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POSSIBLE WAYS TO CORRECT STROKE WITH ANXIETY-DEPRESSIVE DISORDERS

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ABSTRACT
The article is dedicated to the emergence and correction of acute cerebrovascular disorders against the background of anxiety-depressive syndrome. The presented material is the result of the authors' own research based on the use of a "model" of cerebrovascular disease and depressive syndrome.
KEY WORDS: *Anxiety-Depressive Syndrome, Stroke, Ksavron, Teraligen.*

RELEVANCE

Stroke is a dangerous condition characterized by impaired cerebral circulation [1-3]. This type of brain damage is the most common cause of disability and ranks as the leading cause of mortality, second only to myocardial infarction and cancer [16,17]. Depressive disorders and their increasing severity, along with high levels of anxiety, are predictors of acute cerebrovascular accident (ACVA) and fatal outcomes[8]. The comorbidity of cerebrovascular diseases (CVD) and anxiety-depressive disorders (ADD) complicates the clinical picture, with each disorder contributing to the progression of the other. For example, generalized anxiety disorder (GAD) is observed in 98.6% of patients with arterial hypertension and 89.6% of patients with chronic heart failure [6]. The presence of increased anxiety raises the likelihood of sudden death by 4.5 times. There are two models of comorbidity: A model based on descriptive diagnostic classes (i.e., the presence of more than one disorder in a person within a specific period). A model considering the relative risk of developing another disorder when one disease is already present [10,11]. Secondary anxiety disorder following a stroke occurs later, with the time of its development depending on the severity of the disease. Anxiety significantly worsens the psycho-emotional state of patients, intensifies neurotic personality traits, complicates social adaptation, and reduces quality of life. Thus, in this situation, anxiety becomes an independent pathogenic factor. At the same time, ADD as an independent cause of stroke deserves greater attention and further study from clinical, pathogenetic, and neurochemical perspectives. The presence of depression symptoms in individuals over 55 years old increases the risk of stroke by 2.3–2.7 times. The direct impact of ADD on stroke risk includes the body's immediate reaction to "psychological aggression." Psychological stress may lead to overstrain of cortical processes, disinhibition of subcortical hypothalamic centers, and disruption of nervous regulation of homeostasis. The excitability of the sympathoadrenal system increases, leading to a higher secretion of catecholamines, clinically manifested by increased heart rate and blood pressure. When psychological stress is not mitigated by psychological defense mechanisms, the functional stage of brain damage transitions into destructive morphological changes in the central nervous system (CNS). It can be argued that neurohumoral imbalance is one of the main pathogenetic mechanisms of cerebral ischemia, and ADD in this case represents a manifestation of systemic dysregulation of neurotransmitters [14].

A key role in the development of this imbalance is played by the GABAergic system. Dysfunction of GABA-dependent processes in the CNS mediates disturbances in serotonin, catecholamine, and peptidergic systems, ultimately leading to maladaptation caused by anxiety disorder [15]. The role of corticotropin-releasing factor (CRF) in anxiety-related stress conditions has also been established. Indirect effects of ADD involve behavioral disorders in patients, such as ignoring dietary recommendations, failure to take antihypertensive medications on time, etc., which can also contribute to stroke development. Due to the complexity and multifactorial nature of ADD pathogenesis, there is a need for multiple therapeutic agents targeting different pathological mechanisms. This leads to polypharmacy, often accompanied by complications. Therefore, the search for new treatment approaches for patients with ADD remains relevant.

AIM

To explore methods for correcting stroke with anxiety-depressive disorders.

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MATERIALS AND METHODS

Over a 3-month observation period, 120 cases of stroke were registered in the neurology department of the Samarkand Branch of the Republican Scientific Center for Emergency Medical Care. Patients were hospitalized during the acute phase of their first cerebral stroke. Neurological and psychiatric status assessments were conducted on standardized days: 1–3, 7 of the acute period in the hospital, 14, 21, 28 days post-stroke, and after 3, 6, and 12 months. All study participants were assessed for other stroke risk factors and described their stressors and lifestyle factors that might have influenced stroke risk, including alcohol consumption, coffee, energy drinks, and smoking.

Patients who had suffered a stroke were divided into two groups: primary group - 65 patients, control group - 55 patients.
The average age of participants was 57 years. Medications were selected based on stroke type and the patient's condition upon hospitalization. The study employed psychopathological analysis, clinical observation, and anamnesis review.

Diagnostic scales used included: MMSE (Mini-Mental State Examination) for standardized cognitive function assessment [9]. Hamilton Anxiety Rating Scale (HAM-A 14), consisting of 14 indicators, each rated on a 5-point scale (0 = absent, 4 = severe). Patients in the primary group who suffered an ischemic stroke received Xavron as part of basic therapy, while the control group received standard treatment. The efficacy of Teraligen in managing anxiety-depressive states was evaluated in the primary group.

RESULTS AND ITS DISCUSSION

According to the MMSE scale, cognitive function was assessed as follows: 28–30 points: No cognitive impairment, 25–27 points: Mild cognitive impairment, 20–24 points: Mild dementia, 11–19 points: Moderate dementia. 0–10 points: Severe dementia.

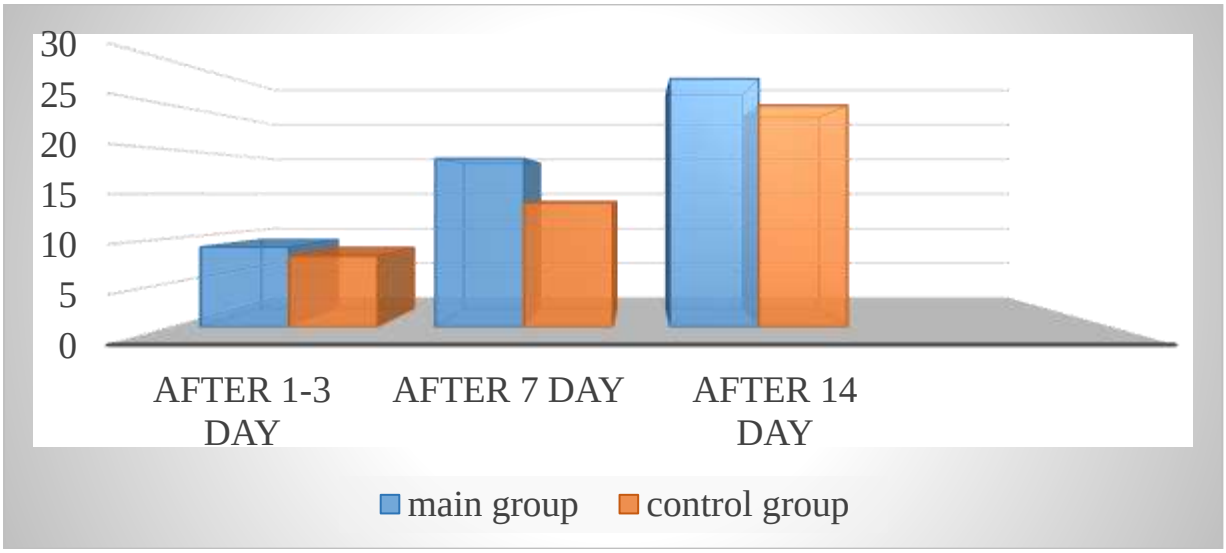

Figure 1. Evaluation of the effectiveness of Xavron based on the MMSE scale before and after treatment.

Analysis showed that cognitive impairments, including dementia, were pronounced in patients within the first 1–3 days of stroke. By day 14, patients receiving Xavron had no cognitive impairments, whereas those in the control group exhibited mild cognitive impairments.

Table 1.
Psycho-neurological changes in stroke patients after taking teraligen (Hamilton Scale).

Criteria	Acute period (n=120)	3 months (n=80)	6 months (n=72)	12 months (n=58)
Anxious mood, %	70.8% (85)	33.8% (27)	26.7% (19)	21.8% (13)
Tension, %	30.9% (37)	29.1% (23)	12.3% (9)	11.2% (7)
Fears, %	22.5% (27)	16.4% (13)	8.6% (6)	7.8% (4)
Insomnia, %	23.3% (28)	21.4% (17)	12.3% (9)	10.8% (6)
Intellectual impairments, %	18.2% (22)	14.5% (11)	11.2% (8)	9.3% (5)
Depressive mood, %	69.2% (83)	44.8% (36)	31.7% (23)	22.1% (12)
Somatic complaints (muscular), %	12.5% (15)	46.2% (37)	39.4% (28)	23.6% (14)

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Somatic complaints (sensory), %	10.8% (13)	9.9% (8)	8.7% (6)	7.8% (4)
Cardiovascular symptoms, %	16.7% (20)	15.3% (12)	14.1% (10)	12.3% (7)
Respiratory symptoms, %	9.2% (11)	8.8% (7)	7.5% (5)	5.1% (3)
Gastrointestinal symptoms, %	15.8% (19)	12.2% (9)	9.3% (7)	6.7% (4)
Urogenital symptoms, %	14.2% (17)	13.2% (10)	5.6% (4)	4.3% (2)
Autonomic symptoms, %	35.8% (43)	53.6% (42)	34.2% (25)	16.6% (9)
Behavior in conversation, %	26.7% (32)	19.7% (15)	15.2% (11)	7.3% (4)

A psychological-neurological assessment based on the Hamilton Scale revealed that in the acute period of stroke, the most frequent disorders were: Anxiety (70.8%), Depressive mood (69.2%), Vegetative symptoms (35.8%), Tension (30.9%), Conversational behavior disturbances (26.7%).

After 3 months of Teraligen treatment, the most common symptoms were: Vegetative symptoms (53.6%), Somatic complaints (46.2%), Depression (44.8%), However, by 6 and 12 months post-stroke, the prevalence of depression and anxiety had significantly declined.

CONCLUSION

Thus, in the treatment of anxiety-depressive disorders associated with brain ischemia, preference is given to drugs that have both anxiolytic and neuroprotective properties. The study found that when anxiolytics were combined with CNS neuroprotection, anxiety did not become generalized, and early post-stroke dementia could be prevented.

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