2022 March 23-24 Webinar

35th European Neurology Congress 2022

Features of clinical and some instrumental manifestations of chronic cerebral ischemia in hypertension combined with migraine

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Chronic cerebral ischemia (ChCI) is one of the most common conditions of the brain (occurs up to 75%), which is related is mainly with defect of cerebral vessels. An important etiological factor of ChCI is arterial hypertension (AH), accompanied by suffer to vessels of different calibers. However, there are diseases which affected small vessels, the main representative of this is migraine, accompanied by cerebral angiodistonia. According to WHO (2016), migraine affects up to 30% of the world's population, 1.7-4% of adults have a headache for 15 or more days a month. The extent of vascular and brain damage depends on the duration and intensity of migraine paroxysms, especially when migraine is combined with arterial hypertension. The purpose was to study the features of the clinical manifestations of chronic cerebral ischemia in hypertension comorbid with migraine.

Material and methods: 72 patients with headaches were examined: 28 (38.9%) patients with migraine combined with hypertension (group 1); 17 (23.6%) patients suffering from migraine with aura (group 2); 27 (37.5%) patients with migraine without aura (group 3). Group 2 and group 3 have not been hypertension. We used clinical and neurological examinations with auscultation of the carotid arteries, psychodiagnostics (MMSE, "Memorizing 10 words", "Schulte Tables"), MRI of the brain, duplex scanning of the brain vessels.

Results and discussion: Age of patients group 1 was older than the rest groups (53.4±2.56 years, $P<0.01$); in 2nd group it was 33.8±1.82 years and in 3rd group 26.3±1.23 years. In 1st group hemicrania had unilateral character, however in 2nd group and 3rd group the headache side changed. In 1st group headache was longer than in 2nd and 3rd groups ($P<0.05$), but it decreased when taking analgesic therapy in combination with antihypoxants, while sumatriptans increased the intensity and duration of hemicrania. They main complaints in exacerbation of the migraine paroxysm, there was dizziness, combined with pulsating hemicrania, nausea, shakiness when walking and tinnitus. Blood pressure increased to 170/120 mmHg, while diastolic pressure tended to increase more than systolic, and among patients of the group 1 11 (39.3%) patients had symptoms of visual aura that lasted longer than patients with migraine (up to 28 min) without hypertension (group 2). There were visual (81.8%) and olfactory (18.2%) auras in 2nd group, with a duration of 11.2±2.31 minutes. Nausea and vomiting were noted as additional symptoms to hemicrania, which brought mild relief and decreased after taking NSAIDs and/or sumatriptans. In 3rd group hemicrania, nausea and vomiting were observed without aura, in the groups of 2 and 3, unlike 1st group, there were practically no signs of dizziness, tinnitus and gait instability. The neurological objective status showed the presence of slowness, deterioration of brain activity, changes in the psycho-emotional state (irritability, apathy, hysteria, depression, insomnia), impaired concentration, fatigue, anxiety, dizziness, decreased sensitivity in the extremities, reflex asymmetry, coordination disorders, inhibition of visual and auditory function in 1st group. In 2nd group focal neurological symptoms were detected during aura attacks (impaired sensitivity, hemi hyperreflexia, vision changes), hemicrania was accompanied by additional symptoms: irritability, apathy, depression, insomnia, impaired concentration, fatigue, anxiety and phobic expectation. In 3rd group almost the same additional symptoms were observed together with hemicrania, nausea and vomiting, without focal symptoms. However, diffuse hyperreflexia and hyperesthesia were observed in during and for 30 minutes after seizures. In group 1, there was a decrease in the linear velocity of blood flow, the elastic properties of the vessel walls and an increase in vascular tone in the internal and external carotid arteries; whereas in groups 2 and 3, there was a decrease in vascular tone and elastic properties without changing the linear velocity of blood flow in the internal carotid arteries and an increase in the linear velocity of blood flow in the external carotid artery. In group 1, there were local and diffuse foci of encephalomalacia (up to 3-5 mm), leukoareosis, the severity of which depended on the duration of hypertension, the frequency and intensity of migraine attacks, as well as aura. In group 2, diffuse and local foci of encephalomalacia (up to 3 mm) were observed, some of them were fused like thin threads, but there was almost no leukoareosis. Small and isolated foci of encephalomalacia (0.5-1.5 mm) were found in patients with migraine without aura lasting more than 10 years. Thus, migraine accompanied by hypertension leads to serious damage to the brain of vascular genesis, aggravating the clinical course and developing chronic cerebral ischemia. This requires more thorough and timely examination and therapy, finding ways to prevent vascular dementia early, taking into account encephalomalacia in the brain at a younger age.

Key words: migraine comorbid with hypertension; chronic cerebral ischemia; damage to the brain of vascular genesis.

Biography

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Received: January 18, 2022 | **Accepted:** January 21, 2022 | **Published:** March 24, 2022