Letter to the Editor

Transient global amnesia complicating dobutamine–atropine stress echocardiography

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Pharmacological stress testing using the combination of dobutamine and atropine is a widely accepted standard diagnostic stress modality for evaluating underlying coronary artery disease. This is used in multiple cardiac imaging modalities, including stress echocardiography, nuclear perfusion imaging, and cardiac magnetic resonance imaging. Although rare, occasional neuropsychiatric symptoms, including confusion and disorientation have been reported following dobutamine–atropine stress testing [1]. Transient global amnesia (TGA) is a syndrome characterized by a sudden memory loss of recent and/or remote events with the transient inability to acquire new knowledge. We present a case of a woman who developed transient global amnesia after the completion of a dobutamine–atropine stress echocardiogram.

A 54-year old woman with history of arthritis and remote history of deep vein thrombosis was referred for an outpatient stress test for evaluation of underlying ischemia. She was not on any medications, including prescription or over the counter medication, and did not report allergy to any medications. Her initial vital signs were stable (baseline heart rate 68/min and blood pressure 106/68 mm Hg), while her resting transthoracic echocardiogram was essentially normal with normal left ventricular wall motion and ejection fraction with trivial tricuspid regurgitation. Of note, there was no identifiable patent foramen ovale (PFO). Although initial attempt was made to perform an exercise stress test, this was soon aborted as patient developed worsening of her knee discomfort and the stress modality was changed to pharmacological stress with dobutamine. As per the institutional protocol, dobutamine infusion was initiated at 10 μg/kg/min and the dose was increased in increments of 10 μg/kg/min every 3 min. However, even at a high dose of dobutamine (40 μg/kg/min), the patient was not close to achieving her target heart rate. So she was given 0.25 mg of intravenous atropine. Soon after the injection, her target heart rate was achieved (peak heart rate 168/min), with corresponding increase in blood pressure (158/99 mm Hg). Immediately after achieving her target heart, the dobutamine infusion was stopped. About 2 min after the completion of image acquisition, the patient started complaining of substernal heaviness and sensation of “heart racing”. In view of her elevated heart rate and associated symptoms, initial Valsalva attempts were made followed by administration of 5 mg of intravenous metoprolol tartrate. Her symptoms resolved and her heart rate returned to baseline after the administration of metoprolol. As per protocol she was sent home soon after the test, but about 2 h after the completion of test, she started complaining of intense headache and started repeating the same question over and over again, “Where am I?”. In view of her persistent neurological symptoms she was emergently brought back to the emergency room, where a thorough neurological check and CT scan of the head without contrast was performed. Although the patient was alert and oriented to time and person, her repetition of the question, “where am I?” continued despite repeated reorientation. Her initial serum chemistries and CT scan of the head were within normal limits. She was admitted to the hospital. By the following morning her symptoms had resolved and she had returned to her baseline.

Additional testing including MRI of the brain and MRA of the brain were made followed by administration of 5 μg/kg/min every 3 min. However, even treatment did not have any recurrence of her neurological symptoms. However, her amnesia for what happened around the stress test persists to date.

While dobutamine atropine stress test is commonly performed with an excellent safety profile, it is important for clinicians performing dobutamine–atropine stress test to be aware of this rare but unnerving side effect of TGA, which has not been reported often among literature on cardiac stress testing [2]. TGA is a well-recognized and reported phenomenon in neurological literature with incidence rates of 5.2 cases per 100,000 population, which increases among individuals who...
are older than 50 years of age up to 23.5 cases per 100,000 population [3]. However because of its dramatic onset and presentation, it can cause extreme anxiety among both patients and health care providers.

TGA is a paroxysmal transient amnestic syndrome, whose prominent feature is a loss of memory for recent events and an impaired ability to retain new information. While most of the neurological examination is relatively unremarkable, the patients may appear anxious or agitated, with or without loss of orientation to time or place. Repetitive questioning about the transpiring event, as exhibited by our patient, is another notable feature. During the episode patients are unable to create new memories which cause profound anterograde amnesia. In addition variable duration of retrograde amnesia, varying from hours to years, may also occur. These symptoms typically last less than 24 h. As the syndrome improves, so does the associated amnesia, but since no new memories are created during the syndrome, it is not uncommon for patients to be left with permanent amnesia regarding the event [4].

The etiology behind TGA is still not clear, although a number of hypotheses have been proposed. Some of the proposed hypotheses include, but are not limited to, cerebral arterial ischemia, venous congestion, migraine, seizures, and psychological stressors. An interesting hypothesis is related to the role of Valsalva maneuvers which causes brief retrograde transmission of high venous pressure to the cerebral venous system, especially in patients with underlying incompetent internal jugular valves [3,5,6]. As in our patient, Valsalva maneuvers are often utilized to lower the heart rate after the peak heart rate has been achieved during dobutamine cardiac stress testing. Hence this hypothesis may be a potential explanation for the occurrence of TGA in our patient.

A large study among 750 patients undergoing dobutamine stress echocardiography which evaluated the occurrence of neuropsychiatric symptoms within 24 h of the test, found that 7.1% of the patients experienced some form of neuropsychiatric symptoms, while amnesia was reported in only 0.4% of patients [1]. In the overall cohort, when the atropine dose was > 1.0 mg, the incidence rate of neuropsychiatric symptoms increased up to 19.54%, while it also had the highest odds ratio of being associated with neuropsychiatric symptoms. Although our patient received atropine, it was a low dose of atropine (0.25 mg), and her symptoms were different from those associated atropine use. In the same above mentioned study, atropine dose < 1.0 mg had a similar incidence of neuropsychiatric symptoms as those patients in whom atropine was not utilized for stress testing (2.78% vs. 3.83%, p-value = 0.5). In addition, atropine toxicity causes anti-cholinergic syndrome whose features, may vary from central nervous system depression (somnolence or coma) to excitation (agitation, psychosis, and seizures) or a combination of both, with associated dysarthria and ataxia [7]. This spectrum of clinical features is different from those associated with TGA, and it is important to distinguish these two clinical conditions.

In conclusion, TGA is a rare side effect of dobutamine stress test, which although rare and has a self-limited course with good overall prognosis, because of its dramatic presentation requires differentiation from other potential cerebral ischemic events. Increased awareness of this syndrome can help early diagnosis of this syndrome and prevent unnecessary invasive and noninvasive testing and alleviate patient and health care providers’ anxiety.

Conflict of interest

The authors report no relationships that could be construed as a conflict of interest.

References


