A 37-year-old man presented with loss of hair on asymptomatic erythematous boggy plaques over the scalp, face, trunk and limbs of 8 months duration. A provisional diagnosis of follicular mucinosis was made. It was confirmed by histopathological examination of the skin biopsy (Alcian blue stain) which revealed mucinous deposits in the dermis, especially around hair follicles and sebaceous glands with destruction of these structures. Direct immunofluorescence from peri-lesional skin did not exhibit immune deposits. Routine investigations were within normal limits. There was considerable cosmetic disfigurement due to the lesions. The patient was treated with oral pred-nisolone 40 mg once daily with marked improvement within 6 weeks. The patient was lost to follow-up for about a year and he then presented with fever, malar erythema, periorbital and bilateral pedal oedema and painless palatal erosions. The old lesions of follicular mucinosis were inactive. He had no systemic symptoms except decreased urine output. Urine examination revealed albuminuria and microscopic hæ-maturia. Twenty-four-hour urine demonstrated proteinuria of non-nephrotic range. Serum urea and creatinine were 140 and 2.7 mg/dl, respectively. A systemic lupus erythematosus (SLE) work-up showed the presence of LE cells, strongly positive ANF (homogenous pattern) and decreased serum C3. A kidney biopsy revealed features of diffuse proliferative lupus glomerulonephritis with deposition of IgG, IgM and C3 on direct immunofluorescence. A lupus band test of photopro-tected and photo-exposed areas of skin was negative. Histopathology of the previous skin biopsy site did not reveal any mucin deposit. A diagnosis of SLE with renal involvement was made, and the patient was managed with monthly cyclophosphamide (1 g) given intravenously along with 60 mg of prednisolone daily.

Discussion

Alopecia mucinosa or follicular mucinosis was first described by Pinkus [1] in 1957. It may be associated with a host of benign or lymphoproliferative conditions [2, 3]. Some authors regard follicular mucinosis as a non-specific follicular reaction [4] since mucin deposits have been reported in other disorders, though clinically papular or nodular mucin deposits in these patients are uncommon [5]. Papulonodular mucinosis associated with SLE has been described as a
distinct but rare entity which may precede clinical and serological evidence of SLE in up to one third of cases [6].

It is suspected that some vascular damage or vasculitis stimulates the production of mucin [6]. In our patient, the plaques of follicular mucinosis had completely subsided before he developed SLE, and even in the active stage of SLE the lesions of follicular mucinosis remained quiescent both clinically and histopathologically. Though active vasculitis was extensively present in our patient during florid SLE, the mucinous process was inactive. Could follicular mucinosis be a marker of SLE akin to papulonodular mucinosis?

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References

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insulinemia via activation of insulin-like growth factor 1 receptors on their surface. Skin tags are more closely related to fasting insulin levels, suggesting its relation with insulin resistance [3]. We estimated insulin resistance in 10 patients with multiple skin tags and 10 control subjects matched for age, sex and body weight. The fasting serum insulin to glucose ratio was the measure of insulin resistance. Blood samples were taken after a 10- to 12-hour overnight fast. Serum insulin was estimated by radioimmunoassay (BARC-india kits) and glucose was estimated by the glucose oxidase method. The ages of the patient and control groups were (means ± SD) 47 ± 6.42 and 44.4 ± 7.2 years, respectively. Insulin/glucose ratios for patient and control populations were (means ± SD) 8.11 ± 1.08 and 8.0 ± 2.02 µU/mg and were not significantly different (t = 0.119, p = NS). No patient or control subject was diabetic or obese (> 120% of ideal body weight).

Thus, we conclude from the present study that skin tags are not markers of insulin resistance; however, the following points must be noted: (i) the fasting insulin to glucose ratio is not a sensitive method for estimation of insulin resistance; (ii) there was no NIDDM or obese individual in our study, in whom insulin resistance may have played a role in the pathogenesis of skin tags; (iii) as epidermal growth factor (EGF) receptors are overexpressed on proliferative fibroblasts in skin tags [4], it is possible that EGF or other growth factors may play a role in the pathogenesis of skin tags.

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Chagas’ Disease: A Potential Plague for Europe?
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Key Words
Chagas’ disease · Tripanosomiasis · Trypanosoma cruzi

Tachyarrhythmia
A 52-year-old man, a tour operator, while in central Brazil, developed a flu-like illness. A few weeks later, back to Italy, he had an episode of tachyarrhythmia. A cardiologist diagnosed myocarditis possibly of postviral etiology and prescribed amiodarone. A few days later, an exanthema developed which prompted a dermatological consultation. The exanthema consisted of several erythematous, nonitching, somewhere annular or targetoid macules, particularly on the trunk. The clinical picture evoked erythema multiforme, drug eruption, Lyme disease and, more closely, African trypanosomiasis [1]. Lyme ELISA and Western blot being negative, the diagnosis remained pending and amiodarone was continued. The eruption faded in a couple of weeks.

In apparently good health, the patient returned to Brazil where soon he had another episode of tachyarrhythmia. Blood tests now included Chagas ELISA which proved strongly positive. A diagnosis of Chagas’ cardiopathy was made and the patient, an occasional blood donor, was instructed to interrupt blood donations.

American trypanosomiasis or Chagas’ disease is endemic in South and Central America where it affects some 20 million people with an annual toll of 50,000 deaths [2]. The causative agent is Trypanosoma cruzi, a flagellate protozoan transmitted by hematophagous Hemi-ptera of the family Reduviidae, subfamily Triatominae, the most common species being Triatoma infestans, Rhodnius prolixus and Panstrongylus megistus. Adult Triatominae are 2.5 cm long nocturnal feeders and use to defecate close to their bite. When feces contaminate a bite or the conjuctiva, the organisms invade the host multiplying in the cells, especially those of the muscles (intracellular amastigotes). Eventually they rupture out into the blood (trypomastigotes) to be
taken up by the insect with the blood meal, to multiply in its gut as epimastigotes and pass into the feces.

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