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Transient Amnesic Syndromes

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Transient global amnesia is a transient, benign neurological syndrome, characterized by global loss of memory, preserved consciousness and self-awareness, associated with some behavioral changes (in particular, repetitive questioning). It generally resolves within 24 hs. Mild brain stem symptoms can often be demonstrated during the attack, but major neurological abnormalities never occur. The only sequel is a permanent amnesic gap for the duration of the episode. The precise pathophysiology of TGA is not clear. On positron emission tomography (PET) and diffusion-weighted MRI (DWI), blood flow to specific brain areas that involve memory appears to be disrupted transiently during TGA. This includes the thalamus and/or mesial temporal structures (in particular the amygdala and hippocampus). The incidence of TGA is somewhere between 5 and 30 per 100,000, depending on age and the population studied. More than 1000 cases have been reported in the literature. The disorder occurs most commonly in persons between 50 and 70 years of age, and about 50% of TGA patients are hypertensive. Dizziness, headache, drowsiness, nausea, or vomiting are occasionally noted during attacks, but illusions, delusions, hallucinations, and seizures are not. Reported possible precipitating factors include emotionally intense or stressful events, sexual intercourse, physical exertion, painful stimuli, bathing, heat exposure, or Valsalva's maneuver. TGA has also been reported in association with cerebral angiography, CNS infection, polycythemia, cardiac valvular disease, exposure to altitude, and heparin-induced thrombocytopenia. When a patient with suspected TGA is evaluated, one must rule out other causes of CNS dysfunction or amnesia such as head injury, epilepsy, brain tumor, CVA, Wernicke-Korsakoff syndrome, migraine, CVA, meningitis, encephalitis, hypoglycemia, medications (eg, benzodiazepines), drugs of abuse, and psychogenic fugue state. TGA is generally a benign condition with an excellent prognosis. Overnight hospitalization has been recommended for repeated neurological examinations and observation until the episode has resolved. No specific treatment is recommended. It might be speculated that anti-migraine agents might be useful DURING an attack, but this is at present not supported by any data. Likewise, low-dose aspirin after the attack is probably reasonable anyway in most patients in the TGA patient's typical age range.

Amnesia episodes lacking other cognitive or ictal phenomena and attributable to temporal lobe epilepsy have been interpreted as a distinct entity termed "Transient Epileptic Amnesia" (TEA). Transient epileptic amnesia (TEA) is a recently described condition, in which temporal lobe epilepsy manifests as episodes of amnesia, often without other cognitive or ictal phenomena The main characteristics of TEA are as follows:[1] recurrent episodes of sudden memory loss which are usually brief in duration (typically less than an hour);[2] the occurrence of amnesic episodes on waking from a period of sleep;[3] a disproportionately severe retrograde component to some attacks, so that patients may 'remember not being able to remember';[4] evidence of inter-ictal EEG abnormality in the temporal lobe; and [5] the reduction or cessation of amnesic episodes by the introduction of anti-epileptic medication.

In a recent study (Zeman et al, 1998), seven out of ten patients with TEA complained of a distinctive and unusual variety of persistent amnesia for salient life events. This amnesia amounted to a partial, but substantial, depletion of the patients' stock of autobiographical recollections. In five of these patients the retrograde amnesia affected their recall for periods of their lives preceding the clinical onset of epilepsy, sometimes by as much as 30 years. Their amnesia often became evident when looking at holiday albums or discussing other highly salient life events with family members. These difficulties with remote memory contrasted with the patients' normal performance on standard anterograde memory tests. Despite the symptoms of remote memory loss, the patients performed well on tests of famous faces and events suggesting that the deficit might selectively affect autobiographical memory.

We performed a single-case study of a 68-years-old man (R.G.) with TEA, who reported very striking autobiographical memory gaps (Manes et al 2001). R.G. underwent a battery of tests of anterograde and remote memory including knowledge of people, events and his own autobiography. He was unable to evoke detailed recollections from a larger part of his life. In contrast, he performed well on tests of new learning and general knowledge and had good personal semantic information about his past. We have replicated this finding in a group study and shown, in addition, that such patients have accelerated forgetting or what has sometimes been termed delayed-onset amnesia.

We have shown, in a group study (Manes et al. JNNP in press), that patients with TEA exhibit clear deficits in autobiographical memory, as measured by the Crovitz-Galton method, and a pattern of accelerated forgetting on tests of long-term recall of verbal material. We showed that an individual may perform well on standard tests of new learning, but have marked difficulty retrieving information over a more extended period (6 weeks).

Transient global amnesia (TGA) is distinguished from TEA by a number of features. Episodes of TGA typically last for hours with gradual resolution. They frequently follow physical exertion or emotional stress, and are rarely recurrent. During the attack both TGA and TEA patients are densely amnesic and repetitive but in TGA the retrograde amnesia is temporally limited. In TEA, by contrast, retrograde memory loss may predominate. Recovery from TGA is protracted with minimal, if any, persistent cognitive impairment but TEA appears to be associated with both, accelerated forgetting of new material and an extensive loss of remote personal memory. In this conference the clinical, neuropsychological and pathophysiology features and the management of TGA and TEA will be discussed.

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