Guillain-Barré syndrome following acute viral hepatitis A

Sir,

A 28-year-old male patient presented with progressive symmetric ascending distal followed by proximal weakness over two days, preceded by low grade fever, malaise, nausea and vomiting along with anorexia of two week duration. His initial evaluation at local hospital revealed features of acute hepatitis with rising levels of serum bilirubin (5.6 mg/dl), transaminitis (SGOT/SGPT - 1877/3058) without any features of hepatic decompensation. He was conscious, alert with jaundice and hepatomegaly without features of hepatic failure. His neurological examination revealed bifacial lower motor neuron weakness, generalized hypotonia with areflexia, neck muscle weakness, and upper and lower limb power of medical research council grade (MRC) 2 with distal more than proximal weakness. In view of rapidly progressive ascending flaccid areflexic quadriparesis along with bifacial weakness, the diagnosis of Guillain-Barré syndrome (GBS) was considered. His motor nerve conduction study showed significantly reduced or inelicitable CMAP from bilateral tibial and peroneal nerves without any improvement on proximal stimulation with mild prolongation of distal latencies and conduction velocities but not amounting to a coexisting demyelinating process. The F waves were either inelicitable or prolonged from all four limbs. Sensory nerve conduction study showed inelicitable SNAP amplitudes from all tested nerves except from bilateral median nerves, consistent with acute motor and sensory axonal neuropathy (AMSAN) [Table 1]. He was treated with IV IgG of 400 mg/kg/day. His biochemical evaluation showed high IgM hepatitis A antibody titer by ELISA in serum and negative serological investigations for hepatitis B, hepatitis C, hepatitis E and negative anti-ganglioside antibody. His CSF showed albuminocytological dissociation with protein of 90 mg%. After admission, he developed respiratory weakness requiring prolonged mechanical

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Table 1: Motor and sensory nerve conduction study

Nerve and site	Latency (ms)	Duration (ms)	Amplitude (mv)	Segment	Latency difference (ms)	Distance (mm)	Conduction velocity (m/s)
Peroneal nerve (right)							
Ankle	8.5	24.2	2.1	EDB-Ankle	8.5		
Fibula (head)	19.5	35.3	1.5	Ankle-Fibula	11	360	33
Popliteal fossa	22.4	32.5	0.9	Fibula-popliteal fossa	2.9	100	34
Tibial nerve (right and left)							
Ankle			Absent				
Popliteal fossa			Absent				
Peroneal (TA) right							
Fibula (head)	3.6	34.9	2.8				
Popliteal fossa	5.8	35.8	2.1	Fibula-popliteal fossa	2.2	100	35
Tibial (GA) right							
Popliteal fossa	4.8	25.9	2.0				
Peroneal Left							
Ankle	8.4	14.8	4.1	EDB-Ankle	8.4		
Fibula	17.8	16	3.0	Ankle-fibula	9.4	360	38
Popliteal fossa	20.4	17.1	2.6	Fibula-popliteal fossa	2.6	100	38
Peroneal (TA) left							
Fibula	3.9	30.0	2.1				
Popliteal fossa	6.2	30.7	1.8	Fibula-popliteal fossa	2.3		
Tibial (GA) left							
Popliteal fossa	12.0	25.9	1.5				

TA: Tibialis anterior, GA: Gastrocnemius

ventilation. In view of strong serological positivity for hepatitis A, the diagnosis of GBS with acute viral hepatitis A (HA) was made for which he was managed supportively. He improved slowly and is asymptomatic at 12 months follow-up.

GBS has been reported with acute viral hepatitis B, acute hepatitis C, hepatitis D, hepatitis E and rarely with HA.[1] The proposed pathology, as in C.jejuni, is that HA infection evokes an immune response, which in turn cross-reacts with peripheral nerve components because of the sharing of cross-reactive epitopes (molecular mimicry).[2] Xie *et al*., had considered a possible association between the GBS and HAV as out of 292,301 cases of HA in Shanghai between January and March 1988 during an epidemic, 8 had GBS.[3] A review of literature revealed only 15 reports of GBS following hepatitis A infection in adults, majority of the cases were AIDP variant, only two cases consistent with AMSAN.[4,5] GBS with HAV is associated with male preponderance, younger age of onset and has an overall better prognosis.[4] Although few patients have fulminant hepatitis, it did not seem to correlate with the severity of neurological symptoms. The good outcome in cases of GBS with preceding infection with HA could be a marker of a favorable prognosis unlike C. Jejuni.

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