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Transient global amnesia

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Summary. Transient Global Amnesia (TGA) is a clinical syndrome characterized by temporary inability to form new memories described as anterograde amnesia. It is associated with retrograde amnesia and repetitive questioning. During the attack patients remain conscious and communicative and personal identity is preserved. Focal neurological symptoms and epileptic features are absent and general conditions appear intact. The ability to store new memories gradually recovers and subjects return to normal conditions except for a substantial amnestic gap for the duration of the attack. TGA has an incidence of 3-8 per 100 000 people per year. It usually affects patients between the ages of 50 and 70 years, at an average age of 61 years; occurrence in patients younger than 40 years of age is rare. The rate of recurrence is between 6% and 10% per years. No gender prevalence has been recorded. The patients with definite TGA have a very good prognosis; their rate of subsequent major vascular events is less than 1% per year.

Key words: transient global amnesia, episodic memory, hippocampus.

Introduction

TGA is a clinical syndrome first described in 1956 indipendently by Guyotat and Courjon and by Bender, even if in 1882 and 1909, Ribot and Benon had already described transient amnestic states suggestive of a TGA.

In 1964, Fisher and Adams coined the term "Transient Global Amnesia (TGA)" by reporting attacks suffered by 17 patients. According to these authors, TGA usually occurred in middle-aged or elderly people and was characterized by the abrupt onset of anterograde amnesia, accompanied by repetitive questioning. With the exception of the amnesia, no neurological deficits were observed. Attacks lasted few minutes or hours and the ability to lay down new memories was gradually recovered (1).

In 1985 Caplan proposed the criteria to define TGA and reserved the term for witnessed attacks of definite amnesia without disturbance of consciousness, focal neurological symptoms or epileptic features in patients who do not have active epilepsy and a recent head injury, and which resolve within 24 hours (2).

It should be stressed that the original authors were highly restrictive in the diagnosis. Subsequently, however, the term has been applied to a variety of different clinical situations merely sharing the feature of prominent memory loss, and the published studies included a number of patients with lasting neurological symptoms or memory loss, such as those observed in epilepsy or after head injury (3-10). The most widely accepted view was that TGA is due to thromboembolic cerebrovascular disease (3); some authors have advocated an epileptic cause and aetiological claims have also been made for migraine. In addition, single cases have been reported in association with a wide range of conditions including brain tumours, drug overdosage, polycythemia and intracerebral and subarachnoid haemorrhage (2).

It was in 1990 that Hodges and Warlow (2) suggested that the aetiological uncertainty of TGA mainly resulted from the lack of both agreed diagnostic criteria (variation in the use of the term TGA) and well-documented epidemiological studies. They have attempted to address the pitfalls in the literature and to investigate the aetiology and natural history of TGA by means of a case-control study. The specific objectives of their study were to measure the prevalence of various probable risk factors in a well defined TGA population, to compare it to two control population – normal community-based controls and conventional TIA controls - and to compare the outcome in the TGA and TIA groups. The results of the case-control and longitudinal studies do not support a cerebrovascular etiology for TGA.

In the same year, Hodges and Warlow published a second study (11) in which they confirmed the diagnostic criteria proposed by Caplan to carry out a case-control study of TGA. They showed that the presence of the criteria was a significant predictor for a good outcome, whereas the patients who did not meet the criteria for TGA had a significantly worse prognosis with a high incidence of major vascular events.

Since this date, no changes occurred in the diagnostic criteria for TGA.

Diagnostic criteria for defining TGA

The diagnosis of TGA is primarily a clinical one. Diagnosis can be made if the following criteria (by Caplan and Hodges) are fullfilled.

1) Attacks must be witnessed and information available from a trustworthy observer who was present for most of the attack.
2) Anterograde amnesia during the attack.
3) Clouding of consciousness and loss of personal identity must be absent, and the cognitive impairment limited to amnesia (no aphasia, apraxia).
4) No accompanying focal neurological symptoms during the attack and no significant neurological signs afterwards.
5) Epileptic features should be absent.
6) Attacks must resolve within 24 hours.
7) Patients with recent head injury or active epilepsy (that is, remaining on medication or one seizure in the past two years) are excluded.

A temporally graded retrograde amnesia is consistently present during the acute attack in patients with acute TGA, although this feature is not included in the diagnostic criteria. Mild vegetative symptoms such as headache, nausea and dizziness might be present during the acute phase and might be a somatic manifestation of anxiety.

TGA classically presents with a sudden onset of severe anterograde amnesia, usually accompanied by repetitive questioning. All patients are unable to retain new information (verbal and non-verbal) and to recall recent events. Retrograde amnesia is present but is of variable duration, which can range from a few hours up to years. Immediate recall is unaffected as memory relating to personal details. Patients remain alert, attentive, and the episodes occur in clear consciousness; they remain fully communicative, higher cortical functions such as language, calculations, visuospatial skills, reasoning and abstract thinking are intact and often they carry out complex tasks like driving and playing music. They do not have any focal neurological symptoms. They are often described-wrongly-as being confused, while they are disoriented to time and place and they are often agitated or anxious, and may repeat the same questions (mostly relating to orientation) every few minutes. At the cessation of the attack, there is a rapid and apparently complete return of anterograde memory. On formal testing, minor changes in anterograde memory may persist for months, although this is unlikely to be clinically detected. In contrast, retrograd memory is slower to return to normal, with most recent memories returning last.

As patients cannot store new memories during the attack, they will never be able to recall the episode itself (12-15).

The nature of cognitive impairments in transient global amnesia

The most important cognitive dysfunctions during TGA are:

1) a substantial reduction of anterograde episodic long-term memory: patients have difficuty in learning and subsequently recalling novel episodic informations after a variable delay.

2) a partial loss of retrograde episodic long-term memory: patients have difficulty recalling episodic information that was learned hours, days, or months before the onset of the amnestic episode.

By contrast there seems to be a complete sparing of:
1) short-term memory;
2) semantic memory;
3) implicit memory (16-23).

The analysis of potential reductions of executive functions (including the executive component of working memory) is underrepresented in the literature.

The exact nature of the impairment of the episodic memory processes remains unknown. Any system for storing information needs (according to Baddeley):
1) encoding, during which perceptive information is transformed into more or less stable mental representations.
2) storage (or consolidation), during which mnemonic information is associated with other representations and maintained in long term memory.
3) retrieval, during which the subject can momentarily reactivate mnemonic representations.

Regarding the retrograde amnesia of TGA, it should depend on a problem of retrieval of information from episodic memory.

The mechanism responsible for the anterograde amnesia have not been completely elucidated. Probably it is the result of a deficit of both storage or encoding components of episodic memory (24). The disturbance in either encoding or storage during TGA is restricted to explicit memory; even massive anterograde amnesia does not prevent encoding, storage and retrieval of new information when implicit tasks are involved (25).

Differential diagnosis

There is a limited but important range of differential diagnosis that may clinically mimic a TGA, such as transient ischaemic attacks (TIA), epilepsy, complications of head trauma, drug intoxications, psychiatric illness and acute confusional state (Table 1) (26,30-31).

Management of patients with transient global amnesia in the emergency department

Although the diagnosis of TGA is primarily clinical, further investigations should be performed if cer-

Table 1. Most frequent diseases that may clinically mimic a transient global amnesia attack.

DIFFERENTIAL DIAGNOSIS	MAIN DIFFERENCES
Transient Ischaemic Attack (TIA) of the posterior cerebral circulation	Additional focal neurological signs usually accompanied a TIA.
Transient Epileptic Amnesia (TEA)	Distinctive manifestation of temporal lobe epilepsy causing amnesia alone, that usually occurs in middle or old age. The amnestic attacks are characterized by a mixed anterograd and retrograd amnesia, sometimes with repetitive questioning; anterograde component is often incomplete, and patients may report being able to "remember not being able to remember". Attacks commonly occur on waking, their duration is usually less than 1 hour; some episodes may be accompanied by olfactory hallucinations, automatisms, or a brief loss of responsivness. There are epileptiform changes on the EEG, and responsivness to anticonvulsivant medications.
Post-traumatic amnesia in head trauma	Usually accompanied with confusion and/or focal neurological signs; adherence to the criterion that the witness should have observed the episode before making the diagnosis of TGA will markedly reduce the mistakes.
Drug Intoxications, (especially benzodiazepines)	Patients typically show an altered state of consciousness.
Psychiatric disorders	Usually occur in the younger population and there is usually a precipitating psychosocial stressor. It is associated with memory loss for personal identification, indifference to memory loss, and retrograde rather than anterograde amnesia.
Acute confusional state	Patients are unable to maintain a coherent stream of thought, inattention is the key deficit. Repetitive questioning does not usually occur in acute confusional state and this is due to lack of insight in the latter.

ebrovascular risk factors are present, the patient shows repetitive amnestic episodes, the age is inferior to 50 years, the clinical features are ambiguous or no reliable witness is available. No specific treatment is indicated for a typical episode. The most important part of managing an episode of TGA is the psychological support for patients and their relatives. Seeing a once competent and healthy partner, sibling or parent become incapable of remembering what happened only a minute before is very distressing and hence also the relatives may require reassurance (32).

Aetiology and pathogenesis

Recently, focal magnetic resonance (MR) signal (diffusion weighted imaging [DWI]/T2) changes in the hippocampus reflecting restrictive diffusion have been described as the structural correlate of TGA reflecting a focal transient perturbation of hippocampal circuits. The lesions are selectively confined to the CA-1 sector of the hippocampal cornu ammonis, indicating that cellular stress of CA-1 neurons is the metabolic correlate of the diffusion changes seen in TGA. DWI lesions are accompanied by corresponding lesions in T2-weighted images and both are reversible within 14 days after TGA. Structural as well as functional sequelae 4 to 6 months after TGA are lacking. According with the data in the literature, a single unilateral DWI lesion either in the right or left hippocampus is sufficient to produce a transient perturbation of hippocampal memory circuits resulting in TGA.

Clinical and experimental data show that hyppocampal CA-1 neurons are critically involved in the process of memory consolidation in terms of a relay function in direct and polysynaptic intrahippocampal circuits and lesions in this area are critical since they can produce a clinically significant memory impairment.

CA-1 neurons show a selective vulnerability to metabolic and oxidative stress such as during hypoxaemia and ischaemia because in this region there are fewer microvessels than in the other sectors; hypoxia leads to a glutamate and calcium-induced and apoptosis-mediated "delayed neuronal death" of affected neurons 1-3 days after hypoxia (33).

The pathophysiologic substrate of these lesions, however, is largely unknown.

Three main pathogenic hypotheses, ischemia, seizure discharge and migraine, have been suggested to be associated with the pathophysiology of TGA.

However, several systematic case-control studies confirmed that none of these appears completely convincing (34-36).

Although the aetiology remains controversial, the nature of precipitating events just before a TGA episode has been widely studied. In about half of the cases, TGA is precipitated by an emotionally or physically stressful event, such as sudden immersion in cold or hot water, physical exertion, emotional or psycological stress, pain, medical procedures, sexual intercourse.

Acute stress might modulate CA1 synaptic mechanisms involved in learning and memory. The enhanced glutamatergic trasmission and increased calcium influx in CA1 neurons in response to stress is mediated by increased levels of corticotropin-releasing hormone, neurosteroids, beta-adrenoceptor agonists, and corticosterone acting via CA1 mineralcorticoid and glucocorticoid receptors. This potentiated calcium exposure might be a risk factor for CA1 neurons with regard to an increased metabolic vulnerability and thus potentially impairing their structural integrity. These mechanisms might also be involved in the pathophysiological cascade, and could selectively affect hippocampal CA1 neurons with a subsequent perturbation of memory pathways, which might result in acute TGA (37).

These situations might lead to an hyperventilation and decrease in CO2 blood level induced cerebral vasoconstriction, followed by a consecutive hypoperfusion and liberation of excito-toxic neurotransmitters in memory relevant structures (38).

Many precipitating factors relate to a Valsalva manoeuvre. Lewis (39) suggested that a Valsalva manoeuvre (blocking venous return through the superior vena cava) allows brief retrograde transmission of high venous pressure to the cerebral venous system, resulting in venous ischaemia. This hypotesis was confirmed by the finding that the incidence of a retrograde flow pattern in the internal jugular vein as expression of jugular valve insufficiency (39), is elevated in TGA (40,41). Increased venous pression might lead to hip-

pocampal venous congestion and ischaemia contributing to the aetiology of TGA.

However the presence of transient cerebral venous hypertension alone is not enough to explain the development of TGA. The incidence of TGA is low, considering the high prevalence of jugular venous insufficiency in the healty population, and the recurrence rate is small, despite the frequent occurence of an involuntary Valsalva event. The presence of addictional factors which contribute to the development and the persistence of memory disfunction should be assumed (42-70).

Conclusions

The core of any cognitive function, probably of any human behaviour, is the ability to memorize and learn process. A person with a damaged memory ability is prevented from emotional and contingent contacts with the surrounding world and is spoiled of his personal continuity feeling, thus becoming submissive, weak and solitary.

More than 50 years after its initial description, transient global amnesia remains one of the most enigmatic syndromes in clinical neurology. In spite of its typical benign prognosis, TGA is a frightening experience for patients and their relatives. However, the vast majority of patients undergo several costly procedures to establish a diagnosis, usually with negative results. Finally, TGA generates dilemmas about therapeutic choices.

Using strict criteria, TGA remains a clinically distinct syndrome of uncertain aetiology, with good prognosis.

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