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DIAGNOSIS AND TREATMENT OF THROMBOSIS OF CEREBRAL VEINS AND VENOUS SINUSES

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RESUME

Annotation: Cerebral vein and venous sinus thrombosis is a rare disease, accounting for less than 1% of all stroke cases. The most common and often the earliest symptom of cerebral vein and sinus thrombosis is headache. The diagnosis of the disease is significantly complicated by the lack of specific characteristics of this pain and the possible absence of other neurological manifestations. In this regard, headache is often the only complaint of patients with cerebral venous sinus thrombosis. Due to the variability of clinical symptoms, often subacute or slow development, the disease is diagnosed late or remains unrecognized at all. The most common thrombosis is the superior sagittal, lateral and transverse sinuses. In 2/3 of cases, thrombosis of several cerebral veins is observed. This paper presents the anatomical features of the structure of the venous system of the brain, issues of etiology, pathogenesis, clinical picture, diagnosis and treatment of cerebral vein and venous sinus thrombosis.
The case histories of three patients who were treated at different times in the second neurological department of the Scientific Center of Neurology with a diagnosis of "cerebral sinus thrombosis" are presented, demonstrating modern possibilities for the diagnosis and treatment of venous circulation disorders.

Key words: venous sinus thrombosis, cerebral vein thrombosis, headache, diagnostics, anticoagulants

Introduction. The venous system of the brain is characterized by "branching", a large number of anastomoses and the fact that one vein can receive blood from the pools of several arteries.
The cerebral veins are divided into superficial and deep.
The superficial veins – the superior cerebral veins, the superficial middle cerebral vein (Labbe's vein), the inferior anastomotic vein (Trolar's vein), and the inferior cerebral veins – lie in the subarachnoid space and, anastomosing with each other, form a network on the surfaces of the cerebral hemispheres (Fig. 1). The main mass of venous blood from the cortex and white matter flows into the superficial veins and then into the nearby sinus of the dura mater.
The deep cerebral veins receive blood from the veins of the choroid plexus of the lateral ventricles, basal ganglia, thalami, midbrain, pons, medulla oblongata and cerebellum.
The superficial and deep veins drain into the sinuses of the dura mater. The superficial veins drain mainly into the superior sagittal sinus. The main collectors of the deep veins are the great cerebral vein (vein of Galen) and the straight sinus. Blood from the superior sagittal and straight sinuses enters the transverse and sigmoid sinuses, which collect blood from the cranial cavity and drain it into the internal jugular vein.
Two mechanisms that determine the symptoms of the disease are involved in the development of cerebral vein and venous sinus thrombosis. The first is occlusion of the cerebral veins, causing cerebral edema and impaired venous circulation. The second link in the pathogenesis of cerebral vein and venous sinus thrombosis is the development of intracranial hypertension due to occlusion of large venous sinuses. Normally, cerebrospinal fluid is transported from the cerebral ventricles through the subarachnoid space of the lower and upper lateral surfaces of the cerebral hemispheres, adsorbed in the arachnoid plexuses and drained into the superior sagittal sinus. In case of venous sinus thrombosis, venous pressure increases, as a result of which the absorption of cerebrospinal

fluid is impaired, and intracranial hypertension develops [5].

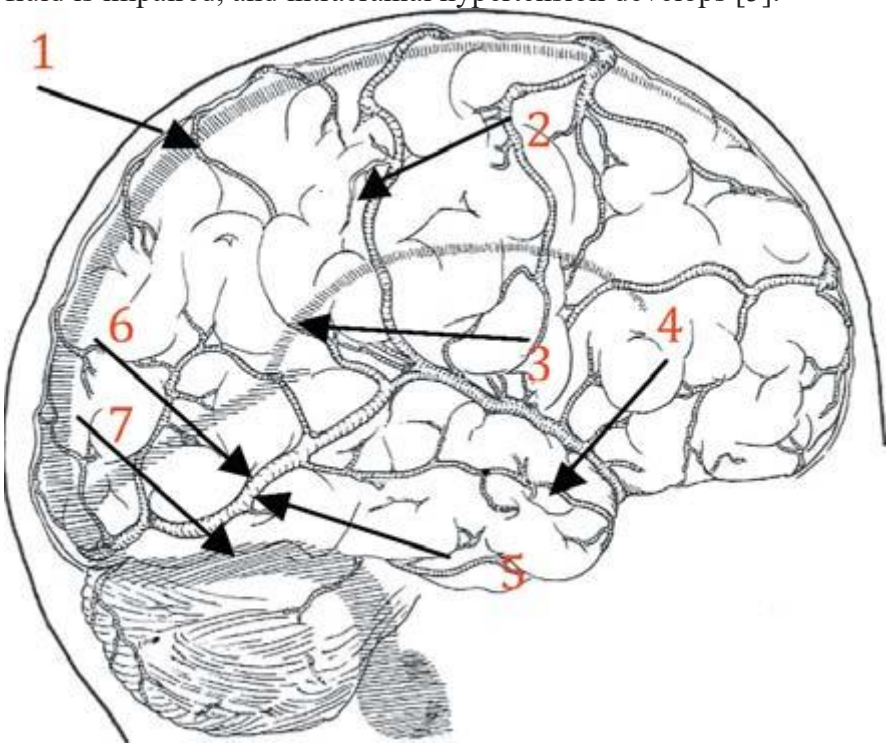


Fig. 1. Venous system of the brain (from the book: Handbook on cerebral venous Thrombosis).1 – superior sagittal sinus, 2 – vein of Trolar, 3 – inferior sagittal sinus, 4 – superficial middle cerebral vein, 5 – vein of Labbe, 6 – straight sinus, 7 – transverse sinus

Clinical picture and diagnostics of thrombosis of cerebral veins and venous sinusesClinical manifestations of thrombosis of the cerebral veins and venous sinuses depend on the location of the thrombosis, the rate of its development and the nature of the underlying disease.

Severe venous circulation disorders are characterized by headache, vomiting, optic disc edema, focal and generalized seizures, and progressive depression of consciousness. However, with early recognition of the process, the clinical picture may be less pronounced. Focal neurological disorders may occur with isolated thrombosis of deep or superficial veins or with the spread of thrombosis from the sinuses to the veins. Meningeal syndrome is considered a rare manifestation of uncomplicated sinus thrombosis. According to most authors, cerebrospinal fluid pressure is normal or moderately elevated.

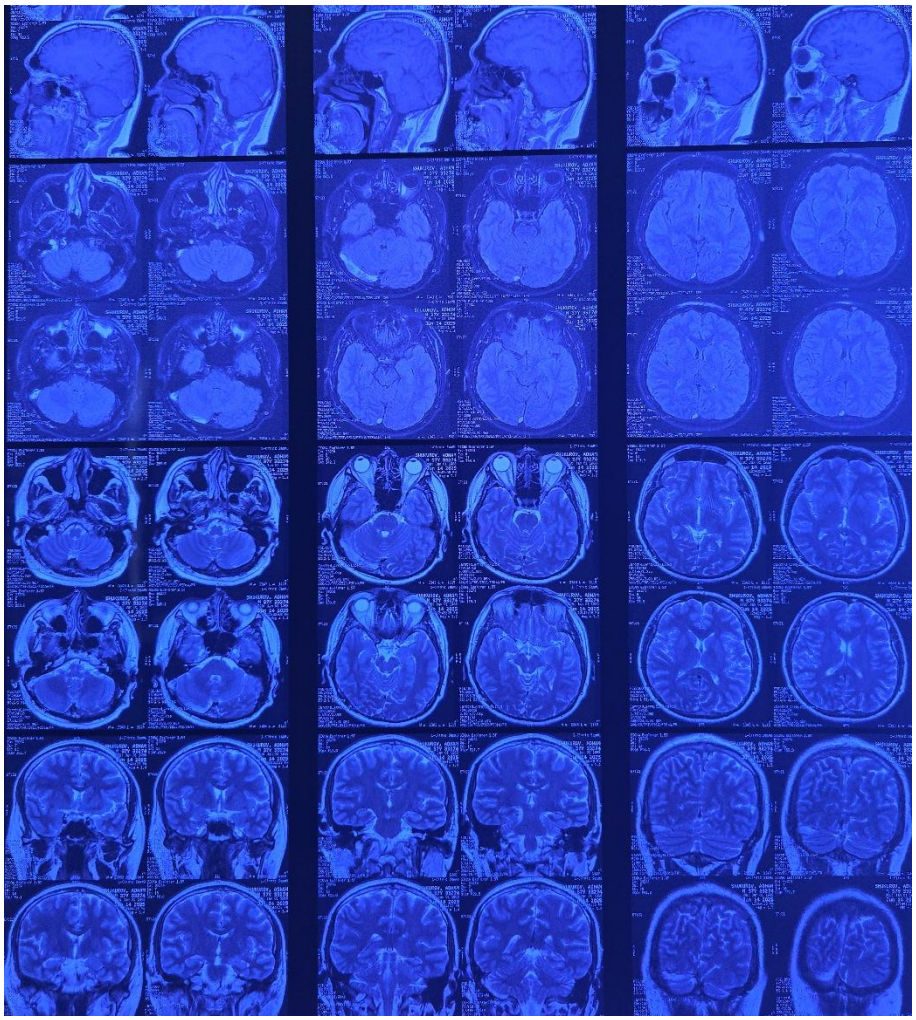
The composition of the cerebrospinal fluid may be either unchanged or with a slightly increased protein content and pleocytosis of no more than 200/3 [7].

The most common symptom of cerebral vein and venous sinus thrombosis is sudden, severe headache, which is usually diffuse (92% of cases) and poorly relieved by analgesics. It is unusual for the patient both in its nature and intensity. Headache may be accompanied by nausea and vomiting, as well as focal neurological symptoms, including dysfunction of the cranial nerves. According to the ISCVT study and other data [9–11], the following symptoms are often detected: • movement disorders – 42%; • convulsive syndrome – 37%; • status epilepticus – 13%; • psychomotor agitation – 25%;

• aphasia – 18%; • visual impairment – 13%; • depression of consciousness (stupor, stupor, coma) – 13%; • cranial nerve dysfunction – 12%; • sensory impairment – 11%; • meningeal syndrome – 5%; • vestibulocerebellar impairment – 1%; • in the long term, the most common symptoms are headache (14%) and convulsive syndrome (11%).

The marginal, cavernous and superior sagittal sinuses are relatively rare foci of infection. More often, the intracranial process is the result of the spread of infection from the middle ear, paranasal sinuses, skin near the upper lip, nose and eyes.

Rice. 2. Thrombosis of the right transverse and sigmoid sinuses with MSCT.



Clinical case.
*Patient*Sh., 38 years old, was admitted to the neurosurgery department of the Multidisciplinary Clinic of the Samarkand Medical University with complaints of severe headache, nausea, and vomiting.
*Medical history:*On 06/10/2025, headache, not relieved by analgesics, nausea, vomiting, and photophobia suddenly appeared. The condition was assessed as intracranial hypertension. Diuretics were prescribed. On 06/12/2025, a generalized epileptic seizure suddenly developed. After treatment (venotonics, glucocorticoids, nootropics), the headache regressed, but on 06/14/2025, headache, nausea, and vomiting suddenly recurred. On 06/15/2025, the patient was hospitalized at the NCN.
*On examination:*pronounced dilation of the subcutaneous veins on the face.
*In neurological status*Mild rigidity of the occipital muscles was detected. There were no focal symptoms.
*Laboratory research methods:*Lupus anticoagulant – 1.10% (negative). Antibodies to cardiolipins IgG – 15.8 U/ml, weakly positive (normal – up to 10 U/ml).
Homocysteine – 16 μmol/l (normal – up to 15 μmol/l). Antigen to von Willebrand factor – 273% (normal – up to 117%). Blood coagulation factors – no deviation from normal values.
Blood test for thrombophilic mutations - negative result. During examination by an ophthalmologist, signs of intracranial hypertension were revealed: hyperemia and swelling of the optic nerve discs, dilation and plethora of veins in the fundus.
*Instrumental research methods:*When performing MRI in T2 mode, an increase in the intensity of the MR signal from the superior sagittal and left sigmoid sinuses was noted (Fig. 4).
When performing MR venosinusography, there is no blood flow in both transverse, superior sagittal and left sigmoid sinuses. Increased blood flow in the superficial cerebral and facial veins is noteworthy (Fig. 5).

*Diagnosis:*thrombosis of both transverse, left sigmoid and superior sagittal sinuses.
*Treatment carried out:*Nadroparin calcium 0.6 ml subcutaneously 2 times a day for 10 days with a transition to warfarin (INR level 2–3), venotonics, carbamazepine (to prevent recurrent epileptic seizures). 10 days after the start of therapy, an improvement in well-being was noted – headache decreased. During MR venosinusography, positive dynamics were noted – blood flow in both transverse sinuses was restored. Four months after treatment, blood flow in the superior sagittal sinus appeared.
Permanent use of anticoagulants is recommended.
Taking into account the thrombosis of the right transverse sinus, deep vein thrombosis of the legs in the anamnesis, and increased homocysteine levels, the patient was recommended long-term therapy with anticoagulants.
ConclusionOver the past two decades, improved awareness of cerebral venous thrombosis and advances in neuroimaging techniques have changed the assessment of its incidence. However, despite improved knowledge, the true incidence of cerebral venous thrombosis remains unknown. The most common and often the earliest symptom of cerebral vein and sinus thrombosis is headache. The diagnosis of the disease is significantly complicated by the lack of specific characteristics of this pain and the possible absence of other neurological manifestations. In this regard, headache is often the only complaint of patients with cerebral venous sinus thrombosis. Early diagnosis of the relationship between headache and cerebral vein and venous sinus thrombosis is very important for the prognosis of the disease.
Due to the variability of clinical symptoms, often subacute or slow development, the disease is diagnosed late or remains unrecognized altogether.
Current therapeutic tactics used in clinical practice include the use of anticoagulants for the treatment of cerebral venous sinus thrombosis. Early diagnosis is critical because the use of anticoagulants can reduce the risk of fatal outcome and severe disability without additionally increasing the risk of intracerebral hemorrhage.
In the three cases shown above, the most likely cause is an increase in the thrombogenic potential of the blood (high levels of von Willebrand factor antigen, hormonal therapy, hyperhomocysteinemia). The thrombophilic state could have triggered the development of venous sinus thrombosis. Thus, in this situation, the main direction
Pathogenetic therapy is the administration of direct-acting anticoagulants with a transition to indirect anticoagulants and maintaining INR within 2–3.
In addition, the importance of a thorough anamnesis is shown, in particular, close attention to the infectious process, craniocerebral trauma, venous thrombosis in the anamnesis, intake of drugs that contribute to the development of a hypercoagulable state. The importance of a physical examination is also emphasized, which can reveal indirect signs of impaired venous outflow through the cerebral veins and venous sinuses (dilation of the facial veins in the first case). In two patients, examination of the fundus revealed signs of impaired venous outflow and intracranial hypertension: congestive, edematous, hyperemic optic discs, dilated, full-blooded veins in the fundus, absence of spontaneous venous pulse. All these symptoms, along with indications of sudden, intense, diffuse headaches that are not relieved by analgesics, should give the clinician a reason to exclude a violation of venous cerebral circulation, which, in turn, is the key to successful treatment of patients and secondary prevention of this type of pathology.

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