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TRANSIENT GLOBAL AMNESIA IN A PATIENT WITH HYPERTENSIVE CRISIS

Abstract

Transient global amnesia was introduced into clinical practice by Fisher and Adams in 1964 to denote a sudden onset of transient disorders in all the types of memory along with loss of memory abilities, retrograde amnesia and inability to recall recent events while still retaining consciousness. The incidence of TGA is 5 to 10 people per 100,000 members of the population per year, but the real incidence is unknown because the episodes of memory loss are temporary and many patients do not consult a doctor at the time when amnesia develops. The triggers of TGA are physical activity, sexual intercourse, pain, Valsalva maneuver, etc. TGA is of interest not only for the neurological practice but therapeutic practice as well since cases of its development are reported in patients with hypertension, patent foramen ovale, cardiac conduction disorders, and mitral valve prolapse. We present a case of a 57-year-old female patient with TGA. She was admitted to the hospital due to hypertensive crisis and an impaired ability to retain new information that started after physical activity. The diagnosis of TGA was based on information from the attack witnesses, the sudden onset of anterograde amnesia, normal consciousness of the patient and short duration of the attack. Also, the patient had no features of stroke, acute hypertensive encephalopathy, epilepsy or alcohol-related blackout. TGA is more typical for females over 50 years with symptoms that start after physical activity and resolve within 24 hours. It is characterized by reversibility of all symptoms and benign outcome according to 2-year follow-up results. This clinical case is considered interesting since it expands the therapeutic concept of cerebral manifestations of hypertensive crisis.

Key words: *transient global amnesia, hypertensive crisis*

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BP — blood pressure, HR — heart rate, ECG — electrocardiogram

A hypertensive crisis can be accompanied by the onset of such cerebrovascular disorders as hypertensive encephalopathy, transient ischemic attack, stroke and subarachnoid hemorrhage. The literature contains data on possible onset of transient global amnesia (TGA) in patients with hypertension [1, 3, 12]. TGA term was introduced into clinical practice by Fisher and Adams in 1964 to denote a sudden onset of transient disorders in all the types of memory along with loss of memory abilities, retrograde amnesia and inability to recall recent events while still retaining consciousness [2]. The incidence of TGA is 5 to 10 people per 100,000 members of the population per year [8]. But the real incidence is unknown because the episodes of memory loss are temporary and many patients do not consult a doctor at the time when amnesia develops. TGA more often develops in women of middle and elderly age; the ratio of female to male patients is 4:1 [2, 8]. Amnesia development can be preceded by physical strain, emotional stress, exposure to extremely intense temperature factor, pain, sexual

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intercourse and Valsalva maneuver [1, 3, 11, 15]. An amnesia episode can last from several hours to several days [1, 4, 8]. The subject of the present discussion is the relation of TGA physiopathological mechanism to local angiospasm, possible microembolism of intracranial vessels and hypoperfusion of brain regions responsible for mnestic functions, and constricted venous outflow from the cranial cavity [3, 4, 9, 16]. The TGA development may be conditioned by a special selective vulnerability to metabolic and oxidative stress of a hippocampus Ca1 region, which is critical for the memory consolidation process [2]. A TGA outcome is favorable in most cases. Repeated amnesia episodes or cerebral strokes are rare [1, 10, 14]. Despite its benign progression, TGA remains one of the most intriguing syndromes in the field of clinical neurology due to its sudden onset and not completely clear pathogenesis [7]. TGA is of interest not only for the neurological practice but therapeutic practice as well since cases of its development are reported in patients with hypertension, patent foramen ovale, cardiac conduction disorders, and mitral valve prolapse [5, 6, 15].

We will discuss a clinical case of TGA in a patient suffering from hypertensive crisis. Patient M., female, 57 years old was admitted to a neurological department with complaints on moderate headache of diffuse pattern. The taken medical history revealed that during the last 3 years the patient has had episodic (no more often than 3 times a year) rises in BP up to 140/90 mm Hg, provoked by stress and overstrain. The patient took antihypertensive drugs (enalapril 10 mg) only in the periods of aggravation. She didn't take the drugs regularly. Being a healthcare professional, the patient sought to keep a healthy lifestyle. She attended callanetics trainings twice a week for three years and did not indulge in any harmful habits. The patient and her relatives indicated that they had no epilepsy in the medical history. On the day of admission, the patient completed callanetics training and decided to stay for aerobics and strength training. At that time she noted hot flashes that went away when she rested. When the patient was back home, her relatives noticed her unusual behavior: she was confused, could not answer the question about the reason for coming 3 hours later than usual, did not remember about the events that occurred at the end of sports classes and after them (how she changed after the training, how she came home). When the same person phoned repeatedly, she did not recall the previous talk they had had few minutes ago and asked the same questions; asked relatives the same questions, while was unable to remember the answers to them. The relatives phoned the trainer and ascertained that she really had completed several trainings at the fitness club. At the recommendation of a healthcare professional who was familiar with the patient's case, they measured BP, which had increased up to 200/140 mm Hg. Therefore, they called the ambulance. A doctor of the emergency team administered enalaprilat at the pre-hospital stage. The patient was admitted to the Neurological Department of the City Hospital, since it was suspected that she had suffered from an acute cerebrovascular accident (CVA). Examination results: patient of normal body build, BMI is 22 kg/m^2; skin (including the head) is of normal color and hydration, and free of damage; heart sounds are rhythmic; heart rate is 68 bpm; BP is 170/100 mm Hg. The patient stated her name, age, and occupation correctly, recognized her relatives but could not describe events after the end of the training; she was disoriented concerning the time and could not state the year, month and date. The retrograde amnesia manifested itself as partial impairment of autobiographical episodic memory with regard to some events of private life. Impairment of verbal and visual memory was notable. The patient repeatedly asked what happened to her, repeatedly asked questions about her health status, and she failed to remember the answers that she received. She failed to recognize doctor on duty during reexaminations. At the same time she correctly carried out habitual actions and the commands of doctor during the examination. She was able to find her way around the room. The patient was critically concerned about her condition, and she clearly understood that she was experiencing problems with her memory. This understanding was accompanied by perplexity and anxiety. An examination for local neurological disorders showed no speech disturbance. TBI was ruled out. Computed tomography scanning of brain was performed to rule out CVA. It showed moderate subatrophy of brain

tissue as well as signs of constricted drainage via superficial veins of the brain. At the extracranial level, triplex scanning of the head major arteries showed signs of diffuse atherosclerotic changes in brachiocephalic artery walls while blood flow values were within the age norm. There were found signs of extravasal influences on the vertebral arteries with impaired venous outflow and ectasia of the right jugular vein. Results of electroencephalography showed disorganization of cerebral bioelectrical activity. Meanwhile, no epileptiform activity or abnormal activity areas were observed. X-ray of the cervical spine showed signs of intervertebral osteochondrosis at C4–C5. The ECG that was taken at admission showed sinus rhythm with heart rate of 75 bpm, and normal electrical axis. Twenty-four hour Holter monitoring that was performed on the third day of the hospital stay registered rare supraventricular polytopic extrasystoles. No conduction disorder was observed. Twenty-four hour monitoring of the blood pressure was performed on the fifth day during the course of antihypertensive therapy. Based on the results of the examination, average values of systolic and diastolic pressure, changes in BP from day to night, and 24-hour index of the systolic pressure were normal (dipper); the 24-hour index of diastolic pressure was classified as over-dipper. Echocardiography showed left ventricular diastolic dysfunction (abnormal relaxation), which can be explained by hypertension. Ophthalmoscopy showed pale pink optic disc, moderately narrowed arteries; moderate myopia of both eyes was detected. Renal ultrasound detected no abnormalities. Laboratory tests carried out in full compliance with current standards showed no abnormalities. Improvement of the patient's condition was observed on treatment with 10 ml 25% magnesium sulphate solution intravenously, 10 ml Cerebrolysin intravenously, 6 ml 5% Mexidol intravenously, 10 mg enalapril, 2.5 mg Arifon per day, 75 mg Curantyl and 75 mg Cardiomagnyl. After 24 hours target BP values were achieved and subsequently remained normal. At the same time, regression of the retrograde amnesia was observed. The patient fully regained the ability to keep recent events in mind, and her aural, visual and gustative memory was completely restored. The memory of the events that occurred during the period of disorder was not restored.

The performed examination helped to exclude CVA, transient epileptic amnesia and TBI. There were no reasons to draw a conclusion of acute hypertensive encephalopathy since there were no distinct clinical signs (intense headache, nausea and vomiting). There were no signs of cerebral edema. Diagnosis of TGA was based on generally accepted criteria [13]: availability of information from episode witnesses, sudden onset of anterograde amnesia (impaired ability to keep new information in mind and no recall of events that took place after the cerebral dysfunction onset), retrograde amnesia (impaired ability to recall information gained before the disease), absence of other cognitive dysfunctions, intact consciousness and personal identity, absence of local neurological symptoms, ruled-out other reasons of amnesia and short duration of the amnesia episode. The hypertensive crisis was classified as a complicated one based on the revised TGA classification as transient cerebral ischemic attack and related syndromes (G45) according to the International Classification of Diseases 10.

After the discharge, the patient has received outpatient follow-up by cardiologist and neurologist for 2 years. Anxiety due to the fear that amnesia would reoccur was observed for the first 2 months. After 2 months the patient overcame the fear and returned to callanetics training. However, she avoids intensive physical exertion. On regular administration of 50 mg losartan on a daily basis, the targeted BP was achieved. The patient feels well. During the follow-up period, no TGA episodes were observed.

It is possible to identify distinct features of TGA development in the presented clinical case: a middle-aged woman who exerted herself excessively, relief of the mnestic function disorder within twenty-four hours without restoration of the memories from the period of amnesia and favorable outcome. The ectasia of the right internal jugular vein is also quite typical for TGA since valve insufficiency of the right internal jugular vein is distinctly related to TGA [5, 6]. Taking into consideration the fact that TGA can develop both after excessive physical exertion and BP rise following a period of no previous physical exertion, it is not absolutely clear whether TGA onset is related to hypertensive

crisis only or whether both TGA and the crisis were provoked by the excessive physical strain and happened at the same time. Nevertheless, this clinical case is considered interesting since it expands the therapeutic concept of cerebral manifestations of hypertensive crisis.

Conflict of interests

The authors declare no conflict of interests.

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