made in our patient because of the presence of highly characteristic melanin deposits in the MRI study.\textsuperscript{1} This is the only method to diagnose CNS melanosis in a living patient.\textsuperscript{3} Secondly, in our patient there were no clinical indicators suggestive of a developing cutaneous melanoma, viz. changes in colour, size, shape, rapid growth rate, proliferative nodules or ulceration, in any of the GCMM. Hence doing a skin biopsy to rule out a developing cutaneous melanoma was not indicated. We would like to mention that malignant transformation within GCMMs is exceptionally rare in the neonate and has been reported in only three neonates.\textsuperscript{4} This recent review has stated that even if clinical indicators of cutaneous melanoma are present, careful histological evaluation may eventually reveal the lesion to be benign.\textsuperscript{4} Also, the melanin deposits detected in our patient’s MRI did not reveal any perilesional edema or necrosis or enhancement with gadolinium contrast; ruling out malignant degeneration.\textsuperscript{3} Thirdly, the presence of melanin deposits in our patient’s amygdala and thalami is not ‘strange’. A recent report has stated that the most common area of involvement in their case series was the amygdala; detected in eight out of 10 MRIs.\textsuperscript{5} Also presence of melanin deposits in thalami in cases of neurocutaneous melanosis is common.\textsuperscript{2}

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References


Clinical characteristics of organophosphate-induced delayed polyneuropathy

Sir,

I read with interest the recent article by Chatterjee et al.\textsuperscript{1} They report a patient with organophosphorus (OP) poisoning who developed intermediate syndrome (IS) and organophosphate-induced delayed polyneuropathy (OPIDP) in succession. However, I would like to make certain comments regarding the diagnosis of OPIDP in this case.

OPIDP as a clinical syndrome is well known and was described even before the association of IS with OP poisoning became known. The prevalence of OPIDP is variable; however, it occurred in 22% of patients with OP poisoning in a recent study.\textsuperscript{2} OPIDP occurs within a period of one week to five-six months of the ingestion of an OP compound, almost exclusively in patients with preceding acute cholinergic toxicity related to severe acute exposure (to an OP compound). However, there are occasional reports of OPIDP developing in a patient without prior cholinergic toxicity following a low-dose chronic exposure to OP.\textsuperscript{3}

The neuropathy in OPIDP is typically a symmetrical sensorimotor neuropathy, with a distal predominance.\textsuperscript{4} Initial symptoms are paraesthesia in the lower limbs and pain in the calves, followed by motor involvement of the lower limbs, manifested by leg weakness, foot drop and muscle hypotonia. The neuropathy in OPIDP is motor-predominant, and pure sensory neuropathy does not occur. If sensory symptoms are present, they are always milder than the motor component. Upper limb symptoms are always preceded by lower limb involvement. OPIDP is typically a “dying-back” neuropathy as revealed by clinical, electrophysiological and nerve biopsy data. The neuropathy has a typical “subacute” course of progression over a two-week period.\textsuperscript{5} In addition, features of pyramidal tract and posterior column involvement may be noted later in the course of illness. The patient reported by Chatterjee et al presented with a wrist drop and had features of right radial nerve palsy alone (even after electrophysiological studies) without any involvement of the lower limbs. This does not fit with the description of OPIDP as mentioned above and an alternative etiology for the same should be considered.

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