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Mosquito Coil (Allethrin) Poisoning in Two Brothers

The pattern of accidental poisoning in children is changing. There is an increasing incidence of poisoning with common household insecticides(1,2). Mosquito (Allethrin) coil, a type of pyrethroid is almost ubiquitous in modern households. We report two brothers brought in unconscious state with alleged history of mosquito coil ingestion.

Two cousin brothers 4 yrs and 3 yrs were brought in department of emergency medicine with history of one episode of generalized tonic-clonic convulsion followed by unconsciousness and violent behaviour. Parents suspected the intake of mosquito coils (they were not sure of quantity of coils ingested). There was no history of pica in the children. At admission both were comatose ($E_2V_2M_4$) with normal pupillary size and reaction. Vitals were normal. There were no meningeal signs or focal neurological deficits. Routine hematological studies, serum bilirubin, ALT, AST and blood sugar were within normal limits. Stomach wash revealed clear fluid. The older child had two episodes of generalized convulsions, which were managed with intravenous diazepam. Both children regained consciousness in 36 hours with supportive

management. They accepted ingestion of one mosquito coil each. On follow up after two weeks both were neurologically normal.

Pyrethroids are common insecticides. There are only few reports of pyrethroid poisoning from India(2,3). Pyrethroids are of two types. Allethrin, present in mosquito coil is a type I pyrethroid which lacks a cyano group and causes repetitive discharges in nerve fibres (by acting on sodium channels) leading to hyperexcitation as compared to Type II pyrethroids (deltamethrin and fenvalerate) which causes nerve membrane depolarization and block leading to paralysis. Allethrin poisoning thus leads to ataxia, loss of coordination, hyperexcitation and convulsions. Recently calcium channels have been also found to be another primary target for allethrin(4). Pyrethroids are generally considered to be safe insecticides in humans because of their rapid biotransformation by ester hydrolysis and hydroxylation to their inactive acids and alcohol components. The fatal dose for allethrin is not known. In our case ingestion of one mosquito coil was sufficient to lead to symptoms. Most cases of acute pyrethroid poisoning recover within 1-6 days with normal neurological outcome on follow up. Deaths have been reported in cases where convulsions increase in duration and frequency and do not stop within 2-3

weeks(5). The fatality with insecticides in India has been reported to be 12.5 - 25%(3). No specific antidote is known either for type I (allethrin) or type II pyrethroids. The treatment is entirely supportive and symptomatic. In conclusion, mosquito coil (allethrin) poisoning though rarely reported can be much frequent in occurrence due to its easy accessibility in households.

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Severe Thrombocytopenia in Association with Hepatitis A

We wish to report an interesting case who came to our attention recently. N, a 12-year-old girl, was referred to us with history of low grade fever, malaise and nausea of 1 week duration followed 2 days later by hematemesis, menorrhagia and purpura. She had attained menarche 6 months prior to this episode and had no history of menorrhagia previously.

On examination, though conscious and alert, she was ill looking with severe hypotension (BP 74/50 mmHg) and a pulse rate and respiratory rate of 124/min and 32/min respectively. Her weight and height were 32 Kg and 141cm (expected 36Kg and

149 cm) respectively. She had mild jaundice, severe pallor and purpuric spots over the face and extremities. There was no lymphadenopathy or bone tenderness. Systemic examination revealed a hepatosplenomegaly of 12 cm and 2 cm respectively and other systems were essentially normal. A slit lamp examination excluded presence of Kayser Fleischer ring. Laboratory data indicated a Hb of 4.5 g/dL, total count of $7.2 \times 10^3/\mu\text{L}$ ($\text{N}_{53\%}$, $\text{L}_{39\%}$, $\text{M}_{6\%}$, $\text{E}_{2\%}$), platelet count of $5 \times 10^3/\mu\text{L}$ and no evidence of abnormal cells on peripheral smear. Aspartate Aminotransferase was 2116 U/L (Normal: 15-45 U/L), Alanine Aminotransferase 1873 U/L (Normal: 7 - 35 U/L) and alkaline phosphatase 328 U/L (Normal: 100-320 U/L). Total serum bilirubin was 5.9 mg/dL (conjugate bilirubin of 2.5 mg/dL).

Serum albumin, glucose, urea, creatinine