

A Case of Hypertensive Retinopathy With Papilledema Manifested as Blurry Vision, Headache, and Dizziness in a Patient With a Recent Diagnosis of Peritoneal Tuberculosis

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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.

Keywords:- Hypertensive Retinopathy, Papilledema, Flame Hemorrhages, Visual Symptoms, Systemic Morbidity, Hypertension, Blurred Vision, Headache.

I. 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) [1]. Ocular manifestations of HTN include choroidopathy, retinopathy, and optic neuropathy [2]. Hypertensive retinopathy (HR) results from elevated blood pressure, culminating in damage to retinal vessels. The severity and duration of hypertension correlate directly with the incidence of HR [1]. 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.

II. CASE HISTORY

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

A. History of Presenting Illness

The patient reported 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. Notably, there was no diplopia, visual distortions, eye pain, recent ocular trauma, blind spots, visual field defects, or colour blindness upon clinical examination. Concomitant headache commenced on the same day of presentation and developed gradually over six hours before the presentation. Characterized as a global headache with progressive pressure-like pain, it involved radiation to the neck and was associated with nausea but not vomiting. Notably, the headache severity escalated significantly in the two hours preceding the consultation, reaching a patient-reported score of 8/10. Light and noise exacerbated the headache (photophobia and phonophobia), with an absence of constitutional symptoms. The patient 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 was scored as 3/10, with an absence of constitutional symptoms.

B. Past Medical History

The patient's past medical history included hypertension, dyslipidemia, ischemic heart disease, diabetes mellitus, and peritoneal tuberculosis. After the patient was diagnosed with diabetes nine years ago, his recent HbA1c level was 7%, and his blood glucose level ranged from 120 to 180 mg/dL. The average systolic blood pressure readings were 160-180 mmHg.

The patient was diagnosed with peritoneal tuberculosis via biopsy, after having constitutional symptoms of low-grade fever and night sweats for two months, for which standard antituberculosis therapy was initiated, and has been on them for the last three weeks, comprising of rifampicin, isoniazid, ethambutol, and pyrazinamide. Concurrently managing cardiovascular and metabolic health, prescribed medications include amlodipine, perindopril, atorvastatin, aspirin, and metformin. Dietary habits revealed a notably high intake of carbohydrates and salt. There was no history of prior surgery or allergies. A comprehensive review of systems was conducted to identify pertinent negatives, as outlined in Table 1.

C. Physical Exam Findings

Upon physical examination, the patient's vital signs were as follows: 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. 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, and was connected to a cardiac monitor with a large bore IV cannula in place. The general examination revealed 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 of hypertensive retinopathy, diabetic retinopathy, optic neuritis, space-occupying lesions, elevated intracranial pressure (ICP), bilateral central retinal vein occlusion (CRVO), optic disc vasculitis, or anterior ischemic optic neuropathy was postulated and a fundoscopic examination was requested. Fundoscopy revealed bilateral papilledema and flame hemorrhages. The patient was scored as 15 on the Glasgow Coma Scale. The cranial nerve examination was unremarkable. Motor function was assessed by power (5/5), tone (normal), and reflexes (normal). There was no abnormality in gait.

D. Diagnosis

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

E. Management

The patient was admitted to the ICU, after which nitroglycerin infusion was initiated to reduce his blood pressure by a maximum of 25% in the first hour to prevent coronary insufficiency and to ensure adequate cerebral perfusion pressure, subsequently, his blood pressure was reduced to approximately 160/100–110 mmHg in the subsequent 2-6 hours. A reduction in blood pressure to the patient's baseline was targeted within 24-48 hours. The patient was also referred for ophthalmology.

F. Follow-Up Results

One week after discharge, the patient presented to the outpatient department for follow-up. His blood pressure had improved when compared to the initial presentation to the ER. The dosage of antihypertensive medications was increased from 5 mg of amlodipine and 5 mg of perindopril to 10 mg each. Hydrochlorothiazide (25 mg OD) was also added.

III. DISCUSSION

Hypertension is widely recognized as a significant risk factor for various diseases collectively referred to as hypertension-mediated organ damage (HMOD), including stroke, disability, myocardial infarction, heart failure, kidney failure, and premature death [3]. The impact of hypertension extends to the eyes, where it induces a series of pathophysiological changes affecting the retinal, choroidal, and optic nerve circulations, leading to retinopathy, choroidopathy, and optic neuropathy, respectively. Among these ocular manifestations, hypertensive retinopathy (HR) emerges as the most prevalent [4]. The association between hypertension and retinal vascular abnormalities was first documented by Marcus Gunn in 1898. HR is characterized by retinal microvascular alterations that develop in response to elevated blood pressure, as defined by Wong and Mitchell [5]. The retinal microvascular signs observed in hypertensive retinopathy (HR) can stem from either an acute surge in systemic blood pressure or chronic, sustained hypertension. HR is linked with several underlying factors, including endothelial cell dysfunction, low-grade systemic inflammation, and oxidative stress [6, 7, 8].

Hypertensive retinopathy (HR) manifests through a range of discernible signs, including arteriolar constriction and tortuosity, vessel narrowing either generally or focally, and a reduction in the normal arteries to vein ratio. Alterations in arteriovenous crossings can be identified by various signs such as Salus's sign, Gunn's sign, and Bonnet's sign, each indicating distinct changes in retinal vascular anatomy. Moreover, HR often presents with accentuated light reflexes along vessel walls, microaneurysms, and various types of retinal hemorrhages including flame-shaped and dot-blot hemorrhages. Additionally, the presence of hard exudates and cotton wool spots are indicative of retinal damage associated with hypertension. In severe cases of malignant hypertension, optic neuropathy may occur, characterized by flame-shaped hemorrhages at the margin of the optic disc, optic disk swelling known as papilledema, congested retinal veins, and the presence of macular exudates

often accompanied by a macular star sign. Furthermore, hypertension can also instigate choroidopathy, a condition

particularly prevalent among younger patients afflicted with malignant hypertension [4].

Table 1 – Review of Systems

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

**Data obtained during the investigation of the patient at International Medical Centre, Jeddah, Saudi Arabia*

The Keith-Wagener-Barker (KWB) classification, introduced in 1939, was the first systematic approach to categorize hypertensive retinopathy (HR) based on observable retinal changes. It delineated HR into four distinct grades, each representing progressive stages of retinal vascular involvement due to hypertension. Grade 1 denoted generalized constriction and tortuosity of retinal arterioles, while Grade 2 included focal arteriolar narrowing accompanied by arteriovenous nicking. Grade 3 extended the classification to include additional features such as flame-shaped hemorrhages, cotton-wool spots, and hard exudates, indicating more advanced retinal pathology. Finally, Grade 4 represented the most severe stage, characterized by Grade 3 retinopathy along with retinal edema and/or papilledema [9].

Various methods for examining lesions in hypertensive retinopathy (HR) have been explored. While routine funduscopy has limitations due to observer variability and may not adequately detect subtle changes [10], studies suggest that semi-automated microdensitometry offers greater utility in diagnosing early vascular modifications [11]. Recent advancements in optical coherence tomography (OCT) have provided enhanced visualization of retinal microvasculature. Optical coherence tomography angiography (OCTA) enables non-invasive assessment of retinal microcirculation, offering valuable insights into vascular changes associated with hypertensive retinopathy [12]. Adaptive optics (AO) retinal imaging has emerged as a valuable tool for precise visualization of retinal vasculature, facilitating the assessment of subtle vascular modifications [13]. These advancements in imaging techniques hold promise for improved diagnosis and monitoring of hypertensive retinopathy.

IV. CONCLUSION

The retina, as a sensorineural tissue, is susceptible to the effects of high blood pressure [14]. Systemic hypertension continues to pose a significant public health challenge, exerting various effects on multiple bodily systems.

The eye stands as the sole organ where alterations in blood vessels resulting from systemic hypertension can be directly observed in vivo. This case highlights the intricate interplay between hypertension and ocular manifestations, emphasizing the significance of prompt diagnosis and management. The patient's presentation underscores the importance of comprehensive evaluation, including detailed history-taking and thorough physical examination, to elucidate underlying pathologies such as hypertensive retinopathy. It also underscores the importance of multidisciplinary care in optimizing patient outcomes and highlights the need for continuous monitoring and treatment adjustments to achieve optimal blood pressure control and prevent target-organ damage.

V. AUTHOR CONTRIBUTIONS

- Concept and design: Hashim Mohamed Siraj
- Acquisition, analysis, or interpretation of data: Hashim Mohamed Siraj, Bakr Faisal AbuSamrah
- Drafting of the manuscript: Hashim Mohamed Siraj, Bakr Faisal AbuSamrah
- Critical review of the manuscript for important intellectual content: Hashim Mohamed Siraj, Bakr Faisal AbuSamrah
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➤ Patient Consent

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