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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