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A Case of Acute Motor-Sensory Axonal Neuropathy after Enteric Fever

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Abstract
We report here a case of enteric fever, which was complicated by Acute Motor Sensory Axonal Neuropathy. A 27-year old man admitted to our hospital with high grade fever and progressive muscle weakness and sensory loss. On evaluation, the patient was diagnosed to have enteric fever. The blood culture was positive for S.typhi. Cerebrospinal fluid analysis showed albumino-cytologic dissociation and nerve conduction velocity test suggested a sensory-motor demyelinating polyneuropathy. Patient was diagnosed with enteric fever complicated with AMSAN and was treated with intravenous immunoglobulin and antibiotics. After 15 days of hospital stay, patient recovered significantly with minimal residual deficit. The clinical course, prognosis and the outcome of neuropathic symptoms of AMSAN following enteric fever were relatively good in our case.
This case report is to highlight a rare neurological complication of enteric fever that is of particular importance to clinicians.

Introduction

Guillain-Barré syndrome (GBS) is an autoimmune disorder of the peripheral nervous system characterized by autoimmune demyelination of peripheral nerves. It is classified into four subtypes: the acute inflammatory demyelinating polyradiculoneuropathy (AIDP), the acute motor axonal neuropathy (AMAN), the acute motor and sensory axonal neuropathy (AMSAN) and Miller Fisher's syndrome[(1)(2)]. GBS results from an aberrant immune response against the nervous tissue. Triggering factors involved in its pathogenesis include surgery, immunizations, or infections[(3)].

The most severe variant, AMSAN is a recently described subtype of Guillain-Barre syndrome. Acute onset of distal weakness, loss of deep tendon reflexes, and other sensory symptoms are

the chief characteristic features of AMSAN[1]. It is distinguished from its other subtypes by fulminant and rapid onset of quadriparesis within 7 days of onset of symptoms, often requiring prolonged respiratory support.

Based on clinical features, laboratory findings, and electro physiologic investigation, here we report a case of a patient diagnosed with AMSAN following enteric fever infection.

Case report

A 27-year-old man with no previous history of any neurological disease was referred to our hospital with 10-day history of high grade fever and progressive muscle weakness in all 4 limbs along with tingling, numbness and loss of sensations for past 3 days. Patient did not provide any history of respiratory or gastrointestinal infection within 1 month before onset of progressive weakness.

On physical examination patient was febrile to touch, vitals were stable, both lung fields were clear on auscultation, single breath count (SBC) was 32 and cardiovascular examination was unremarkable. Mild splenomegaly was found on abdominal palpation. Neurological assessment revealed normal mental status of the patient, cranial nerve examination was normal. By the Medical Research Council (MRC) grading, the patient had weakness with are flexia in both proximal upper limbs (MRC grading 4), both distal upper limbs (MRC grading 2), both proximal lower limbs (MRC grading 4), and both distal lower limbs (MRC grading 2). Pin-prick, vibratory, temperature and joint position senses were reduced in glove and stocking distribution. Weakness was associated with significant wasting of muscles of hands and feet. There was no bladder bowel dysfunction.

Lab investigations revealed a normal hemogram. Liver function test (LFT) and kidney function test (KFT) were within normal limits. Chest X-ray was normal and ultrasound abdomen revealed a splenomegaly of 14 cm. Nerve conduction studies (Table 1) were performed; Motor nerve conduction studies revealed reduced amplitude with prolonged distal latencies(DL) and decreased conduction velocities (CV) in bilateral median, ulnar, peroneal and tibial nerves. The results for Sensory nerve conduction studies and F-wave parameter were not recordable in the nerves tested. Electrophysiological studies suggested sensory-motor demyelinating polyneuropathy.

CSF examination was carried out that revealed 10 leucocytes (85% lymphocytes), raised protein levels of 145mg/dL, normal glucose levels of 65mg/dL, staining with Indian ink and TB PCR were negative. MRI of the brain (figure 1) and the spinal cord (figure 2) did not show any significant pathology. Serologies for human immunodef-iciency virus (HIV), Epstein - Barr virus (EBV), Cytomegalovirus (CMV), Hepatitis-B (HBV) and Hepatitis-C virus (HCV) were also negative. Serum CPK levels were normal.

A diagnosis of AMSAN was retained and the patient was given intravenous immunoglobulin (0.4g/kg/day) for 5 days. Blood culture report received on 3rdday of admission, showed ciprofloxacin resistant *Salmonella typhi*, sensitive to ceftriaxone. Serum was sent for Widal test which was found to be positive ('O' and 'H' titers> 1/160). IgM typhi-dot ELISA was positive in high titres. The patient was given injectable ceftriaxone and became afebrile by 4th day of treatment. The patient showed progressive improvement in weakness and was discharged on 18th day with final diagnosis conclusive of enteric fever with AMSAN and was advised to follow up in OPD for further treatment.

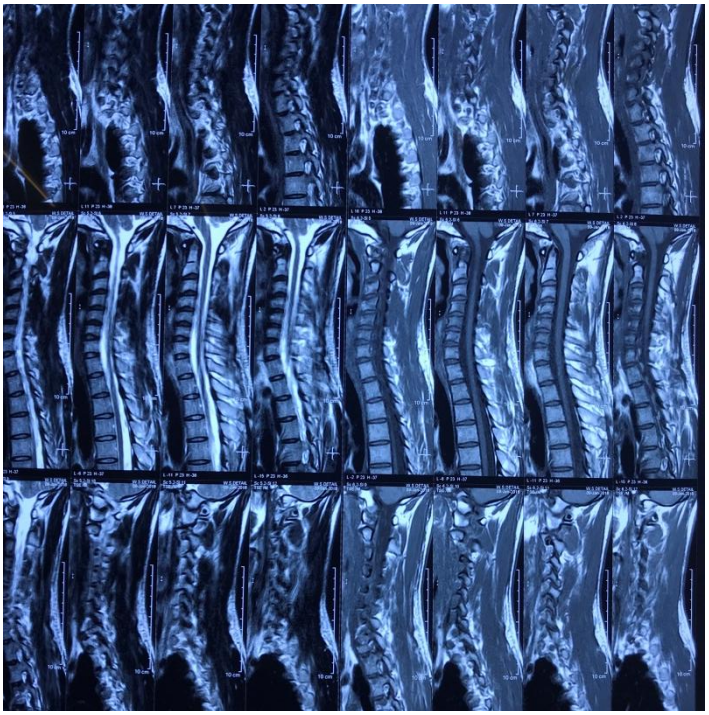

Figure 1: MRI Dorsal Spine: MR imaging does not reveal any significant disc bulge/protrusion or a neural compression.

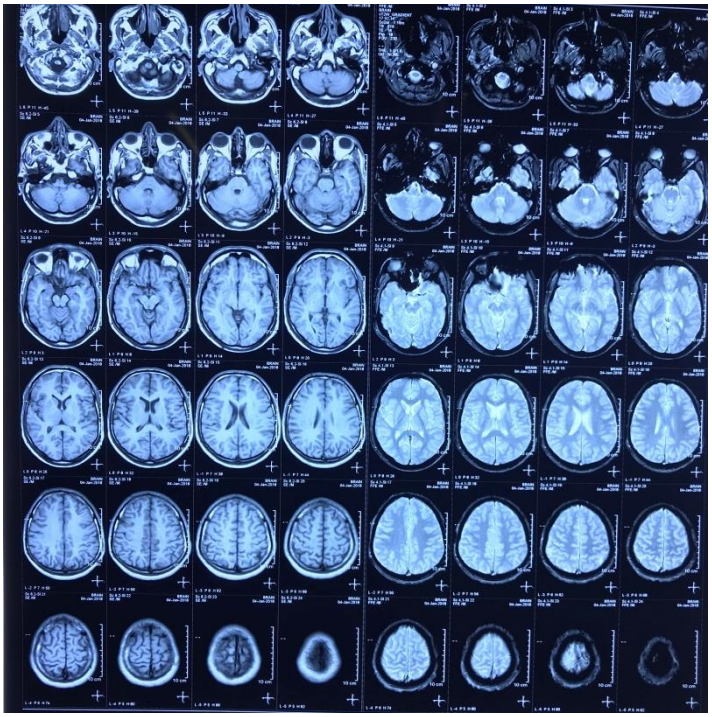

Figure 2: MRI Scan of Brain Plane: MR imaging does not reveal any obvious intracranial abnormality.

Table 1: Electrophysiological studies of nerves; Motor and sensory nerve conduction studies and F-wave parameters.

Nerve Conduction Studies							
Test	**Stimulation site**	**Lat, ms**	**Ampl, mV**	**Dur, ms**	**Dist, mm**	**Time, ms**	**Vel, ms**
MOTOR CV							
right, Median							
	wrist	20.9	1	15.6	80		
	elbow	49.3	1	20.5	220	28.4	7.8
left, Median							
	wrist	19.2	1.5	15.7	80		
	elbow	49	1.5	21.4	220	29.9	7.4
right, Ulnar							
	wrist	15.4	0.9	16.8	80		
	elbow	47.6	1	22.7	220	32.2	6.8
left, Ulnar							
	wrist	13	2.3	15.1	80		
	elbow	43.5	1.6	23.5	220	30.5	7.2
right, Peroneal							
	head of fibula	0					
left, Peroneal							
	sole of foot	19.2	0.7	18.1	70		
	head of fibula	67.7	0.8	23	330	48.5	6.8
right, Tibial							
	medial malleolus	26.7	1.3	28.1	70		
	popliteal fossa	66.5	0.4	33.1	350	39.8	8.8
left, Tibial							
	medial malleolus	25.5	0.5	14.8	70		
		77.8	0.5	21.8	350	52.3	6.7
SENSORY CV							
Test	**Site**	**Lat, ms**	**Ampl., µV**	**Dur., ms**	**Dist.,mm**	**Time, ms**	**Vel. m/s**
right, Median							
	wrist	**0**					
left, Median							
	wrist	0					
right, Ulnar							
	wrist	**0**					
left, Ulnar							
	wrist	**0**					
right,Sural							
	NR	**0**					
left, Sural							
	NR	**0**					
F-Wave parameters							
Test	**Fmin lat, ms**	**M lat, ms**	**Fmin-M lat, ms**				
right, Median							
		21.9					
left, Median							
		21.3					
right, Ulnar							
		15.3					
left, Ulnar							
		15.7					
left, Peroneal							
		22.3					
right, Tibial							
		22.8					
left, Tibial							
		21.6					

Discussion

GBS is a rare neurological complication associated with enteric fever, and only a few cases have been reported[4].

The mechanism of pathogenesis of these neurological conditions is not clear and is under investigation. For GBS to develop in enteric fever, a mechanism attributing to this condition could be the generation of IgM antibodies against the bacterial capsule processed by a non-T cell-mediated mechanism. These IgM might then participate in a cross-reactivity induced inflammatory pathway with myelin gangliosides[6]. Non-specific cerebral changes like edema and hemorrhage along with toxemia and other metabolic dysregulations might be another cause for these neurological manifestations[5][6].

Our patient recovered progressively on treatment with ceftriaxone and IVIG and could walk with mild residual weakness of the left foot within 17 days of treatment. The MRC grading at discharge improved to grade 5 for both proximal and distal upper limbs and for the right proximal and distal lower limb. The grading for the left proximal lower limb improved to grade 5 from 4 and for the left distal lower limb; the grading was MRC grade 4. The sensation (pain, temperature, touch, vibration sense & perception of joint position) was also found to be improved.

The importance of this case report is to highlight upon the fact that a diagnosis of GBS should always be kept in mind whenever a patient of enteric fever develops weakness as a consequence of associated neurological manifestations as seen in our case.

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