

Bilateral Simultaneous Non-Arteritic Anterior Ischemic Optic Neuropathy in Association with Alcoholic Fatty Liver Disease: A Case Report

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Abstract: We present a unique case of bilateral simultaneous non-arteritic anterior ischemic optic neuropathy (NAION) in association with alcoholic fatty liver disease in a 68 year old male. The patient presented with acute visual field loss and jaundice. The history revealed increased short-term alcohol intake. Eye examination, visual field testing and imaging were consistent with anterior ischemic optic neuropathy. Inflammatory markers were normal and there were no vascular risk factors. Liver function tests were acutely deranged and further investigations showed only fatty changes. Here we highlight the importance of alcohol intake enquiry in NAION patients, particularly those presenting with bilateral disease.

Keywords: non-arteritic, ischemic optic neuropathy, simultaneous, alcohol, fatty liver

1. Introduction

Bilateral simultaneous non-arteritic anterior ischemic optic neuropathy (NAION) is exceptionally rare and is usually found in association with severe hypotension resulting from blood loss.[1, 2]

2. CASE REPORT

A 68 year old male retired engineer presented to the eye clinic with a sudden onset inferior visual field defect in both eyes since waking. There were no associated headaches or jaw claudication. The patient had been feeling more lethargic over the past 10 weeks, had been passing dark urine and appeared to be slightly jaundiced in appearance. There was no history of vascular risk-factors such as diabetes or hypertension.

Distance visual acuity was 6/6 in the both eyes. Color vision testing was not possible due to a history of red/green colour-blindness. Temporal artery pulsation was strong and equal on both sides. Formal visual field testing revealed bilateral dense inferior and moderate superior field defects (figure 1 A and B). Dilated fundal examination revealed bilateral optic disc swelling and splinter haemorrhages (figure 1 C and D).

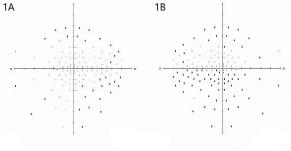






Figure1. Images A and B show 120-point screening visual fields tests of the left and right eye respectively. The patient demonstrated mainly inferior visual field loss. Images C and D show disc swelling and splinter haemorrhages in the left and right eyes respectively on first presentation. The appearance was consistent with acute anterior ischemic optic neuropathy.

Blood tests showed normal full blood count, ESR, CRP and coagulation profile. Given the clinical features, liver function tests were ordered. These were grossly deranged with bilirubin measuring 81 micromol/l, ALP 278 IU/L, AST 140 IU/L, ALT 64 IU/L and GGT 2903 IU/L. The results prompted an inpatient admission under the medical team. Further questioning revealed the patient had been

drinking approximately 16 units of alcohol day for the past year. Medical examination did not identify features of chronic liver disease. Investigations included a negative HIV antibody/p24 test, negative CMV IgM, negative Coxsackie virus PCR and negative Hepatitis A/C/E IgM. The Hepatitis B antibody profile conferred immunity. Nutritional deficiencies were excluded and homocysteine was normal. Ultrasound scanning of the liver showed fatty liver changes with no focal lesions or biliary abnormalities. A full body CT, liver MRI and magnetic resonance cholangiopancreatography showed only fatty nodular deposition in the liver with no evidence of malignancy. Carotid Doppler failed to identify a significant stenosis of either internal carotid artery.

The patient was ultimately given a diagnosis of alcoholic fatty liver disease with acute liver decompensation. On latest follow-up the patient's liver profile has improved but he has developed a right relative afferent pupillary defect and bilateral optic nerve pallor. The progression of his signs and symptoms, optical coherence tomography and visual fields were consistent with a diagnosis of bilateral simultaneous NAION. Formal electrodiagnostic testing identified a reduction of visual evoked potentials (VEP) from both eyes but no significant delay. Final central visual acuity remained 6/6 in both eyes.

3. DISCUSSION

NAION is thought to be multifactorial in origin. Typically the optic disc has a distinct small and crowded anatomy. Patients usually have vascular risk factors such as diabetes, hypertension and hyperlipidemia. No effective treatment exists although there is some evidence for the benefit of steroid treatment for these cases.[3] Most Bilateral simultaneous NAION is exceptionally rare and is usually found in association with severe hypotension resulting from blood loss and acute compromise to the optic nerve circulation.[1, 2]

The main differential that required exclusion in our patient was bilateral optic neuritis. Colorvision testing was not possible in our patient but the history of painless reduction in visual field, no pain on eye movements and no delay in VEPs was consistent with NAION.

NAION has Bilateral simultaneous not previously been reported in association with fatty liver disease and short term increased alcohol intake which were the only identifiable risk factors in our patient. There have been other documented associations between liver disease and the development of this condition. It may be seen as an extra hepatic manifestation of active viral and auto-immune hepatitis and its treatment which were ruled out on serological testing for our patient.[4]Longstanding alcoholic liver cirrhosishas been reported as a cause but our patient lacked these clinical features and did not have a long history of excessive alcohol intake.[5]

There is limited evidence on the mechanism of NAION in liver disease. Despite the wellrecognised bleeding tendency due to lack of coagulation products in end-stage liver disease, patients with liver dysfunction may develop a temporary hypercoagulable state due haemodynamic and secreted factor imbalances.[6] This may lead to acute ischemia of the optic nerves similar to the event that occurred in our patient. Given the association, alcohol intake enquiry in NAION patients is important. Further research is needed on the role anticoagulation or other preventative treatment in at-risk liver disease patients.

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Citation: Dr. SohraabYadav, Ms. Alison Rowlands, Bilateral Simultaneous Non-Arteritic Anterior Ischemic Optic Neuropathy in Association with Alcoholic Fatty Liver Disease: A Case Report. ARC Journal of Ophthalmology. 2018; 3(1): 14-16.

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