Case Report

DOI: 10.7860/JCDR/2022/55673.16716

Malignant Hypertension Leading to Non Arteritis Anterior Ischaemic Optic Neuropathy-Unilateral Involvement an Exigency

Internal Medicine Section

RUCHITA KABRA[1], SOURYA ACHARYA[2], SACHIN DAIGAVANE[3], AVI SHARMA[4], SUNIL KUMAR[5]

ABSTRACT

Non arteritis Anterior Ischaemic Optic Neuropathy (NAION) is mentioned as loss of blood supply to the optic nerve further causing sudden onset and painless vision loss in eye. Exact mechanism, leading to reduced or loss of flow of blood to an optic nerve in NAION, is unknown but there are certain risk factors responsible for NAION like diabetes, malignant hypertension, Hypercholesterolaemia, platelet polymorphism, phosphodiesterase inhibitors, hyperhomocysteinemia, nocturnal hypotension, sleep apnoea, contribute to it. This case report is about a 40-year-old male patient who presented with a complaint of sudden onset vision loss in one eye which was further diagnosed as NAION on fundus examination. This shows association of malignant hypertension and NAION. Early diagnosis of the condition led to complete recovery of vision loss. Early management of blood pressure and timely management of NAION using antihypertensives and steroids in this patient found to be fruitful and patient regained his vision back.

Keywords: Antihypertensive, Blood pressure, Vision

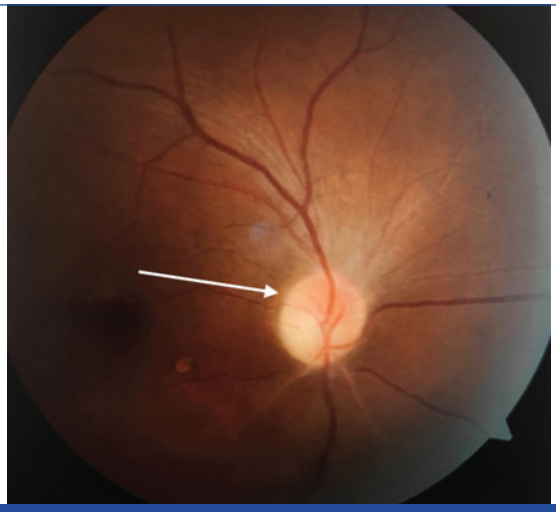
CASE REPORT

A 40-year-old male patient visited the Outpatient Department with complaint of sudden loss of vision in his left eye, noticed since morning. Vision loss was painless in nature. Patient specifically complained of loss of vision particularly in inferior half portion of left eye. On inquiring, he had no complains of floaters, sudden flashes of light, headache, giddiness, light or noise intolerance, pain on eye movements. He was not known to have co-morbidities like hypertension, diabetes mellitus, bronchial asthma, tuberculosis or any chronic systemic disorder or any medications.

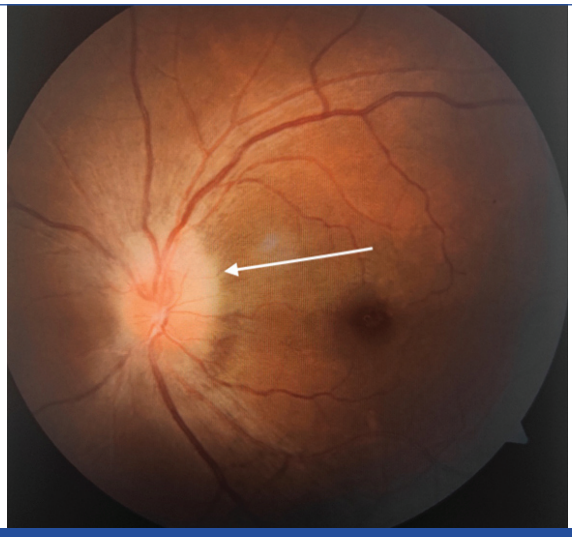
On examination, the patient was conscious and oriented. His vitals were recorded, pulse was 90 beats/min (regular, high volume), blood pressure was 240/146 mmHg, respiratory rate was 18/min. Cardiovascular system examination revealed normal S1, S2 heart sounds, no S3 or any murmur, respiratory and abdominal system revealed no abnormality, Neurological examination was done and was normal. In view of hypertensive emergency, the patient was given injectable labetalol 20 mg intravenous followed by 40 mg, he was monitored for his vitals and Blood Pressure (BP) was monitored at regular interval. Once the BP was normal, he was started on oral antihypertensives like tab. amlodepine 5 mg twice a day, tab. telmisartan 40 mg twice a day, tab. prazosin 5 mg twice a day, tab. metoprolol 50 mg twice a day.

To rule out further complications of malignant hypertension, the patient was evaluated for ophthalmic examination, Electrocardiogram (ECG), 2D Echocardiogram, Magnetic Resonance Imaging (MRI) of brain, chest radiograph. On ophthalmic examination, his best corrected visual acuity was 6/9 in two eyes, colour vision was normal in both eyes with inferior vision loss in left eye with no rapid afferent pupillary defect. Fundus examination with indirect ophthalmoscope showed disc pallor, arteriolar attenuation at posterior pole and retinal pigment epithelium alteration at macula in right eye [Table/Fig-1]. Optic disc oedema with pallor, more superiorly than inferiorly with arteriolar attenuation was seen in the left eye [Table/Fig-2]. Visual field testing showed inferior altitudinal field defect in left eye [Table/Fig-3].

The ECG was suggestive of left ventricular hypertension (Sokolow-Lyon criteria) [1]. The 2D Echocardiogram was suggestive of left ventricular hypertrophy with increased thickness of the posterior wall and septum. Chest radiograph showed displacement of cardiac apex to lateral and downward and rounded appearance of

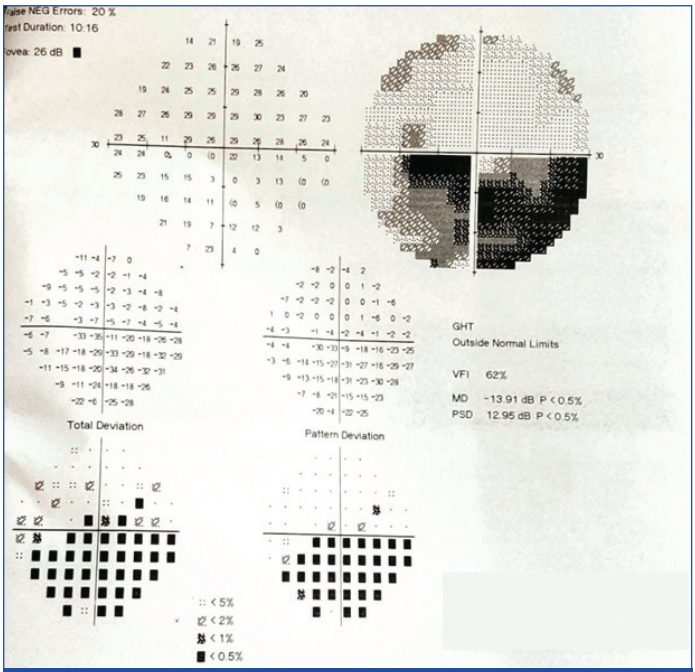


[Table/Fig-1]: Right eye fundus showing disc pallor, arteriolar attenuation at posterior pole (white arrow) and retinal pigment epithelium alteration at macula.

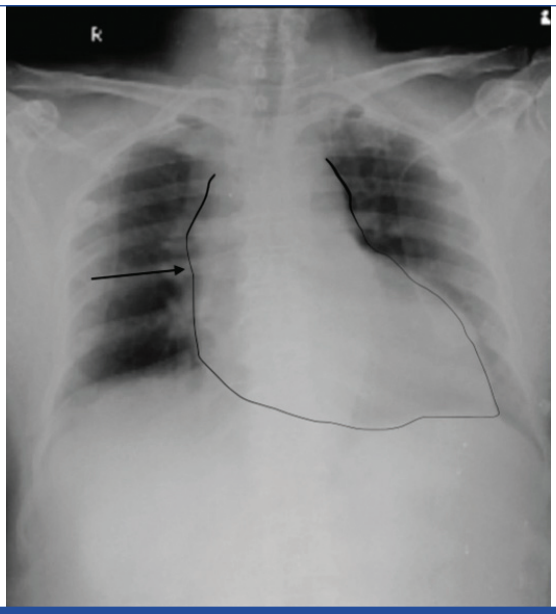


[Table/Fig-2]: Left eye fundus showing optic disc oedema with pallor, more superiorly than inferiorly with arteriolar attenuation (white arrow).

cardiac apex also called as "Shmoo sign" [Table/Fig-4]. The Brain showed chronic lacunar infarct in right side of pons. Laboratory investigations are presented in [Table/Fig-5].



[Table/Fig-3]: Visual field test showing inferior altitudinal field defect in left eye.



[Table/Fig-4]: Chest X-ray showing 'Shmoo' sign (black arrow).

Laboratory investigation	Values
Haemoglobin	14.5 gm% (Normal)
White blood cells counts	6000/cumm (Normal)
Platelet counts	1.7 lakh/cumm (Normal)
Serum creatinine	2.2 mg/dL (Elevated)
Urea	50 (Slight elevated)
Serum sodium	143 mmol/L (Normal)
Serum potassium	4.7 mmol/L (Normal)
Creatine kinase-MB	24 IU/L (Normal)
Troponin-I	18.76 pg/mL (Normal)

[Table/Fig-5]: Laboratory investigations on the day of admission.

The patient was diagnosed to have malignant hypertension causing unilateral non arteritic ischaemic optic neuropathy on the second day of admission. The patient was continued with oral medications to control his blood pressure and regular monitoring of blood pressure was done.

On day 5 of admission, BP was controlled, patient was given injectable methylprednisolone 1 gm in 100 mL normal saline once a day for 3 days for the treatment of NAION. Ophthalmic examination was done daily to look for further progression of the disease and it showed no progression. After 6 days of hospital stay, patient was discharged. Blood pressure on discharge was 130/80 mmHg. Patient was on four antihypertensive drugs on discharge (tab. amlodipine 5 mg twice a day, tab. telmisartan 40 mg twice a day, tab. prasosin 5 mg twice a day, tab. metoprolol 50 mg twice a day).

DISCUSSION

Non arteritic Anterior Ischaemic Optic Neuropathy (NAION) is referred as loss of blood flow to the optic nerve which can lead to sudden painless loss of vision in one eye. It is called "non arteritic" because there is decreased blood flow and without any true inflammation of vessel. It is called "anterior" as reduced blood flow to optic nerve occurs in most front part of the nerve. It is called "ischaemic" because injury is because of reduced blood supply. It is called "optic neuropathy" because it is an injury to the optic nerve. Optic nerve ischaemia may occur at different locations along the course and it is related to various causes which differentiates these syndromes by their causes and different locations and their signs and symptoms along with treatment and outcome also varies [2].

By definition, Anterior Ischaemic Optic Neuropathy (AION) shows involvement of first 1 mm part of the head of optic nerve which is also called as optic disc, and which can be seen as visible disc swelling. The AION is of two types i.e., arteritis and non arteritis [3]. Arteritis type of AION always occurs in association with giant cell arteritis. Non arteritis type of AION are mostly idiopathic but some of them showed specific causes such as malignant hypertension.

The majority of cases of AION are non arteritis type [4]. Non arteritis AION is the most common cause of acute onset neuropathy of optic nerve in patients of age more than 45. Men and women are nearly equally affected [5,6].

The pathogenesis of non arteritis AION is controversy as not a single mechanism is perfectly illustrated. It is assumed that it results from ischaemia or infarct, within the retrolaminar portion of the proximal part of optic nerve head which is supplied by the short branch of posterior ciliary arteries [7-9]. Majority of patients suffering from NAION has small optic discs which measures less than 0.3 mm, which is referred to as crowded disc and prone for ganglion cell death [10-14]. The optic nerve head can autoregulate the blood flow [15]. Risk factors like vasospasm, medications, hypertension or arteriosclerosis may decrease the optic disk capacity to autoregulate the blood flow [16,17].

By definition, malignant hypertension is an acute onset rise of blood pressure that is systolic BP ≥180 mmHg and/or diastolic BP ≥120 mmHg with diffuse necrotising vasculitis, arterial thrombi and fibrin deposition in arterial wall, which clinically can be recognised with papilledema [18]. Malignant hypertension may result because of kidney dysfunction, autonomic dysfunction or non compliance to medications. Ocular involvement of malignant hypertension may include retinopathy, with constriction of arteriole, retinal haemorrhages, cotton wool spots, or disc oedema in severe involvement.

Hypertensive optic neuropathy is a late manifestation of malignant hypertension. Main difference between accelerated hypertension and malignant hypertension is papilledema-It is present in malignant hypertension and absent in accelerated one. Optic disc oedema is commonly detected in patients of malignant hypertension. The cause behind optic disc oedema is variable. It may be due to raised intracranial tension or ischaemia at the optic nerve head [19,20].

Malignant hypertension is associated with acute onset involvement of end-organs which leads to end-organ damage. This includes eye involvement leading to retinopathy, brain involvement leading

to encephalopathy, intracranial haemorrhage, renal involvement leading to renal failure, large vessel involvement leading to aortic dissection [21], rupture of an abdominal aneurysm [22], cardiac and respiratory involvement leading to increased pressure in pulmonary veins ultimately leading to pulmonary oedema, and acute myocardial infarction [23]. These patients require urgent hospitalisation, early diagnosis and early management. Within few months, changes in fundus due to raised blood pressure revert back, but there may be some irreversible loss of vision occurring due to irreversible injury to vascular supply leading to vascular shutdown [19].

CONCLUSION(S)

Simultaneous involvement of bilateral NAION is rare. Simultaneous involvement of eyes due to NAION leads to vision loss in both eyes. Simultaneous involvement occurs because first eye involvement remains unnoticed unless and until second eye is involved. This case report shows unilateral involvement of eye due to NAION secondary to malignant hypertension. Early diagnosis of the condition lead to early treatment and halt the progression of the disease.

REFERENCES

[1] Truong QA, Ptaszek LM, Charipar EM. Performance of electrocardiographic criteria for left ventricular hypertrophy as compared with cardiac computed tomography: From the rule out myocardial infarction using computer assisted tomography trial. J Hypertens. 2010;28:1959-67.
[2] Salgado C, Vilson F, Miller NR, Bernstein SL. Cellular inflammation in nonarteritic anterior ischemic optic neuropathy and its primate model. Archives of Ophthalmology. 2011;129(12):1583-91.
[3] Hayreh SS. Ischemic optic neuropathy. Progress in Retinal And Eye Research. 2009;28(1):34-62.
[4] Guyer DR, Miller NR, Auer CL, Fine SL. The risk of cerebrovascular and cardiovascular disease in patients with anterior ischemic optic neuropathy. Arch Ophthalmol. 1985;103(8):1136-42.
[5] Hattenhauer MG, Leavitt JA, Hodge DO, Grill R, Gray DT. Incidence of nonarteritic anterior ischemic optic neuropathy. Am J Ophthalmol. 1997;123(1):103-07.
[6] Johnson LN, Arnold AC. Incidence of nonarteritic and arteritic anterior ischemic optic neuropathy. Population-based study in the state of Missouri and Los Angeles County, California. J Neuroophthalmol. 1994;14(1):38-44.
[7] Rootman J, Butler D. Ischaemic optic neuropathy-A combined mechanism. Br J Ophthalmol. 1980;64(11):826-31.
[8] Cogan DG. Neurology of the Visual System: Charles C. Thomas Pub Ltd; 1966.
[9] Knox DL, Duke JR. Slowly progressive ischemic optic neuropathy. A clinicopathologic case report. Trans Am Acad Ophthalmol Otolaryngol. 1971;75(5):1065-68.
[10] Jonas JB, Gusek GC, Naumann GO. Anterior ischemic optic neuropathy: Nonarteritic form in small and giant cell arteritis in normal sized optic discs. Int Ophthalmol. 1988;12(2):119-25.
[11] Mansour AM, Shoch D, Logani S. Optic disk size in ischemic optic neuropathy. Am J Ophthalmol. 1988;106(5):587-89.
[12] Feit RH, Tomsak RL, Ellenberger C Jr. Structural factors in the pathogenesis of ischemic optic neuropathy. Am J Ophthalmol. 1984;98(1):105-08.
[13] Beck RW, Savino PJ, Repka MX, Schatz NJ, Sergott RC. Optic disc structure in anterior ischemic optic neuropathy. Ophthalmology. 1984;91(11):1334-37.
[14] Doro S, Lessell S. Cup-disc ratio and ischemic optic neuropathy. Arch Ophthalmol. 1985;103(8):1143-44.
[15] Riva CE, Hero M, Titze P, Petrig B. Autoregulation of human optic nerve head blood flow in response to acute changes in ocular perfusion pressure. Graefes Arch Clin Exp Ophthalmol. 1997;235(10):618-26.
[16] Hayreh SS, Zimmerman MB, Podhajsky P, Alward WL. Nocturnal arterial hypotension and its role in optic nerve head and ocular ischemic disorders. Am J Ophthalmol. 1994;117(5):603-24.
[17] Hayreh SS, Piegors DJ, Heistad DD. Serotonin-induced constriction of ocular arteries in atherosclerotic monkeys. Implications for ischemic disorders of the retina and optic nerve head. Arch Ophthalmol. 1997;115(2):220-28.
[18] Keith NM, Wagener HP, Barker NW. Some different types of essential hypertension: Their course and prognosis. Am J Med Sci. 1974;268:336-45.
[19] Hayreh SS, Servais GE, Virdi PS. Fundus lesions in malignant hypertension, hypertensive optic neuropathy. Ophthalmology. 1986;93:74-87.
[20] Luo BP, Brown GC. Update on the ocular manifestations. Curr Opin Ophthalmol. 2004;15(3):203-10.
[21] Prisant LM, Nalamolu VRP. Aortic dissection. J Clin Hypertens (Greenwich). 2005;7:367-71.
[22] Prisant LM, Mondy JS III. Abdominal aortic aneurysm. J Clin Hypertens (Greenwich). 2004;6:85-89.
[23] Prisant LM, Carr AA, Hawkins DW. Treating hypertensive emergencies. Controlled reduction of blood pressure and protection of target organs. Postgrad Med. 1993;93:92-96,101-104,108-110.

PARTICULARS OF CONTRIBUTORS:
1. Junior Resident, Department of Medicine, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India.
2. Professor and Head, Department of Medicine, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India.
3. Professor and Head, Department of Ophthalmology, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India.
4. Junior Resident, Department of Ophthalmology, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India.
5. Professor, Department of Medicine, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India.

NAME, ADDRESS, E-MAIL ID OF THE CORRESPONDING AUTHOR:
Dr. Ruchita Kabra,
Junior Resident, Department of Medicine, Datta Meghe Institute of Medical Sciences (Deemed to be University), Wardha, Maharashtra, India
E-mail: ruchitapkabra@gmail.com

PLAGIARISM CHECKING METHODS: [Jain H et al.]
- Plagiarism X-checker: Feb 19, 2022
- Manual Googling: Apr 27, 2022
- iThenticate Software: Jul 12, 2022 (5%)

ETYMOLOGY: Author Origin

AUTHOR DECLARATION:
- Financial or Other Competing Interests: None
- Was informed consent obtained from the subjects involved in the study? Yes
- For any images presented appropriate consent has been obtained from the subjects. Yes

Date of Submission: **Feb 13, 2022**
Date of Peer Review: **Mar 21, 2022**
Date of Acceptance: **May 18, 2022**
Date of Publishing: **Aug 01, 2022**