Case Reports

Palatal palsy in a girl with hepatitis A virus infection

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Introduction

Isolated bulbar palsy is a rare entity. There are around 30 reports of isolated bulbar palsy described in the literature, particularly in childhood and adolescence[1]. The aetiology is not exactly known. Isolated bulbar palsy along with hepatitis viral infection, suggest some post viral inflammatory reactions. Here we are describing a case of isolated palatal palsy with hepatitis A virus infection.

Case report

A 10 year old girl presented with nasal regurgitation, difficulty in swallowing and nasal intonation of voice for the past 3 days and jaundice for the past 5 days. She had an episode of high grade fever with vomiting 10 days back. The fever slowly subsided with antipyretics.

On examination, she was conscious, afebrile and icteric, with tender hepatomegaly. There was left ninth and tenth cranial nerve palsy showing uvula pointing towards right with absent left gag reflex. Rest of the neurological examination revealed no abnormality. Blood counts were unremarkable. Urine bile pigments were positive but the rest of the urine examination was normal. Cerebrospinal fluid study was normal. Specific investigations showed deranged liver function tests with raised titres of liver enzymes (aspartate aminotransferase 1513 IU/L, alanine aminotransferase1433 IU/L, alkaline phosphatase 900 IU/L) and raised serum bilirubin (total 10mg/dl, conjugated 5mg/dl). Of viral specific markers, hepatitis A virus (HAV) was reactive (positive for both IgG and IgM) whilst hepatis B surface antigen and hepatitis C virus were non-reactive. Ultrasonography of abdomen showed features of hepatitis with mild hepatomegaly and ascites. Serum caeruloplasmin was normal and slit lamp examination of eyes revealed no Kayser Fleischer rings. Bleeding profile showed normal levels with the prothrombin ratio 1.75. Magnetic resonance imaging (MRI) of the brain showed subtle ischaemia with signal alteration at bilateral symmetrical pontine regions suggestive of encephalomyelitis sequelae. Echocardiography was normal. Patient slowly improved spontaneously from the palsy. During follow-up after 14 days there was complete spontaneous recovery with symptomatic improvement.

Discussion

There were a few case reports of acquired bulbar palsy following viral infection which could be due to varicella-zoster virus[2], herpes simplex virus[3], measles[4] and coxsackie A9 virus[5], but still the aetiology of palsy remains controversial. Previous reports were around 80% in boys [6]. In our case it was a girl which was uncommon. In the cases reported earlier, 1 in 3 only had fever and prodromal symptoms and demonstration of infection was positive for only 20% of cases[2]. Our case had a history of prodromal symptoms with hepatitis A serology positive. Aetiology of this palsy was postulated as molecular mimicry, immune response against the myelin leading to demyelination and cranial mono-neuropathy[7].

One case reported by Prasad P.L. et al with HAV infection followed by palatal palsy showed a normal MRI study and the patient was treated with a short course of steroids[7]. Reports have shown recovery with and without steroids[7]. Our case had a complete spontaneous recovery. MRI showed subtle ischaemia with signal alteration as noted to involve bilateral symmetrical pontine region suggestive of encephalomyelitis sequelae. One limitation in this case study was that we were not able to do nerve conduction studies and other hepatotropic viral screening (EBV, CMV, etc).

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