Kanwar et al reported 20-nail dystrophy in a patient of AA due to LP. Brenner et al reported a case of coincidence of five dermatological disorders: vitiligo, AA, onychodystrophy, morphea and LP. Similarly, ulcerative colitis, myasthenia gravis, LP, AA and vitiligo were present in a single patient reported. Patients with AA were found to be at a higher risk for developing LP (RR=2.7; 95% confidence interval, 1.1 to 6.5). However, co-localization is very rare. Dhar et al had reported one child with co-localization of lesions both conditions. The incidence of AA in the Indian population is 0.7% whereas it is 0.8% for LP. The coexistence of these disorders may be purely coincidental. Gilhar et al found that induction of AA was possible with injection of CD8+ cells cultured with follicular homogenate but not with cultured CD4+ cells. The T lymphocyte is also pivotal in regulating epidermal cell recognition and epithelial destruction in lichen planus. T cells become activated via antigen-presenting cells such as Langerhans cells in conjunction with epidermal keratinocytes and co-stimulatory molecules. Though both CD4+ and CD8+ T cells are found in the lesional skin of LP, progression of disease leads to the preferential accumulation of CD8+ cells. The majority of the lymphocytes in the infiltrate of LP are CD8+ and CD45RO (memory)-positive cells and express the γ/δ T-cell-receptor. The ensuing immune reaction by CD8+ T lymphocytes against activated keratinocytes results in epidermal cell damage and development of the lichenoid reaction that is the hallmark of lichen planus.

Further studies might clarify whether co-localization of lichen planus and alopecia areata is a mere coincidence or represents a common pathogenic mechanism in these two predominantly CD8+ T lymphocyte-mediated disorders.

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REFERENCES


ANA-negative systemic lupus erythematosus

Sir,

We report a case of ANA-negative systemic lupus erythematosus diagnosed on the basis of ARA criteria.

A 27-year-old female presented with fever and bilaterally symmetrical dusky erythematous, scaly papules and patches with pigmentation over the scalp,
The malar area of the face (Figure 1), back, neck and extremities of 2 months’ duration. Her oral cavity showed non-indurated ulcers. Diffuse and patchy alopecia was observed over the parietal and occipital areas of the scalp.

Investigations showed normocytic, hypochromic anemia, leukocytosis, raised ESR, hematuria, pyuria, and raised levels of total serum bilirubin, serum glutamate pyruvate transaminase and serum alkaline phosphatase. An X-ray chest showed right mid-zone consolidation. Ultrasonography of the abdomen revealed hepatomegaly suggestive of a diffuse parenchymal affection, upper abdominal lymphadenopathy with minimal ascites, and changes suggestive of acute renal parenchymatous disease. Serum HBsAg, Elisa for HIV-1 and HIV -2, serum antinuclear antibody (ANA), anti ds-DNA and LE cell tests were negative. Serum anti-Ro test was positive. The serum C3 was 94.3 mg/dl. A skin biopsy showed focal atrophy with keratotic plugging in the epidermis, basal cell liquefaction degeneration with colloid bodies, patchy mononuclear infiltrates in the dermis, and melanin incontinence.

The presence of ANA is one of the criteria for the diagnosis of SLE. In 5-10% of cases of SLE, ANA cannot be demonstrated although the other ARA criteria are fulfilled.¹ About 10% of these cases may eventually become ANA positive.¹ Immunofluorescence (IF) assay or enzyme immunoassay (EIA) can detect ANA. In the IF assays substrates such as Hep2 cells, a human laryngeal cell line, are used. They give a higher incidence of positive results.¹ However, in view of the drawbacks like difficulty of reproducibility and requirement for higher quality control, EIA is a better method for routine use. In our patient, we could eventually detect ANA by using EIA designed by Bio-Rad Laboratories. This is a qualitative immunoassay using tetra methyl benzidine in dilute hydrogen peroxide buffer as substrate. The test was repeated in a couple of other laboratories using the same kit.

Circulating antibodies to DNA are almost always present in active disease, and may occur in the absence of antinuclear factor.¹ Antibodies to soluble cellular antigens include anti-Sm antibody, found in 15-25% of patients with SLE, particularly in patients with renal involvement, CNS disease and vasculitis. Anti-RNP antibody occurs in 25% of patients with characteristics of mixed connective tissue disease. Anti-Ro antibody occurs in 30% of patients who will have increased tendency to photosensitivity, renal disease or Sjögren’s syndrome.² Anti-Ro antibody is also found in patients with subacute cutaneous lupus erythematosus (SCLE). In one Indian study, all 7 patients of SCLE were ANA-negative.²

The presence of 6 ARA criteria in our patient, along with classical histopathological findings in the skin biopsy, strongly suggest the diagnosis of SLE in spite of the absence of ANA. Hence this case can be labeled as ANA-negative SLE.

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REFERENCES

Dexamethasone cyclophosphamide pulse therapy for pemphigus

Sir,
The IJDVL September-October 2003 issue covered various aspects of dexamethasone-cyclophosphamide pulse) DCP pulse therapy.¹²³

I would like to make the following important points related to DCP therapy: