TYPE 2 DIABETES MELLITUS IN A PATIENT WITH LICHEN PLANUS

A CASE REPORT

*Akinlade AT *Mosuro OR *Adeniji OA *Ajose FOA

*Department of Medicine,

Lagos State University Teaching Hospital,

Ikeja, Lagos

Correspondence: Dr. Akinlade AT

Email: dratakinlade@yahoo.com

Summary

Lichen planus is a papulo-squamous skin disorder characterized by extremely pruritic shiny, flattopped and violacious papules (ash grey in blacks), with white lacy markings on the surface (Wickham's striae). It may affect the skin, scalp, nails, and mucous membranes.

We present the case of a 42 year-old teacher with generalized lichen planus, in whom a diagnosis of type 2 diabetes mellitus was made at the same time.

This case is reported because of the rare association between the two conditions, the challenge of managing lichen planus with steroids in a patient with diabetes mellitus and also the dramatic resolution of symptoms with therapy.

Keywords: Lichen planus, Diabetes Mellitus, Steroid therapy

Introduction

Lichen planus is a papulo-squamous skin disorder characterized by extremely pruritic shiny, flattopped and violacious (ash-grey in blacks) papules with white lacy markings on the surface (Wickham's striae). It may affect the skin, scalp, nails, and mucous membranes. Lichen planus occurs at any age but usually develops in middle age with a female preponderance.¹

The prevalence of lichen planus in the USA is estimated to be less than1%. In India, it constitutes about 0.38% of all the patients attending the dermatology clinics. Most patients with lichen planus present with the cutaneous form while mucosal involvement is observed in less than 20%.

Although many different causes have been implicated such as emotional stress, infection, trauma, malnutrition, allergy and heredity, the etiology remains uncertain¹. In addition, many studies have investigated and supported an immunologic pathogenesis. Lymphocytes, particularly T-cells, play a major role^{2,3}. Other factors include antigen-presenting cells, adhesion molecules and inflammatory cytokines. While most cases of lichen planus are idiopathic, some are linked to medication use or hepatitis C virus (HCV) infection. The most commonly implicated drugs include gold, antimalarial agents, penicillamine, thiazide diuretics, beta blockers⁴, nonsteroidal anti-inflammatory drugs, quinidine and angiotensin-converting enzyme inhibitors⁵.

At the Dermatology clinic of the Lagos State University Teaching Hospital (LASUTH), Lagos, Nigeria, 3143 patients were seen over a three-year period. Of these, 64 (2%) had lichen planus. The female-male ratio was 2:1. Diabetes mellitus was seen in only 3 (4.7%) of these patients. Prevalence studies on the co-association of diabetes mellitus and lichen planus were not found in our literature search.

We present the case of a 42 year-old teacher with generalized lichen planus, in whom a diagnosis of type 2 diabetes mellitus was made at the same time.

Case Report

Mrs. A.F is a 40 year-old secondary school teacher who presented at the dermatology clinic with a 6-week history of generalized, intensely itchy, hyperpigmented skin papules and plaques. She also noticed about two weeks after that she was having polyuria and polydipsia.

The skin lesions were not photosensitive but the itching was aggravated by a warm bath. No prior history of fever, insect bites, ingestion of suspicious medications or exposure to chemicals. She did not have associated oral or genital ulcers, joint pains or hair loss or symptoms suggestive of autonomic dysfunction.

She was found to have systemic hypertension and type 2 diabetes mellitus during the course of her illness and was adherent in taking the metformin, chlorpropamide and nifedipine tablets she was placed on before her presentation at our clinic. She had no history of atopy, macrosomic babies, recurrent spontaneous abortions or still births. No history of hypertension or diabetes mellitus in her first degree relatives.

Physical examination showed an obese young woman who was mildly pale but anicteric, afebrile and had no alopecia or nail abnormalities.

She had generalized hyperpigmented (grayish black) skin papules and plaques, only sparing her antecubital and popliteal fossae.

Her pulse rate was 92beats/minute, and blood pressure was 130/70mmHg. No other system abnormalities were found.

Retroviral screening, anti-hepatitis C virus and hepatitis B surface antigen were negative. Full blood count was normal, except for mild normocytic, normochromic anemia, as was urinalysis. Fasting blood glucose at presentation was 249mg/dl and the 2-hours postprandial result was

261mg/dl. The erythrocyte sedimentation rate was 35mm in the first hour.

Skin biopsy and histology showed moderate hyperkeratosis and irregular acanthosis with partly pointed elongation of rete ridges. There was also marked spongiosis in the lower half of the epidermis, with conspicuous melanin deposits in the reticular dermis and scarce lymphocytes in the epidermis.

She was managed with insulin (discharged on metformin tablets), lisinopril and steroids (parenteral, oral and topical). The skin plaques flattened while itching subsided by the 4th day of treatment. She has done well on out-patient follow-up.

Discussion

Lichen planus is a rare skin disorder, which is commoner in females. Lesions are extremely pruritic and it was as a result of this symptom that our patient presented. It is an autoimmune disease⁶ which can be primary or may accompany other autoimmune conditions such as: alopecia areata, vitiligo, dermatitis herpetiformis, pemphigus, scleroderma, primary biliary cirrhosis, autoimmune hepatitis, ulcerative colitis, thyroiditis, systemic lupus erythematosus (SLE), diabetes mellitus, and pernicious anemia⁷.

There is an increased incidence of diabetes mellitus and abnormal insulin response to glucose challenge in patients with lichen planus⁸. This association is reported to occur more commonly in those with oral lichen planus⁹. Our patient, however, did not have oral lesions. Various studies on lichen planus and its relationship with diabetes mellitus, have led to the conclusion that there are two types of lichen planus, i.e. the immunogenic type and the metabolic-defect type, both associated with diabetes mellitus¹⁰. One of the earliest pathological events is the appearance of colloid bodies at the basement membrane zone¹¹, which are coated with immunoglobulins, complement and fibrin^{12, 13}. There are also evidences of humoral immuno-deficiency in these patients.^{14,15,16} Genetic abnormalities of glucose-6-phosphate dehydrogenase (G6PD) enzyme in the skin with increased frequency of glucose intolerance and defective insulin secretions as seen in type 2 diabetes mellitus have been also demonstrated in lichen planus¹⁷⁻²².

The classic appearance of skin lesions includes violaceous polygonal flat-topped papules and plaques. Close examination reveals a reticulated pattern of white scale known as Wickham's striae. Early cutaneous lesions may be difficult to diagnose, often appearing as scattered

erythematous papules. More developed and extensive lesions may mimic discoid lupus, psoriasis or secondary syphilis. The flexor surfaces of the extremities, particularly the wrists, are common locations for lichen planus. Another clue to the diagnosis is that lesions may occur in areas exposed to trauma, such as lacerations. This tendency is known as an isomorphic response, or Koebner's phenomenon. Lesions often resolve with intense hyperpigmentation.

In more than 50% of patients with cutaneous disease, the lesions resolve within 6 months, and 85% of cases subside within 18 months. On the other hand, oral lichen planus had been reported to have a mean duration of 5 years. Large, annular, hypertrophic lesions and mucous membrane involvement are more likely to become chronic. Our patient showed remarkable improvement in a short period of time (within a week of commencing treatment).

A diagnosis of lichen planus is made on the basis of the typical clinical appearance and a punch biopsy of the lesion. Histopathology reveals characteristic findings of a band-like infiltrate of lymphocytes at the epidermo-dermal junction and damage to the basal cell layer. Epidermal changes include wedge-shaped hypergranulosis and irregular acanthosis leading to a saw-toothed appearance²³. Work up should also be aimed at excluding the other diseases known to be associated with such as diabetes mellitus and pernicious anemia. The histology report of the punch biopsy of our patient's lesions showed the presence of moderate hyperkeratosis and irregular acanthosis with partly pointed elongation of rete ridges. There was also marked spongiosis in the lower half of the epidermis, with conspicuous melanin deposits in the reticular dermis and scarce lymphocytes in the epidermis. The scarce lymphocytes seen in this case may be the effect of the steroid therapy she had started before the biopsy was done. The treatment of lichen planus should be individualized, particularly since it is a self-limiting disease that usually resolves within 18 months. Mild cases can be treated with fluorinated topical steroids but more severe cases, especially those with scalp, nail, and mucous membrane involvement, may need more intensive therapy.

Conclusion

Patients with lichen planus can also develop diabetes mellitus (either type 1 or type 2), even though the association is rare. It is therefore important to screen patients with lichen planus for diabetes mellitus.

References

- Andreason JO. Oral lichen planus 1: A clinical evaluation of 115 cases. *Oral Surg.* 1968a; 25: 31
- 2. Boyd AS, Neldner KH. Lichen planus. J Am Acad Dermatol. 1991; 25:593-619
- Porter SR, Kirby A, Olsen I, Barrett W. Immunologic aspects of dermal and oral lichen planus: a review. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997; 83:358-66.
- 4. Halevy S, Shai A. Lichenoid drug eruptions. J Am Acad Dermatol 1993; 29:249-55.
- Ellgehausen P, Elsner P, Burg G. Drug-induced lichen planus. *Clin Dermatol* 1998;16:325-32
- Nigam PK, Sharma L, Agrawal JK. Glucose tolerance studies in lichen planus, Dermatologica, 1987;7175:284-289
- Jolly M. Lichen planus and its association with diabetes mellitus. *Med J Australia*, 1972; 1:990-993
- Lundscrom IM. Incidence of DM in patients with oral lichen planus. *Int J Oral Surg* 1983; 12: 147-152
- Halevy S, Zamir R, Gazit E. HLA system in relationship to carbohydrate metabolism in lichen planus. *Br J Dermatol* 1979; 100: 83-86
- 10. Lisi P, Giommoni U. A study on the carbohydrate metabolism in lichen planus patients in time. *Ann Ital Dermatol Clin* 1983; 37: 29-33
- Medenica M, Lorincz A. Lichen planus : an ultrastructural study, *Acta Dermatovenereol* (Stockholm), 1977; 57:55-62
- 12. Abell E, Presbury DGC, Marks R. The diagnostic significance of immunoglobulin and fibrin deposition in lichen planus, *Brit J Dermatol*, 1975; 93: 17-24

- Failley-Kuyper EHB, Failley HB. An immunofluorescence study of lichen planus, *Brit J Dermatol*, 1974; 90: 365 -371.
- Stankler, L. Deficiency of circulating IgA and IgM in adult patients with lichen planus, *Brit J Dermatol*, 1975; 93:25-27
- 15. Jacyk WK and Greenwood BM. Serum immunoglobulins in Nigerian patients with lichen planus, *Clin Exp Dermatol*, 1978; 3:83-86
- Nigam PK, Sharma L, Singh G. Humoral immunodeficiency in lichen planus, *Ind J Dermatol Venereol Leprol*, 1988; 54:244-246
- 17. Cotton DWK, Van den hurk JJMA, Van der stak WBJM. Lichen planus: An inborn error of metabolism, *Brit J Dermatol*, 1972; 87:341-346.
- Jolly M. Lichen planus and its association with diabetes mellitus, *Med J Australia*, 1972; 1:990-993.
- 19. Lowe NJ, Cudworth AG, Clough SA. Carbohydrate metabolism in lichen planus, *Brit J Dermatol*, 1976; 95:9-13.
- Nigam PK, Sharma L, Agrawal JK. Glucose tolerance studies in lichen planus, Dermatologica, 1987; 175:284-289.
- Nigam PK, Singh G, Agrawal JK. Plasma insulin response to oral hyperglycemic stimulus in lichen planus, *Brit J Dermatol*, 1988; 119:128-129.
- Assatoor N, King EJ. Blood sugar estimation, in: Micro-analysis in Medical Biochemistry, Editors, KingEJ and Wooton IDP: J and A Churchill, Gloucester, 1954; 325-354.
- 23. Lever WF. Histopathology of the skin. In Elder, David E. Lever's histopathology of the skin. 8th ed. Elder D, ed-in-chief; Elenitsas R, Jaworsky C, Johnson B, Jr, eds. Philadelphia: Lippincott-Raven, 1997