

Steroid abuse; two wrongs don't make a right: A case report

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Abstract

Steroid abuse among patients with rheumatic symptoms is prevalent in developing countries. The sources of the steroids vary with a significant proportion of patients self-medicating. Chronic steroid abuse results in multiple adverse effects and rapid withdrawal in such patients leads to acute adrenal insufficiency. We present a case report of a 47 year old lady who self-medicated her joint pains with steroids for 17 years and subsequently developed acute adrenal insufficiency on their rapid withdrawal.

Introduction

Corticosteroids play a key role in the management of rheumatic diseases. Judiciously used, they are invaluable in the management of several inflammatory diseases and minimum doses should be given for the shortest time¹. However, corticosteroids becomes dangerous when available over-the-counter and used in an unregulated manner. Increasing trends of steroid abuse have been reported in developing countries due to loosely audited health care systems and lack of sufficient rheumatological training and facilities to cater for the ever increasing rheumatic diseases^{2,3}. Studies done in developing countries such as Pakistan, report a prevalence of steroid abuse of 42.5% among patients with rheumatic problems³. Such studies have found that 38.2% of the patients on steroids develop side effects, with 29% developing Cushing's syndrome³. Rapid withdrawal of steroids in patients with Cushing's syndrome results in acute adrenal insufficiency. This case report is an interplay of the acute and chronic complications of steroid abuse in the background of an undiagnosed rheumatological disease.

Case Report

A forty seven year old lady was admitted at Kenyatta National Hospital with a three day history of facial swelling and one day history of generalized body weakness.

The lady had been unwell for seventeen years with generalized joint pains mainly involving the wrist and knee joints. At the first instance of joint symptoms she had sought medical assistance at a pharmacy where prednisolone 10mg twice a day was prescribed. She experienced immediate relief on the medication and as a result self-medicated at the same dosage for seventeen years. Over the seventeen years she had adequate relief of symptoms and hence did not seek further medical assistance. Six years after initiating steroid use, she gained approximately 30 kilograms in weight. In addition, she had emotional lability and muscle weakness especially of the thighs. Six months prior to her admission she was diagnosed to be hypertensive at a pharmacy and initiated on antihypertensives.

On further inquiry she revealed that the right sided facial swelling was recurrent over the past two years. The swelling which started after a tooth extraction persisted despite constant use of various antibiotics. At the time of admission the swelling was discharging frank pus.

Two weeks prior to her admission she sought medical assistance at a local clinic due to worsening joint pains. She reported that the doctor immediately stopped her prednisolone and prescribed new medications for control of blood pressure. She was also instructed not to take the steroids again. One day prior to her admission she reported sudden generalized body weakness and inability to walk or stand without support. In addition she felt dizzy and nauseated.

Physical examination revealed a middle aged lady with cushingoid appearance. She had an abscess on the right maxillary region that was discharging frank pus (Figure 1). Her skin was atrophic with generalized striae. Her blood pressure was reduced at 86/46mmHg. Her pulse and respiratory rates were elevated at 110 beats/min and 26 breaths/min respectively. There was grade 5 weakness of the lower limb proximal muscles. Her joints were neither deformed, nor swollen and had full range of movement. Abdominal examination was normal.

Figure 1: The patient demonstrating the cushingoid appearance and right maxillary abscess



Given the history of long standing steroid use followed by sudden withdrawal a diagnosis of acute adrenal insufficiency was made. In addition, this patient with undiagnosed polyarticular pains had other complications of chronic steroid use- Cushing's syndrome and reduced immunity. A therapeutic trial of glucocorticoid was instituted with 100mg intravenous hydrocortisone. Her blood results revealed slightly reduced random cortisol levels, markedly elevated sugars and leucocytosis (Table 1).

Table 1: Results of the laboratory investigations

Test	Result	Reference range
Serum cortisol	9µg/l	>10µg/l
Potassium	5.0	3.5-5.0mmol/l
Sodium	134.3	135-140
Random blood sugar	23.3	7.8-11.1mmol/l
White blood cells counts	12.5	4-12
Urinalysis	Glycosuria, no ketonuria	
ANA, Rh Factor	Negative	

Radiograph of the right maxillary bone was normal, specifically there was no evidence of osteomyelitis. The patient was continued on glucocorticoid supplementation while in the ward. Titrating doses of soluble insulin were used to control the hyperglycemia. On the third day her blood pressure was found to be elevated and she was started on hydrochlorothiazide 25mg once a day and losartan 50mg once a day (Table 2).

Table 2: Blood pressure recording and fasting blood sugars while in the ward

Day of admission	1	3	4	6	10
BP (mmhg)	86/46	160/100	160/110	150/100	130/95
FBS (mmol/l)	23.3	14.1	4.0	9.0	6.9

The abscess was incised and drained. Samples were taken for histology and microbiology analysis including atypical organisms such as mycobacteria and fungi. The histology was reported as chronic inflammatory reaction whereas no growth was obtained on the cultures. The patient was empirically treated with intravenous ceftriaxone 2g once a day and metronidazole 500mg thrice a day.

By the time of discharge ten days later, the patient had markedly improved. Her abscess was resolving and the blood pressure and blood sugar were well controlled. The patient was discharged through the rheumatology clinic on prednisolone 20mg once a day that was to be gradually tapered off. Further investigations to diagnose her underlying rheumatological condition were to be done at the outpatient rheumatology clinic.

Discussion

This case report highlights some complications of chronic steroid abuse. In most patients the signs of steroid abuse are not as overt as in this case. Management of these complications overburdens an already overstretched health care system. It is important that appropriate regulatory measures are put in place to control over-the-counter access of steroids as it is currently estimated that 1-3% of the adults worldwide report long-term steroid use⁴.

Chronic steroid use results in two major complications that this patient had developed -Cushing's syndrome and Hypothalamic Pituitary Axis (HPA) suppression. Patients who have been on prednisolone 20mg /day for more than three weeks, have received an evening/bedtime dose of prednisolone for more than a few weeks and have cushingoid features are likely to have HPA suppression⁵. These patients do not need testing to evaluate their HPA function, but should be treated like any patient with secondary adrenal insufficiency, including the wearing of a medical alert bracelet or necklace and carrying an emergency medical information card. Such patients like in this case should have gradual withdrawal of the steroids to avoid developing acute adrenal insufficiency. The goal of tapering is to use a rate of change that will prevent both recurrent activity of the underlying disease and symptoms of cortisol deficiency due to persistent HPA suppression. A suggested regimen involves a 10-20% reduction every 1-2 weeks while accommodating convenience and individual patient's response until the patient is waned off the steroids. Abrupt withdrawal of steroids is only recommended in patients with herpes virus induced-corneal ulceration or steroid abuse psychosis which have been shown not to respond to treatment in the presence of the steroids⁵.

A commonly used empiric approach to tapering corticosteroid therapy bases the tapering program on the current daily steroid dose:

- (i) At greater than 40 mg/d, one tapers by 10 mg/d every 1 to 2 weeks.
 - (ii) At 40 mg/d, one tapers by 5 mg every 1 to 2 weeks.
 - (iii) At 20 mg/d, one tapers by 2.5 mg every 1 to 2 weeks.
- Tapering continues until a physiologic dose of prednisone is reached (5 to 7.5 mg/d). The patient can then be switched to 1-mg prednisone tablets or the equivalent dose of hydrocortisone, so that further reductions in dose can be made in smaller steps than is possible when 5-mg prednisone tablets are used. Weekly or biweekly reductions can then be carried out in steps of 1 mg of prednisone at a time, as permitted by disease activity⁶.

During the tapering process, a steroid withdrawal syndrome develops in some patients, characterized by depression, myalgias, arthralgias, anorexia, headaches, nausea, and lethargy. Studies have failed to show a relationship between these symptoms and low cortisol levels. In most instances, symptoms are reported when levels are normal, or even elevated, but falling rapidly. HPA responsiveness has also been found to be normal in many of these patients. The mechanisms responsible for this syndrome are unknown but seem to be linked to the rapidity with which the dose is tapered⁶.

Another approach involves the use of plasma cortisol measurements to gauge corticosteroids withdrawal⁶. Patients return to the clinic at two to four week intervals for morning plasma cortisol measurement. Tapering is done at a rate of 2.5 mg of hydrocortisone/week down to a single morning dose of 10 mg of hydrocortisone (equivalent to 2 mg of prednisone). Steroid therapy could be discontinued when the morning plasma cortisol concentration rises to greater than 10 mcg/dL. Stress doses of steroids might be needed for infections.

This approach, however, has not gained much popularity and is generally not used.

Management of Cushing's syndrome depends on the specific features present. Hypertension and hyperglycemia are managed in the conventional manner with good control achieved after steroid withdrawal⁵. Fat redistribution in the cushingoid habitus occurs gradually over months after steroid withdrawal. Exercises and healthy diet have been shown to aid in the fat redistribution⁵.

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