



Fig. 2. Sharpened pencil appearance of metatarsals with osteolysis of their heads. Osteolysis and new bone formation at base of phalanges is seen.

in spite of marked deformity and this is a salient clinical feature of AM.

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Reference

1. Moll JMH. The clinical spectrum of psoriatic arthritis. *Clin Orthopaed* 1979; 143: 66-75.

MINOCYCLINE IN CHRONIC FOLLICULITIS OF LEGS

To the Editor,

Chronic folliculitis of legs (CFL) is a chronic and recurrent problem caused by *Staph aureus*. This condition is encountered in young Asian males commonly.¹ Various agents like Cotrimoxazole, Ciprofloxacin² and PUVA therapy have been partially successful. Minocycline which is a broad spectrum

Ind J Dermatol Venereol Leprol 1996; 62

antibiotic with relatively low toxicity is effective against staphylococci which is resistant to other tetracyclines.³ This pilot study was undertaken to know the response of minocycline in CFL.

Ten consecutive patients who attended the OPD of Rajah Muthiah Medical College Hospital and were clinically diagnosed to have CFL were included in the study. All baseline parameters were measured and clinical work up done. Minocycline, in a dose of 100mg once daily 1 hour before food, was given to all patients for 21 days. Two patients were lost to follow up. Among 8 patients, there was 50% clearance at the end of 2 weeks and complete clearance in 3 weeks period. Patients were followed up upto 6 months and only 2 out of 8 patients (25%) showed a mild clinical recurrence.

Since minocycline is effective in resistant cases of *Staph aureus*, this drug may be tried as a first line of therapy in CFL. None of our patients developed any side effects due to minocycline and hence this drug may be considered safe. However, a long term follow up is needed in a larger number of patients to know the effect of this drug.

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References

1. Highet AS, Hay RJ, Roberts SOB. Bacterial infections. In: Champion RH, Burton JL, Ebling FJG, editors. *Textbook of dermatology*. Oxford: Blackwell, 1992: 972-3.
2. Balachandran C, Malpani S, Srinivas CR. Ciprofloxacin therapy in chronic folliculitis of legs. *Ind J Dermatol Venereol Leprol* 1996; 61: 212-3.

- Hay RJ, Champion RH, Greaves MW. Systemic therapy. In: Champion RH, Burton JL, Ebling FJG, editors. Textbook of dermatology. Oxford: Blackwell, 1992: 2947-8.

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, apparently healthy otherwise, came with tinea corporis. She was prescribed 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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References

- Jager BV. Intravenous administration of modified gammaglobulin: several studies on a patient with gammaglobulinemia. Arch Intern Med 1967; 119: 60.
- Schmidty AP. Anaphylactic transfusion reactions associated with anti Ig A antibody. N Engl J Med 1969; 280: 188.

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