Case Report
Volume 8:7, 2020

Journal of Neurological Disorders

ISSN: 2329-6895

Open Access

A Case Report of Quadriparesis and Cranial Polyneuropathy: A Rare Presentation in Idiopathic Intracranial Hypertension

Aravind G[1], Siddharth Maheshwari[1]*, Aldrin Anthony[1], Suman Kushwaha[1] and Monali Chaturvedi[2]

[1]Department of Neurology, IHBAS (Institute of Human Behaviour & Allied Sciences), New Delhi, India
[2]Department of Neuroradiology, IHBAS (Institute of Human Behaviour & Allied Sciences), New Delhi, India

Abstract

Cerebral Venous Thrombosis (CVT) and Idiopathic Intracranial Hypertension (IIH) are always to be considered in patients with isolated intracranial hypertension. We report a patient with progressive visual loss, ophthalmoplegia and polyradiculopathy with are flexic quadriparesis, secondary to raised intracranial pressure (ICP). Quadriparesis due to raised ICP is a rarely reported presentation.

Keywords: Quadriparesis • Raised ICP • Progressive visual loss.

Introduction

Idiopathic Intracranial Hypertension (IIH) is a syndrome where there is raised ICP without any structural cause (space occupying lesion or dural venous sinus thrombosis on brain imaging). This syndrome usually occurs in young, obese females, but may appear in any age. The most common symptom seen in IIH is headache. Other symptoms are diplopia and transient visual blurring. However, children may have an asymptomatic presentation [1]. Various false localizing signs are seen in IIH among which radiculopathy is rarely reported. Many authors have documented subtle features of radiculopathy in patients with isolated IIH. The usual manifestations of radiculopathy in these cases were limb paraesthesias, and radicular pain. Quadriparesis due to radiculopathy caused by intracranial hypertension has rarely been described [2]. We report a young female patient presented with progressive visual loss, ophthalmoplegia and flaccid quadriparesis. Raised ICP due to IIH was the cause behind the clinical manifestations in this patient.

Case History

A 33-year-old obese female came with complaints of headache followed by progressive loss of vision with mild pain for last 1 month. Since last 1week patient had developed weakness of all 4 limbs and also there was associated paraesthesias in all limbs. No bowel or bladder involvement. No h/o fever. No h/o vomiting. No h/o seizures or joint pain or rashes. H/o post-polio residual paralysis in left leg. Her examination revealed no perception of light, complete ophthalmoplegia, dilated pupils not reacting to light. Fundus showed B/l pale discs with haemorrhage. Power 2/5 all 4 limbs, are flexic and bilateral plantars flexor. No sensory findings. Other detailed examination could not be done. Patient's blood counts, Liver function tests and renal function tests and ESR were normal. CSF pressure manometry showed very high pressure (37 cm H_2O) and normal sugar, protein and counts. CSF cryptococcal antigen was negative. Serum Lyme's IgM -ve. S. ACE levels Normal. NCS of all 4 limbs was normal. ENA profile normal. Serum NMO and MOG -ve. CECT Abdomen and thorax Normal. MRI Brain with contrast – Normal, MRI Orbit with contrast –Bilateral Hyperintense T2 signals in intracanicular optic nerves. MRI whole spine screening – normal, MRV - Normal. Ophthalmology opinion taken-nil visual prognosis given. Pt started on Inj. MPS 1 g IV OD for 5 days. Tablet Diamox started at 250 mg tds and increased to 500 mg tds. Tablet Topiramate started. Patient condition drastically improved with patient having minimal gaze restriction and normal power in limbs. But vision showed no improvement. Patient CSF studies repeated after 10 days. Pressure – 20 cm H_2O, and routines were normal. Patient advised Lumboperitoneal shunt but not done in view of negative consent. Patient maintained on Diamox and Topiramate and is in follow-up (Figures 1-6).

Discussion

Our patient had acute onset of bilateral visual loss, total ophthalmoplegia, flaccid quadriparesis and intact sensorium along with raised intracranial pressure (ICP). MRI Brain and orbit with contrast were normal except for hyperintense signals in bilateral optic nerves and MRV and MRI whole spine screening showed no abnormalities that could explain the condition. Reduction of ICP resulted in dramatic recovery, suggesting a causal relation between intracranial hypertension and the presenting manifestation. Blindness and

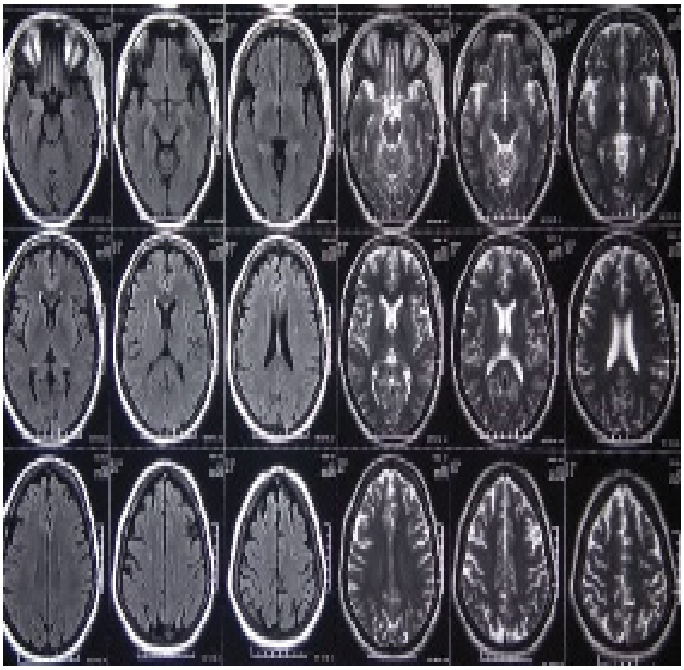

Figure 1. MRI Brain T2WI and FLAIR images show no abnormality.

***Address for Correspondence:** Siddharth Maheshwari, Department of Neurology, IHBAS (Institute of Human Behaviour & Allied Sciences), New Delhi, India Tel: 9868396865; E-mail: drsiddharthvds@gmail.com*

Received 15 November, 2020; **Accepted** 23 November, 2020; **Published** 30 November, 2020

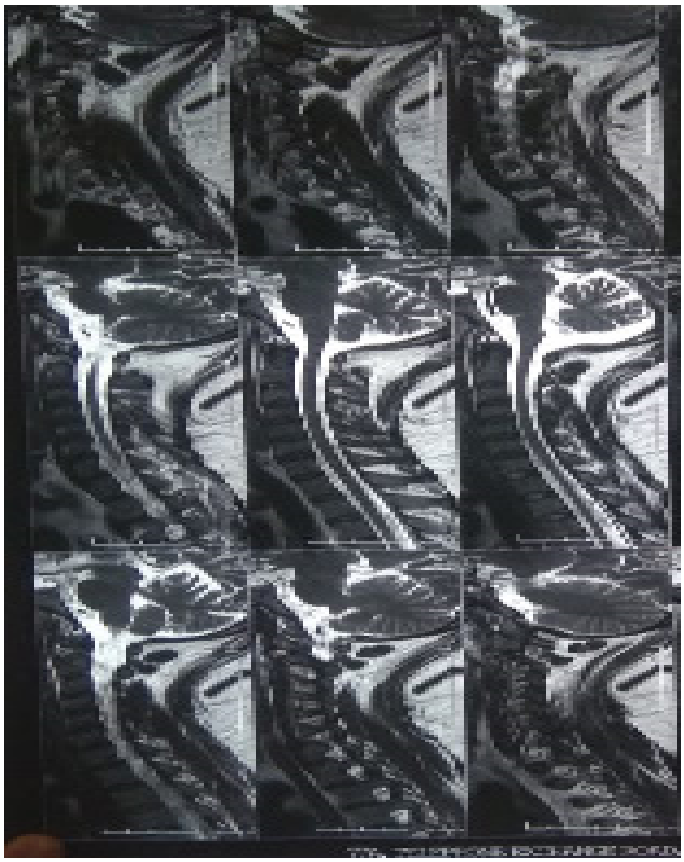

Figure 2. MRI cervicodorsal spine is normal.

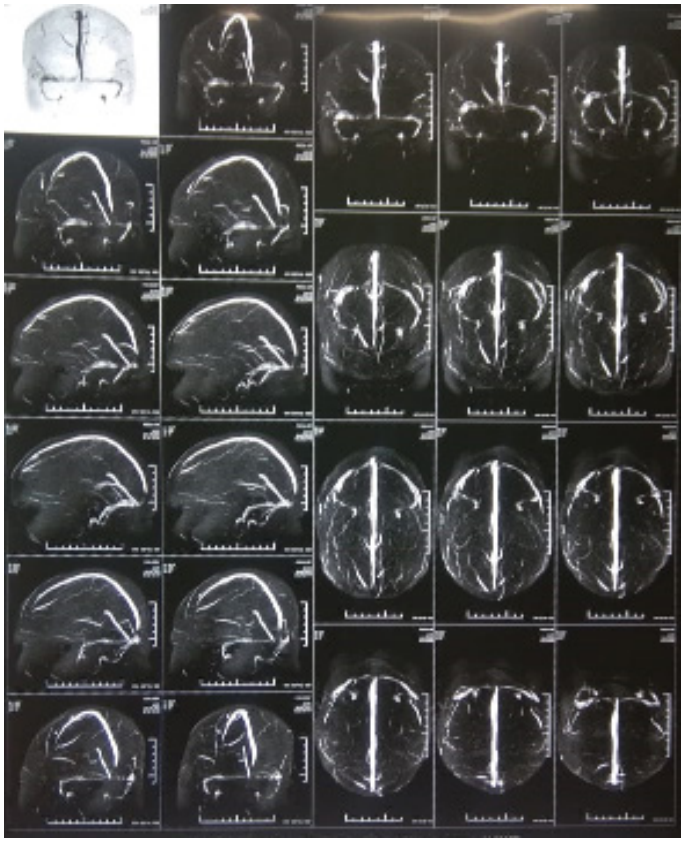

Figure 3. MRV shows normal filling in all sinuses.

ophthalmoplegia are known manifestations of IIH but flaccid quadriparesisis an extremely rare presentation.

Initially localisation was made to midbrain as there are flexic quadriparesis

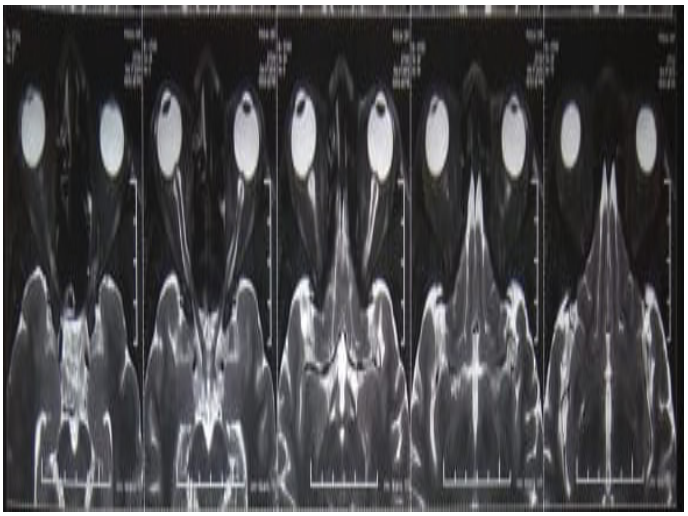

Figure 4. MRI orbit with contrast showing T2 HI in intracanicular optic nerves.

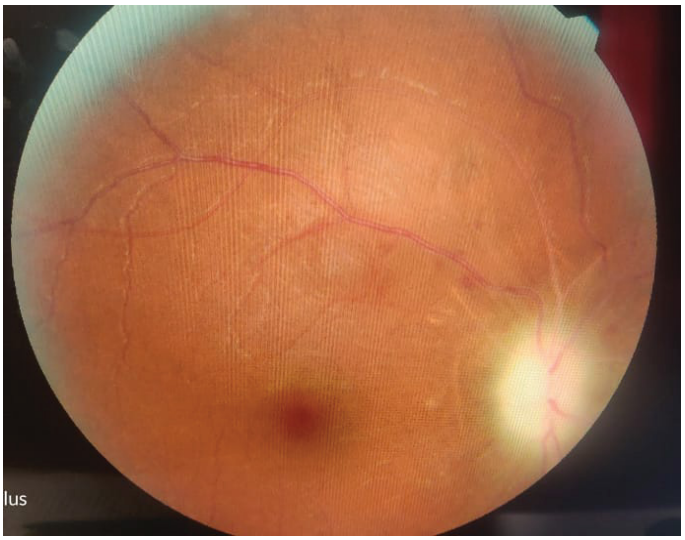

Figure 5. Depicts optic atrophy secondary to disc edema with sclerosed arteries and multiple superficial hemorrhages.

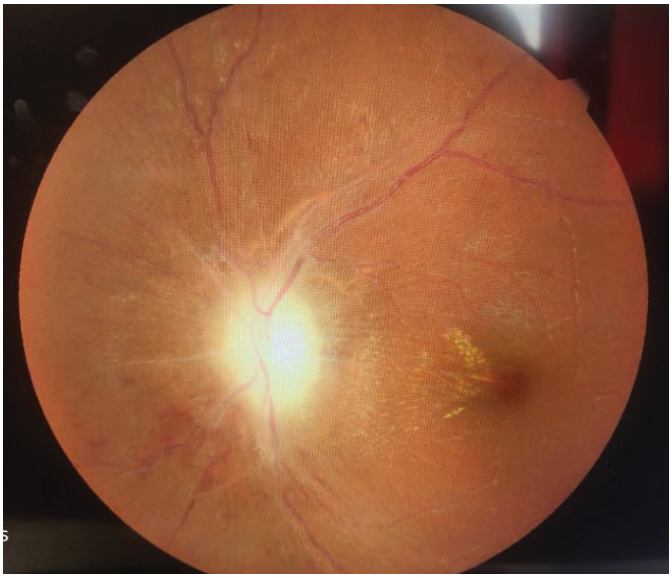

Figure 6. Depicts optic atrophy secondary to disc edema with multiple superficial hemorrhages, sclerosed vessels with macular star.

with ophthalmoplegia but blindness could not be explained by this, and we did a MRI which showed no lesion in the brainstem. Then we thought of Cerebral venous sinus thrombosis causing raised ICP, this was excluded by brain MRV.

We thought of GBS but normal NCV and normal CSF protein ruled it out. Also cases of GBS with papilledema usually had limb weakness then raised ICP features unlike our case. The elevated CSF opening pressure made us think of IIH as probable diagnosis, and patient was started on medical management and she improved drastically. This proves that the clinical features are related to raise ICP. We also ruled out demyelinating pathology, autoimmune cause, Lyme's disease. Moosa et al., reported two cases of intracranial sinus thrombosis who presented with blindness, ophthalmoplegia and flaccid quadriparesis due to extensive radiculopathy [3]. Raised ICP due to intracranial sinus thrombosis was found to be the aetiology for these clinical features Obeid et al., reported two cases with extensive radiculopathy due to raised ICP; one patient had IIH while the other suffered dural venous sinus thrombosis. Both had papilledema and severe visual loss [4]. Nerve conduction study showed features of a diffuse radiculopathy. However, ophthalmoplegia was not there in both the cases. Moosa et al. proposed that mechanical compression of nerve roots by elevated ICP distending the subarachnoid space is the most likely mechanism explaining the presence of radiculopathy in patients with IIH. Reported enlargement of spinal subarachnoid space and distended rootpouches in a patient diagnosed with IIH supports this view [5].

Conclusion

Our patient is a case of IIH presenting with flaccid quadriparesis, ophthalmoplegia and blindness diagnosed after ruling out other probable causes. This presentation of raised ICP presenting as quadriparesis is rare and also no case report is available till date of this presentation improving with medical management. Furthermore T2 hyperintensities in optic nerve resembling an optic neuritis like picture is not usually seen in IIH.

References

1. Soiberman, Uri and Anat Kesler. "Idiopathic intracranial hypertension what is new in 2012" *Harefuah* 152 (2013): 115-118.
2. Moosa, Ahsan, Ma Joy and Ashiwani Kumar. "Extensive radiculopathy: another false localizing sign in intracranial hypertension" *J Neurol Neurosurg Psychiatry* 75 (2004): 1080-1081.
3. Ahsan, Moosa Naduvil, Asha Kishore, AK Gupta and Kurupath Radhakrishnan. "Blindness, ophthalmoplegia and extensive radiculopathy: An unusual clinical syndrome in intracranial sino-venous thrombosis" *Neurol* 52 (2004): 96-98.
4. Obeid, Tony, Adnan Awada, Yahya Mousali and M Nusair, et al. "Extensive radiculopathy: a manifestation of intracranial hypertension" *Eur J Neurol* 7 (2000): 549-553.
5. Moodley, Anand A, Mahlubonke S Dlwati and Miranda Durand. "Intracanalicular Optic Nerve Swelling and Signal Change in Fulminant Untreated Idiopathic Intracranial Hypertension" *Neuroophthalmology* 41 (2016): 84-89.

How to cite this article: Aravind, Siddharth Maheshwari, Aldrin Anthony and Suman Kushwaha. "A Case Report of Quadriparesis and Cranial Polyneuropathy: A Rare Presentation in Idiopathic Intracranial Hypertension." J Neurol Disord 8 (2020): 442