liver and severe impairment of hepatic functions is considered a contraindication to PUVA therapy.[2] We report a case of acute reversible hepatic toxicity by trimethoxy psoralen (trioxsalen).

A 45-year-old Hindu female presented with vitiligo extending to chest and legs. After complete evaluation and baseline investigations like haemogram, blood sugar and LFT, she was given trimethoxy psoralen as 10 mg orally daily and was advised exposure to sunlight after 2 hours. After 7 days she came back with loss of appetite, nausea, vomiting and icterus. Psoralen was stopped and LFT done. Bilirubin was 3 mg%, SGOT 58 IU and SGPT 110 IU. Serum alkaline phosphatase was 27.5 KA units. She was managed for acute liver dysfunction with diet and drugs. Three weeks follow-up investigations revealed normal serum bilirubin, SGOT/SGPT and serum alkaline phosphatase. Patient was asked to discontinue treatment for 2 months. This resulted in increase in vitiligenous lesions. On her insistence she was again put on trioxsalen therapy, but within 3 days she developed nausea, and vomiting followed by icterus. Her investigation revealed increased serum bilirubin levels, SGOT and SGPT were 62 and 132 IU, respectively. Serum alkaline phosphatase was 31 KA units. Trioxsalen was stopped. Presently she is under observation and requires alternate therapy for vitiligo.

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References

1. Pariser DM, Wyles RJ. Toxic hepatitis from oral methoxalen photochemotherapy. J Am Acad Dermatol 1980; 3: 248.
2. Wolf K, Honigsmann H. Clinical aspects of photochemotherapy. Pharmacol Ther 1981; 12: 381.

PRIMARY LOCALISED CUTANEOUS AMYLOIDOSIS

To the Editor,

It was interesting to learn about the effectiveness of colchicine in the treatment of primary cutaneous amyloidosis.[1] In the materials and methods the authors state, "Clinical features were so characteristic that other investigations for confirmation of diagnosis were not considered necessary".

Macular amyloidosis can, at best be suspected on clinical examination. It is not easy to differentiate it from other conditions such as lichen planus pigmentosus, frictional melanosis, ashy dermatosis and numerous other conditions which have an interface dermatitis as histopathological finding. Although I do not doubt the clinical ability of the authors, in my opinion amyloid should have atleast been detected by H & E (a difficult task) if not be special stains. I hope the authors publish another paper confirming the efficacy of the drug after establishing the diagnosis of primary cutaneous amyloidosis.

CR Srinivas
Manipal

Reference

1. Chakravarthy K, Chanda M. Role of colchicine in primary localised cutaneous amyloidosis Ind J Dermatol Venereol Leprol 1995 ; 61 : 268-9.

REPLY

To the Editor,

We thank Dr Srinivas for taking keen interest in our article. I am putting our clarification as follows :

1) We agree with Dr Srinivas that it is a difficult task to detect amyloid by H & E stain. Amyloid can often be recognised in H & E section provided that it is present in sufficiently large amount.[1] As it in not a confirmatory test