



## AN UNUSUAL CAUSE OF POLYRADICULOPATHY

## Neurology

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## ABSTRACT

Idiopathic intracranial hypertension (IIH) is a condition characterised by raised intracranial pressure with normal neurological examination (except cranial nerve abnormalities) with normal neuro-imaging normal cerebrospinal fluid (CSF) composition. IIH presenting with polyradiculopathy is not common. Here we report a case of IIH presenting with polyradiculopathy and cranial neuropathies. The patient had normal normal imaging and normal CSF findings. Patient improved with repeated lumbar puncture (LP) and acetazolamide

## KEYWORDS

Vision, intracranial pressure, Polyradiculopathy, cerebrospinal fluid, quadriplegia

## INTRODUCTION

Idiopathic intracranial hypertension (IIH) is a condition characterised by raised intracranial pressure with normal neurological examination (except cranial nerve abnormalities) with normal neuro-imaging normal cerebrospinal fluid (CSF) composition.<sup>1</sup> Polyradiculopathy secondary to raised intracranial pressure is uncommon.

## Case Presentation

A 16 year old female presented with complaints of headache and vomiting for three weeks. Two weeks later, the patient developed bilateral, painless and gradually progressive blurring of vision. She also developed heaviness and weakness of both of both upper and lower limbs along with neck and upper backache. General physical examination showed abdominal striae, the rest being unremarkable. On neurological examination patient's vision was reduced in both eyes with only bilateral finger counting close to face. There was a pupillary asymmetry, with the right pupil being larger than left. Her fundus examination revealed papilloedema with blurring of disc margins, disc hyperemia and vessel tortuosity in both eyes. She had sixth cranial nerve palsy bilaterally, with left sided partial third cranial nerve (in the form of ptosis) and fifth cranial nerve (sensory, left mandibular division) involvement. The power in bilateral upper limbs was 3/5 at the shoulder joint, 4/5 at elbow and wrist; with left sided hand grip weak. Power in both the lower limbs was 3/5 and 4/5 in proximal and distal muscles respectively. The deep tendon reflexes were absent in all limbs. Her sensory examination was normal. Routine blood investigations were also normal. MRI Brain with contrast showed partial empty sella along with prominence of right optic nerve sheath with bulging of the optic nerve head (figure 1). Contrast venogram and MRI spine were normal. Her lumbar puncture was done which showed CSF opening pressure of 37 cm of H<sub>2</sub>O with no cells, protein – 32mg%, sugar – 44mg %, CSF for india ink and malignant cells was negative. Thyroid function test and serum cortisol level was normal. Nerve conduction studies showed prolonged F wave latency suggestive of polyradiculopathy (table 1). Visual Evoked Potentials were not recordable in right side. In view of markedly high CSF pressures, the lumbar puncture was repeated weekly for three week, following which the patient reported an improvement in vision and weakness. She was started on Acetazolamide in a dose of 1 gm/ day which was gradually up-titrated to 2.5 gm/day. Subsequently, she underwent repeated lumbar punctures with marked improvement in visual acuity and limb weakness. On further follow up patient's vision improved to 6/36 in right eye and 6/12 in left eye but her visual charting still showed severe restriction of peripheral vision (figure 1). The patient was referred for optic nerve fenestration surgery, but the Ophthalmology team opted to

manage the patient conservatively as her visual acuity was improving on medical management alone. She is currently on 2.5 gm/day of acetazolamide and continues to be on close follow up.

## DISCUSSION

Quadriplegia is rare in the setting of IIH and may lead to misdiagnosis of Guillain Barre Syndrome (GBS), especially because papilloedema and cranial neuropathies are known to occur in GBS<sup>2,3</sup>. It may entail a poor outcome for the patient in the form of progressive visual loss, if measures to lower ICP are not initiated urgently. Involvement of third or fifth cranial nerve along with limb weakness due to raised intracranial tension is uncommon.<sup>4,5</sup> Our patient presented with headache, visual diminution, and also had quadriplegia secondary to radiculopathy along with bilateral sixth, and left partial third and fifth cranial nerve paresis. Her symptoms improved with repeated CSF drainage and acetazolamide.

Flaccid quadriplegia in cases of IIH occurs due to radiculopathy probably due to increased CSF pressure distending the subarachnoid space around the nerve roots stretching the nerve roots.<sup>6</sup> This is reversible with timely treatment.

We have presented a case of IIH with poly radiculopathy with areflexic quadriplegia, vision loss and partial third and 5<sup>th</sup> cranial nerve paresis. Most previous cases reported in literature were managed with CSF diversion procedures<sup>7,8</sup>. However, our patient was managed with initially with repeated lumbar punctures to lower intracranial pressure, followed by maintenance therapy with high doses of Acetazolamide, on which she continues to show improvement in visual acuity.

**Table 1. Nerve Conduction Study showing loss of F responses F-wave parameters**

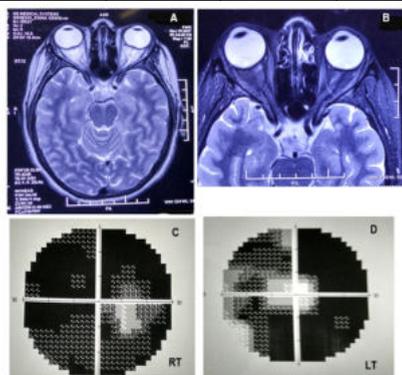
Test	Fmin lat., ms	M lat., ms	Fmin-M lat., ms
<b>right, Median</b>		2.5	
<b>left, Median</b>		2.6	
<b>right, Ulnar</b>		2.05	
<b>left, Ulnar</b>		2.1	
<b>right, Peroneal</b>		3.35	

<b>left, Peroneal</b>			
		3.15	
<b>right, Tibial</b>			
	49.6	3.85	45.7
<b>left, Tibial</b>			
	51.4	4.9	46.5

**Table 2. Follow-up nerve conduction study showing improvement in F response after 2 weeks.**

**F-wave parameters**

Test	Fmin lat., ms	M lat., ms	Fmin-M lat., ms
<b>right, Median</b>			
	26.9	2.6	24.3
<b>left, Median</b>			
	24.5	2.75	21.8
<b>right, Ulnar</b>			
	27.6	2.05	25.5
<b>left, Ulnar</b>			
	27.4	2.1	25.3
<b>right, Peroneal</b>			
	46.6	2.85	43.8
<b>left, Peroneal</b>			
	43.8	3.3	40.5
<b>right, Tibial</b>			
	46.8	3.05	43.7
<b>left, Tibial</b>			
	49.4	3.75	45.6



**Fig 1 A and B** showing prominence of right optic nerve sheath with bulging of the optic Nerve head. **C and D** showing severe peripheral visual field restriction in right and left eye respectively

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