Mosquito Repellent Vaporizer Poisoning – Is the culprit Transfluthrin or Kerosene?

A 3-year-old boy presented with complaints of sudden onset vomiting and difficult breathing following accidental ingestion of a liquid mosquito repellent vaporizer (LMRV). He was alert, respiratory rate was 54/min, and there were subcostal, intercostal retractions, nasal flaring, and fine crepitations over right lower chest. Arterial blood gas was normal on 6 L/min of inhaled oxygen. Chest radiograph done after 6 hours, showed pneumonitis in right middle and lower zones. He improved over 48 hours without antibiotics.

The use of LMRV is common in setting of infections like malaria and dengue. These products contain derivatives of pyrethrins as mosquito repellent and kerosene as the solvent. Pyrethrins have poor dermal absorption and rapid metabolism. Popular brands use 0.88% of the chemical, that was transfluthrin in the present case. Toxicity results mainly in central nervous system (CNS) symptoms like headached, dizziness, drowsiness, status epilepticus and respiratory failure. These chemicals act by prolonging the opening of sodium channels leading to increased influx of sodium ions and thus hyper-excitation of the nervous system [1]. Cardiac dysfunction has also been described, but lung injury is not commonly reported [2,3].

The poisoning of the second component of LMRV – kerosene – is well recognized. Being highly volatile and less viscous, it can cause life threatening lung injury in the form of aspiration pneumonitis and ARDS. Chest radiograph may be normal initially and abnormalities like fine perihilar opacities, basilar infiltrates, and atelectasis may appear after some hours of ingestion. The use of LMRV increases the risk of kerosene poisoning even in setting where it is not used as fuel. We emphasize that LMRV poisoning should be treated as kerosene poisoning with supportive and symptomatic management and recommend that use of household LMRV should be recognized and listed as a risk factor in the etiology of kerosene poisoning.

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Lead Poisoning in an infant

An 11-month-old boy weighing 6.75 kg, resident of a rural area of West Bengal, presented with first episode of generalized convulsions without fever or any other sign of infection. Hemoglobin was 5.2 g/dL with hypochromic microcytic picture, normal reticulocyte count (1.8%), low ferritin (19 ng/mL) and a negative direct Coomb's test. C-reactive protein was negative; hemoglobin electrophoresis and magnetic resonance imaging of brain were within normal limits. *X*-ray of the wrists showed features of rickets along with prominent lines on the metaphyseal ends of the long bones and short bones of the hands. Vitamin D level (13.3 ng/mL) was low. A high blood lead level (121.8 µg/dL) and urine lead level (44.8 µg/dL) along with metaphyseal lead lines documented lead poisoning. History revealed that the family had a small lead battery assembly workshop at home. The child also had pica.

Lead poisoning is unusual nowadays. In a recent study [1] from Delhi, 12 % of the school children had high (>10 µg/dL) blood lead levels. This signifies that measures such as prohibition of leaded pipes, leaded petrol, printing ink are not sufficient. Cottage industries, for example, silversmiths, print shops, brass works, small lead battery assembly workshops, radiator repair

workshops, and other such workshops, which are considered as "hot-spots" should be searched for. Most commonly they are in home settings or where young children have ready access, creating more chances of exposure to the children [2], as in our case. An initiative for "hot-spot" investigations and interventions in cottage industries is thereby mandated.

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Splenic Infarction in *Plasmodium vivax* Malaria

This is in response to a recently reported case of splenic infarction due to vivax malaria [1]. We had a similar child, who on further evaluation was found to have an elevated titer of antiphospholipid IgM. We managed the child with low molecular weight heparin followed by oral warfarin.

In recent years, the association between infections and antiphospholipid syndrome has been reported in several epidemiologic and experimental studies that support the idea of infectious induction of aPL [2]. Witmer, *et al.* [3] reported two children with *Mycoplasma pneumoniae* pulmonary infection complicated by the development of splenic infarction and transient antiphospholipid antibodies. Among parasitic infections, malaria and leishmaniasis have been linked with the production of aPL. Avcin and Toplak [4] reviewed 100 antiphospholipid syndrome cases associated with infection and summarized clinical and experimental evidence on the association between aPL and infectious diseases, they also emphasized a possible association with immunizations. Consigny, *et al.* [5] found a high prevalence of serum cofactor independent aCL, which is a type of antiphospholipid antibody in 137 individuals chronically exposed to *Plasmodium falciparum* or *vivax* infections.

We opine that antiphosholipid antibodies should be done in all unusual cases of thrombosis. Anti-phospolipid antibodies usually take 3-6 months to disappear, till that time the patients should be on anticoagulant prophylaxis.

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