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GRISEOFULVIN-INDUCED ACUTE GLOMERULONEPHRITIS

To the Editor,

Griseofulvin is a safe and effective agent for cutaneous dermatophyte infections. Since its introduction in 1958, remarkably few adverse effects have been attributed to it. A 35-year-old female, apparantly healthy otherwise, came with tinea corporis. She was preseribed 500mg/day griseofulvin. A week later she came back with oliguria, pedal oedema, facial puffiness, tiredness and headache. Though her original skin lesions had lessened, patient discontinued the drug after 4 doses attributing these signs to griseofulvin. Examination revealed mild hypertension (150/96mm of mercury), proteinuria (+), cellular casts and microscopic haematuria. A provisional diagnosis of acute glomerulonephritis (AGN) was made. There was no history or evidence of previous streptococcal skin infection, connective tissue disorders or other drug intake. She was advised rest, salt and fluid restriction as well as regular follow up. On the 7th day the puffiness of face and oedema had come down, blood pressure was 140/86mm of mercury and urinary findings were normal. At this point ASO titre was normal. Tests for antinuclear antibodies and rheumatoid factor were negative. Subsequent follow up for 3 weeks revealed normal clinical and investigative findings.

So far known adverse effects of griseofulvin include proteinuria, cylinduria and serum sickness. It has been proved in experimental animal models that necrotizing angitis, due to the deposition of immune complexes and activation of complement, is responsible for many of the manifestations of serum sickness.[1,2] Comparable mechanism has not been demonstrated in drug induced serum sickness but is assumed to be similar. Post streptococcal AGN is known to be mediated by immune complex deposition. Many drugs are associated with the development of glomerular disease. However, it is usually difficult to establish a direct cause and effect relationship. In a few situations association is clear cut and reexposure has led to the recurrence of the disease. In this patient investigations failed to prove any other causes for AGN. Renal biopsy is useful in characterising the nature of the underlying lesions but need not be done in every case. There is a clear history of association with griseofulvin intake. Patient did not give consent for rechallenge of griseofulvin. To the best of my knowledge, AGN induced by griseofulvin has not been reported to date.

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MIXED CONNECTIVE TISSUE DISORDER

To the Editor,

We are herewith reporting a case of overlap syndrome, a form of mixed connective tissue disorder (MCTD). A 15-year-old boy was admitted with a history of irregular fever and joint pains of 3 months duration. He also had productive cough and on a few occasions had

produced blood streaked sputum. Since 15 days, he was having difficulty in getting up form sitting position because of pain and weakness in thigh muscles. On examination there was mild pallor and a temperature of 100^0 F. There was generalised thickening of skin and diffuse alopecia. He had tenderness in both knee joints and restriction of movements (especially extension) of left knee joint because of gross thickening of skin on medial side of thigh from groin right upto the knee joint. The underlying muscles were swollen and extremely tender. Per abdominal examination showed hepatosplenomegaly. Other examination of musculoskeletal system showed a generalised decrease in muscle power but mainly the proxymal muscles were involved. Investigations showed Hb 9.5 gm%, normal total and differential counts, urine (routine and microscopic) was normal. Liver function tests and renal function tests were normal. ECG and X-ray chest were normal. Fundus examination was normal. Ultrasonography of abdomen revealed hepatosplenomegaly. Sputum for AFB was negative. Skin biopsy showed features of scleroderma, muscle biopsy revealed features of chronic myositis.

So this patient was having features of both scleroderma and polymyositis. The joint pains, pulmonary symptoms and diffuse thickening of skin and nonscarring alopecia were features of scleroderma in this patient, while the muscle weakness which was present in the proximal muscles was the feature of polymyositis. Thus our patient was a case of overlap syndrome which is a mixed connective tissue disorder characterised by a combination of features of multiple collagen vascular diseases.

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MYCOLOGICAL ASPECTS OF DERMATOMYCOSIS

To the Editor,

Please refer to Letter to the Editor entitled "Mycological aspects of dermatomycosis in Yavatmal (Maharashtra)" by KV Ingole et al. We want to share our observations in a similar study done in our institution during the period January, 1995 to December, 1995.

From 298 clinically diagnosed cases of tinea infections, skin scrapings/nail clippings/ hair specimens were examined for the presence of fungal elements by direct microscopic examination in 10% KOH solution. 195 cases (65.43%) were KOH positive and they were further subjected to culture study on Sabouraud's dextrose agar media. 140 cases (71.80%) showed culture growths of pathogenic dermatophytes.

The commonest dermatophyte isolated was Trichophyton rubrum (88 isolates, 62.86%) followed by Trichophyton mentagrophytes (50 isolates, 35.71%), Trichophyton tonsurans (1 isolate, 0.7%) and Epidermophyton floccosum (1 isolate, 0.7%).

Trichophyton rubrum was found to be the main aetiological dermatophyte species responsible for dermatophytoses in our region (62.86%), followed by Trichopyton mentagrophytes (35.71%). This is in confirmity with other published reports.[1]

The various clinical types of tinea infections in the 140 culture positive cases were tinea cruris (42 cases, 30%), tinea unguium (33 cases, 23.57%), tinea corporis (30 cases, 21.43%), tinea buttocks (15 cases, 10.71%), tinea pedis (10 cases, 7.14%), tinea manuum (5 cases, 3.57%), tinea capitis (2 cases, 1.43%), tinea barbae (2 cases, 1.43%) and tinea faciei (1 case, 0.7%). This is also in