Transient global amnesia following deliberate self-harm by hanging: Case report and review

A 25-year-old female was hospitalized subsequent to an attempt to hang herself. She was unconscious for a few minutes but responded to initial resuscitative measures. Relevant investigations, including X-ray neck, computed tomography scan brain, and electroencephalogram were normal. Physical examination was consistent with attempted hanging. On mental status examination, speech was relevant. Mood was euthymic. Attention was arousable, but concentration was impaired. Orientation to time and recent memory were impaired. Remote memory, insight, and judgment were unimpaired. Serial Mental Status Examination (MSE) revealed improvement in concentration and orientation. Due to sudden onset memory loss of <24 h in duration without other signs of cognitive impairment or concomitant focal neurological symptoms, she was diagnosed as a case of transient global amnesia. The pathophysiology of the condition is briefly reviewed.

Keywords: Deliberate self-harm, strangulation, transient global amnesia, hanging

CASE REPORT

A 25-year-old married female was hospitalized following an attempt to hang herself with a “dupatta” (a scarf...
worn in India) from the ceiling fan of her bedroom. After initial medical management, she was referred for psychiatric evaluation. As per the patient’s chief complaint being “under a lot of tension” for the past month. The patient was happily married, having a 1-year-old daughter. About a month back, her mother-in-law came to live with her after undergoing a coronary artery bypass graft for follow-up care. She was irritable, finding faults, and passing rude comments. The patient began to feel unhappy and irritated. She developed multiple aches and pains in the body, lethargy, fatigue, and crying spells. On the day of the event, a fight with her husband over a trivial matter, the patient locked herself in her bedroom, at which her husband anticipated that she was once again attempting to harm herself and broke open the door. The patient had hung herself by the neck, but her toes were still touching the ground. Her husband and neighbors brought her down and took her to the hospital. Following ½ h of emergency resuscitation, her blood pressure and respiration improved. The patient was coughing intermittently. An hour later, she was found to be very restless and required sedation with injectable benzodiazepine. The patient also vomited twice, bringing out brownish-colored vomitus, probably from a tongue bite.

No past or family history of psychiatric disorders. The patient was sensitive to minor criticisms and angry remarks and would get irritated very quickly. The patient had a history of frequent deliberate self-harm since childhood. In her childhood whenever her parents or teachers used to scold her/correct her, she used to cut her hand/finger with a blade. These used to be impulsive acts and were never attempts to die. There was no known history of sexual abuse. The patient was an extraverted and cheerful female who enjoyed being the center of attention. On examination, the patient had ligature marks on the neck, petechiae, and purpura on the right cheek, subconjunctival hemorrhage on the right-eye lower margin, swollen eyelids, puffy face, anterior tongue bite. Initial central nervous system examination revealed occasional eye-opening to verbal commands and spontaneous moans. Mental status evaluation could not be done on admission as the patient was drowsy and was not able to cooperate. She frequently enquired about what had happened to her and whereabouts of her family. MSE on the second day showed a cooperative and respectful female denying all events of the past, claiming to be in the hospital for having spilled tea on herself. Mood she said that she was “under a lot of tension,” her affect was euthymic. Attention was aroused with ease. Concentration was impaired. Memory was impaired. She could not repeat names of three objects after 5 min of being told. She could not recall the previous day’s events or her last meal. She totally denied having attempted to hang herself and could not provide a reason for the ligature marks on her neck. Serial MSE revealed improvement in concentration and orientation. The patient could never recall events of the day of the incident. Relevant investigations, including X-ray cervical spine, computed tomography scan head and electroencephalogram were within normal limits. After 2 days on PGI memory scale immediate, recent, and remote memory was intact. Beck depression inventory score was 24 indicating a moderate level of depression.

Her 16 PF personality profile revealed low score on factor “C” thereby indicating lower ego strength. On factor Q4 tendency to get fatigued and frustrated was noted.

Rorschach test findings were characterized by marked emotional lability and dysphoric affect.

DISCUSSION

TGA is a sudden, transient memory disturbance that typically resolves within 24 h leaving no long-term sequelae; recurrences are rare. During the episode of TGA, alertness, attention, and personal identity are preserved. TGA can be diagnosed by using Caplan’s criteria[7,8] which include: Presence of anterograde amnesia confirmed by an observer; Absence of clouding of consciousness or loss of personal identity; Apart from amnesia, there is no cognitive impairment; focal neurological or epileptic signs are absent; no recent history of head trauma or seizures; symptoms resolve within 24 h; and mild headache, nausea, dizziness, or other vegetative symptoms may or may not be present during the acute phase. Our patients met all the above diagnostic criteria for TGA. The condition is typically seen in middle-aged to elderly individuals[9] but our patient was a young adult.

REVIEW

Ictus amnésique was first mentioned by Jean Alfred Fournier, who in 1879 reported amnesic spells in patients with tabes and general paresis. Benon in 1909 was probably the first to report a typical case of what we now call TGA. The clinical and epidemiological features of transient amnesic attacks were described by Morris Bender and independently by Guyotat and Courjon, in 1956. The term TGA, however, was introduced by Fisher and Adams in 1958.[10,12] They described attacks characterized by the abrupt onset of anterograde amnesia and lasting for a matter of minutes or hours. The subjects frequently asked about what had happened to them and about the whereabouts of their family members. The retrograde amnesia was initially for days but diminished gradually
during the subsequent few hours. There were no other perceptual or cognitive defects. Personal identity was preserved. There were no other neurological signs during or after the attacks.\textsuperscript{[10-12]} By the next day, patients report that their memory is back to normal with the exception of a dense amnesia for the attack itself.\textsuperscript{[9]}

Over half a century after its original description, the pathophysiology of TGA remains elusive, with three major hypotheses: A transient ischemia, a seizure process, or a migrainous phenomenon. Likewise, despite extensive neurological accounts, only 35 cases in whom formal (although usually rather limited) neuropsychological assessment was performed during the acute stage have been reported.\textsuperscript{[13]} Brain lesions producing TGA have been reported in the hippocampal CA-1 neurons of Sommer’s sector of Ammon’s horn, amygdala, thalamus, caudate nucleus, and neocortex.\textsuperscript{[14]}

Fisher and Adams rejected psychiatric explanations and stated that epilepsy too was unlikely in view of the very infrequent recurrence of attacks and the absence of other epileptic features.\textsuperscript{[12]} Certain common precipitating situations have emerged as possible clues, for example, pain, anxiety, exercise, running a half marathon,\textsuperscript{[13]} temperature changes, swimming in cold water, taking a hot shower, sexual intercourse, Valsalva manoeuvres, diagnostic tests or certain drugs, emotional stress, pain, angiography, and driving.\textsuperscript{[15]} Other contributory etiological associations include migraine, epilepsy, venous congestion, ischemic heart disease, glutamate toxicity, cortical spreading depression,\textsuperscript{[16]} and first symptom of primary antiphospholipid syndrome.\textsuperscript{[17]}

Thus there are a number of potential etiologies that may be responsible for TGA, including arterial ischemia, venous congestion, migraine, and psychogenic disorders, but the exact cause remains elusive. The fact remains that the determination of the specific cause may not alter the management or the outcome since the disease process is self-limiting. However, it may help in preventing a recurrence.

Declaration of patient consent
The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Markowitsch HJ. The neuropsychology of hanging: An historical perspective. J Neurol Neurosurg Psychiatry 1992;55:507.
2. Markowitsch H, Stanisoiu A. History of Memory. The Oxford Handbook of History of Clinical Neuropsychology. William B. Barr and Linas A. Bielauskas. Editors. Oxford:Oxford University Press. 2016
3. Mengech HNK. Strangulation as a cause of Korsakoff psychosis. East African Medical journal 1983;60:343-5.
4. Ayugi J, Bojksi K, Onodo P, Molebatsi K, Olashore A. Neuropsychological profile following suicide Attempt by hanging in Botswana: Three case reports. Journal of Natural Sciences Research, 2018;8:74-78.
5. Meyer E. Psychische storungen nach strangulation. Med Klinik 1910;38:1482-6.
6. Medalia AA, Merriam AE, Ehrenreich JH. The neuropsychological sequelae of attempted hanging. J Neurol Neurosurg Psychiatry 1991;54:546-8.
7. Caplan LR. Transient global amnesia: Criteria and classification. Neurology 1986;36:441-2.
8. Quinette P, Guillery-Girard B, Dayan J, de la Sayette V, Marquis S, Vadler F, et al. What does transient global amnesia really mean? Review of the literature and thorough study of 142 cases. Brain 2006;129:1640-58.
9. Arena JE, Rabinstein AA. Transient global amnesia. Mayo Clin Proc 2015;90:264-72.
10. Romero JR, Mercado M, Beiser AS, Pikula A, Sehadr S, Kelly-Hayes M, et al. Transient global amnesia and neurological events: The Framingham heart study. Front Neurol 2013;4:47.
11. Fisher CM, Adams RD. Transient global amnesia. Trans Am Neurol Assoc 1958;83:143-6.
12. Fisher CM, Adams RD. Transient global amnesia. Acta Neurol Scand Suppl 1964;40:91-83.
13. Zidda F, Griebe M, Ebert A, Ruttorm C, Rossmanith C, Gass A, et al. Resting-state connectivity alterations during transient global amnesia. Neuroimage Clin 2019;23:101869.
14. Kim HJ, Kim H, Lim SM, Moon WJ, Hahn SH. An acute tiny left putamenal lesion presenting with transient global amnesia. Neurologist 2012;18:80-2.
15. Magazi D. Amnesia after a half marathon – A case study. Clin J Sport Med 2012;22:448-9.
16. Ding X, Peng D. Transient global amnesia: An electrophysiological disorder based on cortical spreading depression-transient global amnesia model. Front Hum Neurosci 2020;14:602496.
17. Zardi EM, Zardi DM, Lazarevic Z, Santucci S, D’Errico F, Aferlta A, et al. Transient global amnesia as the first symptom of primary antiphospholipid syndrome: A case report. Int J Immunopathol Pharmacol 2012;25:275-80.