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 l49 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 $7.2 \times 10^3/\mu L$ ($N_{53\%}$, $L_{39\%}$, $M_{6\%}$, $E_{2\%}$), platelet count of $5 \times 10^3/\mu L$ 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 bilurubin was 5.9 mg/dL (conjugate bilurubin of 2.5 mg/dL).

Serum albumin, glucose, urea, creatnine

and electrolytes were normal, though prothrombin time was prolonged to 30 seconds (Normal: 10.2-12.0 seconds). Ultrasound of the abdomen showed hepatosplenomegaly but no evidence of biliary tree dilatation, ascitis or space occupying lesion.

Serum IgM for antihepatitis A antibody (by radioimmuno assay) was strongly positive (2.1 OD ratio; normal: up to 0.9 OD ratio) though it was negative for Hepatitis B and C. IgM and IgG titres for Infectious mononucleosis were equivocal (titre of 1:28). Serum ceruloplasmin was 50 mg/dL (Normal: 30-50 mg/dL). The bone marrow study was normal.

The child was transfused with 1 unit of packed cells, 4 units of platelet concentrate along with oral progesterone and other supportive therapy which included IV fluids, ranitidine, antibiotics and vitamin K. Steroids and intravenous immunoglobulins were withheld owing to doubtful efficacy and prohibitive cost respectively.

Over a period of 14 days of hospital stay , her general condition improved and her liver enzymes and platelet count reverted back to normal. She is currently doing well and is under follow up for the last 5 months and she has had no further history of menorrhagia since then.

The clinical profile and serology associated with normal peripheral smear made us conclude that the thrombocytopenia was indeed a result of HAV infection(1). Equivocal (1:28 titer) results of IgM and IgG for EBV was considered amnestic in nature(2) and absence of Kayser Fleisher ring with normal Ceruloplasmin further supported our diagnosis.

Abnormal hematological findings especially thrombocytopenia has been reported following hepatitis (mainly with HBV and HCV infection in adults)(3). However, thrombocytopenia as a result of HAV infection in children has rarely been reported. The pathogenesis of thrombocytopenia has been postulated to be caused as a result of deposition of immune complexes on the platelet surface(4) or to the development of anticardiolipin antibodies(5). We were unable to pinpoint the cause owing to financial constraints.

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