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Case Report

Guillain Barre Syndrome in Hepatitis A; A Case Report with Review of Literature

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Abstract

There are a very few case reports of Guillain Barre syndrome (GBS) associated with hepatitis A. Here were report such a case of 20 years old male who presented with fever, anorexia and icterus of 10 days duration who subsequently developed acute onset areflexic ascending quadriparesis with facial, bulbar and respiratory muscle weakness. Electrophysiological study revealed reduced nerve conduction velocity, prolonged distal latencies and non-recordable F-wave response. These features were suggestive of Guillain-Barre syndrome, acute inflammatory (AIDP) variant. Cerebrospinal fluid (CSF) study showed albumino-cytological dissociation. Patient also had deranged liver function and definite evidence of acute hepatitis A. The patient was treated with intravenous immune-globulins (IVIG) and other supportive treatment. He improved with treatment and liver function test normalized after 3 weeks.

Keywords: *Guillain-Barre syndrome; Hepatitis A; Poly-neuropathy*

Introduction

Guillain- Barré syndrome is an acute inflammatory poly-radiculo-neuropathy. It is characterized by acute onset rapidly progressive symmetrical ascending paralysis. Viral infections with human immunodeficiency virus (HIV), *Epstein Barr* virus (EBV) and *Cytomegalovirus* (CMV) have been linked with the etio-pathogenesis, but it is very rare with Hepatitis A. Symptoms usually occur 1 - 3 weeks after the viral infection. There are only a handful of cases being reported till now. Here we report a case of GBS with Hepatitis A.

Case Report

A 20 years old male presented with acute onset rapidly progressive symmetrical ascending proximal as well as distal weakness and inability to close eyes over two days which was preceded by history of high grade fever, malaise, loss of appetite, nausea, vomiting and yellowish discoloration of urine and eyes of 10 days duration. There was no history of recent vaccination, drug intake, blood transfusion and needle injury. On examination, he had a pulse rate of 84/min, temperature-36.8 Celsius, blood pressure-120/80mmHg, respiratory rate-28/min and single breath count of 12. He had no pallor, but icterus was present without any features of hepatic de-compensation. On nervous system examination higher mental function were normal, muscle power by Medical Research Council (MRC) grading was II/V in

lower limbs, III/V in upper limbs with absent deep tendon reflexes in all limbs and bilateral planters were mute. On second day his muscle power grade had reduced to O/V in lower limb and I/V in upper limb with single breath count of 4. Rest of the systemic examination was within normal limits.

Laboratory evaluation revealed normal complete blood count, renal function tests, serum electrolytes and arterial blood gas analysis. Liver function test showed raised trans-aminase (SGOT- 680 U/L, SGPT- 310 U/L and ALP- 380 U/L) and bilirubin levels (total- 13.47 mg/dL, direct- 7.10 mg/dl and indirect- 6.37 mg/dl). Markers for HIV, hepatitis-B, C, E and *Dengue* virus were negative and IgM antibody for hepatitis-A was positive. Electrophysiological study was suggestive of pure motor demyelinating with secondary axonal polyneuropathy in bilateral median ulnar, common peroneal, posterior tibial nerves with sural sparing. CSF study showed albumin-cytological dissociation (protein- 64.8 mg/dL, glucose- 64 mg/dL and total count- 5/cmm). Clinical, laboratory and electrophysiological test suggested GBS- acute demyelinating variant with simultaneous acute infection of hepatitis A virus. Patient was further managed in intensive care unit with ventilatory support. Patient was given intravenous immunoglobulin (IVIG) in the dose of 2 gm/kg in divided doses over 5 days. He started recovering gradually and was extubated after 10 days. After three weeks, liver profile returned to normal and muscle power grade was II/V in lower limb and II/V in upper limb with single breath count of 24. At 2 months follow up patient was ambulatory without support.

Discussion

Guillain-Barré syndrome (GBS) is an acute post-infectious poly-radiculoneuropathy with an incidence of 0.6 - 4.0/100,000 person/year worldwide [1]. Commonly it presents as acute onset rapidly progressive areflexic demyelinating polyradiculoneuropathy with or without sensory involvement, which is autoimmune in nature. The pathogenesis of GBS has been postulated to be due to molecular mimicry - infection evokes an immune response, which in turn cross-reacts with peripheral nerve components because of the sharing of cross-reactive epitopes [2]. GBS has been seen in association with viral infections such hepatitis B, C, D but rarely with hepatitis A and E. A review of literature revealed only 20 reports of GBS following hepatitis A infection in adults, the characteristics of which are being summarized in tabulated form with their separate references, majority of the cases were AIDP variant, only three cases consistent with acute motor sensory axonal neuropathy (AMSAN) [3-5]. Clinical features of GBS following hepatitis A can be summarized as follows: 1) Most of the patients are men. 2) The interval between the onset of hepatitis and the development of neuropathic symptoms is less than 14 days. 3) There is a frequent association with facial nerve palsy. 4) Joint position and vibration sense are frequently impaired, in addition to superficial sensation. 5) A uniformly good outcome of the neuropathic symptoms is independent of the level of ALT, which corresponds to the severity of liver dysfunction [4].

Author	Patient's age and sex	Onset of GBS (days after onset of HA)	Maximum level of ALT (U/l)	Neurological evaluation				Maximum level of CSF protein (mg/dL)	Treatment	Outcome
				Cranial nerves	Muscle weakness	Superficial sensation	Proprioceptive sense			
Johnston 1981 [6]	37 M	14	617	-	-	+	+++	29	NA	Ambulatory 2 weeks later
Dunk 1982 [7]	48 M	3	273 (AST)	VII Dysarthria	++	+	+	30	NA	Ambulatory 12 days later
Bosch 1983 [8]	25 M 28 M	? ?	2700 129	? ?	? ?	? ?	? ?	? ?	NA	? ?
Igarashi 1983 [9]	49 M	7	412	VII Dysarthria	++	++	++	165	NA	Ambulatory 28 days later
Grover 1986 [10]	31 M	7	8760	VII	-	+++	+++	?	NA	Recovery 30 days later
Mares-Segura 1986 [11]	34 F	7	314	VII	+	+	+	156	NA	Recovery 3 months later
Endoh 1991 [12]	39 M	14	503	Bulbar palsy	++	+	-	108	Prednisolone	Recovery 50 days later
Ono 1994 [4]	62 M	11	5062	VII	++	++	+++	181	Prednisolone	Ambulatory 70 days later
Lee 1996 [13]	43 F	5	1748	-	Paraparesis	-	-	normal	Plasmapheresis	Recovery 19 days later
Mihori 1998 [14]	46 M	2	NA	-	+	+	+++	760	Plasmapheresis	Recovery 176 days later
Azuri 1999 [15]	3.5 M	None	Normal	?	?	?	?	?	IVIG	Recovery 30 days later
Breuer 2001 [16]	28 F	4	868	VII Bulbar palsy	+	?	?	300	IVIG	Recovery 21 days later

Chitambar 2006 [17]	17 M	20	95	-	+++	-	-	100	Conservative treatment	Recovery 90 days later
Bae 2007 [3]	32 M	9	981	?	++	-	-	115	IVIG	Ambulatory 90 days later
Kocabas 2004 [18]	06 M	14	900	-	++	?	?	82	IVIG	Recovery 10 days later
Ratnasari 2002 [19]	19 M	21	197	-	+	+	-	53	?	?
	43 M	16	342	-	+++	+	-	19	?	Recovery 12 days later
Khan 2012 [20]	17 M	7	111	-	++	-	-	86	Plasmapheresis	?
Menon 2014 [5]	28 M	14	3058	VII	++	-	-	90	IVIG	Ambulatory 12 months later
Patel 2015 [21]	25 F	7	1206	VII Bulbar palsy	+++	-	-	?	IVIG	Recovery 21 days later
Comoglu 2006 [22]	22 M	14	181	-	++	+	-	66	Prednisolone	Recovery 30 days later
Present case 2017	20 M	10	310	VII Bulbar palsy	+++	-	-	65	IVIG	Recovery 21 days later

Table 1: *The comparative summary of GBS patient's case reports with Acute Hepatitis A.*

Conclusion

Although GBS in Hepatitis A is very rare, its differential should always be kept in patients of hepatitis who develop weakness out of proportion to the systemic features.

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Conflict of Interest

None.

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