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# Transient Global Amnesia: Complication of Cerebral Angiography

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Transient global amnesia is a disorder of mentation in which there is an abrupt onset of memory loss and disorientation without change in consciousness. The disorder involves events and activities of the present and recent past, but leaves personal identification intact. There is no associated focal or sensory deficit [1]. An episode usually lasts for several hours. Memory returns to normal with residual amnesia for the period of time from the onset to recovery. No consistent precipitating factors have been reported. While series have been published associating the syndrome to a number of possible causative mechanisms [1-3], we know of no previous reports in the radiologic literature describing total global amnesia as a complication of cerebral angiography. Our experience with this syndrome forms the basis of this communication.

## Case Reports

### Case 1

A 27-year-old woman was referred for progressive neurologic symptoms of right-sided numbness, weakness, and "tingling." Symptoms of objective vertigo, marked proximal muscle weakness, and decreased coordination were noted. Her medical history and a general physical examination were unremarkable. Neurologic examination revealed right hemiparesis and subjective sensory disturbance. Unenhanced and enhanced computed tomography and metrizamide (CT) cisternography revealed a low density right cerebellopontine angle mass causing deformity of the brain stem and quadrigeminal plate cistern.

Cerebral angiography was performed from the femoral approach with a 5 French polyethylene catheter with frequent flushing with heparinized saline using a closed system. The right vertebral artery was catheterized and rapid serial filming was performed in the anteroposterior, lateral, and base projections. Three injections of Conray-60 were made at the rate of 6 ml/sec for a total of 9 ml. Subsequently, in catheterizing the left carotid artery, the left vertebral artery was transiently entered on two occasions and small test injections given. Separate anteroposterior and lateral filming of the left external carotid artery was performed with an injection rate of 2 ml/sec for a total of 7 ml. The left internal carotid artery was filmed in the anteroposterior and lateral projections, each with an

injection of 6 ml/sec for 11 ml. The patient received a total of 80 ml of Conray-60 including test injections.

Several minutes later, the patient looked at the examiners and asked who they were and where she was. She did not remember why she was in the hospital or why she was undergoing the study. Transient slurring of speech was noted which cleared immediately. The catheter was withdrawn on recognition of the complication.

Neurologic examination after angiography was unchanged with the exception of amnesia. The patient was confused and upset. She was unable to recall the date or year, to retain numbers, or recall the name of the hospital or objects shown to her for more than a few minutes. Her automatic memory regarding her age, name, and address was fully intact. Her preangiographic neurologic status returned within 24 hr.

Angiography revealed an avascular extraaxial mass in the right cerebellopontine angle. There were no findings of atherosclerosis, spasm, or emboli. Surgery revealed a cholesteatoma. She had an uneventful recovery with return of strength on the right and no further memory deficits.

### Case 2

A 62-year-old man had a right upper lobe squamous cell carcinoma. No metastatic disease was found. Four months after radiation therapy, he developed Jacksonian seizures of the right arm which progressed over a short period to right hemiparesis. Physical examination revealed right hemiparesis, right facial weakness, and dysarthria. Decadron was begun. A CT scan revealed a left parietal mass with surrounding edema and minimal mass effect.

As resection was contemplated, cerebral arteriography was performed via the femoral approach using a 5 French polyethylene catheter. The right common carotid, left common carotid, and left vertebral arteries were studied with a common carotid injection rate of 6 ml/sec for 12 ml. The vertebral artery was injected at a rate of 6 ml/sec for 9 ml. The total amount of contrast material (Conray-60) administered during this study was 80 ml with duration of examination being 55 min.

Shortly after retraction of the catheter, the patient began questioning the angiographic team as to where he was and who the examiners were. He was disoriented to time and place. He did not remember the angiographic procedure or the reason for his current admission. The patient did remember his prior hospitalization and the diagnosis of lung carcinoma. Even after careful explanation,

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confusion recurred within minutes. Automatic memory for name, age, and address were intact. He was fully alert. Neurologic examination in the angiographic suite revealed only his original neurologic deficit and amnesia. Several hours after the procedure, his retrograde amnesia resolved.

Angiographic findings demonstrated a left parietal avascular mass. No macroscopic emboli or spasm were noted. The patient received radiation therapy but died 2 months later of diffuse metastatic disease. An autopsy was not performed.

## Discussion

The neuroanatomy of amnesia is complex and controversial [4]. Bilateral lesions in the medial temporal lobe in humans produce a severe amnesic syndrome with permanent anterograde amnesia (impaired ability to acquire information, that is, to learn or form new memories [5]) and retrograde amnesia (impaired ability to recall events and other information that had been well established for a short period prior to the injury [6–8]). Early memories and memories for technical skills remain intact and there is no loss of general intelligence or complex perceptual abilities [7].

Scoville and Milner [7] found that medial temporal lobe resection produced memory defects only when the hippocampus was included. The pathologic studies of Korsakoff syndrome and posterior cerebral artery occlusion supported the hippocampus as an important structure in amnesic syndromes, however animal studies failed to support this interpretation [4]. Horel [4] recently reviewed the literature on amnesia and proposed that the hippocampus, fornix, and mamillary bodies are not the source of amnesia in brain injury. He proposes that the symptoms are produced by involvement of a system closely associated with the temporal neocortex. This includes the temporal cortex and its white matter extensions. These tracts connect the brain stem as well as the basal ganglia, medial thalamus, and orbitofrontal cortex. The vascular supply to this region is predominantly via the posterior cerebral artery.

Clinically, disorders of memory have been associated with a wide variety of conditions. Amnesia has been a manifestation of bilateral hippocampal infarction, trauma to the diencephalotemporal regions, spontaneous subarachnoid hemorrhage and Wernicke-Korsakoff syndrome, among many others [5, 9]. Amnesia of sudden onset and transitory duration occurs with temporal lobe seizures, electroconvulsive treatment, postconcussive states, and the syndrome of total global amnesia; all but the first and last are easily distinguishable on clinical grounds [5, 9].

Total global amnesia is a syndrome which has elicited numerous reports [9–16] including a detailed description of 17 patients by Fisher and Adams [1]. The clinical presentation in their cases is rather stereotyped. The patient is usually a man in the fifth or sixth decade who is suddenly unable to form new memories. He is unable to perform routine tasks, and characteristically shows concern over the disability, incessantly repeating the same questions. There is a striking inability to learn new material without impairment of remote memory. The patient is well aware of his own identity and the neurologic examination is unremarkable. The episode usually lasts from 30 min to several hours. After recovery, there is amnesia for the period of the attack. No

consistent precipitating factors have been noted [1, 2, 12].

In a recent series by Mathew and Meyer [3], 14 patients with transient global amnesia, aged 40–92 years, were studied for a mean interval of 30 months. Thirteen of their patients had one or more risk factor for cerebral vascular disease (hypertension, cardiac abnormalities, diabetes mellitus, and hyperlipidemia). Clinical evidence for vertebral basilar insufficiency was demonstrated in 11 cases. Cerebral angiography in 12 patients demonstrated lesions predominantly in the vertebral basilar system. In contradistinction to some earlier studies, eight of their patients had recurrent attacks. Other studies have also reported multiple attacks of total global amnesia [16, 17]. In the Mathew and Meyer [3] study, the patients with recurrent attacks showed permanent memory impairment on follow-up examination.

The type and incidence of complications secondary to catheter cerebral arteriography have been well described [18–21]. In a recent review of 5,000 cases, Mani et al. [18] noted that 43 (93%) of the 46 complications related to the central nervous system were transient. The major complications were visual, motor, or cerebellar.

The presumed mechanism for most complications has been embolism from the catheter and guide wire, atheromatous debris, or thrombus from the cardiac wall [18, 19]. A report of memory disturbance during angiography [22] described transient visual abnormalities in six with two cases of isolated memory disturbance. Two cases of total global amnesia occurred during coronary angiography while the catheter was in the aortic arch. Amnesia ensued presumably secondary to embolization of catheter thrombus after arterial pressure through the catheter became clamped and aspiration was performed to clear it [23]. No obvious problems in catheter technique were noted during our cases. Atheromatous emboli are a well known complication of catheter angiography [24, 25]. This is a possible cause in case 2.

The recent finding of intrinsic particles in five commonly used water-soluble contrast media in Europe suggests another possible source of emboli [26]. The multiplicity (the mean number of particles greater than 5  $\mu\text{m}$  was 302/ml contrast medium) of small emboli would more likely affect the territories supplied by both posterior cerebral arteries, the region thought by Horel [4] to be responsible for amnesia. Both our cases occurred within 10 days using the same lot of contrast media. Due to the retrospective recognition of this by separate angiographers, we did not have a chance to analyze the contrast agent. No other neurologic complications were noted in the time period where this lot of contrast material was used. Reviewing our clinical material for 10 years, no other cases of total global amnesia were identified. Spasm related to catheter manipulation or contrast material would seem unlikely as a mechanism in our patients, as spasm was not seen on the angiograms.

Toxic effect of contrast material on the brain was reported with early contrast media and very high-dose modern agents [27, 28]. Opening of interendothelial junctions at the capillary level with alteration in the blood-brain barrier and leakage of contrast material into the parenchyma [29] was noted. Microscopic findings included swelling of perivascular astrocytic filaments, ballooning of mitochondria, and lesions of myelin fibers [28].



Our two cases of an extremely rare, relatively localized neurologic deficit with a normal contrast load of a modern agent would mitigate against contrast toxicity as the cause. A unilateral lesion of one temporal region would more easily explain this syndrome resulting from one or several small emboli affecting the opposite side. However, CT scans revealed the temporal region to be grossly normal in both cases.

Of the three mechanisms postulated for total global amnesia (emboli, spasm, or contrast toxicity), we think emboli the most probable. On the basis of the neuroanatomy of amnesic syndrome, bilateral distribution is necessary. Two cases of total global amnesia within a 10 day period using the same lot of contrast material would suggest the latter as an agent. In light of the recent report regarding intrinsic particulate matter in contrast media, emboli offer the most plausible explanation for this phenomenon. More work needs to be done on contrast agents used in the United States to see if intrinsic particles are present in them, as has been described in Europe.

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