

Transient Global Amnesia Resulting from Mild Trauma

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Abstract *Two cases are presented with clinical manifestations and neuropsychological evaluations which confirm the diagnosis of transient global amnesia (TGA). Their interest lies in the fact that in both cases the episodes were a result of light traumas. It was proposed that TGA was triggered by vascular changes resulting from a mild (whiplash or head) trauma.*

Keywords: Transient global amnesia, whiplash amnesia, mild head trauma.

The syndrome of sudden amnesia and confusion was first described by Bender in 1956 and has been given the name of transient global amnesia (TGA) (Fisher & Adams, 1964). It is clinically characterized by an abrupt deficit in memory for recent events usually associated with retrograde amnesia of hours, days, and even months, and is generally associated with people over 50. During the episode, the patient maintains a personal identity and can perform everyday activities adequately, although he/she usually shows signs of anxiety. Awareness of something strange happening is present (Steinmetz & Vroom, 1972; Heathfield, Croft, & Swash, 1973; Caplan, 1985; Kusher & Hauser, 1985). The most outstanding characteristic is very severe anterograde amnesia, which often causes a patient to ask the same question repeatedly.

TGA has been reported in connection with angiography, migraine, arterial embolism, bilateral infarction of the mesial temporal lobe, and epileptic attacks, among others (Shuttleworth & Wise, 1973; Greene & Bennett, 1974; Fogelholm, Kivalo, & Bergstrom, 1975; Caplan et al., 1981; Cochran et al., 1982; Crowell et al., 1984; Kushner & Hauser, 1985). Several factors have been proposed in relation to its etiology: Most authors have considered it to be a case of a reversible ischemic accident in the region of the posterior cerebral artery with a deficit in circulation in the fornico-hippocampal system (e.g., Poser & Ziegler, 1960; Mathew & Meyer, 1974). This explanation has, however, been criticized (e.g., Caplan, 1985).

In some patients who have exhibited TGA episodes, one or more cerebrovascular illness risk factors have been found, such as hyperlipidemia, arterial hypertension, diabetes, and cardiopathy (Mathew & Meyer, 1974; Dugan, Nordgren, & O'Leary, 1981; Kushner & Hauser, 1985). On occasions, signs or symptoms of vertebrobasilar insufficiency have also been found, such as ataxia, vertigo, diplopia, nystagmus, and blurred vision. Deficits such as homonymous hemianopia, visual agnosia, alexia, and anomia are also compatible with ischemia in the posterior arterial territory (Caplan, 1985).

Some precipitant factors have been related to episode onset, such as emotional experi-

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ence, fatigue, sexual activity, showers with hot or cold water, anxiety, stress, migraine, pain, and trauma (Ardila & Sanchez, 1988; Heathfield, Croft, & Swash; 1973; Fisher, 1982a; Merriam, 1988). In a series of 35 TGA cases, Fogelholm et al. (1975) mentioned a case which is quite clearly related to a sudden extension of the neck, and Fisher (1982b) described a case of whiplash injury, not obviously involving the head, associated with amnesia. A truck crashed into the back of a stationary car in which a 67-year-old female patient was sitting in the front seat. The patient was wearing a safety belt and exhibited only a whiplash effect followed by an amnesic period of approximately 72 hours. Hofstad and Gierde (1985) described a case of TGA very clearly related to a whiplash trauma in a 68-year-old woman. The patient was standing in a bus near the driver facing the back of the bus, when the bus stopped abruptly; the report was based on an accurate eyewitness report; the patient presented an amnesia lasting several hours. Matias-Guiu, Buenaventura, Cervera, & Codina (1985) reported a new case of whiplash amnesia. A 44-year-old man jumped to avoid walking on a just-washed floor; in checking the leap, he made an extension-flexion movement of the neck and felt a crack, and presented an amnesia of about 12 hours. Haas and Ross (1986) reported nine patients who had TGA attacks triggered by a mild blow on the head; the authors proposed that they represented a variety of TGA released by a traumatic migraine (for a review of the problem, see Haas and Lourie, 1988).

Over a period of about $2\frac{1}{2}$ years, opportunities to observe two TGA cases in teenagers resulting from light traumas, probably not caused by the trauma itself (posttraumatic amnesia), have occurred. This paper describes the observations, and offers interpretations of TGA resulting from either a whiplash effect which in some way may have produced a vascular disequilibrium in the posterior arterial region or a mild head injury as described by Haas and Lourie (1988).

Case 1

An 18-year-old patient with 12 years of schooling fell from a horse while jumping over obstacles and without losing consciousness fell into a state of amnesia with no evident external signs of trauma. The patient remembered riding toward an obstacle and thereafter remembered nothing. According to a report given by a cousin who was with him, the patient fell off the horse, got up immediately, and went back leading the horse. The patient reported that from 9 a.m. (the time at which he fell from the horse) to 4 p.m. that same day he could remember only minor details. During that lapse of time, the patient showed adequate behavioral integrity, but repeatedly asked the same questions, for example, what he studied (at the time, he had just started at university, but had not yet begun classes) and what had happened. He did not recognize his car, the number plates, or meaningful telephone numbers of family members and friends. For this reason, he was taken to the Neurologic Institute of Colombia.

Clinical admittance examination, 9 hours postinjury, was normal. The patient showed no signs of having suffered a blow to the head, however slight, at that moment. His only complaint was of a slight pain in the neck and dorsolumbar region. No evident anterograde amnesia was observed: digits, words, and sentences were memorized; faces and places were remembered in a normal way. He did, however, have a lacunal amnesia of several hours (since the moment he fell down, about 9 a.m., until approximately 4 p.m.) and was able to remember what had happened the previous days only with difficulty. A nonenhanced CT scan taken 2 days later was within normal limits.

Six months later, a new evaluation was carried out. It was observed that the patient's memories of the day of the accident were very poor, although he was able to mention some details. No memory defects were observed according to the Wechsler Memory Scale (Wechsler, 1945). He did not present a history of vascular headaches. Currently, he is completing the first semester in engineering and shows a normal academic performance.

Case 2

A 19-year-old patient with 12 years of schooling was admitted to the Neurologic Institute of Colombia; he had fallen while practicing motocross but remained conscious. After the fall, the patient maintained normal behavior and talked and played cards with friends for about 2 hours. His friends decided to take him to the Neurologic Institute because he could not remember what had happened and continuously asked about previous events. On admittance, the patient showed no signs of trauma, except for some scratches on the right arm. Clinical neurological examination was normal. He mentioned only a widely extended headache and pain to the neck. The neuropsychological examination showed him to be an alert patient with retrograde amnesia involving memory traces immediately before the fall and partially involving memory of the previous days. Anterograde amnesia at that moment was quite severe: he could not memorize words, sentences, short stories, and figures; he repeatedly asked the same questions.

The patient could remember absolutely nothing of what had happened over a period of approximately 6 hours, but progressively recovered memory. During all this time, the patient maintained adequate behavior but constantly repeated the same questions. A non-enhanced CT scan taken 2 days later was within normal limits. Two years later he still presented a lacunal amnesia of several hours for the day of the accident.

Conclusion

These two cases are remarkably similar. Both patients were teenagers who suffered light traumas on falling from moving objects. Neither of them suffered a transient loss of consciousness, and both continued performing activities in an apparently normal way. Their amnesia therefore cannot be considered to have resulted from a state of confusion. The second patient was still in an amnesic period when he was taken to the Neurological Institute. Mild head trauma without loss of consciousness can be accompanied eventually by a mild posttraumatic amnesia of a few minutes and a minimal retrograde amnesia (Gennarelli, 1982). In the first patient, amnesia lasted for some 7 hours, and in the second one, for about 6 hours.

Curiously, both patients complained of pain in the neck which might indicate a possible whiplash effect. Whiplash movements are usually expected when the head is free to move in relation to the trunk. The failure to find evidence of head injury in the scalp does not exclude a mild blow to the head, especially when the head has struck the relatively soft earth. In other words, it is reasonable to assume that head trauma occurred in both cases. No history of similar amnesic phenomena was found in either patient. Although neither patient presented a definite history of headache, Case 2 mentioned extended headache during examination.

It seems feasible to propose that amnesia was not due to the trauma itself, but to either the sharp head movement during the fall or the TGA attack triggered by a mild

head trauma. An extension-flexion movement of the neck and head could have caused a transient insult to the central brain modifying in some way vascular activity dynamic in the posterior cerebral arterial region.

Caplan (1985) mentioned four criteria which must be fulfilled in making a TGA diagnosis: (1) information must be obtained concerning episode onset; (2) the patient must be examined during the episode (ideally by a neurologist, but if this is not possible, then by a reliable observer); (3) there should be no important accompanying neurological signs; and (4) memory loss must be transient. These two patients fulfill all these criteria satisfactorily, and therefore it would be acceptable to say that the TGA episodes were due either to the whiplash effect, as has been proposed by Hofstad and Gierde (1985), or to a mild head trauma, as proposed by Haas and Ross (1986). Both patients could have presented both the whiplash effect (pain in the neck) and, likely, a mild head trauma. The association with age (young people) favored the explanation for the TGA resulting from mild head trauma (Haas & Lourie, 1988).

In any case, TGA was triggered in both cases by a mild (whiplash or head) trauma on falling from moving objects. Both hypotheses are plausible, and they are not contradictory. Both suppose that a mild (whiplash or head) trauma can trigger some vascular changes (some disequilibrium in the posterior vascular territory or a traumatic migraine) responsible for the TGA episode.

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References

- Ardila, A., & Sanchez, E. (1988). Neuropsychologic symptoms in the migraine syndrome. *Cephalalgia*, 8, 67–70.
- Bender, M. (1956). Syndrome of isolated confusion with amnesia. *Journal of Hillside Hospital*, 5, 212–215.
- Caplan, L.B. (1985). Transient global amnesia. In J.A.M. Fredriks (Ed.), *Handbook of neurology: Vol. 45. Clinical neuropsychology* (pp. 205–218). Amsterdam: Elsevier.
- Caplan, L.B., Chedru, F., Lhermitte, F., & Mayman, C. (1981). Transient global amnesia and migraine. *Neurology*, 31, 1167–1170.
- Cochran, V., Morrell, F., Huckman, M., & Cochran, E. (1982). Transient global amnesia after cerebral angiography. *Archives of Neurology*, 39, 593–594.
- Crowell, F.G., Stump, D.A., Biller, J., McHenry, L.C., & Toole, J.F. (1984). The transient global amnesia-migraine connection. *Archives of Neurology*, 41, 75–79.
- Dugan, T.M., Nordgren, R.R., & O'Leary, P. (1981). Transient global amnesia associated with bradycardia and temporal lobe spikes. *Cortex*, 17, 633–638.
- Fisher, C.M. (1982a). Transient global amnesia: Precipitating activities and other observations. *Archives of Neurology*, 39, 605–608.
- Fisher, C.M. (1982b). Whiplash amnesia. *Neurology*, 32, 667–668.
- Fisher, C.M., & Adams, R.D. (1964). Transient global amnesia. *Acta Neurologica Scandinava*, 40, 1–83.
- Fogelholm, R., Kivalo, E., & Bergstrom, L. (1975). The transient global amnesia. *European Neurology*, 13, 72–84.
- Gennarelli, T.A. (1982). Cerebral concussion and diffuse brain injuries. In P.R. Cooper (Ed.), *Head injury* (pp. 83–97). Baltimore and London: William and Wilkins.
- Greene, H., & Bennett, D. (1979). Transient global amnesia with a previously unreported abnormality. *EEG and Clinical Neurophysiology*, 36, 409–413.

- Haas, D.C., & Lourie, M.D. (1988). Trauma-triggered migraine: An explanation for common neurological attacks after mild head trauma. *Journal of Neurosurgery*, *68*, 181–188.
- Haas, D.C., & Ross, G.S. (1986). Transient global amnesia triggered by mild head trauma. *Brain*, *109*, 251–257.
- Heathfield, K., Croft, P., & Swash, M. (1973). The syndrome of transient global amnesia. *Brain*, *96*, 729–736.
- Hofstad, H., & Gierde, I.O. (1985). Transient global amnesia after whiplash trauma. *Journal of Neurology, Neurosurgery and Psychiatry*, *48*, 956.
- Kushner, M.J., & Hauser, A. (1985). Transient global amnesia: A case control study. *Annals of Neurology*, *18*, 684–691.
- Mathew, N., & Meyer, I. (1974). Pathogenesis and natural history of transient global amnesia. *Stroke*, *5*, 303–311.
- Matias-Guiu, J., Buenaventura, I., Cervera, C., & Codina, A. (1985). Whiplash amnesia. *Neurology*, *35*, 1225.
- Merriam, A.E. (1988). Emotional arousal-induced transient global amnesia: Case report, differentiation from hysterical amnesia, and an etiological hypothesis. *Neuropsychiatry, Neuropsychology, and Behavioral Neurology*, *1*, 73–78.
- Poser, C., & Ziegler, D. (1960). Temporary amnesia as a manifestation of cerebrovascular insufficiency. *American Neurological Association*, *1*, 221–223.
- Shuttleworth, E., & Wise, G. (1973). Transient global amnesia due to arterial embolism. *Archives of Neurology*, *23*, 340–342.
- Steinmetz, E., & Vroom, F. (1972). Transient global amnesia. *Neurology*, *22*, 1193–1200.
- Wechsler, D. (1945). A standardized memory scale for clinical use. *Journal of Psychology*, *19*, 87–95.