CASE REPORTS
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INSULIN RESISTANCE SYNDROME: A CASE REPORT

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Abstract

Insulin resistance is impaired ability of plasma insulin to perform its actions at usual concentrations. It can be acquired or genetic. Here we report a case of insulin resistance.

Key words: insulin resistance syndromes, acanthosis nigricans

Introduction

Insulin resistance is defined as an impaired ability of plasma insulin at usual concentrations to adequately promote peripheral glucose disposal, suppress hepatic glucose, and inhibit very low density lipoprotein (VLDL) output. It can be acquired or genetic. Insulin resistance is associated with many cutaneous and systemic manifestations[1]. Here we report a case of insulin resistance.

Case Report

A 12 year old male boy born of non consanguineous marriage presented to our OPD with hypertrichosis and severe acanthosis nigricans. On examination, there was abnormal facies with low frontal hair line, large ears with hypertrichosis, large lips, prognathism, hypertelorism. There was severe acanthosis nigricans involving the neck, axillae and flexures with blackening and thickening of skin over trunk. There was generalized hypertrichosis (fig 1-4). Oral mucosa, nails and teeth were normal.

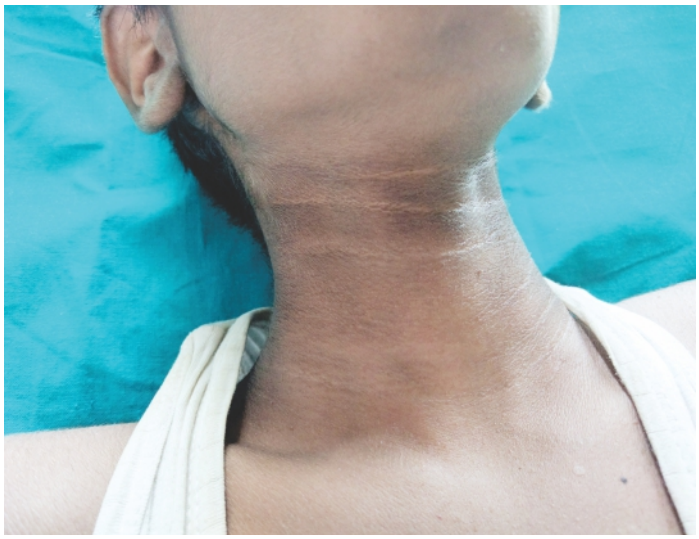

Figure 1 : Acanthosis nigricans over neck and hypertrichosis over ear

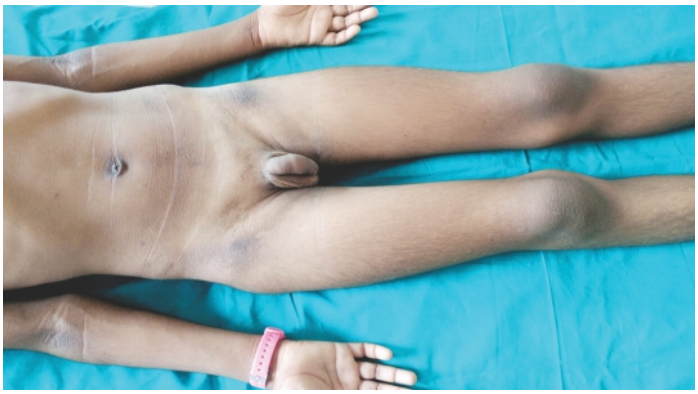

Figure 2 : Acanthosis nigricans over flexor aspect of elbow and blackening and thickening of skin over trunk

A primary diagnosis of insulin resistance syndrome was kept and patient was worked up. His complete blood count, urine examination, renal function test were normal. Liver function test showed elevated enzymes and fasting insulin was remarkably raised with values 65.60 µIU/ml. His fasting blood sugar was 108mg/dl, Hb1Ac was 9.34% and lipid profile was normal. On USG of abdomen there were bilateral bright kidneys. His echocardiography was normal.

Discussion

The pathogenesis of insulin resistance is multifactorial. Thus, several molecular pathways in energy homeostasis, lipid metabolism, insulin receptor signaling pathway, cytokines, hormone-binding proteins including those that are serine protease inhibitors (SERPINS), and other protease regulators are responsible for the development of IR, obesity, or lipodystrophy. On review of literature the above patient seemed to be affected by defect in the insulin-signaling pathway, which may cause mutations in insulin receptors, development of insulin receptor autoantibodies or defects in plasma cell membrane glycoprotein-1 and glucose transporter 4 (GLUT4) molecules are reported. The syndromes reported with this pathway defect are Type A syndrome, Donohue syndrome

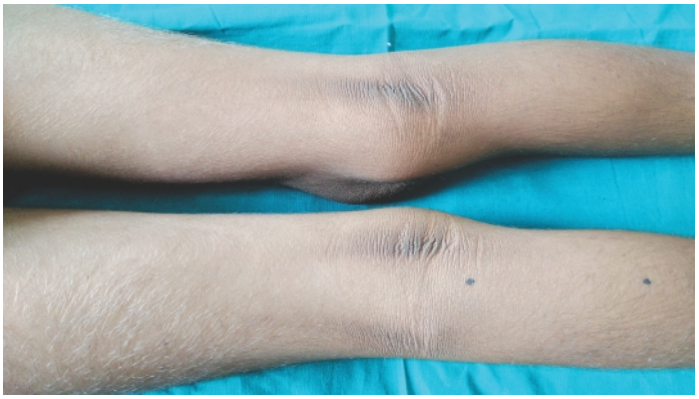

Figure 3 : Acanthosis nigricans over flexor aspect of knee

(Leprechaunism)[2], Rabson-Mendenhall syndrome[3] and Polymorphism in plasma cell membrane glycoprotein-1 (PC-1)[1].

The features present in this patient suggestive of insulin resistance were acanthosis nigricans, hypertrichosis, hypertelorism, large ears, prominent lips, prognathism, steatohepatitis and bilateral bright kidneys which might be due to glomerulonephritis. The patient also had very high fasting insulin although his blood sugar was normal.

In children, insulin resistance is usually well compensated by hyperinsulinemia. However it increases risk for fatty liver, atherosclerosis and increased cancer risk. Thus an early intervention is necessary. This involves regular exercise, restricted calorie, carbohydrate and triglyceride dietary intake. Fibrates may be required, especially when TG levels exceed 500 mg/dl, at which point acute pancreatitis and gall bladder disease become real risks. Metformin can also be used for prophylaxis. Laparoscopic surgery can be used in obese cases.

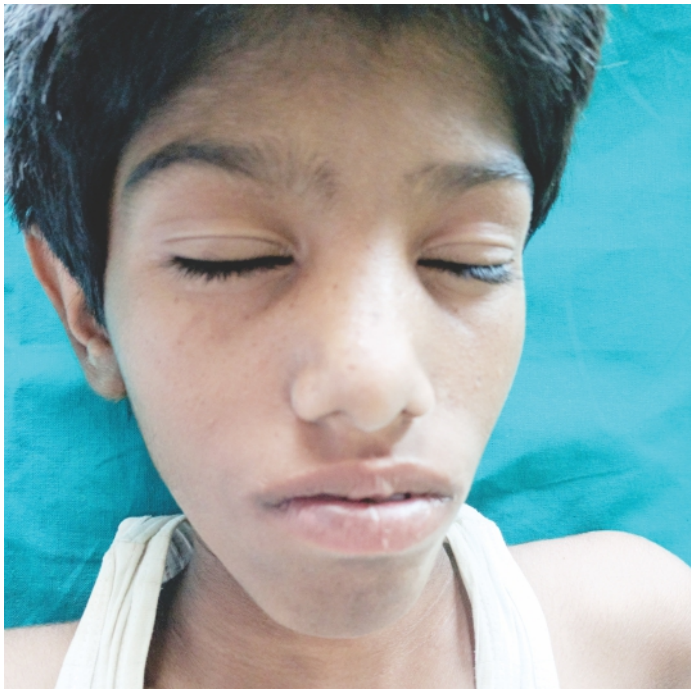

Figure 4 : Showing thick lips, prognathism, hypertrichosis over scalp, hypertelorism

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