Original Research Paper

Volume - 12 | Issue - 03 | March - 2022 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Ophthalmology

A CASE OF FULMINANT INCREASED INTRACRANIAL HYPERTENSION-LESSONS LEARNED

Dr.Vidhya Devi.R*	Assistant professor , Department of Ophthalmology, PSGIMSR, Coimbatore. *Corresponding Author
Dr. S.Saudhamini	Senior resident, Department of Ophthalmology, PSGIMSR, Coimbatore.
Dr. Padmanaban.S	Associate Professor, Department of Ophthalmology, PSGIMSR, Coimbatore.
Dr. Aswatha Sree	Junior resident, Department of Ophthalmology, PSGIMSR, Coimbatore.

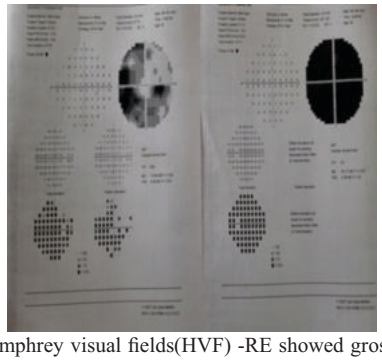
ABSTRACT Idiopathic Intracranial Hypertension (IIH) is defined as an elevated intracranial pressure but no clinical, laboratory or radiological evidence of hydrocephalus, infection, tumor or vascular abnormality.

IIH usually occurs in obese women in childbearing years [1]. The symptoms of increased intracranial pressure are headache, pulse-synchronous tinnitus(pulsatile tinnitus), transient visual obscuration and visual loss. Signs of IIH are diplopia due to sixth cranial nerve paresis and papilledema with its associated loss of sensory visual function [1].

Fulminant IIH" was defined as the acute onset of symptoms and signs of intracranial hypertension (less than 4 weeks between onset of initial symptoms and severe visual loss), rapid worsening of visual loss over a few days, and normal brain MRI and MR venography (or CT venogram). [3]

KEYWORDS : Increased intracranial hypertension(IIH) , Fulminant increased intracranial hypertension , Papilledema.

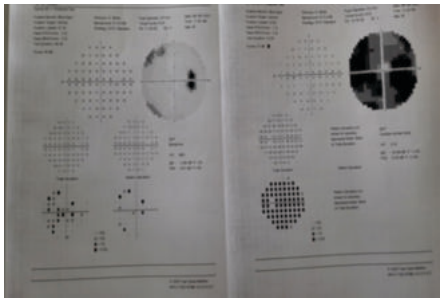
Figures And Tables



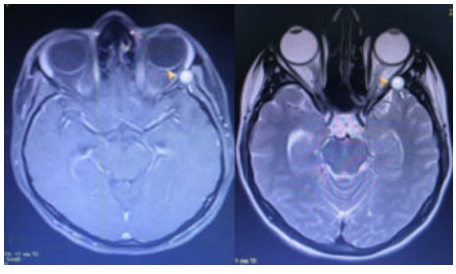
1st day: Humphrey visual fields(HVF) -RE showed gross defects in supero nasal,inferior quadrants.

LE showed gross field defects in all quadrants.

Be Oct Rnfl Showed Edema.



On 3rd day: Vision-RE improved to 6/6.
LE improved from 1/60 to 6/9.
Both eyes(BE): Pupil reaction, HVF improved.
OCT RNFL showed reduced edema.



MRI brain was normal.

MRI orbit showed widening of perioptic space with tortuous course of bilateral optic nerves, flattening of posterior sclera on both sides-suggestive of IIH.

Case Report:

A 32year old female came with complaints of headache associated with blurring of vision in left eye since 3days.

On examination right eye vision 6/12 with pinhole 6/6,near vision N6, colour vision-normal, contrast sensitivity normal, lids normal, conjunctiva-quiet, cornea-clear, A/C-normal depth, pupil 3mm RAPD grade 1 , lens-clear, fundus-papilledema grade1.

Left eye vision 1/60 with pinhole NIF, near vision N36, colour vision-normal, contrast sensitivity could not be assessed, lids normal, conjunctiva-quiet, cornea-clear, A/C-normal depth, pupil 3.5mm RAPD grade 2, lens-clear, fundus-papilledema grade4 .

On Examination	Right eye	Left eye
1.Vision	6/12 with PH 6/6, N6	1/60 not improving.
2.Colour vision	Normal	Normal
3.Contrast sensitivity	Normal	Could not be assessed
4.Lids	Normal	Normal
5.Conjunctiva	Quiet	Quiet
6.Cornea	Clear	Clear
7.A/C	Normal depth	Normal depth
8.Pupil	3mm,RAPD grade 1	3.5 mm, RAPD grade 2
9.Lens	Clear	Clear
10.Fundus	Papilledema grade 1	Papilledema grade 4
11.Extra ocular movements	Full	Full

Humphrey visual fields were taken. Right eye showed gross defects in supero nasal,inferior quadrant, left eye showed gross field defects in all quadrants.

MRI brain was normal, Orbit showed widening of perioptic space with tortuous course of bilateral optic nerves, flattening of posterior sclera on both sides.

Patient underwent lumbar puncture and 30ml cerebrospinal fluid was drained.CSF opening pressure was noted to be 20cm of H2O, showed neutrophils and culture was negative.

Patient was started on tablet acetazolamide 500mg and intravenous methyl prednisolone 1mg/kg body weight.

3days later patient vision in left eye improved from 1/60 to 6/9. HVF also improved and became normal.

Our case of fulminant IIH improved with steroids and carbonic anhydrase inhibitors and vision is maintained.

DISCUSSION:

"Fulminant IIH" was defined as the acute onset of symptoms and signs of intracranial hypertension (less than 4 weeks between onset of initial symptoms and severe visual loss), rapid worsening of visual loss over a few days, and normal brain MRI and MR venography (or CT venogram). [1]

Thambisetty M et al[3] presented ,16 cases with "fulminant IIH" (16 women, mean age 23.8 years [range 14 to 39 years]). Severe and rapidly progressive visual loss suggests "fulminant idiopathic intracranial hypertension" and should prompt aggressive management. Urgent surgery may be required in these patients, and temporizing measures such as repeat lumbar punctures, lumbar drainage, and IV steroids considered. The median delay between evaluation in neuro-ophthalmology and surgery was 3 days (range a few hours to 37 days). All patients reported dramatic improvement of headaches and vomiting following surgery. Visual function improved in 14 cases, although 8 patients (50%) remained legally blind. Visual fields remained severely altered in all cases.

Bouffard MA et al[4] described the presenting features of fulminant idiopathic intracranial hypertension (IIH) and outlined the multimodal approach to its treatment. Prompt surgical intervention with optic nerve sheath fenestration, cerebrospinal fluid shunting, or venous sinus stenting minimizes the chance of poor visual outcome. If a delay is anticipated, serial lumbar punctures or temporary cerebrospinal fluid drainage and medical therapy may forestall irreversible vision loss.

Rapid recognition of the fulminant phenotype of IIH by emergency department physicians, neurologists, and ophthalmologists is critical.[4] Only limited case reports or studies explaining the severity of this condition are available.

Hence we presented a case of fulminant IIH that improved with timely management of steroids and carbonic anhydrase inhibitors and vision is maintained.

CONCLUSION:

Early diagnosis and management of fulminant IIH can prevent irreversible visual loss.

ACKNOWLEDGMENT:

Dr.Balakrishnan, Professor, Head of the Department, Department of Neurology, PSGIMSR, Coimbatore.

Dr.Sundar.D, Professor, Department of Ophthalmology, PSGIMSR, Coimbatore.

REFERENCES:

1. Wall M (2008) Idiopathic intracranial hypertension (Pseudotumor cerebri). Curr Neurol Neurosci Rep 8: 87-93.
2. Bjerre P, Lindholm J, Gyldensted C (1982) Pseudotumor cerebri: A theory on etiology and pathogenesis. Acta Neurol Scand 66: 472-481.
3. Thambisetty M, Lavin PJ, Newman NJ, Biousse V. Fulminant idiopathic intracranial hypertension. Neurology. 2007 Jan 16;68(3):229-32. doi: 10.1212/01.wnl. 0000 251 312.19452.ec. PMID: 17224579.
4. Bouffard MA. Fulminant Idiopathic Intracranial Hypertension. Curr Neurol Neurosci Rep. 2020 Mar 26;20(4):8. doi: 10.1007/s11910-020-1026-8. PMID: 32219578.