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Terson's Syndrome: Case Report

Adil Elkhoyaali^{1*}, Imane Jeddou¹, Ilias Benchafai², Jawad Laaguili³, Yassin Mouzarii¹, Karimreda¹ and Abdelbaroubaaz¹

¹Service D'ophtalmologie, Hopital Militaire D'instruction Mohammed V, Rabat, Morocco.
²Service D'oto-Rhino-Laryngologie, Hopital Militaire D'instruction Mohammed V, Rabat, Morocco.
³Service De Neurochirurgie, Hopital Militaire D'instruction Mohammed V, Rabat, Morocco.

Authors' contributions

This work was carried out in collaboration among all authors. Author AE designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors IJ, IB and JL managed the analyses of the study. Author YM managed the literature searches. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Introduction: Terson's Syndrome, defined since 1900 as the association of vitreous or retinal hemorrhage with subarachnoid hemorrhage, has been extended to include all intraocular hemorrhages secondary to other causes acute intracranial hypertension.

Observation: We report the observation of a 50-year-old woman who presented with bilateral Terson's syndrome following a rupture of a cerebral aneurysm.

The patient was admitted for disturbance of consciousness and neurologic deficit. Ophthalmoscope examination of the fundus of the eye found pre, intra, and subretinal hemorrhages in the left eye masking the papilla and macula and a small hemorrhagic spot in the pre-retina right eye. This aspect motivated the performance of an angio-MRI which revealed a subarachnoid hemorrhage secondary to the rupture of an aneurysm. The treatment consisted of embolization by coils by arteriography. The evolution was marked by the resorption of the subarachnoid hemorrhage and ocular hemorrhages with good visual recovery: 1 in the right eye and 0,8 in the left eye.

Discussion: The pathophysiology of Terson's syndrome is very controversial. It is associated with SAH in 2 to 27% of cases. Most of these hemorrhages are due to ruptures of aneurysms located in the lower part of the polygon of Willis. TS retinal hemorrhages can sit under, intra or pre-retinal, or

diffuse in the vitreous and are easily detected by an examination of the fundus. Vitrectomy allows rapid visual recovery when spontaneous resorption is not obtained after a period of observation. **Conclusion:** The early diagnosis of TS in all cases of SAH and IH will improve diagnostic and therapeutic management, and therefore the visual and vital prognosis of these patients. Hence the value of an examination of the fundus in any patient with disturbance of consciousness without obvious etiological context.

Keywords: Syndrome; terson; sub arachnoid hemorrhage; cerebral aneurysm; retinal hemorrhage; vitreous.

1. INTRODUCTION

Terson's syndrome (TS) is usually associated with intracranial hemorrhage. In the majority of cases, it takes the form of an intravitreal hemorrhage combined with subarachnoid hemorrhage (SAH) in the setting of an aneurysmal rupture.

We present the case of Terson's syndrome occurring in a woman with subarachnoid hemorrhage from a ruptured aneurysm, suggested by the discovery of bilateral retinal hemorrhages in the fundus which gradually resolved.

This work aims to present the physiopathological and therapeutic features of Terson's syndrome and demonstrate the benefit of the fundus examination in an unconscious patient admitted to the emergency room.

2. CASE REPORT

We present the case of a 50-year-old patient with no notable pathological history; admitted to the emeraencv room for disturbances of consciousness with generalized convulsions; general examination objectified meningeal syndrome afebrile with a Glasgow coma score (GCS) = 10/15. Suspecting intracranial hypertension, a fundus examination was requested and it

revealedbilateral pre, intra, and subretinal hemorrhages. The optic papillae were normal.

This funduscopic aspect motivated the realization of a cerebral angio-tomodensitometry which demonstrated a Fisher grade III subarachnoid hemorrhage. This hemorrhage was secondary to the rupture of a wide neck aneurysm in the left posterior communicating artery. Diagnostic and therapeutic arteriography was performed showing an aneurysm of the left posterior communicating artery measuring 6 x 3.4 mm. The aneurysm was embolized by coil without complications (Fig.1).

The patient was seen three days later in an ophthalmologic consultation for a profound drop in visual acuity (VA) in both eyes: right eye (RE) 0,5 and left eye (LE) count the fingers; intrinsic and extrinsic ocular motility were normal; and the funduscopic examination showed almost the same aspect of the retinal hemorrhages(Fig. 2).

After six weeks, the VA rose to 1 in the RE and 0,6 in the LE. At the fundus, the hemorrhages have resolved with macular exudate persistency in the left eye (Fig. 3).

3. DISCUSSION

Terson's syndrome was first described in 1900 by a French ophthalmologist Albert Terson. It associates intravitreal hemorrhage with acute subarachnoid hemorrhage [1]. Since then, Terson's syndrome (TS) has evolved to include types of presentation of intraocular all hemorrhage (intraretinal, retrohyaloid, preretinal or intravitreal) caused bv intracranial hemorrhages (subdural or subarachnoid (SAH)) that usually occur as a result of a ruptured brain aneurysm. Terson's syndrome remained an unrecognized condition until Fahmy, Vanderliden and Chisholm pointed out its relatively high frequency [2,3]. In the literature, we find variable incidences of occurrence of TS ranging from 2 to 27% of subarachnoid hemorrhages [4]. However, vitreous hemorrhages that constitute true TS are seen in less than 10% of cases of ruptured intracranial aneurysms. The frequency of bilateral TS cases is variously estimated: 14% to 60% of cases [5].

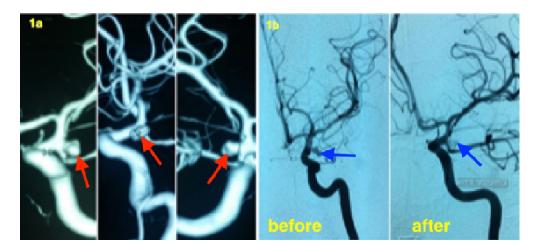


Fig. 1. a- Image of angio-tomodensitometry 3D reconstruction objectifyingthe aneurysm of the left posterior communicating artery (red Arrow); b-Cerebral arteriography showing the aneurysm of the left posterior communicating artery(blue arrow) before and afterembolization

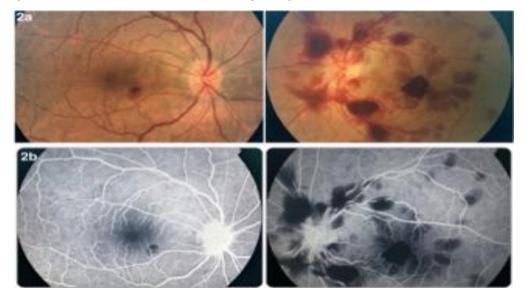


Fig. 2. a- Images of funduscopic examination showing retinal hemorrhages in both eyes; b-Fluorescein angiography showing retinal hemorrhages in both eyes and papillary edema of the left eye

This incidence is underestimated cause the diagnosis is difficult in a neurosurgical emergency context. Some patients die even before an ophthalmologic examination can be performed.

About 85% of hemorrhages are due to ruptures of aneurysms located in the lower part of the polygon of Willis. The three most frequent locations are the intracranial internal carotid artery, the bifurcation of the medial cerebral artery, and the upper part of the basilar arter. The anatomical location of the aneurysm was not linked to the side on which TS appears. It has also been shown that anatomical proximity between the aneurysm and the vitreous cavity is not required for intraocular hemorrhage. Fountas has found a more frequent non-significant occurrence of TS in patients with ruptured aneurysms of the anterior circulation. Others authors have described that even basilar aneurysms can lead to retrohyaloid and vitreous hemorrhages [6]. In our case, the hemorrhages were bilateral and more important on the side of the aneurysm.



Fig. 3. Images of funduscopic examination after 6 weeks

The anatomical location of these retinal hemorrhages is variable. They most often occur pre-retinal due to bleeding under the internal limiting membrane and they can extend into the vitreous cavity, or they can occur deeply into the peri- and juxta-papillary regions [7].

The mechanism of vitreous hemorrhages during cerebromeningeal hemorrhage has long been controversial. Two main physiopathological models have been proposed to explain this syndrome. The first hypothesis asserts that after a ruptured meningeal aneurysm, blood is pressed directly through the optic nerve sheath into the anterior eye sockets.

The second and most widely accepted hypothesis states that the sudden increase of intracranial pressure leads to retinal venous hypertension by compression of the central retinal vein in the intervaginal space. The retinal veins and capillaries are unable to absorb this influx of blood, resulting in papillary and retinal venous ruptures, hence hemorrhage in the vitreous [8].

Since TS is associated with low Glasgow scores on admission, a careful ophthalmologic examination should be performed in all unconscious or confused patients admitted to the hospital. Stienen reports loss of consciousness in most TS patients admitted to the emergency room. In conscious patients, the incidence of TS is much lower and the visual disturbances could be easily communicated.

For the diagnosis of TS, a precise fundus remains the most sensitive method showing in its

posterior pole retinal and pre-retinal hemorrhages, alterations in the pigmentation of the macula and the adjacent retina, and the formation of the pre-retinal membrane. These and other changes can involve one or both eyes and can occur with or without papillary edema.

Early diagnosis of TS is important because the decline in visual acuity, leading to functional blindness in bilateral cases, can be very troublesome for the patient. This blindness considerably hinders the rehabilitation process, even when the evolution of motor skills is favorable.

Although in most cases, a "wait and see" policy is initially proposed for spontaneous resolution of TS bleeding. Early vitrectomy is required in visually immature children and adults with bilateral vitreous bleedings, which are unlikely to resolve within a reasonable time.

Epiretinal membranes are the most common complications of TS, occurring in 63% of cases after the intraocular bleeding has resolved, whether or not it was treated with vitrectomy [9]. The vitreoretinal proliferation that can accompany the formation of these membranes is then the cause of retinal detachments. Other complications can occur such as macular degeneration, premacular cellophane fibrosis, macular scar, macular hole, retinoschisis, and retinal folds [10].

It is a well-known fact that the presence of TS is an indicator of subarachnoid hemorrhage(diagnostic and therapeutic urgency). Most patients with TS remain unconscious for a long time. Pfauser found very high mortality rates in patients with TS [6]. The diagnosis of TS is indicative of a very poor prognosis and frequently associated with re-bleeding from the aneurysm.

The visual prognosis after subretinal hemorrhage is variable. It depends on the amount of blood and the cause of the hemorrhage.

4. CONCLUSION

Performing a fundus examination in patients with subarachnoid hemorrhage may provide information on the prognosis of this condition.

Therefore, the early diagnosis of TS, in all cases of SAH and intracranial hemorrhage, will improve the therapeutic management and the visual and vital prognosis of these patients.

It is essential to perform a fundus examination in any patient presenting to the emergency room with a disturbance of consciousness without an obvious etiological context.

CONSENT

As per international standard or university standard, patients' written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

 TErson A. L'hémorragie dans le corps vitré au cours de l 'hémorragie cérébrale. Clin Ophtalmol 1900;6:309-12. Clin Ophtalmol. 1900;309–12.

- Fahmy JA. Fundal haemorrhages in ruptured intracranial aneurysms: II. Correlation with the Clinical Course. Acta Ophthalmologica. 2009;5(3):299–304.
- Vanderlinden RG, Chisholm LD. Vitreous hemorrhages and sudden increased intracranial pressure. Journal of Neurosurgery. 1974;41(2):167–176.
- Fountas KN, Kapsalaki EZ, Lee GP, Machinis TG, Grigorian AA, Robinson JS, et al. Terson hemorrhage in patients suffering aneurysmal subarachnoid hemorrhage: predisposing factors and prognostic significance. JNS. 2008;109 (3):439–444.
- Perennou D, Pélissier J, Beaufrère L, Laurent E, Bénaim C, Belso L, et al. Baisse de l'acuité visuelle après hémorragie cérébroméningée: six cas de syndrome de Terson. Annales de Réadaptation et de Médecine Physique. 2000;43(4):184–192.
- Pfausler B, Belcl R, Metzler R, Mohsenipour I, Schmutzhard E. Terson's syndrome in spontaneous subarachnoid hemorrhage: a prospective study in 60 consecutive patients. Journal of Neurosurgery. 1996;85(3):392–394.
- Schultz PN, Sobol WM, Weingeist TA. Long-term Visual Outcome in Terson Syndrome. Ophthalmology. 1991;98(12): 1814–1819.
- Ducourneau D, Coulon JJ, Ballereau L. [Treatment of vitreous hemorrhages. Anatomo-clinical classification and diagnostic methods]. Bull Soc Ophtalmol Fr. 1987;Spec No:7–8, 13–28, 30.
- 9. Yokoi M. Epiretinal membrane formation in Terson syndrome. Japanese Journal of Ophthalmology. 1997;41(3):168–173.
- Van Rens GH, Bos PJM, Van Dalen JTW. Vitrectomy in two cases of bilateral Terson syndrome. Doc Ophthalmol. 1983;56(1– 2):155–159.

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