Case Report

Clarithromycin Culprit of Benign Intracranial Hypertension

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Abstract

Benign intracranial hypertension is characterized with increase in CSF opening pressure with no specific etiology. It is predominantly found in women of child bearing age and particularly in individuals with obesity. Visual disturbances or loss and associated headaches are common and can lead to blindness if left untreated. Diagnosis can be achieved once other causes of visual loss, headaches and high opening pressures are excluded. Management consists of serial optic disc assessments although no specific treatment is available despite recent trials using carbonic anhydrase inhibitors. Diet modification and weight management can help in therapy.

Key Words

Benign intracranial hypertension, blurred vision, clarithromycin, headache, psedotumor cerebri

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Ann Indian Acad Neurol 2015;18:475-477

Introduction

A 27-year-old lady presented to the hospital with headache, blurring of vision, and tunnel vision for 4 days. She gave a history of having flu-like illness 3 weeks previously. At the time of the flu-like illness, she did not have a headache, no blurring of vision, no neck stiffness, and no pyrexia She visited her general practitioner and was prescribed Clarithyromycin 500 mg once a day for 1 week. She was never prescribed Clarithromycin previously for any other illness. She noticed the headaches to begin about 5 days after the initiation of Clarithyromycin. The headache was frontal and gradually getting worse over the last 14 days along with visual symptoms. The blurring of vision and constricted field of vision started 2 weeks after the initiation of Clarithyromycin.

Ophthalmologic examination revealed papilloedema bilaterally and haziness of the nasal aspect of the optic nerve [Figures 1 and 2]. Visual acuity in both eyes was normal and visual field examination was normal []. No focal deficits were noted on detailed neurological examination. Computerized tomography (CT) of the brain was arranged, and no space-occupying lesions or any other cranial causes were located [Figure 3]. A lumbar puncture was performed showing an opening pressure of 42 mmHg and cytology was normal [Table 1]. Blood tests revealed normal full blood count, urea and electrolytes (U&E), and liver function tests (LFT) parameters. Erythrocyte sedimentation rate (ESR) was 11 and C-reactive protein (CRP) was < 10. Systemic examination was normal.

The diagnosis was of benign intracranial hypertension (BIH) was suggested and therapeutic lumbar puncture performed. She was commenced on Acetozolamide 250 mg twice a day for 4 weeks. On follow-up 8 weeks from discharge, her ophthalmology examination was normal. She did not have any headaches, or further blurring of vision on further routine follow-up with no medical treatment.

Table 1: Cerebro-spinal fluid (CSF) analysis

Analysis of CSF	CSF sample 1	CSF sample 2	CSF sample 3
Consistency	Clear	Clear	Clear
White cells	4	4	2
Red cells	10	1	1
CSF glucose	4.1		
CSF protein	0.2 g/L		
CSF culture	No growth		
CSF pressure	42 mmHg		

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DOI: 10.4103/0972-2327.165477

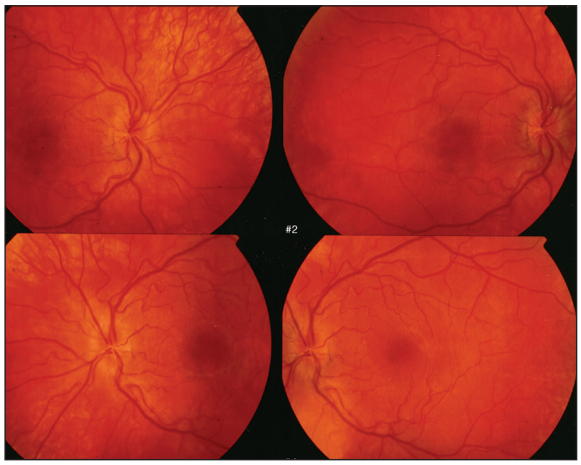

Figure 1: Fundoscopy of right and left eye

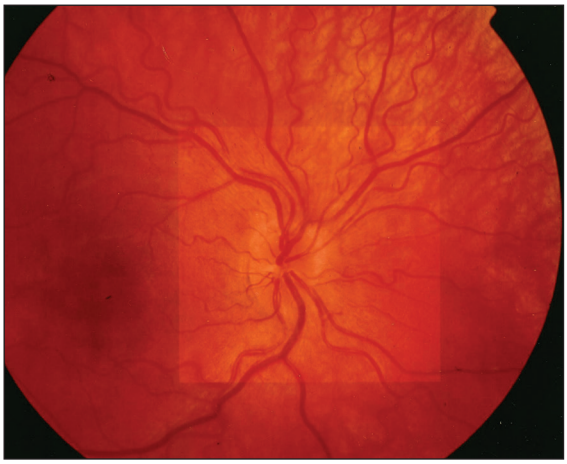

Figure 2: Severe papilloedema (highlighted area)

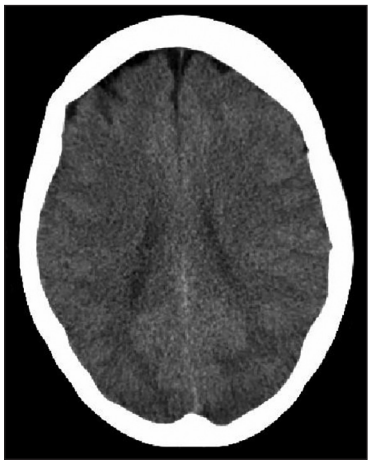

Figure 3: Computed tomography of brain

Discussion

BIH direct etiology is currently unknown. In normal patients, the cerebro-spinal fluid (CSF) production is equal to reabsorption rate. CSF is produced by four choroid plexuses located each ventricles of human brain. In patients with BIH, the rate is equal; however, a greater pressure is required against increased resistance at the Arachnoid granulations. The raised intracranial hypertension is transmitted to the optic nerve sheath. The raised pressure alters the flow in the optic nerve resulting in swelling of the nerve with leak of protein, water along with other cellular contents into the extracellular space. There are no race differences, and affects obese females, with highest incidence in child bearing ages[1,2,3,8,9,12].

The initial symptoms are of increased intra-cranial hypertension (ICP) and papilloedema. The symptoms of ICP include headache of various areas, non-specific type and frequency; diplopia due to opthalmoplegia most commonly due to cranial nerve VI (CN VI). Symptoms of papilloedema include transient visual defects mostly orthostatic; progressive loss of peripheral vision in any eye, initially of nasal side; blurring of central vision; and rarely sudden vision loss[4-8].

The important examinations are fundoscopy, visual fields, visual acuity, color vision, and ocular movements[1-12].

Differential diagnosis would include arteriovenous (AV) malformations, aseptic meningitis, hydrocephalus, intracranial abscess, intracranial hemorrhage, meningioma, Lyme disease, migraine headache, systemic lupus erythematosus (SLE), subarachanoid hemorrhage, malignant hypertension, diabetic papillopathy[9,12,13,14].

Diagnosis is by performing opthalmological examination, exclusion of differential diagnosis by neuroimaging, and lumbar puncture. Magnetic resonance imaging (MRI) is the neuroimaging modality of choice[14-19].

Treatment of benign intracranial hypertension is to remove any causative or associated factor. If no obvious cause is found, then pharmacological strategies have shown some benefit. In acute settings, particularly with visual disturbances, therapeutic lumbar puncture tap of about 20 ml improves acute symptoms. Pharmacological therapy consists of reduction in CSF production by carbonic anhydrase inhibitors, reduced salt intake, and sometimes diuretics are effective. Carbonic anhydrase inhibitors block bicarbonate ($HCO3^-$) formation by the enzyme carbonic anhydrase and reduce CSF production. Corticosteroids are effective but the mechanism is unknown[16-19]. Lumboperitoneal shunting is recommended if patients do not respond to pharmacological therapy. Regular follow-up is recommended with opthalmological examinations. Complication of most importance is optic atrophy if not cured at early presentation.

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How to cite this article: Khan HR, Mason C, Mulcahy R. Clarithromycin Culprit of Benign Intracranial Hypertension. Ann Indian Acad Neurol 2015;18:475-7.
Received: 26-08-14, **Revised:** 20-11-14, **Accepted:** 03-12-14
Source of Support: Nil, **Conflicts of Interest:** None declared