A case of blurry vision, headache and dizziness manifested as hypertensive retinopathy with papilledema in a patient with a recent diagnosis of peritoneal tuberculosis

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Case Report

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Abstract
Hypertension poses significant risks to various organ systems, including the eyes, leading to target-organ damage known as hypertensive retinopathy (HR). This case report explores the case of a 60-year-old male presenting with blurred vision, headache, and dizziness, ultimately diagnosed with grade IV hypertensive retinopathy. The patient had a complex medical history including hypertension, dyslipidemia, ischemic heart disease, diabetes mellitus, and peritoneal tuberculosis, with recent initiation of anti-tuberculosis therapy. Differential diagnoses encompassed various ocular and systemic conditions, highlighting the importance of a thorough evaluation. Fundoscopic examination revealed bilateral papilledema and flame hemorrhages consistent with hypertensive retinopathy. Management involved meticulous blood pressure control and ophthalmological referral. Collaboration between healthcare providers facilitated comprehensive care. Following treatment, the patient's blood pressure improved, necessitating adjustments in antihypertensive medications. This case underscores the critical role of recognizing ocular manifestations in hypertensive patients and the need for interdisciplinary management to mitigate systemic morbidity and mortality. Ongoing research is crucial to enhance diagnostic and therapeutic strategies for hypertensive retinopathy, ensuring optimal patient outcomes.

Introduction
Poorly controlled hypertension (HTN) exerts deleterious effects across various organ systems, including cardiovascular, renal, cerebrovascular, and ocular structures, collectively referred to as target-organ damage (TOD).

Ocular implications of HTN manifest as choroidopathy, retinopathy, and optic neuropathy. Hypertensive retinopathy (HR) ensues from elevated blood pressure, culminating in damage to retinal vessels. The severity and duration of hypertension correlate directly with HR incidence.

This case involves a patient with multiple comorbidities and medications that could lead to ocular manifestations, necessitating a thorough differential diagnosis to pinpoint hypertensive retinopathy resulting from uncontrolled hypertension.

Case History
A 60 year old male presented to the emergency department with complaints of blurred vision, headache, and dizziness.

History of presenting illness
The patient reports a sudden onset of bilateral blurry vision occurring five days before the initiation of a headache. The nature of the visual disturbance is continuous, with a slightly blurry effect on both near and far vision. Noteworthy is the absence of diplopia, visual distortions, eye pain, recent ocular trauma, blind spots, visual field defects, or colour blindness upon clinical examination.

The concomitant headache commenced on the same day of the presentation, developing gradually over six hours before the presentation. Characterized as a global headache with progressive pressure-like pain,
it exhibits radiation to the neck and is associated with nausea but no vomiting. Notably, the headache's severity escalated significantly in the two hours preceding the consultation, reaching a patient-reported score of 8/10. Light and noise exacerbate the headache (photophobia and phonophobia), with an absence of constitutional symptoms.

The patient has also experienced dizziness for the two days before presentation, marked by sudden, brief episodes triggered by movement, particularly upon standing, promptly relieved upon resuming a seated position. The severity of the dizziness is scored at 3/10, with an absence of constitutional symptoms.

Past medical history

The patient’s past medical history includes hypertension, dyslipidemia, ischemic heart disease, diabetes mellitus, and peritoneal tuberculosis. Diagnosed with diabetes nine years ago, recent HbA1c levels were recorded at 7%, with blood glucose ranging from 120 to 180 mg/dL. Home blood pressure readings average 160–180 mmHg systolic blood pressure. The patient was diagnosed with peritoneal tuberculosis with biopsy, after having constitutional symptoms of low-grade fever and night sweats for two months, for which the standard anti-tuberculosis therapy was initiated, and has been on them for the last three weeks, comprising Rifampicin, Isoniazid, Ethambutol, and Pyrazinamide. Concurrently managing cardiovascular and metabolic health, prescribed medications include Amlodipine, Perindopril, Atorvastatin, Aspirin, and Metformin. Dietary habits reveal a notable high intake of carbohydrates and salt. There is no history of prior surgery or allergies.
### Table 1
Review of Systems (Data obtained during investigation of the patient at International Medical Centre, Jeddah, Saudi Arabia)

<table>
<thead>
<tr>
<th>Central Nervous System</th>
<th>Cardiovascular System</th>
<th>Respiratory System</th>
<th>Urinary System</th>
<th>Gastro-intestinal Tract</th>
<th>Skin</th>
</tr>
</thead>
<tbody>
<tr>
<td>No slurred speech</td>
<td>No Paroxysmal nocturnal dyspnea (Heart failure)</td>
<td>No sleep disturbances (Obstructive sleep apnea)</td>
<td>No proteinuria</td>
<td>No increased abdominal girth</td>
<td>No flushed appearance</td>
</tr>
<tr>
<td>No focal weakness</td>
<td>No chest pain (Myocardial Infarction)</td>
<td>No shortness of breath (Flash pulmonary edema)</td>
<td>No oliguria</td>
<td>No abdominal pain</td>
<td>No rashes</td>
</tr>
<tr>
<td>No behavioral changes</td>
<td>No palpitations (Arrhythmia)</td>
<td>No flank pain</td>
<td>No constipation or diarrhea</td>
<td>No petechiae</td>
<td></td>
</tr>
<tr>
<td>No disturbance of gait</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>No abnormal movements (Convulsions)</td>
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</tbody>
</table>

**Physical exam findings**

Upon physical examination, the patient’s vital signs were heart rate – 90 bpm, respiratory rate – 20 breaths per minute, blood pressure – 220/110 mmHg, spo2 – 95% on room air, and blood glucose – 200 mg/dL, taken at random. While afebrile, the patient exhibited discomfort from the headache but showed no signs of respiratory distress. The patient remained conscious, alert, and oriented to time, place, and person, connected to a cardiac monitor with a large bore IV cannula in place. General examination was unremarkable hands with no nail changes, a strong and regular pulse, and no specific characteristics. The neck examination showed no lymph node enlargement or jugular venous distension. Eyes exhibited no signs of jaundice or pallor, with noted xanthelasma around the eyes. Oral hygiene was good, and respiratory, cardiac, abdominal, and musculoskeletal examinations revealed no significant abnormalities.

A differential diagnosis including hypertensive retinopathy, diabetic retinopathy, optic neuritis, space-occupying lesions, elevated intracranial pressure (ICP), bilateral central retinal vein occlusion (CRVO), optic disc vasculitis, and anterior ischemic optic neuropathy was postulated and a fundoscopic examination was requested. Fundoscopy showed bilateral papilledema and flame hemorrhages. The patient was scored as 15 on Glasgow coma scale. The cranial nerve examination was unremarkable. Motor function was assessed by power (5/5), tone (normal), and reflexes (normal). No abnormality in gait.
Diagnosis

The findings of fundoscopic examination coupled with the elevated blood pressure led to a diagnosis of grade IV hypertensive retinopathy.

Management

The patient was admitted to the ICU and initiated on nitroglycerin infusion, to reduce blood pressure by max 25% in the first hour to prevent coronary insufficiency and to ensure adequate cerebral perfusion pressure, and then to reduce the blood pressure to around 160/100–110 mmHg in the subsequent 2–6 hours. Reduction of the blood pressure to the patient's baseline was targeted in the next 24–48 hours. The patient was also referred to ophthalmology.

Follow-up results

After one week of discharge, the patient presented to the outpatient department for a follow-up. The blood pressure had improved when compared to the initial presentation to the ER. The dosage of anti-hypertensive medications was increased from Amlodipine 5 mg and Perindopril 5 mg to 10 mg each. Hydrochlorothiazide (25 mg OD) was also added.

Discussion

Hypertension can affect the eyes in various ways, including the development of retinopathy, optic neuropathy, and choroidopathy, with hypertensive retinopathy (HR) being the most common ocular manifestation.[3, 4] The harmful effects of increased blood pressure can cause not only hypertensive retinopathy but also worsen diabetic retinopathy and increase intraocular pressure.[5] The correct diagnosis and classification of the disease require a dilated fundoscopic exam and consideration of coexisting hypertension.[6] Ophthalmologists and primary care physicians should collaborate to efficiently screen and manage hypertensive patients, controlling blood pressure to reduce the risk of ocular and systemic morbidity and mortality.[7]

Declarations

Patient consent

The patient consented to the submission of this case report to the journal.

References


