Transient Global Amnesia Following General Anesthesia

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Transient global amnesia (TGA) is characterized by sudden, transient loss of memory for recent events with preservation of personal identity, immediate recall, and remote memory.1,2 TGA usually occurs in middle-aged or older patients3 and can last for minutes to hours but no longer than 1 day.3 Other neurologic deficits are absent,2 the electroencephalogram (EEG) usually although not always remains unchanged,4 and no systemic abnormalities are detected. The cause of the disorder is believed to be episodic vascular insufficiency of the medial temporal lobes.5 TGA has been associated with migraine headaches,5,6 intracerebral tumor,7 diazepam overdose,8 and cardiac dysrhythmias,8 and has been described after cerebral angiography9 and spinal anesthesia.10 Following is the description of a patient who exhibited the characteristic signs and symptoms of TGA for several hours following a general anesthetic for a neurosurgical procedure.

REPORT OF A CASE

The patient is a 60-year-old man with trigeminal neuralgia involving primarily the distribution of the maxillary portion of the left trigeminal nerve. He had received medical therapy and underwent a microvascular decompression of the fifth cranial nerve 2 years previously, but neither provided permanent pain relief. He received general anesthesia for his previous surgery, and his postoperative course was reportedly uncomplicated. A repeat microvascular decompression was planned for this hospitalization.

His history included hypertension well controlled by diuretic therapy, asymptomatic peptic ulcer disease treated with cimetidine, and a 20 pack-year smoking history. An old inferior wall myocardial infarction and first degree atrioventricular block were present. Complete blood count, serum electrolytes, and urinalysis were within normal limits.

The patient received diazepam 10 mg po with his usual morning medications including carbamazepine, Dyzide®, and cimetidine 1.5 h prior to transport to the operating room. A peripheral intravenous catheter, a radial arterial line, and a central venous line were inserted prior to induction of anesthesia; correct placement of the tip of the central venous catheter was confirmed by chest roentgenogram.

Anesthesia was induced with thiopental 275 mg and fentanyl 150 μg iv with an FIO₂ of 1.0. Intubation of the trachea was performed following atracurium 0.5 mg/kg iv. The patient’s legs were wrapped with elastic bandages, and the head of the table gradually elevated to 80 degrees. The patient’s neck was slightly flexed, care being taken to ensure that the airway as well as the cerebral venous pathways remained unobstructed. After the patient was placed in the seated position, a precordial Doppler transducer was applied and the accuracy of position checked by injecting 5 ml of saline through the central venous catheter. A left suboccipital craniectomy and microvascular decompression of the left trigeminal nerve were performed.

Anesthetic maintenance for the 4.5 h case included 50% nitrous oxide and 1% inspired isoflurane. End-tidal carbon dioxide was maintained at 23–26 mmHg until closure of the dura was begun; this corresponded to a Pao₂ of 29–32 mmHg. Repeated intraoperative analysis of arterial blood gases revealed adequate oxygenation. Vital signs remained stable with normotension and sinus rhythm throughout surgery, and urine output was approximately 1 ml·kg⁻¹·hr⁻¹. No surgical complications were encountered, and blood loss was minimal. At the conclusion of surgery, adequate reversal of neuromuscular
relaxation was achieved with neostigmine 2.5 mg and glycopyrrolate 0.5 mg IV, and the trachea was extubated in the operating room.

Upon arrival in the recovery room, the patient was alert and demonstrated no focal neurologic abnormalities but was obviously emotionally distressed and verbalized his confusion as to place and time. He repeatedly asked “Where am I, and what day is it?” Although he was reassured and appeared to be reassured each time he asked, in a few minutes he would plaintively repeat the questions. He had no recollection of being admitted to the hospital 14 days previously or of events that had transpired during his hospitalization. His sense of personal identity was unimpaired, and he remembered such remote details as his address and the fact that he was divorced. Throughout his stay in the recovery room, the neurologic examination was unremarkable and laboratory studies including arterial blood gases, pH, complete blood count and serum electrolytes were normal. The amnesia and attendant emotional distress began to resolve in approximately 8 h. He was discharged to the Neurosurgical Intensive Care Unit 4 h after admission to the recovery room. Two hours later he was alert and fully oriented.

The following morning the patient was reevaluated and had no recall of his amnesic episode. He also demonstrated complete recovery of his memory loss. He has been followed for 8 months and has had no recurrence of his TGA, nor has he had other symptoms consistent with a transient cerebral ischemic attack (TIA).

DISCUSSION

TGA has not been previously reported after general anesthesia, perhaps because some degree of memory impairment is not infrequent following a general anesthetic. Amnesia for the postoperative period lasting for as long as 8 h or more has been reported.11 This amnesic period often is characterized by rational conversation and complaints about pain and other discomforts, but subsequently the individual has either no or only vague recollection of the incidents or has “islands” of memory.11 This occurs more frequently and is usually of longer duration in elderly patients than in the young. The immediate postanesthetic period is also characterized by an inability to recall new material after a time lapse of 1–30 min (i.e., delayed recall), while immediate recall and remote memory are not markedly affected.12,13 This makes the differentiation between postoperative amnesia and TGA somewhat difficult as it is also delayed recall that is affected during episodes of transient global amnesia, while immediate recall and remote memory remain intact except for a varying period of retrograde amnesia.6,14 A lengthy period of retrograde amnesia is not common following general anesthesia. Indeed Bahman et al.15 were unable to demonstrate any incidence of retrograde amnesia in healthy, young, unmedicated patients anesthetized with halothane, isoflurane, nitrous oxide, or thiopental.

The retrograde amnesia evidenced by this patient, which covered at least the previous 14 days of hospitalization, as well as his awareness of and the distress occasioned by his circumstances, and the frequent repetition of questions such as “Where am I?” are more characteristic of transient global amnesia than of the memory impairment, which often follows general anesthesia.3–5,11,14 The patient is in the age group in which TGA most often occurs, and he exhibited two of the most common risk factors associated with TGA, i.e., hypertension and ischemic cardiac disease.14

Some investigators have stressed the benign prognosis of TGA, with a low incidence of recurrence and an even lower incidence of progression to completed stroke.3,14,16 Others, however, cited the occurrence of repeated attacks,17,18 persistent defects in long-term memory and verbal IQ following even a single episode,19 and a relatively high incidence of recurrent transient ischemic attacks and stroke in patients followed after an episode of transient global amnesia,20 have emphasized the need for careful follow-up. Thus, it is important to attempt to determine the cause of an episode of TGA when it does occur so that appropriate therapy and follow-up can be initiated.

The mechanism of TGA is presumed to be transient vascular insufficiency in the vertebrobasilar system, leading to ischemia of the medial temporal lobes, particularly the hippocampus, mammillary bodies, fornix, and thalamus. The exact cause of the vascular insufficiency is often unknown.

Possible causes for TGA in the patient reported here might include drug effects, hypoxia, and decreased cerebral perfusion secondary to the seated position, obstruction of the extracranial cerebral vessels during flexion or extension of the neck, or surgical retraction. The patient was receiving cimetidine, which was given the morning of surgery along with diazepam. The syndrome of TGA has previously been reported in association with an overdose of diazepam,5 and cimetidine delays the plasma clearance of diazepam.21 Diazepam blood levels were not obtained in our patient, so this possibility cannot definitely be included or discarded.

Global cerebral ischemia—hypoxia can produce transient memory loss, as the hippocampus is extremely sensitive to hypoxia. The memory loss resolves, if at all, over a period of days rather than hours, however, distinguishing it from TGA.22 Arterial blood pressure and oxygenation were closely followed intraoperatively and postoperatively in our patient, and neither hypoxia nor hypotension was detected. However, undetected focal ischemia in the branches of the vertebrobasilar system occurred. Whether this resulted from direct pressure in the area secondary to retraction or to a focal reduction in perfusion pressure due to positioning, hyperventilation, or alterations in cerebral blood flow distribution secondary to the anesthetic agents is purely speculative. Care was taken to avoid extreme flexion or
extension of the neck, extreme hyperventilation was avoided, and the surgical approach was a standard one for the procedure in which no difficulty with exposure was experienced.

Fisher found precipitating events for 26 of 85 episodes of TGA in 78 patients. Of these, two occurred with stimulation of the trigeminal ganglion during a therapeutic procedure for trigeminal neuralgia. Fisher grouped these with painful stimuli as triggers for the syndrome. Our patient did not have his trigeminal ganglion stimulated purposefully, although some dissection near the nerve was performed and traction may have been exerted. While this should not have produced pain under general anesthesia, cardiovascular changes can occur with traction on the nerve under anesthesia. None were apparent in this case, but perhaps focal vascular changes occurred.

It would be difficult, although desirable, to monitor patients for the occurrence of TGA intraoperatively, since changes in the routine EEG are frequently not evident during the episode; recordings from nasopharyngeal electrodes appear to yield a higher percentage of abnormal EEG findings. At present there is no definitive therapy recommended for the patient having an episode of TGA, and indeed these, by definition, resolve spontaneously. In view of the findings of some series regarding the long-term results in such patients, they should be referred for appropriate follow-up so that further evaluation and therapy may be instituted if episodes recur or other symptoms of cerebral ischemia become evident.

In summary, this case demonstrates an unusual presentation of amnesia in the immediate postoperative period, with features more suggestive of transient global amnesia, which is thought to occur on a vascular basis, than of an amnesic effect of general anesthesia. While the precise cause for the TGA in this patient is not known, several possible causes have been discussed.

REFERENCES


