Prallethrin poisoning that taxes the brain

Sir,

We reviewed the case report of Chandra *et al*.[1] from the point of clinical, toxicological and pharmacological aspects with great interest. The pyrethroids are commonly used group of insecticides to which there is significant human exposure. Despite the original belief that they have low human toxicity, there have been few reports of human fatalities. Their acute toxicity is dominated by pharmacological actions upon the central nervous system (CNS), which is pre-dominantly mediated by prolongation of the kinetics of voltage-gated sodium channels, although other mechanisms operate at higher dose.[2] On the basis of chemical structure and manifestations, clinical features of pyrethroid compounds are classified as type I and II syndromes. Type I presents as reflex hyperexcitability and fine tremor and type II as choreoathetosis, salivation, seizures and potent sympathetic activation.[3] Pyrethroids also decrease the influx of chloride currents through voltage-dependent chloride channels and thus responsible for the typical clinical features of poisoning.[4] The chaotic electrical discharge in the CNS results in seizure activity. In epileptic patients, often there will be a focus in an abnormal cortical area; while pyrethroid induced seizure usually originates in previously normal neurons. Phenytoin is widely used in idiopathic seizure disorders or patients with defined structural or electrical foci of seizure activity. However, pyrethroid induced seizure involve diffusely lowering the seizure threshold due to the proconvulsant activity. In an animal model, pre-treatment with phenytoin did not alter the pyrethroid-induced proconvulsant activity and the results suggest that the effects of pyrethroids on pentylenetetrazol seizure threshold are mediated via an interaction with peripheral-type benzodiazepine binding sites.[5] Furthermore, apart from being not effective as observed in this case also, phenytoin may worsen the overall toxicity. We have previously reported generalized tonic-clonic seizures in four patients with propofol.[6] It is reported that in high doses propofol act as anticonvulsant by depressing both cortex and subcortex, but in low doses it may act as proconvulsant by inhibiting the inhibitory subcortex, which results in the "release" of normal hyperexcitability in the cortex.[7] Hence, benzodiazepines and barbiturates are useful in this setting. Pentobarbitone is almost always effective against type II pyrethroid poisoning, probably due to its dual action as a chloride channel agonist as well as a membrane stabilizer[8] and controls pyrethroid-evoked foci. From the point of patient safety, it is worth to recall toxicological causes for convulsions in emergency room if patients do not respond to conventional anticonvulsants; recognize specific conditions based on coexisting clinical signs and symptoms and/or laboratory data; and respond with pharmacological antidotes or supportive measures without delay or harm.

As pyrethroids were widely used in our area during the recent dengue epidemic, number of pyrethroid poisoning was reported more compared with last year. Hence, practitioners working in emergency room shall be sensitized on clinical manifestations, course of poisoning, therapeutic modalities and outcome.

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