are symmetrically involved. The lesions are wrinkled or depressed, scaly or smooth, yellowish waxy papules, which may coalesce to form band-like firm linear plaques that extend from near the base of the thumb around the web distally to the side of the index finger at the junction of the palmar and dorsal skin. The ulnar sides of the hands, and less commonly the medial and lateral sides of the middle and ring fingers, may be involved in a similar fashion.[3] Histologically, a distinctive deposition of dense collagen and degenerated elastic fibers in the reticular dermis is seen.[8] Because of different pathogenic interpretations, this entity has also been reported as limiting or marginal keratodermas of the palms, keratoelastoidosis marginalis of the hands, marginal collagen degeneration, and digital papular calcific elastosis.

The exact pathogenesis of this condition is unknown, but probably a combination of long-term trauma and pressure together with some degree of actinic damage is responsible for the localization of lesions in these sites.[9] In our case, these factors have not only produced lesions on the border of the fingers and palms, but also the thumbnail beds, leading to V-shaped nicking of the nails. To the best of our knowledge, such a nail change in association with this disorder has not been described earlier.

REFERENCES

1. Rongioletti F, Betti R, Crosti C, Rebora A. Marginal papular acrokeratodermas: a unified nosography for focal acral hyperkeratosis, acrokeratosis, acrokeratoelastoidosis and related disorders. Dermatology 1994;88:28-31.
2. Griffiths WAD, Judge MR, Leigh IM. Disorders of keratinization. In: Champion RH, Burton JL, Burns DA, Breathnach SM, editors. Textbook of dermatology. 6th ed. Oxford: Blackwell Science; 1998. p. 1483-588.
3. Burks JW, Wise LJ, Clark WH. Degenerative collagenous plaques of the hands. Arch Dermatol 1960;82:362-6.
4. Ritchie EB, Williams HM Jr. Degenerative collagenous plaques of the hands. Arch Dermatol 1966;93:202-3.
5. Sehgal VN, Singh M, Korrane RV, Nayyar M, Chandra M. Degenerative collagenous plaque of the hand (linear keratoelastoidosis of the hands). A variant of acrokeratoelastosis. Dermatologica 1980;161:200-4.
6. Mittal R, Chopra A, Gupta K. Degenerative collagenous plaques of the hands. Indian J Dermatol 1984;29:47-51.
7. Johnson B Jr, Honig P. Congenital diseases (Genodermatoses). In: Elder D, Elenitsas R, Jaworsky C, Johnson B Jr, editors. Histopathology of the skin. 8th ed. Philadelphia: Lippincott-Raven; 1997. p. 117-50.
8. Abulafia J, Vignale RA. Degenerative collagenous plaques of the hands and acrokeratoelastoidosis: pathogenesis and relationship with knuckle pads. Int J Dermatol 2000;39: 424-32.
9. Mehregan AH. Degenerative collagenous plaques of the hands. Arch Dermatol 1966;93:633.

B. Jeevankumar, D. M. Thappa, S. Jayanthi*

Departments of Dermatology and STD, and *Pathology, Jawaharlal Institute of Postgraduate Medical Education and Research (JIPMER), Pondicherry - 605006, India.

Address for correspondence: Dr. D. M. Thappa, Professor and Head, Departments of Dermatology and STD, JIPMER, Pondicherry - 605006, India. E-mail: dmthappa@vsnl.net

Insulin induced lipoatrophy

Sir,

Insulin induced lipoatrophy is becoming increasingly uncommon.[1] We came across a 14-year-old girl with insulin induced lipoatrophy presenting as depressed atrophic plaques over thighs and abdomen at the site of repeated self-administered insulin injections.

The patient suffered from insulin dependent diabetes mellitus (DM) since the age of 6 years. Her random blood sugar level at first presentation was 380 mg%. She was initially treated with bovine insulin injections, initially short acting, then long acting, for 3 years. However she developed dimpling and depressed plaques over the anterior aspect of both thighs at the site of the injections, suggestive of insulin lipoatrophy. Since the therapeutic response to bovine insulin injection was unsatisfactory, she was treated with a long acting porcine insulin preparation for the next 2 years. However, she continued to develop bilaterally symmetrical, unsightly depressed plaques over both thighs (Figure 1). She was advised to change the injection site to the abdomen to avoid repeated injections in the thighs. She then developed similar atrophy of the subcutaneous fat on either side of the midline on the abdomen (Figure 1). She has subsequently been using a purified human insulin preparation (a mixture of long acting and short acting insulins) since the

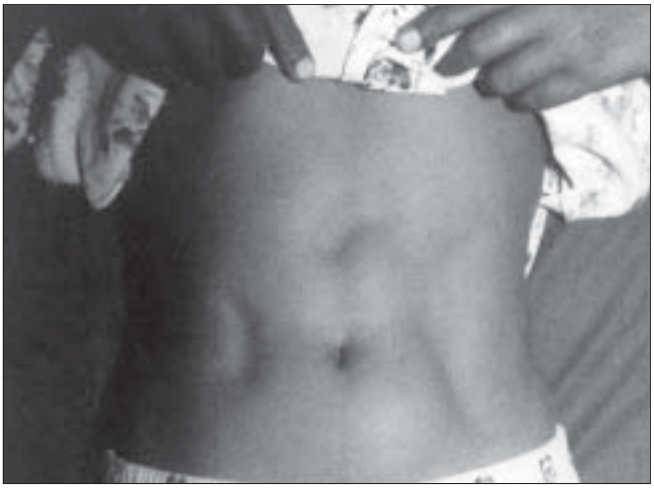

Figure 1: Atrophy of the subcutaneous fat as seen on abdomen

last 3 years. The lipoatrophy has not progressed further, and she has achieved better control of her blood sugar level with purified insulin injections and strict adherence to dietary advice.

Insulin lipoatrophy presents as depressed plaques, histologically showing atrophy of the subcutaneous fat.[2] It is an allergic phenomenon, thought to be due to immune complex deposition. IgG and insulin have been demonstrated in lipoatrophic tissues, and circulating anti-insulin antibody titres are commonly high. It is treated by injections of highly purified soluble insulin, which floods the site with antigen and solubilizes the complexes.[1]

Lipoatrophy localized to injection sites occurs particularly with longer acting preparations. It is becoming more and more infrequent with the use of modern insulins.[1]

REFERENCES

1. Pickup JC, Williams G. Insulin injection treatment and its complications. In: Pickup JC, Williams G, editors. Textbook of Diabetes. 2nd ed. Oxford: Blackwell Science; 1998. p. 33.1-33.20.
2. Fleming MG, Simon SI. Cutaneous insulin reaction resembling acanthosis nigricans. Arch Dermatol 1986;122:1054-6.

Kshiteendra Krishna, Rajendra P. Mane, K. Kavita

Department of Medicine, Bharati Vidyapeeth Deemed University Medical College & Bharati Hospital, Dhankawadi, Pune - 411043, India.

Address for correspondence: Dr. Kshiteendra Krishna, 23, Sopan Baug Cooperative Housing Society, Pune - 411001, India.
E-mail: kavitakcube@rediffmail.com

Treatment of Schamberg's disease with pentoxifylline

Sir,

I read with great interest article by Gandhi et al[1] and would like to offer some comments.

The authors have cited the report of Kano et al and have presumably designed their trial on its basis.[2] Kano et al administered pentoxifylline 300 mg daily for 8 weeks to 3 patients with Schamberg's disease and reported significant clinical improvement after 2-3 weeks. Despite its anecdotal nature, this report attracted wide attention. But, subsequent studies have reported different results. Based on their findings on 2 patients, Basak and Ergin found both the suggested dosage and the duration of therapy (pentoxifylline 400 mg daily for 3 weeks, subsequently increased to 600 mg daily up to 8 weeks) to be inadequate and unable to induce clearing of lesions.[3]

Based on these reports, we performed a multicentric, randomized, investigator-blinded, parallel group, therapeutic trial to compare the effects of oral pentoxifylline 1200mg daily in 3 equally divided doses versus topical betamethasone dipropionate cream 0.05% applied as a thin film locally twice daily on 112 patients, since topical steroids are the traditional therapeutic modality in this disorder.[4] The total treatment period was 8 weeks and follow up was for another 24 weeks. We found that pentoxifylline had a beneficial effect and was more effective than topical betamethasone. However, its effect diminished in many patients at follow-up at six months and we concluded that a 2 month period of therapy was inadequate.

Hence we recommend an even longer period of therapy. This seems to be rational because the dosage regimen followed in our trial was similar to the conventional dosage of pentoxifylline in vascular disorders.[5] Though the cause of Schamberg's disease is not known in almost 75% of cases,[6] capillary stasis has been proposed as a major factor behind the capillaritis. Gravity and