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Case Report

Rare neurological manifestation following hepatitis A infection: a report of 2 cases

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ABSTRACT

Hepatitis A is the leading cause of acute viral hepatitis in developing countries. Extra-hepatic manifestation is very rare occurrence in Hepatitis A. We report 2 cases, one developing Acute Cerebellar Ataxia and another Opsoclonus Myoclonus Ataxia syndrome (OMAS). The former presented with Ataxia of acute onset one week after Hepatitis A infection. The patient did not have any neurological complication and responded to conservative management. The second patient was a 7-year-old girl presented with behavioral abnormality, jerky eye movements and ataxia with myoclonus following a Hepatitis A infection one week ago. Patient was treated with pulse dose methyl prednisolone followed by oral prednisolone and showed a prompt response. Neurological manifestation following Hepatitis A is a rare phenomenon.

Keywords: Opsoclonus myoclonus ataxia syndrome, Hepatitis A, Ataxia, Acute cerebellar ataxia, Postinfectious opsoclonus

INTRODUCTION

Opsoclonus myoclonus ataxia syndrome (OMAS) is an immune mediated inflammatory central nervous system disorder. Opsoclonus is defined by the random jerky chaotic movement of the eyes whereas myoclonus is characterized by jerky shock like contraction of the muscle. OMAS can be paraneoplastic, autoimmune or parainfectious.

A diagnosis of OMAS can be made if three of the following four features are present in a patient, opsoclonus, ataxia or myoclonus, behavior changes or sleep disturbances and neuroblastoma.[1] Acute cerebellar ataxia (ACA) is a self-limiting disease that occurs post viral infection mainly following varicella.

ACA following Hepatitis A infection has been rarely reported. Here we report 2 cases one of ACA and another of OMAS following hepatitis A infection.

CASE REPORTS

Case 1

6-year-old girl presented with jaundice proceeded by fever for two days. Examination showed icterus without signs of encephalopathy. Investigation showed conjugated hyperbilirubinemia (total bilirubin-7 mg/dl, direct fraction- 5.8 mg/dl) with transaminitis (AST-172 Iu/ml. ALT-280 Iu/ml). Hepatitis A IGM was positive. Hbsag, Hepatitis E IGM anti HCV was negative. She had good recovery with conservative treatment.

After1 week from discharge patient developed ataxia. Other neurological examination was normal. cerebellar signs were present. MRI Brain and CSF examination was normal. CSF polymerase chain reaction (PCR) for viruses was done ruling out cytomegalovirus, ebstein barr virus, herpes simplex infection (HSV). Routine blood examination was normal. Hence, patient was diagnosed

as a case of ACA following hepatitis A infection and was treated with multivitamin. Patient was followed up without further neurological complication.

Case 2

7-year-old girl presented with jaundice with soft hepatomegaly, conjugated hyperbillirubinemia (Billirubin 12 mg/dl, direct 8 mg/dl) and transaminitis (AST 500 IU/l, ALT 790 IU/l), was diagnosed as acute Hepatitis A (Hepatitis A IGM >20 IU/l), was managed conservatively. After 1 week patient developed myoclonic jerks (lower limb > upper limb) which progressed and was associated with irregular jerky movements of the eyeball. Ocular movements were fragmented during pursuit intermixed with jerky oscillatory movements in all directions (OPSOCLONUS). She developed ataxia which was more prominent during walking. The patient was diagnosed as a case of OMAS. CBC, liver profile was normal. (Bill-0.5 mg/dl, AST- 51IU/ml, ALT 50 IU/ml). CSF analysis-cell count (5 cells), sugar (90 mg/dl) protein (55 mg/dl). CSF NMDA/ LGI1 LGI2/ CASPR2 antibodies were negative ruling out autoimmune encephalitis. CECT thorax, CECT abdomen and Urinary VMA excluded neuroblastoma. CSF PCR showed no evidence of CMV/ herpes simplex/ EBV. MRI brain, CSF IGG index, Oligoclonal band, MOG antibody and serum Ceruloplasmin (29ug/dl) were not significant.

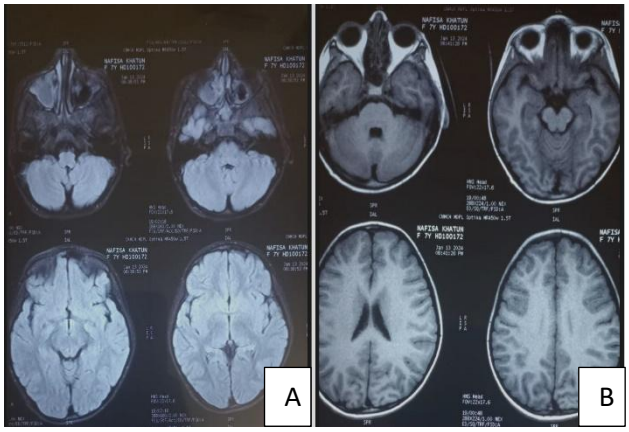

Figure 1 (A and B): MRI brain showing no abnormal changes in the child having opsoclonus; ruling out any possibilities of cerebellitis or demyelination.

Due to worsening ataxia and increasing myoclonus patient was started on I/V methylprednisolone @ 30 mg/kg/day for 5 days followed by oral prednisolone @ 2 mg/kg/day with slow taper over 6 weeks. Myoclonus and ataxia showed resolution at day 3 and day 5 of treatment respectively. Opsoclonus resolved on second week. Ophthalmological examination, VEP, BERA and MRI orbit were normal. Since other causes of OMAS were negative she was diagnosed as para-infectious OMAS post Hepatitis A infection.

Table 1: Cases of OMAS reported in medical literature.

S. no.	Child/adult	Infection	Treatment	Reference
1.	Adult	Streptococcal infection	Oral steroids	Dassan et al[9]
2.	Adult	Lyme disease	Antibiotics	Peter et al[10]
3.	Child	Para-influenza type 3		McCarthy et al[4]
5.	Child	Adenovirus		Syrbe et al[11]
6.	Child	Herpes-virus 6		Naselli et al[7]
7.	Adult	Mumps virus	Intravenous immunoglobulin	Kang and Kim[6]
9.	Adult	Influenza A	Intravenous immunoglobulin and corticosteroids	Morita et al[12]

DISCUSSION

Cerebellar ataxia is divided into acquired, idiopathic and congenital causes. ACA commonly affects previously well children under the age of 6 years but can also impact adolescents and older children.[2] ACA is an inflammatory syndrome which occurs due to direct infection or post infectious autoimmune mechanism.[3] Pathogens causing ACA include viruses like Varicella, Enterovirus, EBV, Herpes simplex, Influenza and a few bacterial infections like Legionella, Mycoplasma, and Salmonella.[4] Neuroimaging and CSF is critical.[5,6] All the structural causes were ruled out by MRI brain, infective etiologies were excluded by CSF PCR. No infection was documented after Hepatitis A infection, making it most likely cause of the ACA in our patient which was treated conservatively. Infection associated opsoclonus (IAO), a rare entity, occurs either due to invasion of the pathogen or infection triggered autoimmunity or molecular mimicry affecting tegmentum of pons or the cerebellum or both.[7] OMAS associated with neuroblastoma, autoimmune encephalitis, other infectious etiologies EBV/ CMV/ Coxsackie virus/HSV/Hepatitis B were also negative in our case. Nothing compatible for Wilson disease, Tb meningitis, SSPE were found. There was microbiological evidence of Hepatitis A 1 week before without evidence of any other infection between the jaundice and OMAS. CNS lesion or demyelination was not documented. OMAS had a strong temporal relationship with Hepatitis A. Therefore, by exclusion this was diagnosed as a case of OMAS following Hepatitis A infection. By far only 2 case reports showing neurological manifestation of Hepatitis A has been reported. Tuhill et al, reported Acute cerebellar ataxia

post hepatitis A infection.[8] Kaninde et al, reported a similar case of ACA in a 3-year-old child affected by hepatitis A.[2] Sharma et al, reported hepatitis A associated with acute cerebellitis and ataxia that was managed with iv methylprednisolone and short course of oral steroids.[3]

The patient developed OMAS after 1 week of after Hepatitis A. The patient was treated with immunotherapy and showed a very good response. Para-infectious OMAS has been reported in multiple studies (Table 1). Most cases of OMAS after 6 days of acute infection requiring low degree of immunosuppression and healing without disability which matches our case. Myoclonus and ataxia responded early and opsoclonus took 2 weeks to resolve.

CONCLUSION

Acute cerebellar ataxia and OMAS form a spectrum one being a benign disease while other having a progressive and debilitating course. Identifying OMAS and managing is mandatory to avoid life threatening complication. Neurological involvement following hepatitis A infection must be managed carefully

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