Brief Report

“Abu-Najma” Syndrome: exogenous steroid induced Cushing’s with serious consequences. A report of two cases.

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Abstract
We describe and report two cases of what we called Abu-Najma Syndrome, caused by the ingestion of ‘Abu-Najma’ tablets: dexamethasone containing tablets promoted as “beauty tablets”. We believe the use of Abu-Najma is fast spreading among young ladies in Sudan. The syndrome has very serious consequences; a concerted effort is required to increase public awareness and prevent the problem.

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became so weak and was unable to stand or even lift her head from the pillow. She was brought on a wheelchair. She gave a recent history of amenorrhea; her periods were previously normal and regular.

Examination revealed a very ill young lady with oral thrush, Cushingoid features, puffy face, central adiposity, marked proximal muscle wasting, thin skin, subcutaneous bruises and extensive striae in the abdomen, thighs, legs, shoulders and upper arms (Fig. 1).

Fig. 1: Extensive abdominal striae

Pulse was 90/min, BP 100/60, she was too weak to stand or sit for postural BP measurement. She had weak flexion and extension of neck muscles and severe proximal muscle weakness in her upper and lower limbs, otherwise examination of CNS, cardiovascular and respiratory systems was unremarkable.

Initial investigations revealed haemoglobin of 12 gm/dl, leucocytes of 12 x 10⁹/L, Platelets 348 x 10⁹/L, random blood glucose 108mg/dl, Na 120 mmol/l, K⁺ 1.8 mmol/l, urea 30 mg/dl, serum creatinine 1.08 mg/dl. Liver functions test, Creatine kinase, and abdominal ultrasound were normal, HIV test was negative. X-ray of the lumbo-sacral spine showed features of osteoporosis. Upper gastrointestinal tract endoscopy showed mild gastritis.

Plasma cortisol measured at 08:00 AM was low at 23 nmol/l [130-650], PM cortisol measured at 18:00 hrs was also 23 nmol/l [80 – 350], although this is sufficient to diagnose cortisol deficiency a short synacthen test was performed, this confirmed a flat cortisol response with 30 min. value of only 79 nmol/l [normal response is >600 nmol/l ]. Thyroid functions test, leutinizing hormone, follicle stimulating hormone and prolactin were within normal limits.

She has been married for 4 years with history of early miscarriage soon after her marriage, she has no children. Eight months earlier she started taking “Abu-Najma”, a minimum of 6 tablets per day, to shape-up because her husband, an expatriate worker in the Gulf, was coming back on holiday! Few weeks after the return of her husband she noticed amenorrhea, but pregnancy test was negative. Subsequently, she stopped the Abu-Najma tablets after taking them for 5 months, mainly because her legs have become too weak.

Abu-Najma is a tablet containing dexamethasone 0.5mg; it is available at local beauty shops at a low price.

She was treated initially with intravenous normal saline, potassium replacement and intravenous dexamethasone; she was then switched to oral prednisolone. Her general condition improved, nausea and vomiting settled, appetite improved. Electrolytes returned to normal, however proximal muscles remained weak, she left the hospital on the wheelchair. Prednisolone dose gradually reduced to 5 mg AM and 2.5 mg PM prior to discharge then to 5 mg od. On review, 5 months later she looked better, her periods resumed; but she remained on the wheelchair because of continuing severe steroid induced proximal myopathy (Fig. 2).
Our patient believed the tablet would boost her beauty by helping her gain some weight and lighten her skin colour! As a consequence she suffered severe proximal myopathy and osteoporosis as well as adrenal crisis after stopping Abu-Najma tablets.

**Case 2**

A young lady sadly lost her life earlier in the same year in our hospital, she was a 20-year-old lady who took 10 Abu-Najma tablets per day for two months then stopped. Seven days later she presented with general ill health, body aches and arthralgia. She was ill with cushinoid features, and features of severe septicaemia, she sadly died of her illness despite active resuscitation and treatment with intravenous steroids.

“Abu-Najma” syndrome is a “self inflicted”, exogenous steroid induced Cushing’s with serious consequences.

**Discussion**

Health and beauty products are money generating commodities and are often vigorously marketed putting vulnerable girls and young ladies at risk of serious side effects. The concept of beauty varies from one culture to another, while slim built and tanned skin are fashionable in western countries, in Sudan a plump and curvy built and fair skin are preferred by some. Abu-Najma induces weight gain by causing fluid retention and can lead to pallor of skin via suppression of release of melanocortins from the pituitary gland.

The use of Abu-Najma tablets is widespread among girls and young ladies in Sudan, our index case reported that most of her peers use the tablets and are not aware of any ill effects or harmful consequences. Anecdotally, one of the authors of this paper had informal discussions with several young high school age girls; a number of them used Abu-Najma. Also, screening of a number of Sudanese web based discussion groups confirmed the widespread use of Abu-Najma as beauty preparations. “Najma” in Sudanese/Arabic means a star; Abu-Najma is a reference to the star trade mark label on the package of the tablet (Fig. 3).
Some local beauty shops use the attractive, yet misleading, slogan “Be a Star” to sell this product. Our index patient nearly lost her life from acute adrenal crisis and was crippled by severe steroid induced myopathy, she also has osteoporosis with increased risk of fractures. On the second occasion, Abu-Najma lead to the tragic loss of a young life.

Glucocorticoids may be taken via the oral, ocular, inhaled, transdermal, rectal or parenteral routes. Steroids may be present in bleaching creams and herbal preparations and may not be reported by patients as a "medication" unless specifically queried\(^1\). A thorough history regarding medications, creams and supplements should be obtained in every patient with apparent Cushing’s syndrome or secondary or tertiary adrenal insufficiency. Abu-Najma was promoted to our patients as a “beauty” tablet, we would recommend that female patients with proximal myopathy, suspected Cushing's or adrenal suppression are specifically asked about the use of “beauty” or weight boosting tablets; and in Sudan specifically asked about the use of Abu-Najma tablets.

Suppression of hypothalamic-pituitary-adrenal function by chronic administration of high doses of glucocorticoids is the most common cause of adrenal insufficiency\(^2\). In the absence of ACTH stimulation, the zona fasciculata of the adrenal cortex atrophies with reduced ability to produce cortisol. Cortisol production may be restored by prolonged ACTH administration, a feature that has been used in the past to distinguish secondary or tertiary from primary adrenal insufficiency. Another important difference is that the adrenal glands in secondary or tertiary adrenal insufficiency are capable of nearly normal mineralocorticoid secretion, because this function depends mostly on the renin-angiotensin system rather than on ACTH. Hence, hyperkalaemia is not a feature of secondary hypoaldrenalism. Our patient had severe hypokalaemia at presentation and this may have contributed to the severe muscle weakness. The hypokalaemia in this case is due to metabolic alkalosis secondary to the chronic repeated vomiting.

The time required to achieve HPA-axis suppression depends upon the dosage and varies among patients, probably because of differences in their rates of glucocorticoid metabolism. Our two patients used Abu-Najma tablets for five and two months respectively. However, suppression can occur in only 2-3 weeks after starting exogenous steroids\(^3\), therefore any patient who receives glucocorticoid for more than three weeks should be considered at risk, and advised accordingly. Alternate-day glucocorticoid therapy is less likely to cause HPA axis suppression\(^4,5,6\). Glucocorticoids have a direct catabolic effect on skeletal muscle leading to myopathy\(^7,8\). Steroid-induced myopathy can occur when systemic glucocorticoid treatment is first begun, or may appear in a patient on chronic maintenance therapy whose dose is suddenly increased because of an exacerbation of the underlying disease. There is wide variation in the dose and duration of treatment prior to the onset of muscle weakness. Some patients become weak after a low dose of steroids for a few weeks, while others never develop myopathy despite receiving large doses of steroids for months or years. Despite this variability, there is a general dose relationship
Glucocorticoid myopathy is very unusual in patients treated with less than 10 mg/day of prednisolone or its equivalent. Large daily doses in excess of 40 to 60 mg/day almost always result in some degree of muscle weakness when continued for more than one month. The higher the dose of glucocorticoid is, the higher the risk of myopathy. High dose therapy can induce clinically important weakness within two weeks\(^{(10)}\). Muscle strength begins to improve within three to four weeks after appropriate dose reduction and eventually resolves in virtually all patients if glucocorticoid therapy can be discontinued\(^{(9)}\). Fluorinated steroid preparation, such as dexamethasone are associated with higher risk of myopathy than a nonfluorinated preparation, such as prednisolone, if the patient cannot be weaned from glucocorticoid therapy a switch from dexamethasone to prednisolone may help. In our patient, significant weakness persisted despite replacement with “smaller” dose of 5 mg/day of prednisolone (equivalent to 20 mg of hydrocortisone). However, even this dose is supra-physiological, currently the physiological dose is believed to be in the order of 10 to 15 mg of hydrocortisone day. Large doses of steroids are usually used for inflammatory or autoimmune conditions to suppress immunity or inflammatory cytokines. The severe consequences suffered by our two Abu-Najma victims raise the question of whether the absence of an inflammatory response prior to administration of steroids is to blame; the other and more likely explanation is that we are just seeing a tip of an iceberg: the severe end of a wide spectrum of problems caused by Abu-Najma tablets.

In conclusion, we aimed to describe and draw attention to “Abu-Najma” Syndrome, this is a syndrome with very serious consequences; it involves ingestion of a large dose of dexamethasone to boost beauty and body shape, putting the individual at risk of serious side effects of steroid as well as adrenal crisis on withdrawal. Studies are needed to investigate the magnitude of what we believe is a fast spreading problem among young girls and ladies in Sudan. A concerted campaign of public awareness and education is needed to prevent the problem, together with enforcement of appropriate regulation for prescription and dispensing of drugs.

References