There are several points against Hirayama’s disease including the age, sex, onset and distribution of weakness. Also, there is no characteristic worsening of weakness when exposed to cold. MRI spine did not show any localized atrophy of the anterior aspect of C7, C8, T1 segment of the spinal cord. EMG and muscle biopsy findings were also not absolutely characteristic.

Overall the body of evidence suggests the diagnosis in the case to be lead neuropathy, and not Hirayama’s disease. I request the authors now to contact the affected child and her parents to look for the factors for lead excess in her. May be in that process many others in the community can be detected to have lead toxicity.

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Retinal hemorrhages in children are important clinical findings. They may be associated with trauma or systemic diseases(1). In the absence of bleeding disorders, retinal hemorrhage most often result from non-accidental injuries such as direct streaking of the head, severe shaking causing acceleration - deceleration and thoracic compressions. Sever contraction of chest muscles in convulsion, crying or vomiting may cause retinal hemorrhages(2,3).

The prevalence of retinal hemorrhage after convulsions is unclear, and there are few and small studies in this relation(2-4). So, we conducted this study to evaluate the incidence, nature and distribution of retinal hemorrhage in children after a convulsion which required hospitalization and to help clinicians how to deal with this problem.

Thirty one children aged 2 months to 5 years who hospitalized in pediatric ward (of Shahid Beheshti hospital) due to acute seizure attack, in 2002 were included in the study. All
children underwent dilated fundus examination with indirect ophthalmoscope within 48 hours after attacks. Informed consent was obtained from their parents. A detailed history of nature, duration of seizure and any associated history of trauma or vomiting was taken, then complete neurological examination was done by pediatric neurologist. Classification of seizure was done on the basis of nature and type of epilepsy. The study was designed on type, pattern, distribution, and rate of resorption with appropriate follow up in the out-patient department of those children who were noted to have retinal hemorrhage.

Thirty one children with convulsion were examined including 19 boys (61.3%) and 12 girls (38.7%). Their ages ranged from 2 months to 5 years and a median of 18 months. Ten (32.2%) children were less than 2 years old. 29 (93.5%) had generalized seizures, 15 (48.4%) had febrile seizure, and 2 (6.5%) had complex seizure. In two cases with generalized seizure, we noticed streak retinal hemorrhage that disappeared 2 weeks later in both of them.

Retinal hemorrhages occur when blood extravasates from blood vessels due to variety of causes from benign to life-threatening conditions. It is usually seen in children with trauma (including child abuse), vomiting, coughing or cardiopulmonary resuscitation. Theoretical mechanism is sudden rise of retinal venous pressure secondary to forceful thoracoabdominal muscle contraction. Such a mechanism is also possible after convulsion. There are a number of reports in literature about retinal hemorrhages after convulsion(3,4).

The prevalence of retinal hemorrhage after convulsion has not been established in review of literature. Sandramouli, et al. in a study of 33 children with convulsion find no retinal hemorrhage(2), but Mei-Zahav in 153 children with convulsion attacks noticed one case with retinal hemorrhage(4).

Our study in 31 children with convulsion revealed 2 patients (7%) with streak unilateral retinal hemorrhage in first 48 hours after convulsion attacks. Although theoretically convulsion can cause retinal hemorrhage in children, but its occurrence is very low and a direct connection between the convulsion attacks and retinal hemorrhages could not be proved.

Due to low occurrence of retinal hemorrhages with convulsion, we can not rule out its occurrence. Statistically, it has been shown that proving prevalence less than 1% requires examining 300 children and finding normal fundi in all of them(5). So, the finding of a retinal hemorrhage in a child after a convulsive episode should therefore trigger an extensive search for other causes, such as non-accidental injuries including child abuse.

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Biliary Ascariasis

Ascariasis, affecting 60% of children in endemic areas, is not generally a life threatening problem. But it can at times produce serious biliary or pancreatic complications, which need early detection and treatment.

An 8-year-old girl from a low socio economic background was admitted with history of fever with chills and rigors of one-month duration, intermittent abdominal pain and bilious vomiting of 15 days duration. There was history of passing round worms in the stools and constipation. She was febrile, anemic, ill looking, toxic, dehydrated with icterus and had dry coated tongue. Pulse rate was 100/minute, and B.P 110/70 mmHg. The abdomen was tender with guarding, hyperesthesia and moderate hepatomegaly. She had no splenomegaly or ascites. Other systems were normal. Clinically, enteric fever was suspected.

Investigations: Hemoglobin 7 g/dL; peripheral blood smear: normocytic hypochromic anemia, with no malarial parasite. Blood counts: TLC 4650/cumm, DLC: N - 72%, L - 20%, E - 3%, ESR - 26 mm at first hr; blood Widal was positive with a titre of TO - 1:240, TH - 1:480, TAH - 1:30 and TBH - 1:30; Blood culture for Salmonella typhi was negative; Serum bilirubin 10 mg/dL (direct 7.8 and indirect 2.2 mg/dL); Serum AL T: 300 IU/L and serum alkaline phosphatase 350 IU/L. Stool examination was normal.

Ultrasound abdomen revealed a well distended gall bladder with wall thickness of 2mm and no evidence of calculi. The common bile duct was 6mm and was mildly dilated. There was a linear echo genic tubular structure with a central lumen within the distal CBD suggests Biliary Ascariasis (Fig. 1).

![Fig. 1. Mildly dilated CBD with a linear echogenic tubular structure with central lumen within the distal CBD-Biliary ascariasis.](image-url)