

REVERSIBLE ONYCHOMADESIS INDUCED BY CARBAMAZEPINE

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An epileptic teenager put on carbamazepine therapy presented with onychomadesis 6 months later. Carbamazepine was substituted with phenytoin. Affected nails were shed and healthy nails regrew in 5 months.

Key Words : Onychomadesis, Carbamazepine

Introduction

Spontaneous separation of nails from the matrix area is called onychomadesis which results from a limited lesion of the proximal matrix.¹ Besides some diseases, various drugs like penicillin, arsenic, lead, parathormone and retinoids have been implicated as causative agents.^{1,2} There is only one report of carbamazepine (CBZ) induced onychomadesis earlier.¹

Case Report

A 14-year-old boy presented with the complaint of asymptomatic nail changes of 4 months duration. Neurologists had made a diagnosis of right temporal lobe epilepsy with mild mental retardation and had put him on carbamazepine 200 mg bid for past 6 months. No other relevant history was present.

On examination, all the finger and toe nails showed onychomadesis with the thumb and great toe nails being most prominently involved (Fig.1). The detachment was upto 5 mm short of free edge of the thumb nail. He also had a Becker's naevus over the right side the trunk and right thigh, and also 3 café-au-lait-macules each measuring less than 1cm in diameter.

All the routine investigations were within normal limits. The KOH preparation and

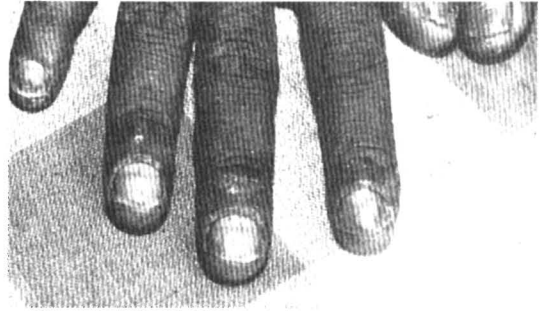


Fig.1. Finger nails showing onychomadesis.

culture for fungus were negative.

The patient was started on phenytoin 100 mg per day and carbamazepine was tapered over 2 weeks. Total dose of carbamazepine received was 64.2 g. No specific therapy was given for the nails. The patient came for review after 5 months. He reported that all the nails had shed a month after stopping CBZ and there was regrowth of the normal nails. This time, the left thumb nail showed a greyish linear, pigmented band 1 mm in width extending through the whole of its length in the centre.

Discussion

CBZ is an anticonvulsant structurally related to imipramine. 3% of patients on CBZ develop cutaneous eruptions.¹ In our patient, with the absence of any other apparent aetiologic factors and the sequence of events, appearance of onychomadesis with CBZ therapy and regeneration of healthy nails following cessation of CBZ therapy, we implicate CBZ as the agent inducing onychomadesis.

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It is possible that CBZ or its metabolites inhibited matrix function, and the rare occurrence of this adverse reaction could have a pharmacogenetic basis. The longitudinal greyish band might have been due to melanocyte stimulation similar to that caused by adriamycin.¹

References

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