CASE REPORT

Transient global amnesia: Minor inconvenience or early warning sign?

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1. Introduction

Transient global amnesia (TGA) is a clinical diagnosis characterized by the sudden and reversible onset of anterograde amnesia accompanied by repetitive questioning without associated focal neurological deficits, usually occurring in the middle-aged or elderly population. Although TGA is relatively benign, underlying life-threatening medical conditions (e.g., myocardial infarction [1–3], dissecting aortic aneurysm [4,5], arrhythmias [6], tumor [7]) have been associated with TGA and should be considered to prevent catastrophic outcomes. We report a case of TGA with underlying non-ST-elevation myocardial infarction (NSTEMI).

2. Case report

A 69-year-old female with past medical history of hypertension being treated with metoprolol, presented with an acute episode of altered mental status. Emergency medical service was called once the staff at the facility noticed she was suddenly confused. She was noted as having a ''moment of memory lapse'', then appeared disoriented with repetitive queries. No other neurological symptoms or signs were reported. At baseline, she is fully conversant and entirely independent in activities of daily living. She recalled waking up in her usual state of health and going to the nearby senior center, as she frequently does for daytime activity since the passing of her husband this past year. She previously spent all her time caring for him because of his advanced Alzheimer's. She also reported increased stress recently due to her computer classes at a local public school. In addition, she had been having suboccipital pressure sensation and notably had migraines in the past, but denied any migraines or headaches for several years now. She also denied any dizziness, anxiety, nausea, paresthesia, chest pain, shortness of breath, abdominal pain, dysuria, or any other symptoms.

Upon arrival to the emergency department, she was found to be hypertensive to 171/79 mmHg. On examination, she continued perseveration of the same questions/statements (What happened? Where did I come from? Who brought me here?).

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Otherwise alert and oriented to person, place, and time with no aphasia or cognitive deficits. Physical examination revealed no focal neurological or epileptic signs or symptoms. A brain computed tomography without contrast showed diffuse cerebral atrophy, otherwise no acute findings or prior infarcts. Biological parameters and diagnostic tests ruled out metabolic disorders (i.e., hypoglycemia, hypocalcemia), infection, fluid/electrolyte derangement as underlying etiology of her acute presentation and the lack of disturbance of consciousness and altered cognition excluded delirium. However, her troponin was elevated to 0.79 ng/mL (normal $\leq$0.04 ng/mL). ASA was given and patient was admitted to medicine floor for elevated troponin. Repeat troponin began to trend up so she was placed on a heparin drip. Troponin peaked to 3.06 ng/mL and electrocardiogram revealed T-wave inversions in the anteroseptal leads (more prominent in V1) and mild ST-segment (<1 mm) elevations in the inferior leads, therefore cardiology was consulted.

Given the elevated troponin as a sign for stress and the preservation, neurology was consulted to evaluate for possible TGA. A neurologist evaluated the patient and she demonstrated poor short-term memory with anterograde amnesia. Immediate recall was 3/3; delayed recall 0/3 which improved to 2/3 after many prompts/guessing. According to neurology, the clinical picture was consistent with anterograde amnesia of short-term memory and given psychosocial stressors including recent loss of husband, and current occipital headache with prior history of migraines. TGA appeared most likely; however, neurology also considered the possibility of complex partial seizure(s), and could not rule out small new infarct.

On follow-up evaluation the next morning, 12 hours after presentation, the patient remained hemodynamically stable. When asked why she was brought to the hospital, she stated ''my mind wasn't working'' and was told that she was asking the same questions repeatedly. The patient remembered going to the senior center to practice a salsa dance performance for an upcoming event. She stated having a vague recollection of being there but had no idea how she got to the hospital or where her belongings were. On mini-mental status examination, she was awake, alert, and oriented to person, place, time, and could state her birthday but indicated that she was 48 years old. Her speech was fluent without dysarthria. She could name, repeat, and follow complex commands crossing the midline, able to say the months of the year in reverse, and did not repeat questions. Immediate recall was 3/3, delayed recall 1/3. The amnesic gap resolved within 18 hours from the onset of symptoms.

Close work-up unveiled non-ST-elevation myocardial infarction and the patient subsequently was admitted to the cardiac care unit where a transthoracic echocardiogram estimated global left ventricular ejection fraction at 25% with severe left ventricular systolic dysfunction and delayed diastolic relaxation. A coronary angiogram performed later demonstrated nonobstructive coronary artery disease.

Nearly 24 hours after the initial presentation; the patient appeared to be making new memories and returning to baseline. There was no indication for further neurologic workup. Troponin I down trended to 2.92 ng/mL and repeated electrocardiograms were unchanged from admission. She was safely discharged from the hospital on the 2nd day with routine post myocardial infarction treatment and follow-up.

3. Discussion

This case of TGA appeared to be secondary to an acute myocardial infarction. To date, three previous cases of TGA presenting along with or as a manifestation of acute myocardial infarction have been reported in the literature [1–3].

We report a case on a 69-year-old female with sudden onset altered mental status with anterograde amnesia and repetitive queries. Other than suboccipital headache, there were no focal neurological or epileptic deficits. There were no cardiac symptoms on admission or at any time during her hospital stay. She recovered from her amnestic state within 24 hours of presentation of symptoms and the findings were consistent with the diagnostic criteria for TGA as established by Caplan and Hodges [8]. Therefore, NSTEMI and psychosocial stressors leading to TGA was postulated.

The pathogenesis of transient global amnesia remains ambiguous and the leading hypotheses centers on ischemia, seizure/epileptic discharge, TIA, and a migrainous phenomenon as possible etiologies [9]. It was previously surmised that arterial thromboembolism in patients with vascular risk factors could have attributed to the ischemic events leading to TGA [10]. These cardiovascular risk factors potentiate events such as atrial fibrillation and myocardial infarction, which are associated with intracardiac thrombus formation, and are potential sources of emboli [11,12]. Although it is plausible that the left ventricular dysfunction

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