Acanthosis nigricans following nicotinic acid therapy

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Summary

Drug induced Acanthosis Nigricans is described in a 67 year old year old male who was taking nicotinic acid as a lipid lowering drug. The condition disappeared completely on withdrawal of nicotinic acid. The need to be aware of this complication is stressed as nicotinic acid is being increasingly used in the treatment of hyperlipidaemias.

Introduction

Acanthosis Nigricans characterised by pigmentation, hyperkeratosis, papillomatous elevations giving a velvety texture and appearance is well known to dermatologists. However drug induced Acanthosis Nigricans is by far the least common of the many causes of this condition. The drugs incriminated are high dosage nicotinic acid, stilbesterol in young males, oral contraceptives and triazinate therapy [1]. The pathogenesis of acanthosis nigricans is now thought to be linked to insulin resistance[2].

Case report

A 67 year old male retired from an international agency in New York was seen in March 1995. He has had coronary artery bypass surgery in 1986. His medication for ischaemic heart disease and hyperlipidaemia were atenelol and bezafibrate since 1988. In June 1993 nicotinic acid was added to his medication as a lipid lowering agent. The initial dose of 100 mg three times daily was gradually increased and at the time of examination he was taking 750 mg daily. On examination he was found to be of linear build and in good health. There was diffuse macular pigmentation of the face. His neck, axillae, cubital fossae and the groin were deeply pigmented with hyperkeratosis having a velvety appearance. Rest of the physical examination was unremarkable. The diagnosis of acanthosis nigricans was confirmed by a skin biopsy of the right anticubital fossa which showed hyperkeratosis with irregular spiky papillomatosis. The following investigations were done. ESR 10 mm, Haemoglobin 11.4 g/dl, White cell count 6800 with normal differential count and platelet count. Electrocardiogram showed evidence of previous myocardial infarction. Stools were negative for occult blood. He was further investigated and the following were found to be within normal limits. Bone marrow examination, CT scan of the abdomen, upper gastrointestinal endoscopy and colonoscopy. Nicotinic acid was discontinued at the time of investigations. The pigmentation started to fade soon after stopping nocotinic acid and by the end of 8 weeks the hyperkeratosis and the pigmentation disappeared completely.

Discussion

The velvety hyperkeratotic and pigmented skin of Acanthosis Nigricans is usually seen mainly in flexural areas of the body as seen in our patient. Acanthosis Nigricans has been classified into five main types. The benign forms being hereditary, endocrine, pseudo acanthosis nigricans in the obese and drug induced acanthosis nigricans. Although a rarity in the literature our patient clearly had a drug induced acanthosis nigricans as the lesions disappeared completely on withdrawal of nicotinic acid. Acanthosis Nigricans is now believed to be associated with insulin resistance but the role of niacin in the causation of this disorder is not presently understood[3].

We believe that the intensive investigations done in our patient are justified as the delay of 6 to 8 weeks for the disappearance of drug induced acanthosis nigricans would have meant a further

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spread of a maligancy if that was the cause of his condition. Since nicotinic acid is being increasingly used as a lipid lowering agent it is important to be aware of this complication so that the drug could be discontinued at the earliest sign of increased pigmentation.

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