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VENOUS ENCEPHALOPATHY: EARLY DIAGNOSIS (literature review)

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Annotation: Venous encephalopathy (including acute cerebral sinus thrombosis syndrome, chronic forms of venous insufficiency, and venous collagenoses) is a significant cause of neurological impairment, potentially leading to parenchymal changes, cerebral edema, and hemorrhagic complications. Early diagnosis determines the outcome and enables timely initiation of anticoagulant or interventional therapy. This article discusses the pathophysiology, clinical presentation, laboratory and instrumental markers, and a practical diagnostic algorithm, focusing on the role of MRI/MRV, CT/CTV, SWI, and laboratory tests (D- dimer and thrombotic panels). Risk assessment schemes, an algorithm for action in emergency and elective situations, and recommendations for monitoring and selection of additional tests are proposed.

Venous encephalopathy is a collective term that includes brain damage resulting from impaired venous outflow. This condition may be a consequence of acute cerebral venous thrombosis (cerebral venous thrombosis (CVT), chronic venous insufficiency (venous collagenosis , perivenous fibrosis), jugular vein stenosis, and other causes. Unlike arterial strokes, venous lesions have a wide range of clinical manifestations (from headache to focal neurological symptoms and coma) and are often diagnosed with a delay, which worsens the prognosis. Modern imaging techniques and laboratory tests make early detection and differential diagnosis possible (2).

The pathogenesis of venous encephalopathies is based on impaired venous outflow, which leads to increased venous pressure, blood retention, decreased cerebral perfusion pressure, interstitial and cytotoxic edema, and, in severe hypertension, to rupture of thin-walled veins and intracerebral hemorrhage. Chronic venous hypertension is accompanied by remodeling of the venous wall, sometimes with collagen deposition (venous collagenosis) - a factor associated with the formation of periventricular white brain changes (white matter hyperintensities). These mechanisms are discussed in recent papers on venous pathological anatomy and correlation with changes on MRI (1,2).

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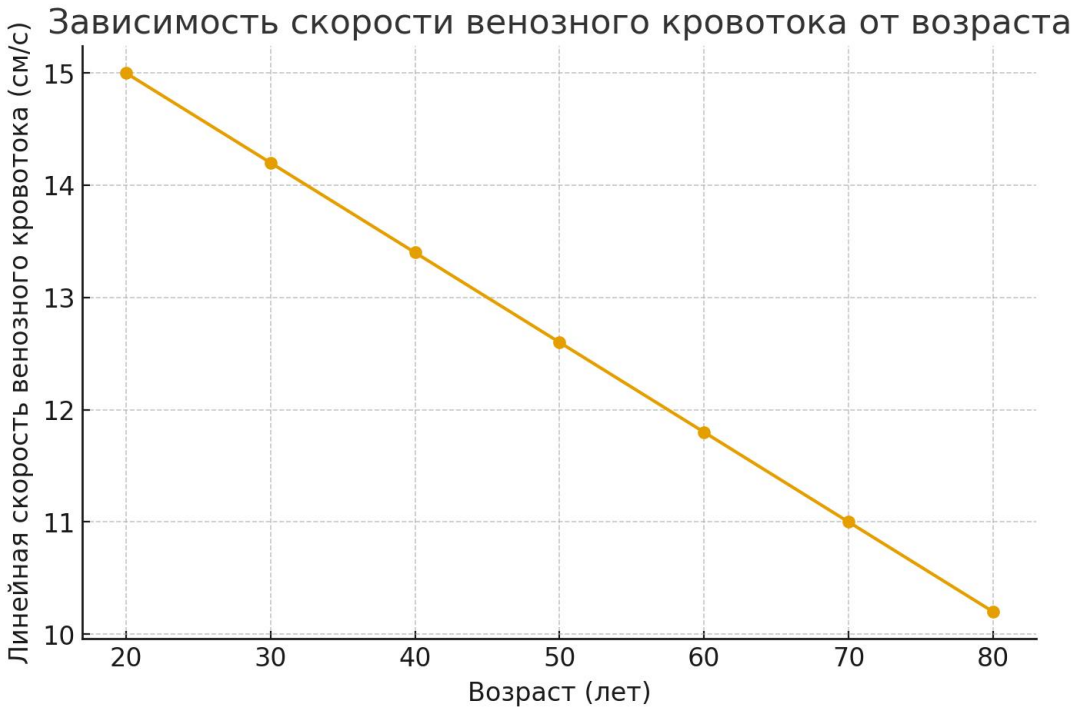

Clinically:

- A common and early complaint is headache (intense, progressive).
- Focal neurological symptoms (weakness, aphasia, ataxia), seizures.
- Vague symptoms in elderly patients: cognitive impairment, progressive leukoaraiosis symptoms in chronic venous insufficiency.
- In the acute period: loss of consciousness, meningeal symptoms, focal and generalized seizures are possible (3).

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Early diagnosis: approach and components

1) Pre-test risk assessment and clinical stratification

The classic strategy is to consider age, thromboembolism risk factors (hormonal therapy, pregnancy/postpartum period, infections, cancer, hereditary coagulopathies), the rate of symptom onset, and the presence of focal neurological disorders and seizures. A low pretest risk plus normal D- dimer significantly reduces the likelihood of CVT; however, interpretation depends on the time since symptom onset (3,4).

2) Laboratory markers

- D- dimer is widely used as a screening test: it has a high negative predictive value with a low pre-test risk, but sensitivity decreases with symptom duration of more than a few days and with localized thromboses. Therefore, a normal D- dimer does not always exclude CVT. Additional markers (TAT - thrombin - antithrombin complexes and others) are discussed as promising, but have not yet entered into widespread practice (5).
- thrombophilia testing - if CVT is confirmed, it is advisable to look for provoking factors (VWF Leiden , protein C/S, antithrombin III, antiphospholipid antibodies, etc.) (1,3).

3) Magnetic resonance imaging combined with venography (MRI/MRV) is the current "gold standard" for confirming CVT: visualization of the thrombus itself, assessment of parenchymal changes, edema, and focal hemorrhage. MRV demonstrates the absence of sinus/vein filling (2,3). CT + CT- venography (CTV) is a rapid and highly accessible test in emergency situations,

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with sensitivity close to MRI/MRV in the acute period; it is often the first tool in emergency departments. Conventional CT is more useful for assessing the presence of parenchymal hemorrhage or significant edema, but is less sensitive for direct visualization of the thrombus (4,5). Susceptibility-weighted SWI imaging is valuable for the early detection of deoxyhemoglobin in the vein/thrombus lumen, microbleeds, and venous bed assessment; it is useful as an additional sequence to MRI. SWI also helps to recognize areas of venous hypoxia (3). Digital subtraction angiography (DSA) is an invasive technique indicated when noninvasive imaging results are inconclusive or when planning endovascular intervention; it has very high accuracy (2,4).

4) Early practical action plan (algorithm)
Briefly: if suspected, perform emergency CT with CT venography (if available) and laboratory tests (including D- dimer). If results are positive/suspicious, initiate anticoagulation immediately (in the absence of absolute contraindications) and perform MRI+MRV with SWI/ASL to clarify the extent of the lesion and plan further therapy. If data are unclear, perform DSA and multidisciplinary discussion. (A detailed flowchart of the algorithm is provided in the illustrations) (1,4).

The methodology and clinical utility of D- dimer in CVT are controversial: D -dimer levels are often elevated in early, generalized thromboses , but sensitivity decreases in localized and/or subacute forms. D- dimer is often recommended as a screening test in conjunction with clinical assessment; a negative test in the setting of low pre-test risk may allow for delaying urgent imaging, but with high clinical suspicion, imaging is mandatory. New markers of thromboembolism (TAT and others) have shown promising properties in individual studies, but their widespread clinical use is still limited (5,4).

Chronic venous pathology associated with small vein wall degeneration and collagen deposition can manifest as progressive white medulla pathology, cognitive impairment, and gradually increasing encephalopathy. Pathological correlation and modern MR techniques (including SWI and advanced perfusion methods) allow us to differentiate venous changes from arterial and degenerative ones. These mechanisms have been described in recent studies (venous collagenosis is an important contribution to the pathogenesis of WM).

Visualization guidelines and protocol (practical guide):

1. Emergency stage: urgent CT + CTV if CVT is suspected.
2. Confirmation and detailed assessment: MRI (T1, T2, FLAIR, DWI) + MRV; be sure to include SWI and, if possible, ASL/perfusion to assess ischemia/ penumbra .
3. Additional methods: in case of diagnostic uncertainty or planning of intervention – DSA.
4. Monitoring: follow-up imaging after 1–2 weeks and then after 3 months if clinically indicated.

Differential diagnosis includes arterial ischemic strokes (especially with subcortical lesions), demyelinating processes, infectious/post-infectious encephalitis, neoplastic processes, and

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normal- / normal-pressure hydrocephalus. Key distinctions are made based on MRI/MRV imaging and the dynamics of clinical symptoms. SWI and perfusion methods help distinguish the venous component (venous congestive hyperintensity , the presence of a thrombus in the veins) from other causes.

Early diagnosis allows for the earliest possible initiation of anticoagulant therapy (often low-molecular-weight heparins in the acute phase), which reduces the risk of thrombosis progression and improves outcomes. In recurrent/refractory cases, endovascular methods (thrombectomy / lavalization) are used. Correction of predisposing factors (cancer, infections, hormonal therapy, fluid/electrolyte status, etc.) is important.

Prospects and new directions of research

- Development and validation of new biomarkers (TAT, etc.) to improve early sensitivity.
- Use of SWI and advanced perfusion for earlier detection of venous hypoxia and prediction of areas susceptible to reversible damage.
- Studies of the role of venous collagenosis in the development of age-related white marrow changes and dementia.

Conclusions: Venous encephalopathy is a clinically heterogeneous group of conditions that requires high awareness for early diagnosis. A combination of clinical stratification, laboratory screening (including D- dimer), and prioritized imaging (emergency CT/CTV followed by MRI, MRV, and SWI) provides the best approach to early detection. New markers and advanced MR sequences promise improved early diagnosis and risk stratification, but require further validation and implementation into clinical protocols.

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