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EC NEUROLOGY
Case Report

Acute Inflammatory Demyelinating Polyradiculoneuropathy Associated with *Mycobacterium tuberculosis*

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Abstract

We report the case of a 65-year-old gentleman with 1 month history of low grade fever, weight loss and anorexia along with productive cough, admitted to our hospital with the acute development of rapidly progressing, symmetrical ascending weakness of all 4 limbs. The weakness had started in the lower limbs and spread to the upper limbs in 5 days, along with urinary symptoms. He had marked flaccid weakness of all 4 limbs; the weakness being more marked in the distal muscle groups. Within 2 days the weakness ascended to involve the proximal and bulbar muscles as well. He had three sputum samples positive for acid-fast bacilli. Electrodiagnostic studies revealed markedly prolonged distal latencies and absent H-reflexes and F-latencies. The patient received I/V IG along with ATT, to which he responded well and his weakness started improving in 1 week with complete ambulation in 2 months.

Keywords: *Gullain-Barre Syndrome (GBS); Polyradiculoneuropathy; Mycobacterium tuberculosis*

Background

Gullain-Barre Syndrome (GBS) is an acute post-infectious immune-mediated polyradiculoneuropathy. Different predisposing factors are known, of which respiratory and gastrointestinal infections i.e. with *Campylobacter jejuni* are the most prevalent. The underlying pathological mechanisms are not yet fully elucidated, however, an infection-induced aberrant immune response and following T-cell activation seems to be crucial.

The reason for reporting this case is that there have been only 10 cases so far reported, featuring association of GBS with pulmonary Tuberculosis. Secondly, the age old belief that GBS is the result of a T-cell mediated response does not seem to apply in this case as Tuberculosis is a B-cell mediated response. Thirdly, as there is a very high incidence and prevalence of Tuberculosis in Pakistan, one should also look for TB in patient with GBS.

Case Presentation

65-year-old gentleman with no known comorbids, non-smoker, was admitted to the hospital with 1 month history of low grade fever, weight loss and anorexia along with productive cough. He came to our hospital with 20 days history of weakness of all 4 limbs. The weakness had started in the lower limbs and spread to the upper limbs in 5 days, along with urinary symptoms. He was experiencing severe pain and numbness in his bilateral lower limbs. Due to his pain and weakness he had become bed bound. On further inquiring he reported

that he had been having low grade fever and cough since 3 months along with scanty expectoration. His past medical and surgical history were unremarkable. He had no family history of tuberculosis, polio or other familial neurologic disorders.

On examination, he was grossly emaciated. He had marked flaccid weakness of all 4 limb; the weakness being more marked in the distal muscle groups. Power was grade II/V in the proximal muscle groups of both upper and lower limbs and grade 1/V in distal muscles of both lower limbs. The tendon reflexes were diminished in the upper limbs and absent in the lower limbs. The planter response was flexor. In the chest there were crepatations in the upper and mid-zones bilaterally.

Investigations

Laboratory investigation revealed no grossly abnormal findings. Chest X-Ray showed multiple confluent, ill-defined and ring shadows in both upper and mid-zones suggestive of pulmonary tuberculosis. MRI-D Spine was done which was normal. Three sputum samples were positive for acid-fast-bacilli.

His FVC (forced vital capacity) was 1.2 L/min.

Electrodiagnostic studies were indicative of sensory-motor demyelinating polyneuropathy with markedly prolonged latencies and absent H-Reflex and F-Latencies.

Motor Nerve Conduction Studies									
S.No.	Nerve	Sum-Site	Latency (ros)	Dist. (cm)	Amplitude (my)	ND/ (rols)	Fun (ns)	Duration	Area (mVms)
1	RMAPB	Wrist	8.6	70	400	-	NR	16	
2	RMAPB	Elbow	12.5	23	500	59	-	17	
3	.								
4	RUADQ	Wrist	5.9	7	1.4	-	NR	13.5	
5	RUADQ	D.ELBOW	10	22	1.2	54	-	15.8	
6	RUADQ	P.ELBOW	11.8	10	1.1	56	-	14	
7	.								
8	R.POST'T	Ankle	18.3	8	200	-	NR		
9	R.POST'T	Knee	36.4	45	200	25	-	12	
10	.								
11	R.PERONEAL			NR					
12	R.PER'T.A	DFH	4.1	12	1.9	-	26.4		
13	R.PER'T.A	PFH	6.9*	10	1.7	36		25.9	
14	.								
15	R.H.REFLEX	POP.FOSSA		NR					
16	L.H.REFLEX	POP.FOSSA		NR					

Sensory Nerve Conduction Studies							
S. No.	**Nerve**	**Record. Site**	**STIH.SITE**	**Latency (m/s)**	**DIST. (cm)**	**AMP (UV)**	**NCV(m/s)**
1	RM	F2	Wrist	NR			
2	RU	F5	Wrist	NR			
3	RR	Snuffbox	Forearm	2.8	10	17	35
4	R. SURAL	Ankle	Calf	NR			
5	L. SURAL	Ankle	Calf	If			
6	L. RADIAL	Snuffbox	Forearm	2A	10	27	

Treatment

The patient was started on standard dose of human Immunoglobulins (IV/IG) in a dose of 0.4 g/kg/d for 5 days. He was also given chemotherapy with isoniazid 300 mg, rifampicin 600 mg, ethambutol 1200 mg, pyrazinamide 1500 mg, pyridoxine 50 mg and levofloxacin 750 mg. Multidisciplinary teams were involved in his management including Neurology Critical Care, Pulmonary, Infectious Disease and Rehabilitation team. Physiotherapy had been started at the same time along with care of calories, diet, bowel and bladder care.

Outcome and follow-up

He responded well to I/V IG, with improvement in his forced vital capacity along with no further progression in weakness. His weakness started improving in 1 week and was able to ambulate in 2 months, with support.

His cough improved and his sputum became negative for AFB in 2 months. However, he was continued on ATT for a total of 8 months.

Discussion and Conclusion

GBS is characterized by heterogenous clinical spectrum. It is a diffuse predominantly Motor Polyneuropathy and is known to follow a variety of viral, mycoplasma and chlamydial infections besides there being reports of post-vaccine GBS. Worldwide, it is the most frequent cause of acute flaccid paralysis (AFP), with an incidence of 1.2 to 2.3 per 100,000 persons per year [1]. Despite medical treatment, GBS often remains a severe disease; 3 - 10% of patients die, while 85% patients recover spontaneously but 10% patients need hospitalization and 20% are still unable to walk after 6 months [1]. Its prevalence has been reported to vary from region to region. A study conducted in Hong Kong, during 1997 to 2002, reported a prevalence rate of 42% [2]. 45% prevalence rate from Oman has been reported [2]. Similarly in a study conducted in Hazara division, Pakistan GBS was found in 47% of patients [3]. The prevalence rate for GBS in Pakistan by WHO is 1,591/159,196,336 estimated population [4].

Electrophysiological studies are key for diagnosis and IVIG is the standard treatment of choice.

Pulmonary tuberculosis is a worldwide problem and WHO declared Tuberculosis Emergency. According to the WHO 2005 report, the incidence of pulmonary tuberculosis in Pakistan is 181cases/100,000/yr whereas prevalence is 297/100,000, with a high incidence of mortality; 37/100,000, with a dramatic increase in multidrug resistant Tuberculosis [5,6].

With this high prevalence rate of pulmonary tuberculosis it is likely that GBS may also be prevalent but is unrecognized or treated as possible tuberculous radiculitis and therefore it is imperative to know the likely association of this communicable disease with this highly treatable immune condition.

The clinical and radiological features in this case were sufficiently characteristic to enable a diagnosis of pulmonary tuberculosis to be made and this was confirmed by sputum positive AFB smears on three occasions.

This case is interesting in the sense that apart from pulmonary tuberculosis which was evident, the patient presented with a neurological picture of peripheral neuropathy with nerve conduction studies showing a demyelinating picture, supporting the diagnosis of AIDP rather than tuberculous polyradiculitis.

Felix Leneman in 1966 reviewed 1100 cases of Guillain Barre' Syndrome [7] of which 365 seemed to have occurred *de novo* without any mention of antecedent or under-lying illnesses. The other 735 cases were associated with some condition or other of which tuberculosis (of the lung and brain) was observed in 8 cases (approximately 1%) as associated illness. Since this association may have been fortuitous these conditions are offered only as probable or possible etiological factors [8].

Similarly, 2 cases occurring in association with chronic pulmonary tuberculosis were reported from Sri Lanka in 1983 [9]. Both had a history of weakness of all 4 limbs of acute onset over a background of chronic cough, low grade fever and weight loss. Chest X-Ray showed multiple ill-defined confluent shadows in the upper zones strongly suggestive of pulmonary tuberculosis. They had marked flaccid paralysis of all 4 limbs with diminished tendon reflexes and loss of all sensory modalities in a glove and stocking distribution. Both patients were given anti-tuberculous chemotherapy along with steroids with improvement of symptoms in one.

A cell-mediated delayed hypersensitivity reaction to, or invasion of the nerve roots by tubercle bacilli would seem to be the likely explanation of the neuropathy.

Learning Points/Take Home Messages

1. GBS is not just associated with respiratory or gastrointestinal infections. It can also occur in association with other B-cell mediated disorders.
2. Not all cases associated with complicated tuberculosis presenting with limb weakness and bladder and bowel dysfunction are due to Pott's disease, because these conditions if not recognized early can result in significant morbidity.
3. With the high prevalence of Tuberculosis in South Asia, it is necessary to recognize the possible treatable but rare complic ations of this communicable infection.

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