence of direct product analysis represents an

important limitation. Future cases would benefit from collaboration with toxicology laboratories capable of screening for synthetic corticosteroids. Finally, our findings also resonate with broader public health concerns highlighted across the literature. Multiple authors, including those reporting on *Artri King* and other unlabeled “herbal” products, highlights that social determinants of health substantially increase the risk of accidental steroid exposure(8–10, 14). Taken together, the striking clinical presentation in our patient, together with similarities and distinctions identified in previous reports, strongly supports that the unlabeled powder was the likely source of his exogenous Cushing’s syndrome. Despite the limitation of lacking product analysis, this case adds to growing evidence that unregulated OTC “herbal” formulations can contain potent corticosteroids capable of causing life-threatening systemic complications.

Conclusion

This case illustrates the serious risks caused by unregulated “herbal” weight-gain products, which may contain undisclosed corticosteroids capable of inducing exogenous Cushing’s syndrome and its complications. Although product analysis was not feasible due to resource limitations, the clinical picture strongly supports this etiology. This case highlights the need for clinicians to consider hidden glucocorticoid exposure in patients using non-prescription supplements and underscores the importance of regulatory oversight and public education to prevent such preventable and potentially life-threatening outcomes.

Declarations

Author Contributions

Fatemeh Sarv involved in patient management, reviewed final manuscript, and had full access to all of the data in this study, taking complete responsibility for the integrity of the data and the accuracy of the data analysis. Nasrin Asgari Soran, Haleh Chehrehgosha, Hossein Samadanifard and Amir Ziaee was involved in patient management and reviewed the final manuscript. Mohammad Hossein Teymoori wrote original draft and editing.

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Disclosure

All authors have read and approved the final version of the manuscript.

Ethics Statement

Ethical approval was obtained from the Research and Ethical Review and Approval Committee, Iran University of Medical Sciences, Iran, Tehran (IR.IUMS.REC.1404.1002).

Consent

Written informed consent was obtained from patient, and all identifying information has been anonymized.

Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability Statement

The data are available from the corresponding author. The data that support the findings of this study are available in the Supporting Information section of this article.

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Figures



Figure 1

Clinical photograph showcasing the striae on the patient’s abdomen.