

Acute Onset Chronic Inflammatory Demyelinating Polyneuropathy with Prostatic Malignancy

Sir,

Prostate cancer is the second most common cancer diagnosis made in men.^[1] Neurological manifestations described in prostate cancer are usually related to metastasis, local pelvic growth, or treatment-related. However, Chronic inflammatory demyelinating polyneuropathy (CIDP) has rarely been reported with prostatic adenocarcinoma. In this report, we describe a case of acute onset CIDP in a patient with prostatic adenocarcinoma.

A 70-year-old gentleman with obstructive urinary symptoms was diagnosed with prostate adenocarcinoma after prostatic biopsy in our hospital. After one month, he experienced progressive weakness of all four limbs over 2 weeks without any perceived sensory loss or bowel bladder symptoms. Neurological examination revealed bilateral mild lower motor neuron (LMN) facial palsy. Power testing (MRC grading) revealed a grade of 3 in all limbs with absent deep tendon

reflexes. The vibration test showed early decay in both lower limbs till the knee. However, proprioception, pain, and touch sensations were intact. The rest of the neurological and systemic examination was normal.

On the basis of clinical finding of flaccid quadriparesis with areflexia alongwith LMN facial palsy progressing over 2 weeks, the diagnosis of acute onset neuropathy was kept. Myopathy and neuromuscular disorders were ruled out initially due to sensory involvement, areflexia and muscle weakness without diurnal variation.

Her initial laboratory workup showed pancytopenia with haemoglobin 12.0 g/dL, white cell count $6 \times 10^3/\mu\text{L}$, and platelets $220 \times 10^3/\mu\text{L}$. The patient's aspartate aminotransferase was 27, alanine aminotransferase was 25 and alkaline phosphatase was 30. The coagulation profile and kidney function tests (urea of 18 mg/dl and creatinine of 0.9) were

normal. HbA1c was 6% and erythrocyte sedimentation rate and thyroid function tests were normal. Serology for viral hepatitis, Human immunodeficiency virus, antinuclear antibody, and rheumatoid factor were negative. The serum and urine immunoelectrophoresis and immunofixation were normal. Prostatic specific antigen was 28.2 ng/ml. CSF examination showed albuminocytological dissociation with no malignant cells. The MRI brain was normal. However, the MRI spine showed post-contrast enhancement of lumbosacral roots. MRI pelvis revealed an enlarged prostate with 1.9*1.0 cm, T2 hypointense area in the peripheral zone with loss of fat planes with bilateral seminal vesicles. [Figure 1] Prostatic biopsy revealed adenocarcinoma with Gleason score of 9 (4 + 5). Nerve conduction study (NCS) showed prolonged F wave

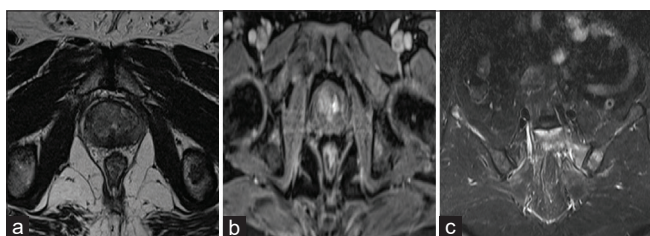


Figure 1: (a) MRI Pelvis axial section T2W images showing enlarged prostate gland. (b) MRI pelvis axial section post-contrast T1 images showing heterogeneous enhancement in the peripheral zone of the prostate. (c) MRI spine coronal section STIR (short TI inversion-recovery) images showing root enhancement

latency and prolonged motor distal latency of both common peroneal nerves. The left posterior tibial and bilateral common peroneal nerve showed prolonged motor distal latency with reduced conduction velocity while the right posterior tibial nerve was non-recordable. Both the sural nerves were spared. Upper limbs NCS showed prolonged F wave and distal latency of bilateral ulnar, median and radial nerves. Conduction velocity of all recorded nerves of upper limbs were reduced. Sensory studies showed reduced conduction velocity of bilateral median nerves. [Tables 1 and 2] The paraneoplastic panel for onconeural antibodies (anti-Hu, anti- Yo, anti-Ma, anti-CV2/CRMP5, anti-Ri, anti-amphiphysin, anti-GAD, anti-Ta) were negative.

A diagnosis of Guillain barre syndrome (GBS) was made on basis of CSF and NCS findings (as per Brighton Criteria) and intravenous immunoglobulin (4 mg/kg/day) was administered for 5 days. During the next week, the limb weakness remained static. Subsequently, he went home because of the sudden demise of his wife and henceforth he became lost to follow up for the next one month. Thereafter he was admitted for the second time with respiratory distress (respiratory rate of 30 and 88% oxygen saturation). Meanwhile in the lost to follow up period his motor weakness kept progressing (MRC grade 1) in all limbs. He developed bulbar symptoms in form of difficulty in swallowing. He also developed proprioception loss in the distal part of all limbs.

Based on disease progression over 8 weeks, the diagnosis was revised to acute onset CIDP as per European Federation

Table 1: Nerve Conduction Study of lower extremities

NCS	NERVE	LATENCY		AMPLITUDE (mV)		NCV (m/s)	F-MIN (ms)
		D	D	P	P		
Motor	Left CPN	11.66	3.19	3.72		28.08	70.25
	Left PTN	13.53	4.59	4.62		26.31	68.75
	Right PTN	NR	NR	NR		NR	-
	Right CPN	13.24	3.87	4.15		26.16	69.12
Sensory	Right Sural	2.32		16.51 μ V		60.34	
	Left Sural	2.4		13.26 μ V		58.33	

Table 2: Table showing Nerve Conduction Study of Upper Extremities

NCS	NERVE	LATENCY		AMPLITUDE (mV)		NCV (m/s)	F-MIN (ms)
		D	D	P	P		
Motor	Left Radial	6.56	4.94	5.12		32.48	41.47
	Left Median	8.63	5.09	5.21		33.49	36.75
	Left ulnar	7.86	5.13	5.22		31.87	30.56
	Right radial	8.44	5.74	5.55		44.50	33.7
	Right median	13.24	5.86	5.25		40.37	40.8
	Right Ulnar	7.22	5.87	5.15		33.40	69.12
Sensory	Left Radial	4.73		5.6 μ V		38.3	
	Left Median	4.55		10.3 μ V		61.9	
	Left Ulnar	2.60		7.9 μ V		40.5	
	Right Radial	4.78		6.14 μ V		33.6	
	Right Median	3.35		6.5 μ V		61.9	
	Right Ulnar	2.80		2.8 μ V		40.2	

of Neurological Societies (EFNS) criteria. He was started on intravenous methylprednisolone (MPS) (1 gram/day) for 5 days. After the 2nd dose of MPS, his bulbar and respiratory weakness started improving, and by the end of the week resolved completely. Subsequently, he was discharged on oral steroids (prednisolone 60 mg/day). Over the next month, he regained the power of 4-/5 in upper limbs and 3/5 in lower limbs. Repeat NCS revealed improvement in F wave latencies in bilateral common peroneal nerves, radial and median nerves. He maintained the response for the next 1 month but subsequently got admitted with liver metastasis and succumbed to his illness within a week.

The present case illustrates two important aspects of CIDP. Firstly, the patient had acute onset CIDP (A-CIDP) which contributes to 16% of cases of CIDP with typical progression over 8 weeks.^[2] Secondly, although the detailed workup for CIDP was negative, the recent diagnosis of prostatic malignancy in the patient made us think of the possible association. Although, it was difficult to determine whether CIDP was related to prostatic malignancy or it was a coincidence.

Demyelinating neuropathies (GBS, CIDP, multifocal motor neuropathy) are rarely associated with solid malignancy. CIDP has been described with solid malignancy like melanoma, gastrointestinal/hepatic carcinomas, breast carcinoma, lung malignancy, and very rarely with prostatic malignancy.^[3] Only one report showing association of prostatic malignancy with CIDP has been published in the last decade to the best of our knowledge.^[4] The pathophysiology of malignancy-associated CIDP is incompletely understood. However, some studies suggest the mechanism of molecular mimicry by expression of antigens of Schwann cells by some malignancies.^[5]

Paraneoplastic neuropathies can also mimic and fulfill the diagnostic criteria of demyelinating neuropathies. On applying diagnostic criteria for paraneoplastic neurological syndromes, we kept the probability of possible paraneoplastic neurological disorder secondary to prostatic malignancy.^[6] However, onconeural antibodies were negative in our case. In a literature review on 37 patients of prostatic malignancy with paraneoplastic neurological manifestation, none of them had CIDP. Onconeural antibodies were absent in 14 patients (37.8%).^[7]

This case expands the spectrum of neurological complications of prostatic malignancy with the association of acute onset CIDP and suggests its possible paraneoplastic origin. It also highlights the need for further research for the association, pathogenesis, and the possibility of another onconeural antibody that has not been identified yet in association with prostatic malignancy.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. The patient and his relatives have given consent for his images and other clinical information to be reported in the journal. They understand that his name and initials will not be published, and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed

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Conflicts of interest

There are no conflicts of interest.

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