Section: Critical Care

Case Report

NON-ARTERITIC BILATERAL ISCHEMIC HYPOXIC OPTIC NEUROPATHY: A CASE REPORT OF POST CARDIOPULMONARY RESUSCITATION PATIENT

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Abstract

P Older adults with vasculopathy conditions are likely to be affected by non-arteritic ischemic optic neuropathy (N-AION), which is thought to be brought on by a reduction in blood supply to the optic nerve. We reported a case of a 63-year-old female patient brought to the hospital with chief complaints of vision loss following post-cardiopulmonary resuscitation. Considering the clinical examination and other investigations, we established the diagnosis of N-AION in both eyes. The patient received vasodilator therapy, antithrombotic, and neuroprotector treatment.

INTRODUCTION

An acute ischemia condition in the optic nerve is called ischemic optic neuropathy. There are two variants of ischemic optic neuropathy: Posterior ischemic optic neuropathy (PION) is brought on by ischemia in the vascularized posterior region of the optic nerve. The anterior section of the optic nerve, which is fed by the short posterior ciliary artery circulation, becomes ischemic and results in anterior ischemic optic neuropathy (AION). AION has two distinct etiological and pathogenetic subtypes: arteritic AION (A-AION), caused by giant cell arteritis, and nonarteritic AION (N-AION), caused by all other causes. N-AION often affects the inferior visual field, with vision loss manifesting over hours to days and frequently characterized as blurring, dimming, or cloudiness. N-AION normally does not cause pain; however, 8–12% of those who have it report experiencing some sort of periocular discomfort.[1]

According to estimates, there are 2.3–10.2 incidences of N-AION per 100,000 persons each year in the European population among individuals who are older than 50 years. However, a large Medicare database study found that the annual incidence was as high as 82 cases per 100,000 people, with an annual prevalence of 0.3%. According to the Beijing Eye Study, N-AION affects 1 in 45 000 people over the age of 40.[2]

The vision loss in N-AION normally worsens over the first two weeks and stabilizes in two to three months. After the vision loss has stabilized, there may be an improvement of three or more lines of visual acuity in 13-43 percent of the patients.[3] Acute vision loss can occur sometimes, and some patients report it as a sudden or fleeting fading or blurring of vision, especially in the center of the visual field. Optic disc edema, for example, can result in peripheral vision field abnormalities. Sector or widespread hyperaemic optic disc edema with or without retinal or optic disc hemorrhages are among the early clinical signs of N-AION.[4]

According to the available evidence, ischaemic optic neuropathy, particularly the N-AION, is a multifactorial disease. Each patient may have a special combination of systemic and local factors that all together may have caused the disease. Moreover, it should be emphasized that the management of N-AION remains mainly unsatisfactory due to its uncertain etiopathogenesis and pathophysiology. The present case study aims to highlight a rare case of N-AION who develop bilateral neuropathy following cardiopulmonary resuscitation.

CASE REPORT

The patient was a 63-year-old female brought to the hospital with chief complaints of vision loss over the last three days. Clinical examination revealed a history of an episode of elevated systolic blood pressure one week ago, for which the patient was taken to a local hospital. The patient suffered from a cardiac arrest for which three cycles of cardiopulmonary resuscitation were given to the

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patient. The patient was intubated and remained on mechanical ventilation for three days, after which the patient was stabilized and extubated. A few days following discharge, the patient developed vision loss which was partial in the beginning but progressed to complete blindness.

The patient had no relevant family history or ophthalmological afflictions but was a known case of hypertension, diabetes, atrial fibrillation, and pancreatitis. No significant personal history, family history, or occupational history was observed.

Pharmacologic dilation of the pupil revealed bilateral pupillary dilation characterized by no pupillary constriction to light. Visual acuity and perception of light were present. The optic nerve disc seemed enlarged, strongly suggesting papillary edema. The vessels were arranged in a concentric pattern, the retinal arteries were constricted, and the veins were turgescent. The macula showed no abnormalities.

The working diagnosis of Papillary Edema in both eyes was obtained based on clinical and paraclinical findings. The patient underwent additional testing to determine the cause of the condition and the best course of therapy. We also suggested a number of supplementary clinical, paraclinical, and laboratory studies. We establish the diagnosis as bilateral N-AION by considering all medical examinations. The patient received vasodilator therapy, antithrombotic, and neuroprotector treatment.

Magnetic resonance imaging revealed symmetrical areas of restricted diffusion in bilateral caudate and putamina with small patchy foci of restricted diffusion in the left parietooccipital lobes. 2D-ECHO revealed global hypokinesia of the left ventricle with global LVEF<30%. Follow-up 2D ECHO showed intact ventricular septum jerky and mildly hypokinetic. LVEF was > 50%. Grade 1 diastolic relaxation abnormality was present. No intracardiac clot/mass/pericardial pathology was observed.

HRCT chest highlighted mild bilateral pleural effusion with features likely of pulmonary edema with infective etiology and mild cardiomegaly. CT scan of the head and brain region showed chronic ischemic change and diffuse neuro parenchymal atrophy. Chest x-ray indicates prominent lung markings are noted in bilateral lung fields. CP angles and domes of the diaphragm are normal. Cardiac size and configuration are normal. Aortic knuckle calcification is seen. Trachea is central; no mediastinal shift is seen. The bony thorax and soft tissues of the chest wall are normal. USG carotid doppler showed no significant abnormality.

Biochemical investigation showed Hb-12.1, TLC-12.5, RBC count-4.59, Platelet count-338, Total protein-7.14, Albumin-3.6, Bilirubin (t)-0.72, AST-66, ALT-70, Alkaline phospahate-119, Urea- 38.8, Creatinine-1.00, Sodium-135.0, Potassium-4.20, T3-1.80, T4- 0.85, TSH-14.409, Homocysteine-6.7, Vit-B12>1500, Folate-9.7, Vit-D-18.76, Procal-0.289, HbA1c- 5.8, CKMB-2.3.

DISCUSSION

Although the precise pathogenesis is unknown, it is believed that N-AION results from a circulatory insufficiency of the posterior ciliary arteries supplying the optic nerve. Nearly all N-AION sufferers have at least one underlying vascular risk factor, which may or may not be known at the time of vision loss. Systemic hypertension, diabetes mellitus, and hyperlipidemia are just a few of the disorders that have been linked to N-AION and may increase the risk of reduced optic nerve head perfusion due to microvascular blockage.[1]

The N-AION was most common in the age group of more than 50 years. Previously Bordas et al. reported a case of a 52-year-old male who suffered from the N-AION in his left eye.[1] Golabchi et al. also reported a case of a 52-year-old male who suffered from AION in his left eye as a rare manifestation of COVID-19.[5] Mabaso et al. reported a case of a 56-year-old man who developed the N-AION.[2] Similarly, in our study also, the age of the patients was also more than 50 years.

Kang et al. reported a case of an 80-year-old female with a history of diabetes and hypertension presented with bilateral posterior AION. Similar to the present study, a diffusion restriction in the posterior portion of both optic nerves in the MRI scan was observed. The history of diabetes and hypertension was also a common parameter of our study.[6]

Although our understanding of the risk factors and clinical findings has improved, N-AION still has no known effective treatment. Aspirin to prevent N-AION in the opposite eye, steroid medication, and surgical decompression of the optic nerve were among the previously recommended therapies that were later proved to be unsuccessful.[7] Many medical professionals use aspirin as a treatment for N-AION and for its role in preventing stroke and coronary artery disease despite the lack of conclusive evidence. Some ophthalmologists and neurologists typically inform N-AION patients that there is nothing that can be done to treat their condition.[8] In order to reduce the chance of any more episodes in the afflicted eye or involvement in the unaffected eye, Hayreh thinks that the best course of action is to treat the risk variables, particularly nocturnal arterial hypotension. According to reports, after five years, there is a 5% probability of N-AION recurring in the damaged eye and a 15% chance that it would include the unaffected eye.[8] Unfortunately, in the present case, vision loss has occurred in both eyes.

CONCLUSION

Lack of perfusion to the optic nerve is thought to be the cause of N-AION. Optometrists may use the clinical symptoms and risk factors linked to this disorder to collect pertinent case histories and

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conduct thorough eye exams to differentiate this condition from other causes of vision loss. The risk of vision loss in the unaffected eye and recurrence or advancement in the afflicted eye may be decreased with early identification and prompt referral. Additional investigation is needed into the epidemiology of N-AION, particularly in low-income nations like India, where there is a dearth of information on this condition.

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