
PREVIOUS CASES

Transient Global Amnesia

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The first case of a sudden, isolated episode of amnesia and confusion was described by Bender (1956). In their detailed monograph, Fisher and Adams (1964) termed the syndrome 'transient global amnesia' (TGA), which they attributed to dysfunction of the medial temporal lobes and/or diencephalon. In subsequent years, numerous cases of TGA have been reported in the literature, the characteristic pattern of signs and symptoms refined, and diagnostic criteria proposed (Hodges and Warlow, 1990; Hodges, 1991). This review will outline recent advances in our understanding of the neuropsychology, aetiology and neuroradiology of the syndrome.

Episodes of TGA usually occur in otherwise healthy middle-aged or elderly people. The onset is abrupt, with severe anterograde amnesia and disorientation. Although aware of their own identities, patients are often perplexed as to their surroundings and the identity of those around them. They typically question companions repetitively about where they are, what is happening, etc., forgetting almost immediately the answers they are given. This dense anterograde amnesia is usually the most obvious symptom of TGA, with patients incapable of registering any new verbal or visual information into long-term memory (Hodges and Ward, 1989, see Case P173; Kapur *et al.*, 1998, see Case P704 below). In view of the profound anterograde amnesia, the nature and severity of the retrograde memory deficit are of considerable theoretical interest, yet relatively few studies have addressed this issue. It is clear that some patients have an extensive and temporally graded retrograde amnesia, but others with equally severe anterograde amnesia have very little disruption to past memories, attesting to the dissociation of the processes involved (Hodges and Ward, 1989; Kazui *et al.*, 1996, see Case P705 below; Eustache *et al.*, 1999, see Case P699 below). There is also growing evidence that autobiographical memory, rather than knowledge of public events and famous people from the past, may be differentially impaired (Evans *et al.*, 1993, see Case P168; Guillery, 2000, this issue; Venneri and Caffarra, 1998, see Case P719 below).

Another contentious issue highlighted by studies of TGA is the degree of separation between implicit and explicit memory processes. A number of authors have now reported preservation of perceptual and semantic priming, and of procedural skill learning (Goldenberg, 1995; Kazui *et al.*, 1995, see Case P176; Kapur *et al.*, 1996, see Case P703 below; Eustache *et al.*, 1997, see Case P698 below). Occasional deficits are seen relative to controls, however, which have been explained as reflecting the contamination of implicit memory by impaired explicit memory strategies (Eustache *et al.*, 1997). When experimental techniques are employed that are designed to probe implicit memory 'purely', results confirm that these processes are preserved in the disorder (Beauregard *et al.*, 1997, see Case P693 below). Other components of memory, such as short-term (working) memory (Hodges and Ward, 1989) and semantic memory (Hodges, 1994, see Case P174), are also typically retained in TGA. The syndrome is thought to be largely benign in nature, with cognitive impairments usually resolving after a few hours, leaving only a brief gap in memory for the period of the attack and often some hours before (Kazui *et al.*, 1996; Kapur *et al.*, 1998).

Uncertainty still remains as to the aetiology of TGA (Goldenberg, 1995; Hodges, 1998). Fisher and Adams (1964) postulated three possible causes: thromboembolic cerebrovascular disease, epileptic discharge and migraine. Many subsequent authors have interpreted the clinical features of their patients as supporting one or other of these hypotheses (e.g. Attarian *et al.*, 1995, see Case P691 below; Jung *et al.*, 1996, see Case P702 below; Tosi and Righetti, 1997, see Case P718 below). Others have suggested alternative possible causes, such as transient ischaemia (Kushner and Hauser, 1985; Raffaele *et al.*, 1995, see Case P711 below; Ay *et al.*, 1998, see Case P692 below), spreading cortical depression related to migraine (Olesen and Jorgensen, 1986; Strupp *et al.*, 1998, see Case P715 below), raised intracranial venous pressure due to impaired autoregulation (Lewis, 1998), cerebral angiography (Cochran *et al.*, 1982; Meder

et al., 1997, see Case P708 below), mild head or neck trauma (De Renzi *et al.*, 1995, see Case P696 below; Venneri *et al.*, 1998, see Case P719 below), physical exertion (Caplan, 1990; Richardson *et al.*, 1998, see Case P712 below; Litch and Bishop, 1999, see Case P706 below), and emotional stress (Fisher, 1982; Merriam *et al.*, 1992, see Case P710 below; Inzitari *et al.*, 1997; Durst *et al.*, 1999, see Case P697 below).

In recent years, a distinction has been drawn between cases of 'pure TGA' (Hodges and Warlow, 1990) and those in which there is a distinctive epileptic aetiology, known as transient epileptic amnesia (Kapur, 1993; Zeman *et al.*, 1998, see Case P723 below). By contrast to TGA, transient epileptic amnesia is characterized by recurrent brief episodes of amnesia, which the patients may partially recall, often occurring in the early morning. During the attack, there is severe retrograde amnesia and repeated attacks may lead to permanent loss of remote memories with normal performance on standard anterograde memory tests (Zeman *et al.*, 1998). Another associated syndrome, termed transient semantic amnesia, has been described as being characterized by a selective, temporary impairment of semantic memory with preservation of anterograde episodic and working memory (Hodges, 1997, see Case P700 below). A further variation, involving a selective transient autobiographical amnesia in the context of preserved anterograde memory, has also been reported recently (Venneri and Caffarra, 1998).

With the development of functional imaging techniques, many patients with TGA have undergone computed tomography (CT), magnetic resonance imaging (MRI), single photon emission computed tomography (SPECT) or positron emission tomography (PET) scans during (or soon after) their attack. In some cases, structural or functional changes have been observed in regions such as the medial temporal lobes or thalamus (Volpe *et al.*, 1983; Evans *et al.*, 1993; Jung *et al.*, 1996; Eustache *et al.*, 1997), known to be relevant for memory (Zola-Morgan *et al.*, 1986; Squire, 1992; Aggleton and Brown, 1999). Many cases, however, have been reported as showing normal scans (e.g. Hodges and Warlow, 1990; De Renzi *et al.*, 1995). The advent of diffusion-weighted MRI (DWMRI) has indicated that, in these cases, there may be changes in the medial temporal lobe indicative of ischaemic damage, of the type observed during migraine attacks, which are not apparent using conventional MRI sequences (Woolfenden *et al.*, 1997, see Case P722 below; Ay *et al.*, 1998, see Case P692 below; Strupp *et al.*, 1998). Another recent study used proton magnetic resonance spectroscopy to rule out the metabolic changes associated with ischaemia in their patient (Zorzon *et al.*, 1998, see Case P724 below).

As briefly described in this review, a considerable amount of research has been undertaken into the neuropsychology, aetiology and neuroradiology of TGA in the last few years, and yet there is still a great deal about the syndrome that is uncertain. Recent investigations, such as that by Guillery *et al.* (2000; in this issue), have refined the picture on the patterns of impairment and preservation of memory processes

associated with TGA. Detailed epidemiological studies have greatly improved our appreciation of the possible precipitating factors underlying the disorder. Finally, recent advances in neuroimaging technology have helped elucidate the possible neuroanatomical loci for the pathology involved. There still remains, however, little consensus as to the precise aetiology and pathogenesis of TGA, such that although not quite so shrouded in mystery as it was a decade or so ago, the syndrome still remains rather an enigma.

References

- Aggleton JP, Brown MW. Episodic memory, amnesia, and the hippocampal–anterior thalamic axis. *Behavioral and Brain Sciences* 1999; 22: 425–89.
- Attarian S, Michel B, Delaforte C, Chave B, Gastaut JL. Un cas d'amnésie transitoire par thrombophlébite cérébrale: Apport de la neuro-imagerie à la physiopathologie des amnésies transitoires/A case of transient global amnesia due to cerebral venous thrombosis: The contribution of neuroimaging techniques to the study of the physiopathogenesis of transient amnesias. *Revue Neurologique* 1995; 151: 552–8.
- Ay H, Furie KL, Yamada K, Koroshetz WJ. Diffusion-weighted MRI characterizes the ischemic lesion in transient global amnesia. *Neurology* 1998; 51: 901–3.
- Beaugregard M, Weiner J, Gold D, Chertkow H. Word priming during and after transient global amnesia: A case report. *Neurocase* 1997; 3: 451–9.
- Bender MB. Syndrome of isolated episode of confusion with amnesia. *Journal of Hillside Hospital* 1956; 5: 212–5.
- Caplan LR. Transient global amnesia: Characteristic features and overview. In: Markowitsch HJ, editor. *Transient global amnesia and related disorders*. New York: Hogrefe & Huber, 1990: 15–27.
- Cochran JW, Morell F, Huckman MS, Cochran EJ. Transient global amnesia after cerebral angiography: Report of 7 cases. *Archives of Neurology* 1982; 39: 593–4.
- De Renzi E, Lucchelli F, Muggia S, Spinnler H. Persistent retrograde amnesia following a minor trauma. *Cortex* 1995; 31: 531–42.
- Durst R, Teitelbaum A, Aronson R. Amnesic state in a holocaust survivor patient: Psychogenic versus neurological basis. *Israel Journal of Psychiatry and Related Sciences* 1999; 36: 47–54.
- Eustache F, Desgranges B, Petit Taboue M-C, de la Sayette V, Piot V, Sable C *et al.* Transient global amnesia: Implicit/explicit memory dissociation and PET assessment of brain perfusion and oxygen metabolism in the acute stage. *Journal of Neurology, Neurosurgery and Psychiatry* 1997; 63: 357–67.
- Eustache F, Desgranges B, Laville P, Guillery B, Lalavee C, Schaeffer S *et al.* Episodic memory in transient global amnesia: Encoding, storage, or retrieval deficit? *Journal of Neurology, Neurosurgery and Psychiatry* 1999; 66: 148–54.
- Evans JJ, Wilson BA, Wraight EP, Hodges JR. Neuropsychological and SPECT scan findings during and after transient global amnesia: Evidence for the differential impairment of remote episodic memory. *Journal of Neurology, Neurosurgery and Psychiatry* 1993; 56: 1227–30.
- Fisher CM. Transient global amnesia: Precipitating activities and other observations. *Archives of Neurology* 1982; 39: 605–8.
- Fisher CM, Adams RD. Transient global amnesia. *Acta Neurologica Scandinavica* 1964; 40: 1–83.
- Goldenberg G. Transient global amnesia. In: Baddeley AD, Wilson BA, Watts FN, editors. *Handbook of memory disorders*. Chichester: Wiley, 1995: 109–33.
- Guillery B, Desgranges B, Piolino P, Laville P, de la Sayette V, Eustache F. Extensive temporally graded retrograde amnesia for personal–episodic facts in transient global amnesia. *Neurocase* 2000; 6: 205–10.
- Hodges JR. Transient global amnesia. London: WB Saunders, 1991.
- Hodges JR. Semantic memory and frontal executive function during transient global amnesia. *Journal of Neurology, Neurosurgery and Psychiatry* 1994; 57: 605–8.
- Hodges JR. Transient semantic amnesia: A new syndrome? *Journal of Neurology, Neurosurgery and Psychiatry* 1997; 63: 548–9.
- Hodges JR. Unraveling the enigma of transient global amnesia. *Annals of Neurology* 1998; 43: 151–3.
- Hodges JR, Ward CD. Observations during transient global amnesia. *Brain* 1989; 112: 595–620.

- Hodges JR, Warlow CP. The aetiology of transient global amnesia: A case-control study of 114 cases with prospective follow-up. *Brain* 1990; 113: 639-57.
- Inzitari D, Pantoni L, Lamassa M. Emotional arousal and phobia in transient global amnesia. *Archives of Neurology* 1997; 54: 866-73.
- Jung HH, Baumgartner RW, Burgunder JM, Wielepp JP, Lourens S, Wielepp JP. Reversible hyperperfusion of the right medial temporal lobe in transient global amnesia. *Journal of Neurology, Neurosurgery and Psychiatry* 1996; 61: 654-5.
- Kapur N. Transient epileptic amnesia: A clinical update and a reformulation. *Journal of Neurology, Neurosurgery and Psychiatry* 1993; 56: 1184-90.
- Kapur N, Abbott P, Footitt D, Millar J. Long-term perceptual priming in transient global amnesia. *Brain and Cognition* 1996; 31: 63-74.
- Kapur N, Millar J, Abbott P, Carter M. Recovery of function processes in human amnesia: Evidence from transient global amnesia. *Neuropsychologia* 1998; 36: 99-107.
- Kazui H, Tanabe H, Ikeda M, Nakagawa Y, Shiraishi J, Hashikawa K. Memory and cerebral blood flow in cases of transient global amnesia during and after the attack. *Behavioural Neurology* 1995; 8: 93-101.
- Kazui H, Tanabe H, Ikeda M, Hashimoto M, Yamada N, Okuda J *et al.* Retrograde amnesia during transient global amnesia. *Neurocase* 1996; 2: 127-33.
- Kushner MJ, Hauser WA. Transient global amnesia: A case-control study. *Annals of Neurology* 1985; 18: 684-91.
- Lewis SL. Aetiology of transient global amnesia. *Lancet* 1998; 352: 397-9.
- Litch JA, Bishop RA. Transient global amnesia at high altitude. *New England Journal of Medicine* 1999; 340: 1444.
- Meder J-F, Mourey-Gerosa I, Blustajn J, Lemaiguen H, Devaux B, Fredy D. Transient global amnesia after cerebral angiography: A case report. *Acta Radiologica* 1997; 38: 273-4.
- Merriam AE, Wyszynski B, Betzler T. Emotional arousal-induced transient global amnesia: A clue to the neural transcription of emotion? *Psychosomatics* 1992; 33: 109-13.
- Olesen J, Jorgensen MB. Leao's spreading depression in the hippocampus explains transient global amnesia: A hypothesis. *Acta Neurologica Scandinavica* 1986; 73: 219-20.
- Raffaie R, Tornali C, Genazzani AA, Vecchio I. Transient global amnesia and cerebral infarct: A case report. *Brain Injury* 1995; 9: 815-8.
- Richardson RS, Leek BT, Wagner PD, Kritchevsky M. Transient global amnesia: A complication of incremental exercise testing. *Medicine and Science in Sports and Exercise* 1998; 30: S403-5.
- Squire LR. Memory and the hippocampus: A synthesis from findings with rats, monkeys, and humans. *Psychological Review* 1992; 99: 195-231.
- Strupp M, Bruning R, Wu RH, Deimling M, Reiser M, Brandt T. Diffusion weighted MRI in transient global amnesia: Elevated signal intensity in the left mesial temporal lobe in 7 of 10 patients. *Annals of Neurology* 1998; 43: 164-70.
- Tosi L, Righetti CA. Transient global amnesia and migraine in young people. *Clinical Neurology and Neurosurgery* 1997; 99: 63-5.
- Venneri A, Caffarra P. Transient autobiographical amnesia: EEG and single-photon emission CT evidence of an organic etiology. *Neurology* 1998; 50: 186-91.
- Venneri A, Brazzelli M, Della Sala S. Transient global amnesia triggered by a mild head injury. *Brain Injury* 1998; 12: 605-12.
- Volpe BT, Herscovitch P, Raichle ME, Hirst W, Gazzaniga MS. Cerebral blood flow and metabolism in human amnesia. *Journal of Cerebral Blood Flow and Metabolism* 1983; 3: S5-6.
- Woolfenden AR, O'Brien MW, Schwartzberg RE, Norbash AM, Tong DC. Diffusion-weighted MRI in transient global amnesia precipitated by cerebral angiography. *Stroke* 1997; 28: 2311-4.
- Zeman AZJ, Boniface SJ, Hodges JR. Transient epileptic amnesia: A description of the clinical and neuropsychological features in 10 cases and a review of the literature. *Journal of Neurology, Neurosurgery and Psychiatry* 1998; 64: 435-43.
- Zola-Morgan S, Squire LR, Amaral DG. Human amnesia and the medial temporal region: Enduring memory impairment following a bilateral lesion limited to field CA1 of the hippocampus. *Journal of Neuroscience* 1986; 6: 2950-67.
- Zorzon M, Longo R, Mase G, Biasutti E, Vitrani B, Cazzato G. Proton magnetic resonance spectroscopy during transient global amnesia. *Journal of Neurological Sciences* 1998; 156: 78-82.

Received on 31 January, 2000; resubmitted on 15 February, 2000; accepted on 16 February, 2000

Un cas d'amnésie transitoire par thrombophlébite cérébrale: apport de la neuro-imagerie à la physiopathogénie desamnésies transitoires/A case of transient amnesia associated with cerebral thrombophlebitis: the contribution of neuroimaging techniques to the physiopathogenesis of transient amnesias

S. Attarian, B. Michel, C. Delaforte, B. Chave and J. L. Gastaut

Abstract

Presents a case report on a right-handed 57-year-old woman with transient global amnesia associated with cerebral venous thrombosis. Magnetic resonance imaging and single-photon emission computed tomography revealed a lesion in the right internal temporal region. Implications of this finding for the specialization of each hippocampus are discussed.

Journal

Revue Neurologique 1995; 151: 552–8

Neurocase Reference Number:

P691

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Case no. 13505

Key theoretical issue

- TGA can result from cerebral venous thrombosis

Key words: transient global amnesia; SPECT; hippocampus

Scan, EEG and related measures

MRI, SPECT, cerebral angiography

Standardized assessment

Batterie d'Estimation Mnésique, Mini-Mental State Examination

Other assessment

None specified

Lesion location

- MRI: right temporal lobe; SPECT: right hippocampal region

Lesion type

Cerebral venous thrombosis

Language

French

Diffusion-weighted MRI characterizes the ischaemic lesion in transient global amnesia

H. Ay, K. L. Furie, K. Yamada and W. J. Koroshetz

Abstract

We present a patient with transient global amnesia (TGA) whose diffusion-weighted MRI (DWI) showed increased signal in the splenium of the corpus callosum and in the left parahippocampal gyrus. The absence of high signal on the corresponding apparent diffusion coefficient (ADC) images supports the diagnosis of an acute infarction. This finding provides a temporal relation between cerebral ischaemia and infarction in the territory of posterior cerebral artery and in certain cases of TGA. An early means of detecting ischaemia in TGA by DWI may influence clinical decisions made in patient evaluation and management.

Journal

Neurology 1998; 51: 901–3

Neurocase Reference Number:

P692

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- TGA may be associated with lesions of insufficient size to be seen using CT or MRI. The advent of diffusion-weighted MRI may allow such lesions to be documented

Key words: transient global amnesia; retrograde amnesia; diffusion-weighted magnetic resonance imaging

Scan, EEG and related measures

CT, ECG, diffusion-weighted MRI (DWMRI)

Standardized assessment

Not specified

Other assessment

Questions relating to orientation, anterograde memory, person recognition

Lesion location

- CT, ECG: normal; DWMRI: lesion in left splenium of the corpus callosum; hyperintensity in left parahippocampal gyrus

Lesion type

Ischaemia

Language

English

Word priming during and after transient global amnesia: a case report

M. Beaugregard, J. Weiner, D. Gold and H. Chertkow

Abstract

Many studies have shown relative preservation of word priming in subjects with mild amnesia, but some impairment in severe amnesia. This calls into question the degree of separation between implicit and explicit memory. Possible contamination of implicit memory tasks by impaired explicit memory strategies might be obscuring the actual dissociation between the two memory systems. We have developed a method circumventing explicit memory contamination by using brief duration repeated primes below the awareness threshold of subjects. We have used this approach to evaluate the status of word priming in a 70-year-old woman with transient global amnesia. This subject was examined during her attack as well as 8 months after. She was tested for word priming on a speeded category membership decision task. Implicit or explicit encoding procedures were used in three different experiments. Results indicated that there was no significant difference between the priming effects measured in the implicit and explicit conditions or between the priming effects measured during and after the transient global amnesia attack. Results also confirmed that brief multiple presentation of words can offer a means of producing word priming in the absence of explicit recognition or recall of the primed words in amnesia. These findings demonstrate that word priming is preserved during transient global amnesia. They also suggest that the capacity to encode, store and retrieve information implicitly, e.g. unintentionally, is retained in this neurological condition.

Journal

Neurocase 1997; 3: 451–9

Neurocase Reference Number:

P693

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

HS

Key theoretical issue

- Support is provided for the preservation of word priming during transient global amnesia

Key words: transient global amnesia; word priming; brief multiple presentation

Scan, EEG and related measures

SPECT

Standardized assessment

Folstein Mini-Mental State Examination, Boston Naming Test, Wechsler Memory Scale–Revised

Other assessment

Raven's Coloured Progressive Matrices test, neurological examination

Lesion location

- None

Lesion type

Not known

Language

English

Reduction of visual P300 during transient global amnesia

H. Bokura, S. Yamaguchi, H. Tsuchiya, K. Yamashita *et al.*

Abstract

Reports the case of a 56-year-old man who demonstrated a selective reduction of the visual P300 endogenous event-related potential during and after an attack of transient global amnesia (TGA). The contribution of the short-term memory system to P300 generation was investigated before and after (1 week and 9 months) TGA. Visual target P300 and P300 to novel visual stimuli were reduced in amplitude during TGA and at 1 week after the attack, while auditory P300 was preserved. Visual P300 was recovered at 9 months after TGA. Results support the notion that the neuronal networks responsible for P300 generation are modality dependent and that brain structures perfused by the posterior circulation are involved in visual P300 generation.

Journal

Electroencephalography and Clinical Neurophysiology: Evoked Potentials 1994; 92: 422–5

Neurocase Reference Number:

P694

Primary diagnosis of interest

Transient global amnesia, Wallenberg's syndrome

Author's designation of case

The patient

Key theoretical issue

- Selective reduction of visual P300 event-related potential after TGA may indicate disconnection between visual cortex and memory regions

Key words: P300; transient global amnesia; hippocampus; short-term memory; visual stimuli

Scan, EEG and related measures

CT, MRI

Standardized assessment

None

Other assessment

Tests of retrograde memory and visual and auditory target detection

Lesion location

- CT: normal; MRI: left lateral medulla

Lesion type

Hyperintensity

Language

English

Transient global amnesia and thalamic haemorrhage

W.-H. Chen, J.-S. Liu, S.-C. Wu and Y.-Y. Chang

Abstract

Discusses the case of a left rostral thalamic haematoma found in a 52-year-old hypertensive man who suffered from a 10-h episode of transient global amnesia (TGA). A neuropsychological study revealed no cognitive impairment in a follow-up period for 5 years. The left rostral part of the thalamus appears to be responsible for his TGA, due probably to an interference of the mamillothalamic tract, ventroamygdalofugal pathway or dorsal noradrenergic bundle.

Journal

Clinical Neurology and Neurosurgery 1996; 98: 309–11

Neurocase Reference Number:

P695

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Not specified

Key theoretical issue

- Disruption of the mamillothalamic tract, ventroamygdalofugal pathway or dorsal noradrenergic bundle may underlie TGA

Key words: transient global amnesia; thalamus; cerebral haemorrhage

Scan, EEG and related measures

CT

Standardized assessment

None specified

Other assessment

Tests of retrograde and anterograde memory, semantic knowledge and short-term memory

Lesion location

- CT: left rostral thalamus

Lesion type

Haematoma

Language

English

Persistent retrograde amnesia following a minor trauma

E. De Renzi, F. Lucchelli, S. Muggia and H. Spinnler

Abstract

Reports dense retrograde amnesia (RA) following a motor accident, in a 19-year-old man. Intelligence, anterograde memory (unknown name–activity, famous name–activity congruous, and famous name–activity incongruous associations), and retrograde memory (memory of autobiographical events, public events and famous persons, encyclopaedic notions, verbal and visual semantics, and procedural memory) were examined. Computed tomography (CT), magnetic resonance imaging (MRI) and positron emission tomography scans were negative. Follow-up was conducted 7, 13, 21 and 29 months post-onset. Ability to acquire verbal, visual and spatial information was excellent, in contrast to profound autobiographical RA, and impaired memory of public events and people. Encyclopaedic notions were well preserved. He was able to relearn facts or episodes of his past life, and integrate them in the frame of his autobiography. Since RA persisted unmodified at 29 months, diagnosis of transient global amnesia is suggested.

Journal

Cortex 1995; 31: 531–42

Neurocase Reference Number:

P696

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

MA

Key theoretical issue

- Mild trauma may cause functional inhibition of access to stored memories, leaving intact the ability to encode and retrieve new memories

Key words: transient global amnesia; retrograde memory; episodic memory; semantic memory; PET

Scan, EEG and related measures

CT, MRI, PET

Standardized assessment

Wechsler Adult Intelligence Scale–Revised; Token test; Boston Naming Test; Rey Figure Copy and Recall; Corsi Blocks

Other assessment

Tests of picture naming, letter fluency, anterograde memory, autobiographical memory, memory for public events and people, encyclopaedic knowledge

Lesion location

- CT, MRI, PET: all normal

Lesion type

Not known

Language

English

Amnestic state in a Holocaust survivor patient: psychogenic versus neurological basis

R. Durst, A. Teitelbaum and R. Aronzon

Abstract

Differentiation between psychogenic and organic amnesia is sometimes quite difficult. This paper focuses on the psychogenic and organic components of a complex case of amnesia rooted in remote and prolonged traumatic stress and manifested under circumstances evoking dissociated memories. The neurologically based transient global amnesia (TGA) of a 65-year-old female concentration camp survivor who developed sudden amnesia during a psychiatric intake interview was clearly triggered by the pressure of repressed Holocaust memories. The importance of distinguishing between TGA and dissociative amnesia is emphasized, and the role of psychological upset as a precipitant TGA is stressed.

Journal

Israel Journal of Psychiatry and Related Sciences 1999; 36: 47–54

Neurocase Reference Number:

P697

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- Remote and prolonged traumatic stress can lead to occurrence of TGA

Key words: transient global amnesia; retrograde amnesia

Scan, EEG and related measures

None specified

Standardized assessment

None specified

Other assessment

Assessment of orientation and memory

Lesion location

- Not known

Lesion type

Not known

Language

English

Transient global amnesia: implicit/explicit memory dissociation and PET assessment of brain perfusion and oxygen metabolism in the acute stage

F. Eustache, B. Desgranges, M.-C. Petit Taboue, V. de la Sayette, V. Piot, C. Sable, G. Marchal and J.-C. Baron

Abstract

Assessed explicit memory and two components of implicit memory (perceptual–verbal skill learning and lexical–semantic priming effects) and resting cerebral blood flow (CBF) and oxygen metabolism (CMRO₂) during the acute phase of transient global amnesia. In a 59-year-old woman with transient global amnesia, a neuropsychological protocol was administered, including word learning, story recall, categorical fluency, mirror reading, and word stem completion tasks. Results showed a dissociation between impaired long-term episodic memory and preserved implicit memory for its two components. Categorical fluency was significantly altered, suggesting word retrieval strategy, rather than semantic memory, impairment. The PET study disclosed a reduced CMRO₂ with relatively or fully preserved CBF in the left prefrontotemporal cortex and lentiform nucleus, and the reverse pattern over the left occipital cortex.

Journal

Journal of Neurology, Neurosurgery and Psychiatry 1997; 63: 357–67

Neurocase Reference Number:

P698

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- Patient's selective deficit of explicit episodic memory may be related to hypometabolism in left prefrontal and temporal regions during episode of TGA

Key words: transient global amnesia; implicit/explicit memory dissociation; oxygen metabolism; PET

Scan, EEG and related measures

CT, ECG, EEG, PET, MRI

Standardized assessment

Batterie d'Efficienc Cognitive, Batterie d'Efficienc Mnésique, Raven's Progressive Matrices, Wechsler Memory Scale, Rey Complex Figure

Other assessment

Skill learning, word-stem completion

Lesion location

- CT, ECG, EEG, MRI: normal; PET: hypometabolism in left frontotemporal regions, lenticular nucleus and occipital lobe

Lesion type

Not known

Language

English

Episodic memory in transient global amnesia: encoding, storage, or retrieval deficit?

F. Eustache, B. Desgranges, P. Laville, B. Guillery, C. Lalevee, S. Schaeffer, V. de la Sayette, S. Iglesias, J. C. Baron and F. Viader

Abstract

Assessed episodic memory during the acute phase of transient global amnesia to differentiate an encoding, a storage, or a retrieval deficit in three patients (aged 54–71 years), whose amnesic episode fulfilled all criteria for transient global amnesia. A neuropsychological protocol was administered that included a word learning task. Results show that an encoding deficit was present in one patient, and a storage deficit was present in the two other patients. It is concluded that the encoding/storage impairment concerning anterograde amnesia documented in these patients stands in clear contrast with the impairment in retrieval which must underlie the retrograde amnesia that also characterizes transient global amnesia. This dissociation favours the idea of a functional independence among the cognitive mechanisms that subserve episodic memory.

Journal

Journal of Neurology, Neurosurgery and Psychiatry 1999; 66: 148–54

Neurocase Reference Number:

P699

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Patient A, B and C

Key theoretical issue

- In TGA, anterograde and retrograde amnesia may be caused by different mechanisms: anterograde by encoding and/or storage deficit, retrograde by impairment of retrieval

Key words: transient global amnesia; encoding; storage; retrieval; anterograde amnesia; retrograde amnesia; episodic memory

Scan, EEG and related measures

ECG, EEG, CT, MRI

Standardized assessment

Batterie d'Efficiency Cognitive

Other assessment

Digit span, category and letter fluency, episodic memory tests designed to differentiate encoding, storage and retrieval

Lesion location

- Patient A: ECG, CT: normal
- Patient B: EEG, ECG: normal; CT: mild diffuse atrophy; MRI: normal
- Patient C: ECG, CT: normal; EEG: bilateral frontotemporal slowing

Lesion type

Not known

Language

English

Transient semantic amnesia: a new syndrome?

J. R. Hodges

Abstract

Transient global amnesia describes a clinical syndrome characterized by the abrupt onset of severe amnesia usually accompanied by repetitive questioning, occurring in middle aged or elderly people, and lasting several hours. This report details, for the first time, a patient with transient loss of general (non-person specific) semantic memory. The patient, a 50-year-old male, had what seems to be a temporary reversible impairment of semantic memory with preservation of anterograde memory function.

Journal

Journal of Neurology, Neurosurgery and Psychiatry 1997; 63: 548–9

Neurocase Reference Number:

P700

Primary diagnosis of interest

Transient semantic amnesia

Author's designation of case

The patient

Key theoretical issue

- First report of a transient loss of general semantic memory with preservation of anterograde episodic memory

Key words: transient global amnesia; retrograde amnesia; semantic memory; episodic memory; temporal lobes; semantic dementia

Scan, EEG and related measures

CT, EEG

Standardized assessment

Category fluency, Digit span

Other assessment

Assessment of object naming, orientation, verbal anterograde memory, spontaneous language production

Lesion location

- CT, EEG: normal

Lesion type

Not known

Language

English

Haemorrhagic pituitary adenoma manifesting as transient global amnesia

Y. Honma and S. Nagao

Abstract

A 68-year-old female presented with recurrent transient global amnesia due to haemorrhagic prolactin-producing pituitary adenoma. Magnetic resonance imaging clearly revealed the anatomical relationship between the haematoma, within the parasellar tumour, and the compressed medial temporal lobe of the right (non-dominant) hemisphere. Within 4 weeks after the start of bromocriptine administration, the adenoma had markedly regressed and the affected temporal lobe was decompressed. She has experienced no further episode of transient global amnesia. Magnetic resonance imaging is recommended to detect latent organic lesions responsible for transient global amnesia, although the disorder is considered to be benign and of functional origin.

Journal

Neurologica Medica Chirurgica (Tokyo) 1996; 36: 234–6

Neurocase Reference Number:

P701

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Not specified

Key theoretical issue

- In some cases, TGA may be associated with intracranial lesion as documented using MRI evidence

Key words: transient global amnesia; pituitary adenoma; hippocampus; magnetic resonance imaging

Scan, EEG and related measures

CT, MRI

Standardized assessment

None specified

Other assessment

Questions about orientation, anterograde memory

Lesion location

- CT: parasellar mass; MRI: parasellar pituitary adenoma, compressing right medial temporal lobe

Lesion type

Prolactinoma

Language

English

Reversible hyperperfusion of the right medial temporal lobe in transient global amnesia

H. H. Jung, R. W. Baumgartner, J. M. Burgunder, J. P. Wielepp, S. Lourens and J. P. Wielepp

Abstract

Amnesia can facilitate memory performance: evidence from a patient with dissociated retrograde amnesia.

Journal

Journal of Neurology, Neurosurgery and Psychiatry 1996; 61: 654–5

Neurocase Reference Number:

P702

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- Increased perfusion of medial temporal lobe structures may be observed during TGA, perhaps due to the dynamic nature of seizure activity

Key words: transient global amnesia; retrograde amnesia; SPECT

Scan, EEG and related measures

EEG, MRI, SPECT

Standardized assessment

None mentioned

Other assessment

Assessment of orientation, verbal comprehension and production, fluency, anterograde and retrograde memory

Lesion location

- EEG, MRI: normal; SPECT: increased activity uptake in posterior caudal region of right medial temporal lobe

Lesion type

Not known

Language

English

Long-term perceptual priming in transient global amnesia

N. Kapur, P. Abbott, D. Footitt and J. Millar

Abstract

Presents the case of a 62-year-old female who displayed the classical features of transient global amnesia. During the episode, she completed a task that required perceptual identification of fragmented pictures over a number of learning trials. Seven days later, after recovery from the episode, she was required to identify the same fragmented pictures together with a new set of pictures that she had never seen before. She was significantly better at identifying the old pictures than the new pictures, in spite of having amnesia for the period of the attack. Four matched controls who had not seen either set of pictures before were also tested and performed at a similar level on the old and the new pictures. Findings demonstrate a residual capacity for long-term perceptual priming during an acute episode of apparent total loss of memory.

Journal

Brain and Cognition 1996; 31: 63–74

Neurocase Reference Number:

P703

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

JD

Key theoretical issue

- At level of perceptual priming, long-term consolidation can occur even during a transient episode of acute amnesia

Key words: transient global amnesia; perceptual priming; implicit memory; explicit memory; retrograde amnesia

Scan, EEG and related measures

MRI

Standardized assessment

Wechsler Memory Scale–Revised, Dead-or-Alive test, Wechsler Adult Intelligence Test–Revised

Other assessment

Orientation, memory for visual designs, picture naming, fragmented picture learning task, test of verbal recognition memory

Lesion location

- MRI: normal

Lesion type

Not known

Language

English

Recovery of function processes in human amnesia: evidence from transient global amnesia

N. Kapur, J. Millar, P. Abbott and M. Carter

Abstract

Examined some of the processes underlying recovery of memory function by attempting to answer the following questions: (1) does retrograde amnesia show the same pattern of shrinkage as anterograde amnesia; (2) do different anterograde memory functions recover at the same rate; and (3) does a form of Ribot's law govern the profile of retrograde amnesia, such that early memories are relatively spared compared to more recent memories. Evidence is offered from the perspective of a study of recovery of function during an episode of transient global amnesia (TGA; 29-year-old male) that occurred as a complication of a cerebral angiographic procedure. Shrinkage of anterograde and retrograde memory loss was plotted at four separate intervals throughout the acute recovery process, and also 24 h later. It is proposed that recovery from some types of human amnesia, such as that associated with TGA, follows a lateral-to-medial rule—lateral inferotemporal areas that play a major role in retrograde amnesia recover first from hypometabolism related to the TGA attack, followed by interface areas such as the rhinal and parahippocampal cortices, with the last areas to recover physiological integrity being discrete limbic diencephalic structures such as the hippocampus.

Journal

Neuropsychologia 1998; 36: 99–107

Neurocase Reference Number:

P704

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

SG

Key theoretical issue

- After an episode of TGA, recovery of retrograde memory preceded recovery of anterograde memory, perhaps reflecting earlier recovery from hypometabolism of lateral than medial temporal lobe structures

Key words: transient global amnesia; retrograde amnesia; anterograde amnesia

Scan, EEG and related measures

MRI

Standardized assessment

Rivermead Behavioural Memory Test, Dead-or-Alive Test, Verbal News Events Test, Wechsler Adult Intelligence Test–Revised, Wechsler Memory Scale–Revised, Warrington Recognition Memory Test, Rey Complex Figure, National Adult Reading Test

Other assessment

Tests of picture naming, orientation, Modified Card Sorting Test, verbal fluency

Lesion location

- MRI: (pre-morbid) left temporal arteriovenous malformation

Lesion type

Not known

Language

English

Retrograde amnesia during transient global amnesia

H. Kazui, H. Tanabe, M. Ikeda, M. Hashimoto, N. Yamada, J. Okuda and T. N. Wydell

Abstract

Two patients who met Hodges' clinical criteria for transient global amnesia (TGA) were given anterograde and retrograde memory tests during and after the attack. A SPECT scan was performed during TGA in one case, showing a reduced blood flow confined to the bilateral medial temporal lobes, which resolved on the next day. In both cases, the initial period of retrograde amnesia was within several years. In one case, autobiographical and public retrograde memory were assessed separately. These assessments revealed that autobiographical and public retrograde amnesias were impaired to the same extent during TGA. During recovery, almost all the events that had been forgotten gradually recovered according to a temporal gradient, apart from a single exceptional memory which had made a deep impression at the time of memorizing. In addition, during recovery both cases first remembered the existence of the events and second their contents. This finding indicates that the memory of events themselves may be more easily accessible than their contents. Even when the two patients had recovered from the retrograde amnesia almost completely, definite anterograde amnesia still remained. This implies that there exists a time in the recovery phase where anterograde amnesia is still prominent but retrograde amnesia has already almost disappeared, which may account for the previously reported variation in the extent of retrograde amnesia.

Journal

Neurocase 1996; 2: 127–33

Neurocase Reference Number:

P705

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

II and MY

Key theoretical issue

- Retrograde amnesia (autobiographical and public domain) and anterograde amnesia are dissociable during TGA

Key words: transient global amnesia; retrograde amnesia; public memory; autobiographical memory; hippocampal area

Scan, EEG and related measures

CT, MRI, SPECT, EEG

Standardized assessment

Wechsler Memory Scale–Revised

Other assessment

RMTvt

Lesion location

- Hippocampal area

Lesion type

Transient dysfunction, cause unknown

Language

English

Transient global amnesia at high altitude

J. A. Litch and R. A. Bishop

Abstract

Describes two cases of isolated, transient global amnesia at very high altitude. The first patient, a healthy 21-year-old man with no history of migraine, experienced amnesia after climbing to an altitude of 4400 m. A descent of 450 m brought gradual resolution of the amnesia. He was subsequently able to climb even higher without recurrence of symptoms. A 1-year follow-up examination showed he was in good health. The second patient, a 60-year-old man with no history of migraine, dementia, or cerebral vascular disease, exhibited symptoms of amnesia after climbing to a height of 3760 m. After a 760 m descent, the patient became fully oriented and the amnesia resolved. At 2-month follow-up the patient reported good health.

Journal

New England Journal of Medicine 1999; 340: 1444

Neurocase Reference Number:

P706

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Patient 1 and 2

Key theoretical issue

- Rapid ascent to very high altitude may trigger episode of TGA, resolving upon descent

Key words: transient global amnesia; retrograde amnesia

Scan, EEG and related measures

None specified

Standardized assessment

None specified

Other assessment

Questions relating to orientation, person recognition, semantic memory

Lesion location

- Not known

Lesion type

Not known

Language

English

High-resolution Tc-99m HMPAO SPECT in a patient with transient global amnesia

H. Matsuda, S. Higashi, S. Tsuji, H. Sumiya, T. Miyauchi, K. Hisada and J. Yamashita

Abstract

This is a report of a patient who underwent high-resolution brain perfusion SPECT studies during and after an episode of transient global amnesia. During the attack, SPECT imaging showed increased perfusion in the left medial temporal region involving the amygdala and hippocampus. After recovery from amnesia, a follow-up SPECT examination showed slightly decreased perfusion in this region. These findings support the hypothesis that transient global amnesia is associated with transient hyperperfusion in the medial temporal brain structures, and confirm the utility of high-resolution SPECT imaging for the evaluation of the fine details of functional brain anatomy.

Journal

Clinical Nuclear Medicine 1993; 18: 46–9

Neurocase Reference Number:

P707

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- TGA may be associated with transient increase in perfusion of medial temporal lobe regions

Key words: transient global amnesia; retrograde amnesia; SPECT

Scan, EEG and related measures

CT, MRI; EEG, SPECT

Standardized assessment

None specified

Other assessment

Assessment of retrograde and anterograde memory, verbal expression, comprehension, and repetition

Lesion location

- CT, MRI, EEG: normal; SPECT: hyperperfusion in left medial temporal lobe during attack; hypoperfusion after TGA resolved

Lesion type

Not known

Language

English

Transient global amnesia after cerebral angiography. A case report

J. F. Meder, I. Mourey-Gerosa, J. Blustajn, H. Lemaigen, B. Devau and D. Fredy

Abstract

We report on a case of transient global amnesia after cerebral angiography in which a non-ionic contrast medium was used. The complication observed showed no evidence of any definite cause.

Journal

Acta Radiologica 1997; 38: 273–4

Neurocase Reference Number:

P708

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- Description of occurrence of TGA following cerebral angiography involving a non-ionic contrast agent

Key words: transient global amnesia; cerebral angiography

Scan, EEG and related measures

CT, EEG, MRI

Standardized assessment

None specified

Other assessment

Unspecified assessment leading to diagnosis of TGA

Lesion location

- CT, EEG, MRI: normal

Lesion type

Not known

Language

English

Transient global amnesia of epileptic origin accompanied by fever

R. Meo, L. Bilo, S. Striano, P. Ruosi,
A. Estraneo and C. Nocerino

Abstract

The case of a previously healthy 69-year-old female patient is described who presented, in a period of 6 months, three long-lasting (from 2 h- to 10 h-duration) episodes of transient global amnesia accompanied by a temperature rise. During one of these episodes an EEG was obtained, showing a diffuse alteration, focal slowing, and bitemporal asynchronous paroxysmal activity giving rise to electrical ictal discharges. Interictal EEGs were normal. Cerebral computed tomography was normal. Carbamazepine was given with complete control of the attacks. These episodes may be interpreted as complex partial status with unusual semeiology.

Journal

Seizure 1995; 4: 311–7

Neurocase Reference Number:

P709

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- Episodes of amnesia with fever, especially in an elderly patient, may mark the onset of epilepsy

Key words: transient global amnesia; retrograde amnesia

Scan, EEG and related measures

CT, ECG, EEG

Standardized assessment

None specified

Other assessment

Questions related to retrograde and anterograde memory, orientation, praxis

Lesion location

- CT: normal; ECG: suggested inferolateral myocardial ischaemia; EEG: abnormal activity mainly in frontal and left temporal areas during episode. After TGA resolved, rare small spikes in left temporal areas

Lesion type

Not known

Language

English

Emotional arousal-induced transient global amnesia: a clue to the neural transcription of emotion?

A. E. Merriam, B. Wyszynski and T. Betzler

Abstract

Reports on a case of a 59-year-old woman with transient global amnesia (TGA). Triggered by an emotionally stressful event, the TGA spontaneously remitted within 18 h. It is suggested that the benzodiazepine (BZD) system is a candidate for the neurological substrate of this variety of TGA. Intense emotional events may have the capacity in susceptible individuals to perturb the function of the brain's normal BZD system and thereby induce a transient amnesic syndrome similar to that encountered in individuals who have ingested BZD compounds.

Journal

Psychosomatics 1992; 33: 109–13

Neurocase Reference Number:

P710

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- TGA triggered by emotional events has a distinct aetiology, perhaps involving the benzodiazepine system

Key words: transient global amnesia; retrograde amnesia

Scan, EEG and related measures

CT, MRI, EEG

Standardized assessment

None specified

Other assessment

Assessment of orientation, anterograde and retrograde memory, short-term memory, digit span, arithmetic, diagram copying, object naming, language comprehension, production and repetition

Lesion location

- CT, MRI: normal; EEG: fast activity, no focal features

Lesion type

Not known

Language

English

Transient global amnesia and cerebral infarct: a case report

R. Raffaele, C. Tornali, A. A. Genazzani and I. Vecchio

Abstract

Discusses the relationship between transient global amnesia (TGA) and cerebral infarction (CI), through a case report. TGA refers to a sudden and isolated dysfunction of memory for recent events, lasting a few hours. Its pathogenesis is still uncertain, though attempts to explain the aetiology of TGA have been made using the epileptogenic, migraineous and ischaemic hypothesis. The case of a 59-year-old female is presented, who was diagnosed to be suffering from CI associated with computed tomography (CT) evidence for a hypodense area in left thalamus, 10 days after the transient memory dysfunctions. Clinical evidence for diagnosis of TGA consisted of reversibility of the syndrome, inability to lay down new memories during the attack, variable retrograde amnesia, amnesic lacuna persisting after the episode, and resolution within 24 h. It is concluded that TGA can be the isolated manifestation of a cerebrovascular event involving diencephalic structures.

Journal

Brain Injury 1995; 9: 815–8

Neurocase Reference Number:

P711

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Not specified

Key theoretical issue

- TGA can result from ischaemic lesion to diencephalic regions in the absence of other causative factors (e.g. epilepsy, migraine)

Key words: transient global amnesia; retrograde amnesia

Scan, EEG and related measures

ECG, EEG, CT

Standardized assessment

Wechsler Adult Intelligence Scale–Revised, Temporal Orientation Test, Rey Auditory Visual Learning Test, Wechsler Memory Scale, Boston Famous Faces Test, Corsi's Cube Test, Benton Visual Retention Test, Stroop Test

Other assessment

Other tests of memory, orientation and executive function

Lesion location

- ECG: normal; EEG: some left hemisphere abnormalities; CT: focal lesion in left thalamus; 3 months later: normal

Lesion type

Presumed ischaemia

Language

English

Transient global amnesia: a complication of incremental exercise testing

R. S. Richardson, B. T. Leek, P. D. Wagner and M. Kritchevsky

Abstract

Incremental exercise testing is routinely used for diagnosis, rehabilitation, health screening, and research. The authors report the case of a 71-year-old patient with chronic obstructive pulmonary disease who suffered an episode of transient global amnesia (TGA) several minutes after successfully completing an incremental exercise test on a cycle ergometer. TGA, which is known to be precipitated by physical or emotional stress in about one-third of cases, is a transient neurological disorder in which memory impairment is the prominent deficit. TGA has a benign course and requires no treatment although 24-h observation is recommended. Recognition of TGA as a potential complication of incremental graded exercise testing is important to both aid diagnosis of the amnesia and to spare a patient unnecessary evaluation

Journal

Medicine and Science in Sports and Exercise 1998; 30: S403–5

Neurocase Reference Number:

P712

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- TGA can be precipitated by incremental exercise testing

Key words: transient global amnesia; retrograde amnesia; stress

Scan, EEG and related measures

None specified

Standardized assessment

None specified

Other assessment

Assessment of orientation, anterograde and retrograde memory for verbal and non-verbal material

Lesion location

- Not known

Lesion type

Not known

Language

English

Alterations of peak distribution of auditory ERPs during transient global amnesia: a case report

M. Saito, T. Ishida and M. Asai

Abstract

Conducted an auditory event-related potential study on a 51-year-old right-handed man during and 2 days after an incident of transient global amnesia. These findings are described, including the alterations of amplitude and peak distribution of scalp event-related potentials, which may provide a new viewpoint in the field of transient global amnesia research. The alteration of peak distribution that occurred to the patient may reflect changes in non-specific factors such as affective state, circadian rhythm, blood pressure, and food; however, the frontally distributed P3 is unique in that it is completely relevant to task. This finding suggests that, during the episode, habituation of orienting response was so attenuated that the anterior component of P3 did not decrease throughout the event-related potential session. Presumably, impairment of recognition memory may explain this attenuation of habituation.

Journal

Biological Psychiatry 1997; 41: 237–40

Neurocase Reference Number:

P713

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Not specified

Key theoretical issue

- Observation of ERP abnormalities in a patient with TGA

Key words: transient global amnesia; event-related potentials; orienting response

Scan, EEG and related measures

EEG, CT, ERP

Standardized assessment

Mini-Mental State Examination

Other assessment

Digit span, Syllables memory test

Lesion location

- EEG, CT: normal; ERP: increased frontally distributed P3

Lesion type

Not known

Language

English

Transient global amnesia in a young child

E. P. Silberstein

Abstract

Unprovoked and unexplained sudden loss of memory and inappropriate behaviour in a 5-year-old child is described as fitting into the clinical picture of 'transient global amnesia'. The likely pathophysiology of this condition is discussed with some support for Fisher's suggestion of 'hippocampal-fornical' dysfunction.

Journal

Journal of Paediatrics and Child Health 1994; 30: 366–7

Neurocase Reference Number:

P714

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- Rarely described occurrence of TGA in early childhood

Key words: transient global amnesia

Scan, EEG and related measures

CT, EEG

Standardized assessment

None specified

Other assessment

Questions related to orientation, anterograde and retrograde memory

Lesion location

- CT: normal; EEG: isolated mid-temporal spikes

Lesion type

Not known

Language

English

Diffusion-weighted MRI in transient global amnesia: elevated signal intensity in the left mesial temporal lobe in seven of ten patients

M. Strupp, R. Brüning, R. H. Wu, M. Deimling, M. Reiser and T. Brandt

Abstract

Prompted by the findings of previous studies with positron emission tomography and single photon emission computed tomography, which demonstrated hypoperfusion or hyperperfusion in the left temporal lobe in isolated patients with transient global amnesia (TGA), the authors compared the sensitivity of diffusion-weighted (DW) magnetic resonance imaging (MRI) with that of conventional T1- and T2-weighted MRI in patients with TGA. Ten patients with the typical syndrome of a pure TGA were included in the study. For all patients, a coronal DW sequence, a steady-state free precession (SSFP) sequence, and conventional T1- and T2-weighted turbo spin-echo sequences were obtained. Seven of the 10 patients had elevated signal intensity in the left hippocampal region on DW MRI; moreover, three of these seven patients exhibited bilateral signal abnormality in this sequence. All conventional T1- and T2-weighted images as well as all follow-up studies were normal. The signal elevation in DW MRI correlates with a decrease in the interstitial space and with cellular oedema in the temporal lobe during TGA. The underlying pathomechanism causing this cellular oedema cannot be clearly outlined using DW MRI. The present data are, however, compatible with spreading depression. This is the first study to show that DW MRI is a sensitive method for evaluating TGA, especially in the early stage of the disease.

Journal

Annals of Neurology 1998; 43: 164–70

Neurocase Reference Number:

P715

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Cases 1–10

Key theoretical issue

- Signal changes in early TGA, identified using diffusion-weighted MRI, indicate local nerve cell oedema like that seen in spreading depression

Key words: transient global amnesia; diffusion-weighted magnetic resonance imaging; temporal lobe; hippocampus; spreading depression

Scan, EEG and related measures

T1-, T2- and diffusion-weighted MRI; EEG

Standardized assessment

None specified

Other assessment

None specified

Lesion location

- Conventional MRI: all cases normal; DWMRI: Cases 1–3: bilateral hippocampal; Cases 4–7: left hippocampal; Cases 8–10: normal

Lesion type

Presumed cellular oedema

Language

English

Resting and acetazolamide-challenged technetium-99m-ECD SPECT in transient global amnesia

R. Takeuchi, Y. Yonekura, H. Matsuda, Y. Nishimura, H. Tanaka, H. Ohta, H. Sakahara and J. Konishi

Abstract

Regional resting cerebral blood flow and vascular reserve in a patient with transient global amnesia (TGA) were evaluated during and after a TGA episode using 99mTc-ethyl cysteinate dimer (ECD). The patient had consecutive SPECT studies before and after acetazolamide (ACZ) administration with adjunctive radionuclide angiography using equal-volume-split 99mTc-ECD. SPECT study during TGA episode showed poor vasodilatory reactivity to ACZ in the left medial temporal region involving the hippocampus and resting hypoperfusion in the regions bilaterally. The resting hypoperfusion with reserved vasodilatory reactivity to ACZ also was seen in the bilateral thalami. Abnormal findings in these regions disappeared on the follow-up SPECT study 15 days after the onset. No previous SPECT evaluation of regional abnormalities of both haemodynamic reserve and resting perfusion during and after an episode of TGA has been reported

Journal

Journal of Nuclear Medicine 1998; 39: 1360–2

Neurocase Reference Number:

P716

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- Perfusion abnormalities during TGA may be observable using SPECT methodology

Key words: transient global amnesia; SPECT

Scan, EEG and related measures

CT, EEG, SPECT

Standardized assessment

None specified

Other assessment

Assessment of verbal comprehension and production, anterograde and retrograde memory

Lesion location

- CT, EEG: normal; SPECT: bilateral hypoperfusion in medial temporal lobes and thalami

Lesion type

Not known

Language

English

Hippocampal ischaemia in a patient who experienced transient global amnesia after undergoing cerebral angiography

M. Tanabe, T. Watanabe, M. Ishibashi, N. Hirano, S. Tabuchi and H. Takigawa

Abstract

It is well known that transient global amnesia (TGA) is a complication of cerebral angiography. The authors describe a case of TGA with ischaemia of the bilateral hippocampi that was induced by angiography. A 43-year-old ambidextrous woman was referred for treatment of falx meningioma, which had been found incidentally during an examination for headache. The patient had no history of epilepsy.

Journal

Journal of Neurosurgery 1999; 91: 347

Neurocase Reference Number:

P717

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Not mentioned

Key theoretical issue

- Bilateral hippocampal ischaemia at the time of a TGA episode may be observed using diffusion-weighted MRI

Key words: transient global amnesia; retrograde amnesia

Scan, EEG and related measures

CT, MRI

Standardized assessment

None mentioned

Other assessment

Observations of orientation, anterograde and retrograde memories

Lesion location

- CT: normal; T1- and T2-weighted MRI: normal; diffusion-weighted MRI: bilateral hippocampal lesions

Lesion type

Presumed ischaemia

Language

English

Transient global amnesia and migraine in young people

L. Tosi and C. A. Righetti

Abstract

Describes two cases of transient global amnesia (TGA), one in a 16-year-old male and one in a 13-year-old female. Both cases occurred during a competitive sport and were associated with migraine and a 4- and 3-h amnesic gap, respectively. Neurological examinations and EEGs were used to rule out the possibility of epilepsy. The authors believe that TGA in young people could provide crucial information on the pathogenesis and they suggest that a migrainous mechanism is likely to underlie TGA in young people and 'pure' TGA in general.

Journal

Clinical Neurology and Neurosurgery 1997; 99: 63–5

Neurocase Reference Number:

P718

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

Cases 1 and 2

Key theoretical issue

- Because epilepsy and vascular precipitants could be ruled out, it is likely that a migrainous mechanism was the cause of these patients' episodes of TGA

Key words: transient global amnesia; retrograde amnesia

Scan, EEG and related measures

EEG, CT

Standardized assessment

None mentioned

Other assessment

Observations related to recent and remote memory, anterograde memory, orientation

Lesion location

- EEG, CT: normal

Lesion type

Not known

Language

English

Transient autobiographic amnesia: EEG and single-photon emission CT evidence of an organic aetiology

A. Venneri and P. Caffarra

Abstract

The authors describe a 44-year-old patient who had a transient attack of autobiographic amnesia. When assessed during the attack, her learning abilities were normal, with no sign of anterograde amnesia. In the remote memory domain, she showed a striking dissociation between a detailed knowledge of public events and famous people and a complete loss of autobiographic information. During the attack, EEG recorded bilateral frontotemporal slow waves and single-photon emission CT (SPECT) showed hypoperfusion in the right temporal and parietal lobes; no abnormalities were detected when both EEG and SPECT were repeated 1 week later. This case provides evidence for an organic aetiology for the episode and supports the hypothesis that autobiographic memory is independent of other forms of retrograde memory.

Journal

Neurology 1998; 50: 186–91

Neurocase Reference Number:

P719

Primary diagnosis of interest

Transient autobiographic amnesia

Author's designation of case

TB

Key theoretical issue

- This case provides evidence of a selective transient deficit of autobiographical memory, with preservation of public event and famous person knowledge and anterograde episodic memory

Key words: transient global amnesia; retrograde amnesia; autobiographical memory; SPECT

Scan, EEG and related measures

EEG, SPECT

Standardized assessment

Raven's Progressive Matrices, Borrini autobiographical memory questionnaire

Other assessment

Tests of digit span, visuospatial span, prose memory, paired associate learning, supra-span spatial learning, attention and language. Visual object naming and description, naming to description. Famous faces recognition test. Knowledge of public events

Lesion location

- EEG: bilateral frontotemporal slowing; SPECT: right temporal and parietal hypoperfusion

Lesion type

Not known

Language

English

Transient global amnesia triggered by mild head injury

A. Venneri, M. Brazzelli and S. Della-Sala

Abstract

An episode of transient global memory loss was observed in a 27-year-old woman following a mild head injury in a car accident. Her clinical and neuropsychological profiles were indistinguishable from those of transient global amnesia. This paper argues that a cause-effect relationship may be postulated between head trauma and transient memory loss, perhaps as the result of a very stressful situation such as a car crash.

Journal

Brain Injury 1998; 12: 605–12

Neurocase Reference Number:

P720

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

ER

Key theoretical issue

- Mild head trauma, although not causing observable brain damage, may temporarily disrupt memory mechanisms, leading to TGA

Key words: transient global amnesia

Scan, EEG and related measures

EEG, CT

Standardized assessment

Raven's Progressive Matrices

Other assessment

Disyllabic serial recall, picture naming, category fluency. Assessment of recent and remote autobiographical memory, verbal and spatial span, supra-span verbal and spatial learning, story recall, famous event knowledge, digit cancellation

Lesion location

- EEG, CT: normal

Lesion type

Not known

Language

English

Failure to recall (but not to remember): pure transient amnesia during non-convulsive status epilepticus

P. Vuilleumier, P. A. Despland and F. Regli

Abstract

The authors report a patient with a generalized frontal-predominant non-convulsive status epilepticus without clinically apparent altered consciousness. The patient was examined and EEG performed during and after the episode. Severe retrograde and anterograde amnesia during the seizure, contrasting with a preservation of ongoing memory formation that could be assessed only after its resolution, suggests a transient disconnection of access to stored representations. This unusual memory disorder is both clinically and electrophysiologically dissimilar to other reported cases of transient epileptic amnesia. Although the patient probably had numerous episodes previously, there was no history of overt seizure.

Journal

Neurology 1996; 46: 1036–9

Neurocase Reference Number:

P721

Primary diagnosis of interest

Transient epileptic amnesia

Author's designation of case

The patient

Key theoretical issue

- Transient inability to access memory traces without loss of ongoing acquisition of new memories emphasizes that retrieval and encoding processes can be dissociated from one another

Key words: transient global amnesia; transient epileptic amnesia

Scan, EEG and related measures

EEG

Standardized assessment

Poppelreuter Test; Hooper Visual Organization Test; Copy of Rey Complex Figure

Other assessment

Digit span, word recall and recognition, questions related to retrograde memories (autobiographical, public events, famous faces, famous places), visual naming, category fluency, reading, calculation, visuospatial abilities, alternating gestures and go-no-go tests

Lesion location

- EEG: bilateral frontotemporal spikes

Lesion type

Not known

Language

English

Diffusion-weighted MRI in transient global amnesia precipitated by cerebral angiography

A. R. Woolfenden, M. W. O'Brien, R. E. Schwartzberg, A. M. Norbash and D. C. Tong

Abstract

BACKGROUND: Transient global amnesia is a well-described complication of cerebral angiography. Speculation about the pathophysiology exists but is as yet unsubstantiated. Diffusion-weighted MRI is a new imaging technique that is very sensitive in detecting acute ischaemia. Its use in the evaluation of transient amnesia precipitated by cerebral angiography has not previously been reported. **CASE DESCRIPTION:** A 44-year-old man underwent posterior circulation cerebral angiography for the investigation of episodic vertigo. Shortly after completion of the procedure, he was noted to have symptoms of transient global amnesia. Diffusion-weighted MRI at 6 and 44 h after the procedure demonstrated increased signal in the right hippocampus and other areas within the posterior circulation bilaterally consistent with ischaemia from emboli. Abnormalities on conventional MRI images performed at the same time points were noted only in retrospect. A follow-up MRI at 2 months was normal. **CONCLUSIONS:** Ischaemia from cerebral emboli may cause transient global amnesia precipitated by cerebral angiography. Diffusion-weighted MRI may be useful in defining the pathophysiology.

Journal

Stroke 1997; 28: 2311–4

Neurocase Reference Number:

P722

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- Cerebral angiography may precipitate TGA if embolic ischaemia, observable in some cases using diffusion-weighted MRI, are present

Key words: transient global amnesia; retrograde amnesia; cerebral angiography; diagnostic imaging

Scan, EEG and related measures

MRI

Standardized assessment

None specified

Other assessment

Assessment of orientation, attention, immediate recall, anterograde verbal and non-verbal memory, recent and remote retrograde memory

Lesion location

- T1- and T2-weighted MRI: normal; diffusion-weighted MRI: right hippocampus hyperintensity (also right thalamus and left temporal lobe?)

Lesion type

Presumed ischaemia

Language

English

Transient epileptic amnesia: a description of the clinical and neuropsychological features in 10 cases and a review of the literature

A. Z. J. Zeman, S. J. Boniface and J. R. Hodges

Abstract

To clarify the clinical and neuropsychological aspects of transient epileptic amnesia (TEA), 10 cases were studied and 21 previously published cases reviewed. TEA episodes are brief, lasting less than 1 h, and recurrent, with a mean frequency of three a year. Although repetitive questioning commonly occurs during attacks, the anterograde amnesia is often incomplete so that patient may later be able to 'remember not being able to remember'. The extent of the retrograde amnesia during attacks varies from days to years. Epileptiform abnormalities are most often detected on interictal sleep EEG. Most patients experience other seizure types compatible with an origin in the temporal lobes, but transient amnesia is the only manifestation of epilepsy in about one-third of patients. The authors propose tentative diagnostic criteria for the diagnosis of TEA.

Journal

Journal of Neurology, Neurosurgery and Psychiatry 1998; 64: 435–43

Neurocase Reference Number:

P723

Primary diagnosis of interest

Transient epileptic amnesia

Author's designation of case

Cases 1–10

Key theoretical issue

- TEA is an identifiable syndrome and comprises episodic transient amnesia with an epileptic basis, without impairment of other aspects of cognitive function

Key words: transient global amnesia; transient epileptic amnesia; retrograde amnesia; SPECT

Scan, EEG and related measures

EEG, MRI, CT, SPECT

Standardized assessment

National Adult Reading Test, Wechsler Memory Scale–Revised, Rey–Osterrieth Figure, Warrington Recognition Memory Test, Famous Faces Test, Famous Names Test, Autobiographical Memory Interview

Other assessment

Observations related to retrograde and anterograde amnesia, topographical amnesia, psychiatric history

Lesion location

- EEG: Cases 1, 8–10: normal; Case 2, 3, 5: bitemporal changes; Case 4: left temporal abnormality; Cases 6 and 7: right temporal abnormality; MRI: Cases 1 and 2: normal; Case 5: posterior corpus callosum; Case 10: right hippocampus; CT: Case 3, 4, 6–9: normal; SPECT: Cases 1 and 3: minor frontal hypoperfusion

Lesion type

Case 5: infarction; Case 10: atrophy; Cases 1–4, 6–9: not known

Language

English

Proton magnetic resonance spectroscopy during transient global amnesia

M. Zorzon, R. Longo, G. Mase, E. Biasutti, B. Vitrani and G. Cazzato

Abstract

There is uncertainty about the aetiology of transient global amnesia and none of the pathogenetic hypotheses proposed so far, i.e. transient ischaemia, epileptic discharge and spreading depression of cortical electrical activity, is completely satisfactory. Using water suppressed proton magnetic resonance spectroscopy we studied one patient during a typical episode of transient global amnesia and 2 weeks thereafter in order to investigate the metabolic changes in the hippocampal region. In both hippocampi, spectra of *N*-acetyl-aspartate, creatine-phosphocreatine, compounds containing choline and lactate failed to show changes consistent with cerebral ischaemia, both in the acute phase and in the follow-up. Spreading depression in response to emotional stress seems a likely explanation in this patient, who suffered from migraine in the past.

Journal

Journal of Neurological Science 1998; 156: 78–82

Neurocase Reference Number:

P724

Primary diagnosis of interest

Transient global amnesia

Author's designation of case

The patient

Key theoretical issue

- Study of metabolic changes during acute phase of TGA failed to show spectra consistent with an ischaemic aetiology

Key words: transient global amnesia; proton magnetic resonance spectroscopy

Scan, EEG and related measures

ECG, EEG, MRI, proton nuclear magnetic resonance spectroscopy

Standardized assessment

None

Other assessment

Assessment of remote and recent retrograde memory, anterograde memory, language

Lesion location

- ECG, EEG: normal; MRI: diffuse white matter hyperintensity; proton MRS: no metabolite changes in hippocampi

Lesion type

Not known

Language

English