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Case Report

Terson's syndrome leading to fatal outcome in a 36-year-old woman: A case report ☆,☆☆

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ABSTRACT

Terson's syndrome occurs as a result of intraocular hemorrhage associated with intracranial hemorrhage, but was formerly used to describe vitreous hemorrhage associated with Aneurysmal Subarachnoid Hemorrhage (SAH). We present a case of a 36-year-old woman who was not a known hypertensive but presented with a sudden onset of loss of vision in both eyes and a few hours later became deeply unconscious. A computed tomography (CT) scan of the head revealed massive intracerebral hemorrhage with intraventricular extension secondary to severe hypertension and bilateral acute retinal hemorrhages due to the acute rise in intracranial pressure and the recently described ocular glymphatic system provides a novel perspective on the pathophysiology. A diagnosis of Terson's syndrome was made but unfortunately, her clinical condition deteriorated and she expired a few hours after the CT scan. Terson's syndrome is usually associated with poor clinical outcomes from increased intracranial pressure. Implication for clinical practice is that radiologists should critically examine the orbits during imaging for retinal hemorrhage in the setting of severe intracranial hemorrhage for the necessary ophthalmological and neurosurgical interventions to be made since most patients present with sudden onset of loss of vision.

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Introduction

Terson's syndrome is defined as intraocular hemorrhage associated with intracranial bleeding but was formerly used to describe vitreous hemorrhage associated with aneurysmal subarachnoid hemorrhage (SAH) [1]. It was first described by a German ophthalmologist Moritz Litten in 1881, however, Albert Terson, a French ophthalmologist was credited for first eliciting this sign in 1900 in a patient with subarachnoid hemorrhage [2]. Currently, Terson's syndrome corresponds to the association of any type of intracranial hemorrhage with intraocular hemorrhages like preretinal, retinal, or subretinal and vitreous hemorrhage [3].

The suspected pathophysiology for Terson's syndrome is a sudden increase in intracranial pressure which leads to rapid effusion of Cerebrospinal fluid (CSF) into the optic nerve sheath causing dilatation of the retrobulbar portion of the optic nerve sheath and retinochoroidal veins, pressure on the central retinal vein and resultant venous hypertension and rupture of the retinal vessels [4]. Although the pathophysiology of Terson's syndrome remains controversial, the syndrome has been reported to be associated with conditions like SAH which cause a rapid increase in intracranial pressure [5].

Terson's syndrome is not an uncommon condition, perhaps it is being underdiagnosed [6]. The presence of vitreous and preretinal hemorrhage without associated cerebral hemorrhage creates challenges for ophthalmologists and neurosurgeons and reduces the suspicion of Terson's syndrome [7]. The reported incidence of Terson's syndrome is 12.5%-40.0% [8]. Recent studies have highlighted the need for increased awareness and clinical suspicion of Terson's syndrome. As per a systematic literature review conducted by Aboulhson et al. [4] in 2021, Terson syndrome tends to be frequently overlooked and underdiagnosed. This is attributed to the fact that routine ophthalmologic examinations are not consistently conducted, and some patients are unable to communicate their visual complaints due to their underlying medical conditions. This assertion is corroborated by a study conducted by Lima-Fontes et al. [8] in 2023, wherein the authors underscored that the diagnosis of Terson's syndrome commonly encounters delays. This delay is attributed to the concurrent presence of neurological deficits and a compromised conscious state in affected individuals. These factors restrict the ability to conduct a formal assessment of visual acuity loss.

We present a case of Terson's syndrome in a 36-year-old woman found on head CT scan with emphasis on diagnosis and under diagnosis, since this subtle but crucial finding could be easily missed on routine imaging. By addressing this gap in awareness, our case contributes to a comprehensive understanding of Terson's syndrome and emphasizes the importance of vigilance in its identification in clinical practice.

Case presentation

A 36-year-old woman who was not a known hypertensive presented with sudden onset of visual loss in both eyes and few hours later became unconscious and was rushed to the emergency room of the Korle Bu Teaching Hospital. On admission the blood pressure was very high 210/132 mm Hg, pulse was 98, and respiratory rate 13 breaths per minute. Oxygen saturation was normal. Glasgow's coma score was 3 /15. Urgent head CT scan was requested and done at the radiology department. The noncontrast head CT scan revealed a fairly-defined area of hyperdensity of acute blood attenuation in the left basal ganglia with associated perilesional edema, compression of the third ventricle, and extension of the hemorrhage into the third and the anterior horn of the left lateral ventricle with resultant acute obstructive hydrocephalus (Fig. 1).

Also visualized was focal hyperdense lentiform or nodular collection of acute blood attenuation on the retina of the right and left globes in keeping with Terson's syndrome (Fig. 2). The brainstem and cerebellum were normal. Ophthalmological and neurosurgical evaluation could not be done because unfortunately, the patient expired a few hours (3 hours) after the head CT scan. We are reporting this case to bring to the fore the importance of critical evaluation of the orbits when radiologists are confronted with intracranial hemorrhage as this can easily be missed on imaging even though ophthalmological and neurological intervention can improve the condition of the patient.

Discussion

Terson's syndrome is a relatively common complication of SAH that may require early surgical intervention to prevent long term loss of vision. The incidence of Terson's syndrome varies from 8% to 46% as a result of differences in the diagnostic method. The frequency of Terson's syndrome is relatively high and yet 77% of such cases are overlooked in daily reports [8–10]. In a systematic review of 154 scholarly works on the incidence of Terson's syndrome, McCarron et al. [11] reported a 13% frequency among patients studied prospectively and 3% studied retrospectively. A study done by Czorlich et al. [12] reported an incidence of Terson's syndrome in 19% of patients with SAH, 9% in patients with intracerebral hemorrhages, and 3% in patients with traumatic brain injury. Despite the relatively high incidence of Terson's Syndrome in patients with SAH, the syndrome remains underdiagnosed.

The exact etiology remains unknown. However, an association with Terson's syndrome has been supported by raised intracranial pressure by Czorlich et al. [12] who found that Terson's syndrome patients were more likely to have periods of increased intracranial pressure more than 25 mm Hg. There has not been a definitive conclusion concerning the pathophysiology of this condition. However, in majority of cases, the increased intracranial pressure may result in retinal venous hypertension of the retinal vessels and hemorrhage [13]. The recently described ocular glymphatic system provides a novel perspective on the pathophysiology of Terson's Syndrome [14]. Drainage from the globe into intracranial glymphatics is dependent on the pressure gradient between intraocular and intracranial pressure. This glymphatic pathway serves as the sole extravascular conduit connecting the subarachnoid space to the retina. The mechanism proposes that subarachnoid blood in skull base cisterns near the optic nerve

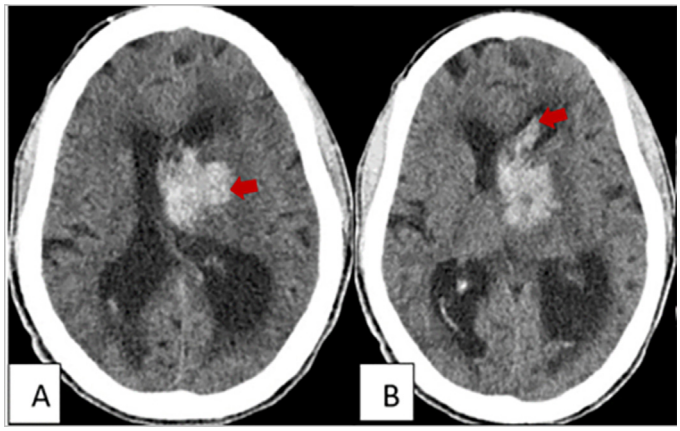

Fig. 1 – Non contrast axial CT Scan of the head revealed a fairly-defined area of hyperdensity of acute blood attenuation in the left basal ganglia with associated perilesional edema, compression of the third ventricle, and extension of the hemorrhage into the third and the anterior horn of the left lateral ventricles (arrows) with resultant acute obstructive hydrocephalus. There is an associated midline shift to the contralateral side (A and B).

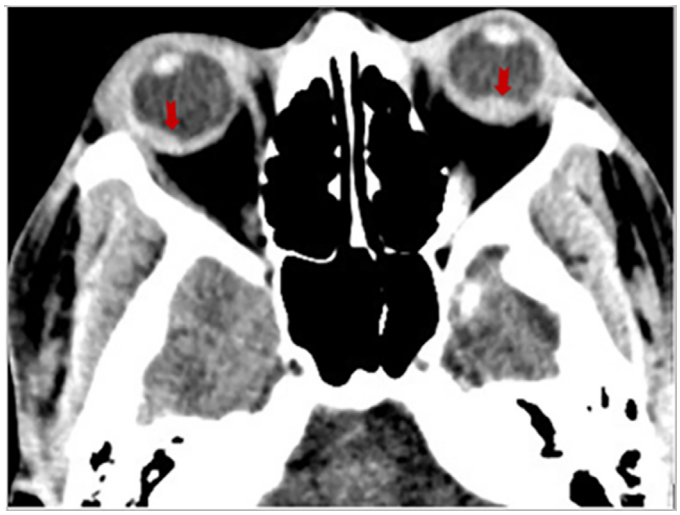

Fig. 2 – Non contrast axial CT scan of the head revealed a fairly-defined focal hyperdense lentiform collection of acute blood attenuation on the retina (red arrows) of the right and left globes in keeping with Terson's syndrome.

serves as the substrate for blood. Raised intracranial pressure causes reflux of this blood through glymphatic channels into the globe, ultimately leading to intraocular hemorrhage [14].

Different forms of intraocular hemorrhage have different prognoses. Minimal retinal hemorrhage has a better prognosis than vitreous or large pre-retinal hemorrhages and intracranial hemorrhage is more massive in patients with bilateral retinal hemorrhages than in patients with unilateral retinal hemorrhage [11]. Our patient had massive intracerebral hemorrhage, and bilateral retinal hemorrhage, and the prognosis was poor. In general, vitreous hemorrhage is known to be a factor of poor prognosis in patients with subarachnoid hemorrhage [14,15]. Hence, Terson's syndrome is highly correlated with morbidity and mortality when compared with intracerebral hemorrhage or SAH without retinal hemorrhage, this was the scenario in our patient.

The symptoms of Terson's syndrome largely depend on the extent and site of the hemorrhage and the neurological state of the patient. Our patient had a massive hemorrhage in the basal ganglia with intraventricular extension and hence had a severe neurological deficit. The majority of patients with severe neurological deficits may not be able to communicate or perceive visual problems [5,15]. Funduscopy is being regarded as the standard for detecting and diagnosing Terson's syndrome but can be challenging when the view is obscured with vitreous hemorrhage [16] therefore imaging plays a significant role. Our patient could not undergo fundoscopy since she, unfortunately, expired just a few hours after the head CT Scan. A decreased red reflex seen during fundoscopy is helpful in evaluating a patient in a coma, and B-scan ultrasonography can also establish the degree of intraocular hemorrhage. Other modalities like head CT scan findings can be used in making the diagnosis. In this case report, we based our diag-

nosis on the head CT scan findings. For instance, in a study of patients with SAH, a B-scan was 100% sensitive and specific in determining vitreous or pre-retinal hemorrhages and 44% sensitive in identifying intra-retinal hemorrhages whilst a head CT scan was 60% sensitive and 96% specific in identifying preretinal hemorrhages [17]. Thus, prior to an eye exam, a head CT scan may be useful in identifying possible Terson's syndrome and also help in diagnosing associated intracranial hemorrhage. Findings on CT scans can be very subtle and may show thickening, nodularity, or crescent-shaped hyperdensity of the surface of the retina compared to the vitreous body usually on the lateral portion of the retina and close to the optic nerve and these findings can be visualized during the first few days after intracranial hemorrhage [13]. Our patient had hyperdense lentiform collection of acute blood attenuation on the retina bilaterally. Volume averaging at the insertions of the rectus muscles and tangential sectioning of the inferior and superior portions of the eye ball can pose diagnostic challenges. Thus, a crescentic or lentiform hyperdensity can be mimicked.

The differential diagnosis includes metastasis, melanoma, hemangiomas, and hypertensive retinopathy. Metastasis is typically associated with a known history of primary malignancy, exhibits solid lesions on imaging. On CT scan, melanoma appears hyperdense and shows mild enhancement after intravenous administration of iodinated contrast medium. Hemangioma on Magnetic Resonance Imaging (MRI) and CT scan reveals an enhancing retinal mass with extensive retinal detachment. Hypertensive retinopathy, however, will show on funduscopy as spots near the optic nerve head, peripapillary region nerve fiber hemorrhages, macula lipid exudates, macular edema, and retinal hemorrhages. In cases of intracerebral hemorrhage or SAH, Terson's syndrome emerges as the most likely diagnosis, warranting a comprehensive diagnostic approach to differentiate it from these potential mimics [13].

Whenever a diagnosis of Terson's syndrome is made, vigilant monitoring becomes paramount. Regular ophthalmological assessments, including funduscopy and imaging studies, are essential for tracking the progression of intraocular hemorrhage. Considering the association with acute intracerebral hemorrhage, monitoring intracranial pressure may guide treatment decisions. Neurosurgical interventions, such as hematoma evacuation, and ophthalmological interventions, like vitrectomy, may be warranted based on the severity of the hemorrhage [18].

Conclusion

Acute retinal hemorrhage may not be appreciated clinically in very ill patients presenting with severe massive intracerebral hemorrhage, therefore retinal hemorrhage in both globes should be sought for on head CT scan by radiologists for the necessary interventions to be made since this subtle but crucial finding could be missed on imaging. The occurrence of Terson's syndrome is closely associated with poor clinical outcomes from the rapid increase in intracranial pressure.

Ethical consideration

The nuclear family of this patient gave informed consent and patient confidentiality was maintained

Authors' contribution

All authors contributed equally to the conception, design, drafting and revision of the manuscript and approved the final draft for publication.

Patient consent

Informed consent was obtained from the nuclear family of this patient.

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