



Case report

Terson syndrome: Two case reports

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ABSTRACT

Purpose: Vitreous or retinal hemorrhage occurring in association with subarachnoid hemorrhage (SAH) is known as Terson syndrome (TS). Its incidence is of the order of 10–50% after subarachnoid hemorrhage. We report two cases of TS with different clinical presentations, different managements, and a good final prognosis, which were managed at the Ibn Rochd hospital in Casablanca.

Observations: We report two cases of TS. The first one occurred in a 55-year-old hypertensive man following a ruptured aneurysm of the internal carotid artery, revealed by a decrease in visual acuity concomitant with the neurological symptomatology. The evolution was favorable after specialized neurological management and spontaneous resorption of the intravitreal hemorrhage was obtained 3 months after the acute episode with good visual recovery. The second case was that of a 45-year-old man who presented with an intravitreal hemorrhage of the right eye, 24 h after severe head trauma responsible for a bifrontal extradural hematoma. The B-mode ocular ultrasound discovery of a retinal detachment with persistent hemorrhage led to the indication of a pars plana vitrectomy with retinal tamponade, which allowed visual rehabilitation without recurrence after 1 year.

Conclusions: Ophthalmologic evaluation by specialized examination and radiology (ocular ultrasound, OCT) is necessary for all patients with TS because early diagnosis and treatment can prevent visual loss and associated complications. Treatment of TS can be conservative and based on periodic monitoring. A pars plana vitrectomy is considered in some cases.

1. Introduction

Terson syndrome (TS) is defined as an intraocular hemorrhage associated with intracranial hemorrhage and was first described by the French ophthalmologist Albert Terson in 1900 [1]. The incidence of TS is variably described in the literature and is in the order of 10 to 50% after subarachnoid hemorrhage [2,3]. The exact pathophysiology of Terson syndrome is not yet well known, but several hypotheses are discussed. A rupture of the peripapillary and retinal capillaries secondary to an abrupt increase in intravascular pressure following a sudden intracranial hypertension event is often suggested [2,3]. The latest studies speak of a mechanism: the theory of lymphatic reflux [4].

Currently, this syndrome corresponds to the association of any type of intracranial hemorrhage with intraocular hemorrhage, including subretinal, retinal, pre-retinal, and vitreous hemorrhages [5]. Clinically, it is an attack made of neurological syndromes according to the type of

lesion, associated with a decrease in visual acuity [2,5]. Its management is multidisciplinary, consisting first of neurosurgical management to treat the brain damage in order to improve the vital prognosis and then, after stabilization, to treat the intraocular hemorrhage, which usually evolves spontaneously, giving a good visual prognosis. Otherwise, a pars plana vitrectomy is considered and should be performed earlier [6].

We report two cases of ST with different clinical presentations that were managed at the Ibn Rochd hospital in Casablanca. We will compare our cases to those reported in the literature while reviewing the different clinical and evolutionary aspects as well as prognostic factors.

2. Case reports

2.1. Case report 1

The patient was 55 years old and had been known to be diabetic for

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16 years on a diet alone and hypertensive for 5 years on beta-blocker monotherapy. He had been suffering from intense headaches with photophobia for 48 h. The situation was aggravated by the appearance of incoercible vomiting with a sudden drop in visual acuity in the right eye, without pain, leading to a consultation in the emergency room. The neurological examination showed a Glasgow score of 13/15, an apyretic meningeal syndrome, and a right hemiparesis with slight motor aphasia. The emergency CT scan revealed a spontaneous meningeal hemorrhage due to a ruptured aneurysm of the right internal carotid artery at the level of its supra-clinoid segment (Fig. 1). Complementary cerebral MRI confirms the subarachnoid hemorrhage and the appearance of the ruptured aneurysm. The patient was managed neurosurgically the same day. The aneurysm was treated by embolization with success. On the second postoperative day, he underwent an ophthalmological examination of the right eye, which showed visual acuity of 6/20 without correction, normal ocular tone, a normal anterior segment, and on the fundus, a stage 2 intravitreal hemorrhage organized inferiorly, with a flat retina superiorly. A B-mode ocular ultrasound confirmed the intravitreal hemorrhage and did not find an associated retinal detachment (Fig. 2). The examination of the left eye was unremarkable. Given the cerebral and ocular involvement, the diagnosis of Terson syndrome was made. The ophthalmological management consisted of medical treatment with hygienic and dietary measures, strict rest with abundant drinks, and periodic follow-up to monitor the disappearance of the vitreous hemorrhage and thus the improvement of visual acuity. Neurologically, the evolution was favorable, with progressive improvement of his clinical condition. The control cerebral scanner, carried out in slices in the different planes, without injection, at D10 post-operation showed no bleeding and no endocranial complications of the operation. The patient was declared discharged and was followed up in ophthalmologic consultation every week with visual acuity measurement and fundus examination. During the second postoperative month, the visual acuity of the right eye began to improve to 15/20, and the fundus examination showed regression of the intravitreal hemorrhage. After 3 months, the hemorrhage was completely cleaned up, with visual acuity up to 18/20 without correction. No recurrence or neurological or ocular complications after 1.5 years.



Fig. 1. Cerebral scanner in axial slices, after injection, showing an Aneurysm of the internal carotid artery in the supra-clinoid segment.

2.2. Case report 2

The patient was 45 years old and had no history of head injury prior to being involved in a traffic accident with a right frontoparietal impact point. He was a cyclist who was hit by a motorcycle. Following the accident, the patient reported an initial loss of consciousness with vomiting. He consulted the emergency department of the Ibn Roch hospital in Casablanca, then was admitted to the neurosurgical emergency room, and the clinical neurological examination revealed a Glasgow score of 14/15 with a frontal syndrome without sensory-motor deficit. He was hemodynamically stable. The CT scan performed in the emergency room revealed the presence of a bi-frontal extradural hematoma (Fig. 3). The rest of the workup did not reveal any other lesions. The patient was hospitalized and managed neurosurgically in the neurosurgical intensive care unit. During his hospitalization, the patient experienced a sudden decrease in visual acuity, without pain in the right eye, on Day 1 (24 h) of his accident. The ophthalmological examination revealed a distance visual acuity of 1/10 in the right eye without correction and 6/10 in the left eye. The ocular tone was normal at 14 mmHg in both eyes. The anterior segment was normal. Fundus analysis on the right eye revealed a stage IV intravitreal hemorrhage (Fig. 4). This was confirmed by ocular ultrasound, which did not reveal any retinal or choroidal detachment. The examination of the adelpic eye fundus was without abnormality. The initial therapeutic attitude consisted of strict rest, good rehydration, and daily monitoring of visual acuity and fundus. Neurologically, the evolution was very favorable, with a Glasgow score of 15/15 and the resorption of the extradural hematoma on the follow-up CT scan after 2 weeks. The patient was declared discharged and followed up on an outpatient basis. Ophthalmologically, the visual acuity remained stable with the persistence of the intravitreal hemorrhage after one month of follow-up. Ocular ultrasound examination showed a dense intravitreal hemorrhage that had become organized, with doubt about a retinal detachment (Fig. 5). A pars plana vitrectomy under general anesthesia was indicated. The procedure consisted of a total vitrectomy that revealed a partial temporal retinal detachment without macular detachment and visible dehiscence. Reapplication of the retina was performed with an endolaser barrier and completed by buffering with C2F6 gas diluted to 17%. The final visual acuity was 5/10 at 3 weeks post-operatively, which was maintained for one year of follow-up without recurrence or complications.

3. Discussion

Terson syndrome (TS) is defined as an intraocular hemorrhage associated with intracranial hemorrhage [1]. The exact pathophysiology of Terson syndrome is not yet known, but several hypotheses are discussed [2]. A rupture of the peripapillary and retinal capillaries secondary to an abrupt increase in intravascular pressure following a sudden intracranial hypertension event is often suggested [2,3,7]. The pathogenesis of the various reported cases of intraocular hemorrhage is thought to be the result of altered circulation in the central nervous system in conjunction with disturbed circulation in the retinal vessels, leading to retinal venous hypertension and ultimately hemorrhage [8].

But the last study by Ashwin Kumaria [4] reports the possibility of “the glymphatic reflux theory”. A new ocular glymphatic system has recently been described, the drainage of which from the globe into intracranial glymphatics is reliant on the pressure gradient between intraocular pressure and intracranial pressure. The glymphatic pathway is the only extravascular anatomical conduit between the subarachnoid space and the retina [4].

According to various studies, there are multiple causes and different clinical presentations are possible [4,5,8]. Terson Syndrome is often observed after rupturing of an aneurysm of the anterior circulation, especially of the anterior communicating artery or the internal carotid artery [9], and this was the case in our first patient who presented with a ruptured aneurysm of the internal carotid artery at the level of its

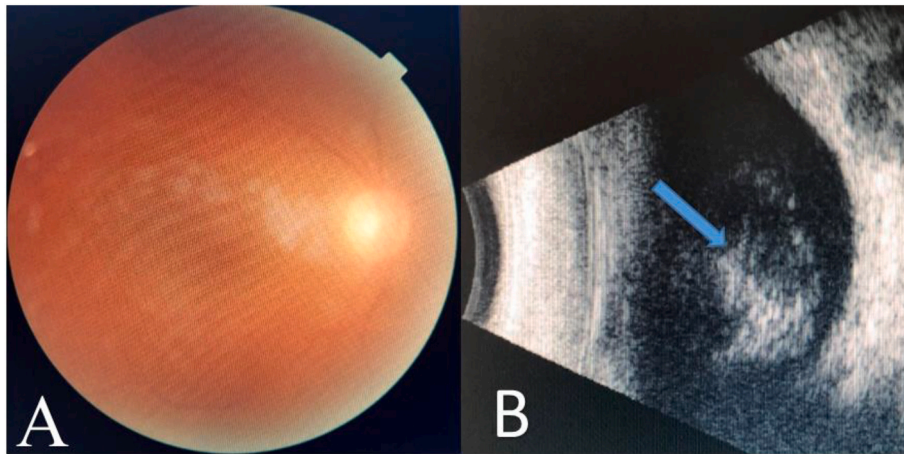


Fig. 2. : stage II intravitreal hemorrhage (A) and its appearance on B-mode ocular ultrasound showing dense, inferiorly organized intravitreal hemorrhage without retinal detachment (B).

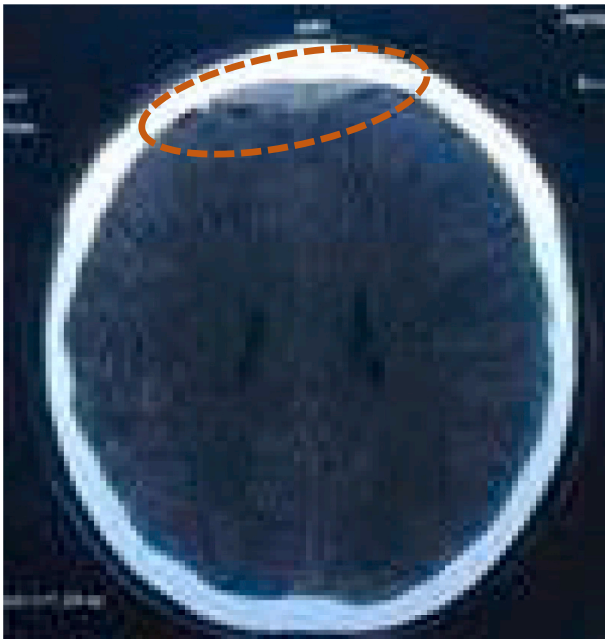


Fig. 3. : Cerebral CT-scan, without injection, in axial sections, showing a bifrontal extradural hematoma.

supraclinoid segment. Other rare causes include aneurysms of the posterior circulation or aneurysmal rupture of the vertebral artery [10], subdural hematoma, and post-traumatic bleeding with severe brain damage [11], as in the case of our second patient, where Terson syndrome followed a bifrontal extradural hematoma of post-traumatic origin. The hypothesis of increased pressure in post-traumatic intracranial and intracerebral abnormalities would explain the occurrence of intravitreal hemorrhage in our second patient. As an interrelated mechanism in this kind of case, the possible alteration of peripapillary structures via the intervaginal space of the optic nerve sheath [12]. However, no correlation has been established between the type of intracranial hemorrhage and the type of intraocular hemorrhage.

The therapeutic management of the TS depends on the location of the intraocular hemorrhage. The clinical course of this hemorrhage is variable. While in some patients, the hemorrhage resolves spontaneously, many will experience vision loss, chronic hematoma, or epiretinal membranes requiring ophthalmologic care, including vitrectomy [2,8]. In one study, it was also suggested that different types of intraocular

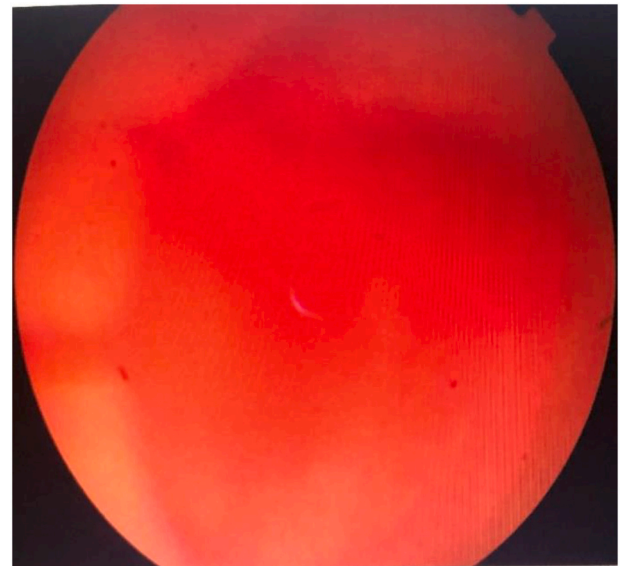


Fig. 4. : intravitreal hemorrhage stage 4 on fundus: presence of massive blood in the vitreous without individualization of the fundus elements.

hemorrhage had different prognostic significance, in that mild retinal hemorrhages were more strongly associated with a better prognosis than large preretinal hemorrhages or vitreous hemorrhages [13]. Most intravitreal hemorrhages resolve spontaneously [14]. Only 40% of cases require vitrectomy, and only half of these also require internal limiting membrane detachment. Treatment can be conservative based on periodic monitoring, i.e., careful evaluation with repeated ophthalmologic examinations, pending spontaneous resolution. Elevation of the head with bed rest, taking copious amounts of fluids, and avoiding anticoagulant medications may be beneficial to patients. Usually, spontaneous resorption occurs over several months. This was the case in our first patient, whose evolution was favorable with spontaneous regression after 3 months, probably favored by the relatively small intravitreal hemorrhage but probably also by the good neurosurgical management of his responsible brain lesion. The main monitoring parameters are visual acuity and the evolution of the intraocular hemorrhage, but above all, the early detection of the appearance of other elements compromising the visual prognosis. Monitoring is then clinical but also radiological by ocular ultrasound or even other explorations such as OCT. [2,14] If the hemorrhage does not resolve or does not progress well, a



Fig. 5. B-mode ultrasound image of a total IVH with suspected retinal detachment.

pars plana vitrectomy is indicated, especially for intravitreal hemorrhage [5,14]. However, a surgical approach is not possible in the presence of intraretinal hemorrhage [15]. It should also be noted that immediate vitrectomy for intraocular hemorrhage is not recommended, except in cases of sub-macular hemorrhage, binocular damage with severe visual loss, or in pediatric patients at risk of amblyopia [5]. Thus, the authors agree on a spontaneous resolution time of 6 to 12 months before considering vitrectomy, although there is current research that states that vitrectomy should be performed earlier [6]. Also at follow-up, persistent vitreous hemorrhage, epiretinal membrane formation, or the occurrence of retinal detachment may result in significant vision loss. In such cases, vitrectomy can improve the visual outcome. In our second patient, the indication for vitrectomy was based on the persistence of intravitreal hemorrhage with suspicion of retinal detachment on ultrasound and a poor evolution of visual acuity during follow-up. This was the right course of action because the operation allowed the management of the retinal detachment and restored better visual acuity.

Terson Syndrome is a rare but often under-diagnosed entity. Therefore, any intracranial hemorrhage should be investigated for intraocular hemorrhage, especially in the presence of warning signs. The treatment remains relatively simple, ranging from surveillance to vitrectomy. Ophthalmologic and radiologic evaluation is necessary for all patients with TS because early diagnosis and treatment can prevent visual loss and associated complications.

Our work consists of two cases case report and has been reported in accordance with SCARE 2020 criteria [16].

4. Conclusion

Terson's syndrome is a complication of cerebral-meningeal bleeding, whether caused by aneurysm rupture or traumatic brain injury. Its pathogenesis is becoming better understood, and the most recent notion of glymphatic reflux appears to be promising.

In the absence of further difficulties and in the case of prompt and proper treatment, the prognosis is still favorable.

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Ethical approval

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Consent

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CRedit authorship contribution statement

Moctar ISSIAKA: as first and corresponding author made substantial contributions to the conception, the design of the work, the acquisition, analysis and interpretation of data.

Adil MCHACHI: the design of the work, analysis and interpretation of data.

Ismael MAHAZOU: writing the paper, analysis and interpretation of data.

Maimouna BANAQ: the acquisition, analysis and interpretation of data, writing the paper.

Rayad RACHID: made substantial contribution to the design and the final revision of the work.

Mohamed EL BELHADJI: made a significant contribution to the final revision of the paper and approved it.

Declaration of competing interest

Authors of this article have no conflict or competing interests. All of the authors approved the final version of the manuscript.

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