

Case Report

Organic Depression and Terson's Syndrome in Adult Polycystic Kidney Disease: Case Report and Review of Literature

Ranganath R. Kulkarni, Pradeep V. Addagadde¹

ABSTRACT

Depressive symptoms are common in neurological diseases, at times posing dilemma in organic or functional origin. Cerebrovascular disease may predispose, precipitate, or perpetuate some geriatric depressive syndromes that resemble primary depressions both clinically and therapeutically in about half of the patients following acute stroke. Terson's syndrome is the direct occurrence of vitreous hemorrhage following subarachnoid/subdural hemorrhage, often overlooked in the acute setting. Autosomal dominant (adult) polycystic kidney disease may be associated with berry aneurysms and hypertension, and may lead to intracranial bleeds. We report an unusual case of organic depression and Terson's syndrome in a 50-year-old female with polycystic kidney disease and hypertension, following anterior communicating artery aneurysmal subarachnoid bleed with bilateral subdural extension. Management included anti-hypertensives, antiepileptics, neodymium: YAG laser photocoagulation, and aneurysmal clipping.

Key words: *Depression, intracranial aneurysm, polycystic kidney disease, subarachnoid hemorrhage, syndrome, vitreous hemorrhage*

INTRODUCTION

Litten first described an intraretinal hemorrhage associated with subarachnoid hemorrhage (SAH) in the German literature.^[1] However, Terson's description of vitreous hemorrhage (VH) following SAH in 1900 is now associated with this syndrome.^[2] In Terson's syndrome (TS), intracranial hemorrhages (ICH) are followed by intraocular hemorrhage (IOH),

classically in the subhyaloidal space, but may also include subretinal, retinal, preretinal, and vitreal collections.^[3] Very few reports of organic depression and TS in autosomal dominant (adult) polycystic kidney disease (ADPKD) are reported in the literature. ADPKD is a common (1:400-1:1000) disease seen in fourth decade, characterized by bilateral multiple expanding renal cysts, associated with hypertension (75%), mitral valve prolapse (20-25%), saccular aneurysms of Circle of Willis (10-30%), and asymptomatic liver cysts (40%).^[4] Such associations may pose patients at risk for SAH or rarely subdural hemorrhage (SDH) and subsequent TS.^[3] Cerebrovascular disease may predispose, precipitate, or perpetuate some geriatric depressive syndromes that resemble primary depressions both clinically and therapeutically in about half of the patients following acute stroke.^[5]

Access this article online	
Website: www.ijpm.info	Quick Response Code 
DOI: 10.4103/0253-7176.127267	

Departments of Psychiatry and ¹Ophthalmology, Sri Dharmasthala Manjunatheshwara (SDM) College of Medical Sciences and Hospital, Dharwad, Karnataka, India

Address for correspondence: Dr. Ranganath R. Kulkarni
Department of Psychiatry, Sri Dharmasthala Manjunatheshwara (SDM) College of Medical Sciences and Hospital, Dharwad - 580 009, Karnataka, India. E-mail: dranant007@rediffmail.com

We report an unusual case of organic depression and TS in a hypertensive middle-aged lady with ADPKD, following anterior communicating artery (ACoA) aneurysmal SAH, with full remission of depressive symptoms after post-neurosurgical interventions. We also summarize the relevant literature as identified via PubMed, EMBASE, and PsycINFO, as well as reference sections of selected articles.

CASE REPORT

A 50-year-old lady, receiving treatment for hypertension for the past 8 years, was diagnosed as having comorbid organic depression as per ICD-10 diagnostic criteria,^[6] and was symptomatic for the past 4 months. She was managed with escitalopram (10 mg/day) and mirtazapine (15 mg/day), but was in partial remission. She presented to the psychiatry out-patient services with exacerbation of symptoms of pervasive depression, apathy, insomnia, anorexia, fatigue, reduced social interactions, and frontal headache for the past 4 months, with diminished vision and vomiting for the past 1 week. Episodic frontal headache was followed by unresponsiveness and sudden fall without tonic — clonic limb movements or injuries, lasting for about 10 min with amnesia for the event. She had no history of head trauma, epilepsy, substance (alcohol, nicotine, or drug) abuse, or psychosocial stressors. She had second-degree consanguineous marriage. Family history of late-onset kidney diseases and hypertension was noted in both her parents and ADPKD was diagnosed in both her sons.

On admission, the Glasgow Coma Scale^[7] score was E4V5M6 and Hunt — Hess scale^[8] was grade-1, with blood pressure of 190/110 mm Hg. Neuromuscular and cardiovascular examination was normal. Mental status examination revealed depressed mood, apathy, negative cognitions, impaired judgment with preserved attention, orientation, memory, and intelligence, with grade-4 insight. Hamilton depression rating^[9] (HAMD-17 item) score was 16 and the score on the mini-mental status examination^[10] was 28/30. Ophthalmological evaluation revealed reduced visual acuity in the right eye (1/60) and normal left eye (6/6), bilaterally normal anterior segments, normal pupils (bilaterally 3 mm, round, regular, and reacting to both direct and consensual light reflexes), and normal intraocular pressures. Fundoscopy showed boat-shaped subhyaloid hemorrhage involving the posterior pole extending below the inferior arcade, with multiple hemorrhages seen around the normal optic disc in the right eye [Figure 1a] and normal left fundus [Figure 1b], with evidence of bilateral grade-1 hypertensive retinopathy [Figure 1]. Hematological and biochemical investigations revealed moderate normocytic and normochromic anemia (hemoglobin 8.7 g/dL), mild leukocytosis (12,240/mm³), raised

serum creatinine (2.5 mg/dL) with normal blood urea (42 mg/dL), serum electrolytes, blood glucose, lipid, liver, and thyroid profiles. Ultrasonography (USG) of abdomen showed bilateral renal ill-defined multiple (>6) cysts and calculi [Figure 2]. Echocardiography revealed mitral valve prolapse along with trivial mitral and aortic regurgitation. Electrocardiograph and carotid Doppler studies were normal. Electroencephalography showed right frontal polyspikes and sharp waves with preserved alpha background activity suggestive of epileptogenic foci. Cranial magnetic resonance imaging (MRI) revealed minimal SAH in the anterior inter-hemispheric fissure and adjacent cortical sulci with edema in adjacent frontal lobes, and hyper-acute SDH in bilateral fronto-temporo-parietal regions along the cerebral convexities (5 mm), with no mass effect or midline shift [Figure 3a], confirming the diagnosis of TS. Cerebral angiography showed small bi-lobed aneurysm (6 × 6 × 4 mm) arising from the ACoA with the neck of aneurysm measuring 1.5 mm in medio-lateral direction [Figure 3b].

Management included anti-hypertensives, antiepileptics, zolpidem, and hematonic. Antidepressants were withheld for the risk of re-bleed. Neodymium: YAG (Nd: YAG) laser posterior hyaloidotomy was performed by transcorneal route using frequency doubled Nd: YAG laser 532 nm (Zeiss-Visulas YAG laser 532 nm; laser setting: 9 spots, 100 msec duration, 50 μm spot size for penetration burn, 130 mW power) delivered via a slit lamp using Goldmann lens.^[11] The aiming beam was focused on the sloping edge of the most dependent portion of posterior hyaloid membrane at the inferior edge of subhyaloid hemorrhage to facilitate gravity-induced drainage. Blood flowed from hyaloidotomy opening and dispersed into inferior vitreous. Patient was advised for head-end elevation, and later discharged with anti-hypertensives, antiepileptics, and zolpidem. A week later, near-complete resolution of subhyaloid hemorrhage [Figure 4a] and ICH, with mild ventriculomegaly [Figure 4b] was noted. A month later, aneurysm clipping was successfully done by the neurosurgeon in liaison with a nephrologist. A month later, patient was asymptomatic with HAMD-17^[9] score of 3, and was regularly followed up for a year following neurosurgical intervention. She remained euthymic without any active psychiatric interventions and was managed with antihypertensives and antiepileptics alone. This sequential presentation with full remission of symptoms following neurosurgical interventions led to the diagnosis of “possible” organic depression due to intracranial ACoA aneurysm as per ICD-10 diagnostic criteria.^[6]

DISCUSSION

In our case, comorbid hypertension, depression, vomiting, akinetic seizures, and the chronology of presentation led to the suspicion of possible organicity.

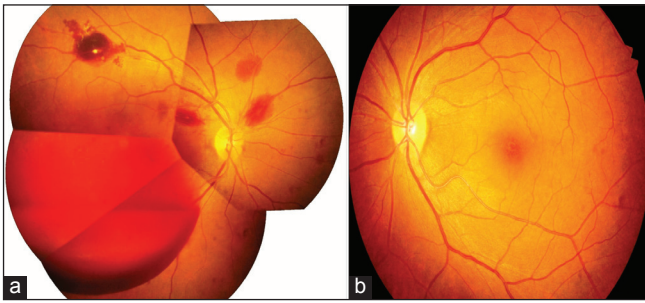


Figure 1: (a) Right fundus montage showing subhyaloid hemorrhage in boat-shaped pattern with multiple blotch hemorrhages and grade-1 hypertensive retinopathy changes. (b) Left fundus showing grade-1 hypertensive retinopathy changes with absence of intraocular hemorrhages

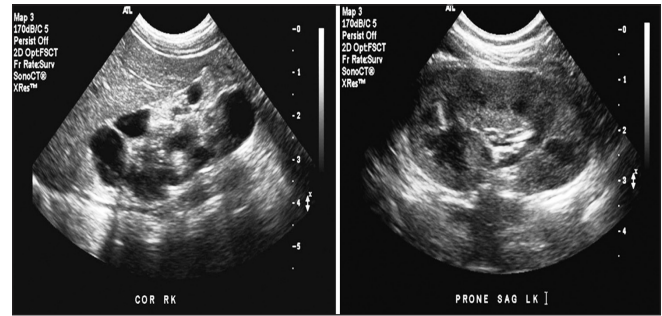


Figure 2: Ultrasonography of abdomen showing bilateral renal ill-defined multiple (>6) cysts and calculi

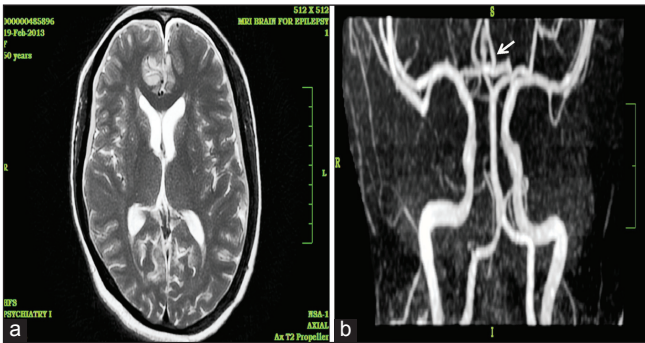


Figure 3: (a) Cranial magnetic resonance imaging showing minimal subarachnoid hemorrhage in the anterior inter-hemispheric fissure and adjacent cortical sulci with edema in adjacent frontal lobes, and hyperacute subdural hemorrhage in bilateral fronto-temporo-parietal regions along the cerebral convexities (5 mm), with no mass effect or midline shift. (b) Magnetic resonance angiography showing a small aneurysm (6 × 6 × 4 mm) arising from the anterior communicating artery, with the neck of aneurysm measuring 1.5 mm in medio-lateral direction

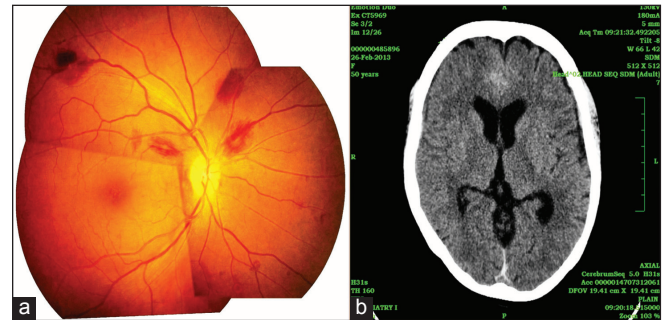


Figure 4: (a) Right fundus montage showing complete resolution of subhyaloid hemorrhage with resolving blotch hemorrhages, a week following Nd:YAG laser photocoagulation. (b) Cranial computed tomography showing near-complete resolution of intracranial hemorrhages with mild enlargement of lateral and third ventricles

Presence of SAH and symmetric bilateral SDH led to the suspicion of ACoA aneurysm, confirmed by neuroimaging. Furthermore, findings of polycystic kidneys on USG, raised creatinine, hypertension, and spontaneous (non-traumatic) SAH with SDH following aneurysmal rupture in the 50-year-old female led to the strong suspicion of a possible case of ADPKD. Temporal association of depressive symptoms with aneurysmal growth and rupture, followed by post-neurosurgical complete remission of depressive symptoms without antidepressant therapy possibly suggests the organic nature of depression. Disruption of prefrontal systems or their modulating pathways by single lesions or by an accumulation of lesions exceeding a threshold is hypothesized to be the central mechanism in “vascular depression.”^[12] Although vascular lesions in the left anterior cortex are found to be associated with a significantly higher frequency of major depression than the lesions in other locations, right-sided lesions may also predispose to retarded depression,^[5] as seen in our case.

The most common cause of spontaneous (non-traumatic) SAH is the rupture of a saccular aneurysm that originates 80% in the anterior and 20% in the posterior circulation. Saccular aneurysms enlarge with time, with the greatest risk for rupture at diameters of 6-10 mm. ACoA or middle cerebral artery bifurcation berry aneurysms may rupture into the adjacent brain or subdural space and sometimes form a hematoma large enough to produce mass effect.^[8] Surprisingly, in our case, there was minimal aneurysmal bleed with absent neurological signs, preserved mental status, and complete resolution of hemorrhage in 2 weeks, but for seizures, depression, and TS. The most common cause of TS is SAH due to ruptured cerebral aneurysm, while other causes include strangulation, traumatic ICH, and post-surgical ICH.^[13,14] Most patients with TS are comatose on initial presentation, depending upon the severity of the SAH as per the Hunt — Hess classification system.^[8,15] IOH may be difficult to diagnose immediately because the ophthalmologist is restricted from dilating the pupil in a comatose patient. A decreased red reflex is helpful in evaluating a comatose patient and B-scan ultrasound can further establish the extent of VH.^[16]

The exact pathophysiological mechanism of TS remains controversial.^[17] Two theories have been

proposed to explain the pathogenesis of VH after SAH. Firstly, subarachnoid blood is pressed through the optic nerve sheath into the interior of the eyeball when a bleeding episode occurs.^[17] The second theory, which has been most accepted, suggests that VH is the result of venous hypertension and rupture of superficial retinal veins, secondary to abruptly raised intracranial pressure, transmitted through the optic nerve sheath to the swollen optic nerve head, thereby occluding the retinal and choroidal anastomoses at the level of the lamina cribrosa. This theory is supported by the clinical findings of IOH and significant papilledema occurring in the eye contralateral to the side on which the ruptured aneurysm occurs.^[18] However, in our case, no such findings were noted on the contralateral side, thus supporting the first theory.

In our case, we preferred frequency doubled Nd:YAG laser (532 nm) over the standard Nd:YAG laser (1064 nm) due to its focusing capability, less scattering in the ocular media, high absorption by hemorrhage, and excellent delivery system. Spontaneous remission of VH or small IOH is most common, while complete resolution of such hemorrhages may take up to 9 months.^[19] Head-end elevation with bed rest and avoidance of anticoagulation medications may benefit. Immediate vitrectomy for IOH is not recommended, except in cases of submacular hemorrhage, monocular with severe visual loss, or in pediatric patients at risk for amblyopia.^[3,19] In patients who refuse surgery, repeated visual testing and ultrasound examinations are recommended to detect the developing retinal detachment and to treat late complications.^[13] Patients with SAH/SDH need a detailed fundoscopic examination for IOH to prevent long-term visual loss. Evaluation of late-onset depression and hypertensive patients for comorbid ADPKD may lead to possible clues of intracranial aneurysms.

REFERENCES

1. Litten M. Über einige vom allgemein-klinischen Standpunkt aus interessante Augenveränderungen. *Berliner Klinische Wochenschrift* 1881;18:23-7. [Article in German]
2. Terson A. De l'hémorragie dans le corps vitré au cours de l'hémorragie cérébrale. *Clinical Ophthalmology* 1990;6:309-15. [Article in French]
3. Kuhn F, Morris R, Witherspoon CD, Mester V. Terson syndrome. Results of vitrectomy and the significance of vitreous hemorrhage in patients with subarachnoid hemorrhage. *Ophthalmology* 1998;105:472-7.

4. Alpers CE. The Kidney. In: Kumar V, Abbas AK, Fausto N, Aster JC, editors. *Robbins and Cotran Pathological Basis of Disease*. 8th ed. Philadelphia: Elsevier-Saunders Publishing Inc.; 2010. p. 905-69.
5. Fleminger S. Cerebrovascular disorders. In: David AS, Fleminger S, Kopelman MD, Lovestone S, Mellers JD, editors. *Lishman's Organic Psychiatry: A Textbook of Neuropsychiatry*. 4th ed. New Jersey: Wiley-Blackwell Publishing Inc.; 2009. p. 473-542.
6. World Health Organization. *The ICD-10 classification of mental and behavioural disorders: Clinical descriptions and diagnostic guidelines*. 10th ed. Geneva: World Health Organization; 1992.
7. Teasdale G, Jennett B. Assessment of coma and impaired consciousness: A practical scale. *Lancet* 1974;2:81-4.
8. Hemphill JC, Smith WS, Gress DR. Neurologic critical care, including hypoxic-ischemic encephalopathy, and subarachnoid hemorrhage. In: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J, editors. *Harrison's Principles of Internal Medicine*. 18th ed. Washington: McGraw-Hill; 2011. p. 2254-65.
9. Hamilton M. A rating scale for depression. *J Neurol Neurosurg Psychiatry* 1960;23:56-62.
10. Folstein MF, Folstein SE, McHugh PR. 'Mini-mental state'. A practical method for grading the cognitive state of patients for the clinician. *J Psychiatry Res* 1975;12:189-98.
11. Raymond LA. Neodymium: YAG laser treatment for hemorrhages under the internal limiting membrane and posterior hyaloid face in the macula. *Ophthalmology* 1995;102:406-11.
12. Alexopoulos GS, Meyers BS, Young RC, Campbell S, Silbersweig D, Charlson M. 'Vascular depression' hypothesis. *Arch Gen Psychiatry* 1997;54:915-22.
13. Medele RJ, Stummer W, Mueller AJ, Steiger HJ, Reulen HJ. Terson's syndrome in subarachnoid hemorrhage and severe brain injury accompanied by acutely raised intracranial pressure. *J Neurosurg* 1998;88:851-4.
14. Pfausler B, Belcl R, Metzler R, Mohsenipour I, Schmutzhard E. Terson's syndrome in spontaneous subarachnoid hemorrhage: A prospective study in 60 consecutive patients. *J Neurosurg* 1996;85:392-4.
15. Frizzell RT, Kuhn F, Morris R, Quinn C, Fisher WS 3rd. Screening for ocular hemorrhages in patients with ruptured cerebral aneurysms: A prospective study of 99 patients. *Neurosurgery* 1997;41:529-33.
16. Sharma R, Shrestha JK. Terson's syndrome. *Nepal J Ophthalmol* 2009;1:77-9.
17. Muller PJ, Deck JH. Intraocular and optic nerve sheath hemorrhage in cases of sudden intracranial hypertension. *J Neurosurg* 1974;41:160-6.
18. Toosi SH, Malton M. Terson's syndrome — Significance of ocular findings. *Ann Ophthalmol* 1987;19:7-12.
19. Ou RJ, Yoshizumi MO. Terson Syndrome. Available from: <http://emedicine.medscape.com/article/1227921-overview#showall>. [Last accessed on 2013 Jun 19].

How to cite this article: Kulkarni RR, Addagadde PV. Organic depression and Terson's syndrome in adult polycystic kidney disease: Case report and review of literature. *Indian J Psychol Med* 2014;36:94-7.

Source of Support: Nil, **Conflict of Interest:** None.