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SCIENTIFIC LETTER

H1N1 Infection Presenting as Acute Liver Failure in an Infant

Vijay Kumar[1] · Soumalya Chakraborty[1] · BC Gowtham[1] · Surjeet Kumar[1] · Suresh Kumar Angurana[1] · Jayashree Muralidharan[1]

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To the Editor: An 8-mo-old boy presented with fever, cough, and rapid breathing for 10 d. At the referring hospital, he was admitted for 4 d and received oxygen, intravenous fluids, and antibiotics. On day 4 of admission, he developed one episode of generalized tonic-clonic seizure followed by encephalopathy and was referred to us. There was no history of aspirin intake. Examination revealed weight 7 kg (−1.06 Z score), pulse 160/min, gasping respiratory efforts, feeble peripheral pulses, SpO_2 82% on room air, pallor, no jaundice, and random blood glucose 43 mg/dl. Central nervous system (CNS) examination revealed Glasgow Coma Scale (GCS) of 8, no cranial nerve deficits, increased tone, brisk deep tendon reflexes, and upgoing planters. Respiratory examination revealed bilateral crepitations. A disseminated viral illness causing respiratory and CNS involvement was considered as the likely possibility. The treatment included positive pressure ventilation, intravenous fluids and antibiotics (ceftriaxone, cloxacillin, acyclovir, and oseltamivir), sedation-analgesia, phenytoin, and 3% saline infusion. Investigations revealed hemoglobin 7.2 g/dl, total leukocyte count (TLC) 8600/cu mm, platelet count 2,50,000/cu mm, Serum glutamic oxaloacetic transaminase (SGOT) 4988 IU/L, Serum glutamic pyruvic transaminase (SGPT) 3323 IU/L, bilirubin 1.5 mg/dl, prothrombin time (PT) 28 s, activated partial thromboplastin time (APTT) 31 s, international normalized ratio (INR) 2.8, serum ammonia 252 μmol/L, and normal renal function test. Chest radiograph revealed interstitial infiltrates. Nasopharyngeal swab tested positive for H1N1 by RT-PCR. Hepatitis A, B, C, E, Epstein Barr virus, and Herpes simplex virus serologies were negative. The CSF analysis (on day 3) and CT head were normal. Further, rifaximin and lactulose were added. He showed gradual improvement in clinical and biochemical parameters, was extubated on day 5, received oseltamivir for 7 d, and discharged on day 8, and had pre-morbid neurological status on 3 mo follow-up.

H1N1 (Influenza A) is usually an acute and self-limited respiratory illness. The severe cases can present with pneumonia, acute respiratory distress syndrome, septic shock, neurological involvement, myocardial dysfunction, and multiorgan failure [1]. H1N1 with liver involvement is rarely reported in children and accounts for <3% of all H1N1 cases [2–5]. The liver involvement in H1N1 is believed to be due to overproduction of pro-inflammatory cytokines leading to changes in hepatic metabolism and enzyme activity, increased hepatic oxidative stress, decreased antioxidant defences, and damage by viral antigens rather than the direct viral invasion [6]. Whitworth et al. reported 4 children (aged 14–39 mo) with influenza A presenting as acute hepatitis and who recovered with supportive therapy [3]. Li et al. reported 2 children with severe influenza A (H3N2) with hepatic dysfunction who were treated with supportive treatment, mechanical ventilation, high dose corticosteroids, and gamma globulin; and one child died [5]. To best of our knowledge, this is the first report of acute liver failure in a child with H1N1 infection from India.

Compliance with Ethical Standards

Conflict of Interest None.

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✉ Suresh Kumar Angurana
sureshangurana@gmail.com

[1] Division of Pediatric Critical Care, Department of Pediatrics, Advanced Pediatrics Centre, Postgraduate Institute of Medical Education and Research (PGIMER), Chandigarh 160012, India

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