CASE REPORT

A 42year old male was presented with recent-onset diabetes mellitus. His disease was diagnosed because of mild weight loss. There was no ketonuria at the time of the onset of the disease.

In the past, patient had not suffered from any significant illness. In this family, father died at the age of 71 years of a myocardial infarction. Mother was alive and healthy. One brother suffered from Type 2 diabetes.

A system review at the time of initial consultation revealed normal vision. Patient gave history of considerable edema of feet, increasing on dependency. He also had exertional dyspnea. There was no paroxysmal nocturnal dyspnea or angina. Appetite was good and bowels were regular. He had nocturia twice every night. He reported normal sexual function. There was mild tingling and numbness of feet.

His initial physical examination revealed a pulse of 95/min, regular. Wt 72 kg, BP 160/95. JVP mildly raised. Edema feet++. Skin was rather dry and cold. There was evidence of mycotic infection in intertrigenous areas. His heart was enlarged. Liver palpable 1" below the costal margin in the mid-clavicular line. Abdomen was protuberant. Abdominal skin and gluteal region showed presence of purpulish striae. There was evidence of balanitis. He had proximal muscle weakness of hip girdle.

His investigation revealed an essentially normal hemogram. Serum Na 138 mEq/l, K 3.2 mEq/liter, FBG 230 mg%, post meal BG 312 mg%; urinalysis revealed occasional pus cells with trace of proteinuria, creatinine, 1.1 mg%, GHb 11.2%, cholesterol 212 mg%, triglycerides 320 mg%.

ECG showed ischemia of lateral wall. Chest x-ray revealed cardiomegaly.

T_3-75 ng%, T_4-5.4 m g%, TSH-2.2 m IU/ml. Serum cortisol at 8 am was 2 m g%. ACTH was 10 pg/ml.

Patient was diagnosed as Type 2 diabetic with coronary artery disease, hypertension, congestive heart and hypercortisolism.

He was put on a 4 gm salt, 1500 cal diabetic diet. He was put on 3 dose insulin therapy which produced fair control of DM as evidenced by a GHb of 9%.

Follow Up:

He was followed on quarterly basis for the next 4 years. His congestive failure worsened in spite of furosemide administration and digitalisation. Hypertension was inadequately controlled with nifedipine. At one point, an ACE inhibitor was exhibited, but distressing dry cough necessitated its withdrawal. His diabetes also remained uncontrolled. A repeal cortisol was 4 m g% at 8 am.

An intensive search was made for the possibility of surreptitious corticosteroid administration. Patient was not on any indigenous drugs. He was not using any corticosterold containing ointments. Only topical sterold used by him was in the form of a 0.05% betamethasone nasal drops.

Discussion:

Topital corticosteroid therapy is often mistakenly considered harmless. It is important to appreciate that concentration of 0.05% is 0.05 gm or 50 mg/100 ml of the compound.

Topical corticosteroids are divided into mild, moderate, potent and very potent in their effects. Typical examples are hydrocortisone (mild), fluandrenolone, desoxy-metasone (moderate), betamethasone, flucinolone (potent). These compounda are absorbed through the skin, especially from occlusive dressings. They are better absorbed through mucus membrances.

Our patient was using 0.05% betamethasone nasal drops and consuming about 2-3 bottles of 10 ml per week. Thus, he was being exposed to 10 to 15 mg of betamethasone per week. The dose used was sufficient to produces cushingoid changes, striae, easy bruisability, hypokalemia, hypertension aggravation of diabetes and HPA-axis suppression. There are two additional reasons for the pronounced clinical manifestations of hypercortisolism in this patient: use of a long acting corticosteroid and near complete absorption of corticosteroid from the nasal mucosa.

H. B. C.