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# Typical course of hippocampal sclerosis. Literature review

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**Abstract.** The article reviews the problem of hippocampal sclerosis as the main cause of pharmacoresistant temporal lobe epilepsy. Clinical manifestations, diagnostic criteria, recommended methods of examination and treatment of hippocampal sclerosis are given. The effectiveness of surgical and medical treatments was analyzed. The relevance of this article is due to a general increase in the detection of this disease, primarily owing the improvement of neuroimaging methods. Given that this disease is relatively rare, and that surgical treatment is often delayed for many years, a review of this topic is useful for early diagnosis and improved treatment outcomes. Modern literary sources on hippocampal sclerosis were studied. The studied material is summarized and presented in the form of a literature review in this article. A search for literary sources was carried out in two main scientific databases: Scopus and PubMed. The review included original articles, research, and official guidelines from medical associations. **Keywords:** epilepsy; hippocampal sclerosis; pharmacoresistant epilepsy; temporal lobe epilepsy; review

# Introduction

Hippocampal sclerosis (HS) is a pathology that is often resistant to anticonvulsant therapy in cases of focal epilepsy, in particular, in mesial temporal lobe epilepsy with HS. This disease is characterised by various electroclinical and pathological features. Despite pharmacoresistance, there is a large proportion of patients in whom surgical treatment can help control seizures, although not always successfully (up to 40 % of patients experience early or late surgical failure). However, awareness of the semiology and timely use of advanced diagnostic methods leads to a better understanding of the disease and timely treatment.

# Definition and epidemiology

The terms "Ammon's horn sclerosis", "hippocampal sclerosis", and "mesial temporal sclerosis" are often used interchangeably, as they all refer to similar processes characterised by a reduction in volume and compaction of the hippocampal structure. HS causes selective neuronal loss accompanied by astroglial proliferation, involving certain areas of the hippocampus to varying degrees; the follo-wing areas are distinguished in the structure of the hippocampus (cornu Ammonis): CA1, CA2, CA3, CA4, dentate gyrus and subiculum (Fig. 1).

It is well known that the most susceptible sectors for sclerosis are CA1, CA3 and CA4, while dentate gyrus granule cells, CA2 sector and subiculum are the most resistant [1-4].

The amygdala, corpus callosum, and parahippocampal gyrus can also be affected, making the term "mesial temporal sclerosis" a more appropriate description than "hippocampal sclerosis". Such volume reduction is often detected on magnetic resonance imaging (MRI), which is an indicator of the corresponding histopathological process [2, 4-8].

HS is recognised as the most common pathological substrate of mesial temporal lobe epilepsy and is found in 60-70 % of patients who have been operated for drug-resistant seizures. Other structural abnormalities, such as hamartomas, glial tumours, vascular and congenital mal-

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formations, and gliotic lesions due to trauma or infection, can also trigger the development of temporal lobe epilepsy [9-11].

Numerous studies have reached a consensus on the classification of HD. The classification allowed us to identify three types of hippocampal sclerosis: ILAE type 1, characterised by significant neuronal cell loss and gliosis predominantly in CA1 and CA4; ILAE type 2, characterised by predominant neuronal cell loss and gliosis in CA1; and ILAE type 3, characterised by predominant neuronal cell loss and gliosis in CA4 (Table 1).

Approximately 1 % of the population suffers from epilepsy. Epilepsies can be classified as generalised or localised (focal). Temporal lobe epilepsy is the most common form of focal epilepsy in adults, and HS is the most common histopathology found in patients with drug-resistant temporal lobe epilepsy [11–16]. However, the prevalence of HS or temporal lobe epilepsy in the general population remains a mystery, as most studies are mainly based on reports and outcomes after surgical interventions. Nevertheless, HS plays a major role in the occurrence of focal seizures in adults, which are usually resistant to antiepileptic drugs (AEDs). It should be noted, however, that some patients with MRI evidence of HS do not have seizures resistant to therapy [17, 18].

The link between epileptic seizures and HS has been studied for over 150 years. Autopsy and neuroimaging studies have shown that patients with HS often have bilateral asymmetric hippocampal lesions. In addition, HS can be both a cause and a consequence of epileptic seizures, a debate that remains controversial to this day. Despite these differences, the clinical manifestations of temporal lobe epilepsy depend on a critical combination of different morphological changes. There is also evidence that even in experimentally induced cases of typical HS, seizures may not occur. These findings, along with some others, suggest that HS and epileptic seizures are symptoms of a more complex common pathological process underlying the disease [10, 19-22].

## Pathogenesis

The mechanisms underlying the development of HS remain largely unclear. Most likely, HS has different causes in different people and may be the result of a complex interaction of genetic and external factors. The role of the initial trauma in the origin of HS has been widely studied. This hypothesis postulates that HS may be the result of vari-



Figure 1. Microscopic anatomy of the human hippocampus [12]

Notes: cresyl violet and luxol violet staining of postmortem human hippocampus, illustrating the use of the International League Against Epilepsy (ILAE) classification terminology: SUB — subiculum; CA1-CA4 sectors of the Ammon's horn; DG — dentate gyrus with outer (DGe) and inner (DGi) edges; HF — hippocampal fissure remnant; ALV — alveolus; FIM — fimbriae. Dotted lines indicate the anatomical boundaries between the CA sectors. Scale bar = 1,000 lm.

ous factors, including a history of obstructed labour, head trauma, and prolonged febrile seizures in early childhood [3, 4, 23-25].

In the early 1950s, Penfield proposed that HS is caused by transtentorial insertion of the mesial temporal lobe at birth. This can lead to an ischaemic lesion called "incisural sclerosis", which can potentially lead to epilepsy over time [26]. In addition to a history of difficult childbirth, Meyer and Falconer identified other predisposing factors for HS, such as head trauma and, in particular, prolonged febrile seizures in early childhood — now called the Meyer hypothesis [27]. However, population-based studies have not demonstrated a significant association between early febrile seizures and later reliable development of temporal lobe epilepsy [28]. The interpretation of these results continues to be the subject of debate. It is possible to assume that early febrile seizures damage the hippocampus and thus cause HS. On the other hand, a child may have prolonged febrile seizures precisely because the hippocampus was previously damaged due to prenatal or perinatal trauma or genetic predisposition

Table 1. ILAE classification of hippocampal sclerosis (consensus) [12]

Class	Pathological pattern (neuronal loss and gliosis)			
	HS ILAE type 1	HS ILAE type 2 (CA1 prevails)	HS ILAE type 3 (CA4 prevails)	No HS Only gliosis
CA1	2	1–2	0-1	0
CA2	0-2	0-1	0-1	0
CA3	0-2	0-1	0-1	0
CA4	2	0-1	1–2	0
Dentate gyrus	0-2	0-1	0-2	0-1

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[25]. One theory suggests that an increase in the frequency of epileptic seizures may exacerbate the course of hippocampal sclerosis, while another suggests that the severity of HS may cause more intense seizures. Despite the fact that retrospective analyses reveal a link between seizure frequency and HS severity, they do not allow us to unequivocally determine what is the cause and the effect of these phenomena.

New research indicates that prolonged and focal febrile seizures can lead to acute hippocampal damage that progresses to atrophy, and that in some children complex febrile seizures may begin in the temporal lobes. Although retrospective studies have shown a high prevalence of complex febrile seizures among patients with mesial temporal sclerosis, it remains unclear whether complex febrile seizures are a clinical manifestation, a causative factor, or both [25]. Chemical and anatomical studies have shown that sensitive areas of the hippocampus are rich in kainate (endophallus and CA3) and NMDA receptors (CA1) [2]. When activated, NMDA receptors and a subclass of kainate receptors contribute to a significant influx of Ca<sup>2+</sup> into postsynaptic neurons. If these neurons are not protected from Ca<sup>2+</sup> during prolonged seizures, they can suffer irreversible damage and die. In the human hippocampus, the primary cells of the vulnerable sector, namely the endophallus and CA3 and CA1, contain virtually no Ca2+-binding proteins (calbindin or parvalbumin), while relatively resistant structures, such as dentate granule cells and sector CA2, are rich in calbindin [2, 3]. Thus, the destruction of neurons in sensitive hippocampal areas characteristic of hippocampal sclerosis may be a consequence of two of their chemical and anatomical features: a) the high content of glutamate receptors that promote Ca<sup>2+</sup> entry into the neuron during an attack, and b) their insufficient protection against Ca<sup>2+</sup> overload due to the almost complete absence of Ca<sup>2+</sup>-binding proteins. A similar pattern of hippocampal vulnerability to seizures, probably caused by the same pathogenetic mechanism, is also observed in some experimental models of epilepsy [2].

However, this pathogenetic mechanism does not explain the common unilateral nature of hippocampal sclerosis. Both prolonged febrile seizures (often unilaterally dominant) and their consequences may be due to preexisting hippocampal damage.

Genetic predisposition seems to be the main causative factor in patients with HS and previous long-standing febrile seizures. Febrile seizures (simple, complex) should be considered as signs of an abnormal brain process that is most likely regulated developmentally, as febrile seizures in HS often stop when the child reaches 5–6 years of age. However, it is worth considering the feasibility of further long-term follow-up to better understand possible negative consequences and associated risks that may occur over time [29].

A study was conducted on the effect of prolonged febrile seizures on gene expression in mesial temporal lobe epilepsy with hippocampal sclerosis, where 515 genes with altered expression were identified, which emphasises the significant impact of febrile seizures on gene activity. Five genes were identified (SNAP25, SLC32A1, SYN1, GRIN1, GRIA1) that were most significantly expressed in mesial temporal lobe epilepsy with hippocampal sclerosis with prolonged febrile seizures [30].

According to experimental and clinical studies, innate immunity is important in the development and progression of temporal lobe epilepsy. Inflammatory processes, detected through the activation of chemokine genes during epilepsy, can directly or indirectly affect nerve excitability. For example, activation of chemokines after viral infection, encephalitis, febrile seizures, trauma, tumours, or other events may be a key mechanism for the development of temporal lobe epilepsy after an initial injury. There is evidence of the role of complement activation and other inflammatory mechanisms in the formation of epileptic states, both in research models and in real clinical situations.

The notion that hippocampal sclerosis can be both a cause and a consequence of epileptic seizures [27] is supported by more recent studies [25]. Surgical findings and subsequent histopathological studies have expanded the concept of primary injury to include any significant medical event that may damage the brain prior to seizures, such as trauma, hypoxia, and intracranial infection [25]. These studies support the idea that hippocampal sclerosis is likely an acquired pathology, with the greatest neuronal loss occurring at the time of the initial injury, but frequent, ongoing seizures contributing to additional progressive hippocampal damage [21, 25].

Recent histopathological studies focusing on hippocampal development have led to two intriguing discoveries in HS samples from temporal lobe epilepsy patients undergoing surgery: 1) the persistence of Cajal-Retzius cells, indicative of early damage and altered conduction pathways, and 2) increased neurogenesis along with abnormal dentate granule cell layer structure, observed in young patients with early hippocampal seizures. These findings point to defects in the development of the hippocampus (inherited or acquired), which, in combination with subsequent life events (such as trauma, infection, febrile seizures), can lead to the development of recurrent seizures.

The presence of neuronal loss, aberrant axons and synaptic reorganisation seem to be associated with HS in humans. Descriptive studies show synaptic reorganisation of mossy fibre systems (axons of dentate granule neurons). The sprouting of mossy fibres involves the formation of new, aberrant synaptic contacts on the proximal dendrites of hippocampal dentate granule neurons. This abnormal pattern may be associated with epileptogenesis due to changes in excitatory and inhibitory processes. Numerous laboratory studies have provided important data for understanding the epileptogenesis of HS. These studies suggest that the affected hippocampus is significantly different from the "normal" hippocampus, demonstrating structural changes, alterations in neurotransmitters and receptors, modification of ion and water channels, changes in mitochondrial and glial function, and signs of inflammation [2, 31].

Consistent data from large series of surgical patients have confirmed the association between epilepsy severity and HS.

The presence of MS detected by MRI often correlates with unsuccessful medical treatment of seizures [17]. However, as discussed earlier, MRI abnormalities found in patients with successful treatment or seizure remission demonstrate that mesial temporal sclerosis is not exclusively found in medically uncontrolled seizures. In addition, some MRI findings confirm the existence of mesial temporal sclerosis that develops in adulthood, which is not always associated with poor seizure control [21, 22, 30].

Studies have shown that the development of neural damage and dysfunction in patients with temporal lobe epilepsy and hippocampal sclerosis gradually increases, but the exact causes, timing and pathways of these changes remain uncertain. The influence of seizure frequency, the ratio of genetic factors, primary brain damage, patient age and environmental conditions are key elements that contribute to the progression of damage, making it difficult to understand the mechanisms of HS.

In addition, the mechanisms responsible for the onset or progression of epilepsy are different from those that cause acute epileptic seizures. Another complicating issue is that seizure-related damage can manifest itself in different ways and does not always represent neuronal loss or atrophy. For example, many patients with HS with treatment-resistant seizures experience progressive memory loss, depression and signs of cognitive impairment, as well as a gradual increase in bilateral epileptiform discharges [2, 3, 24]. These data indicate that focal epileptic discharges can cause dysfunction of neurons distant from the seizure focus.

# Epilepsy and hippocampal sclerosis Risk factors for HS

As mentioned earlier, family history is one of the risk factors, along with prolonged febrile seizures and other brain damage in early childhood. However, these factors have been mainly documented in surgical series [10] and have not been conclusively confirmed in population-based studies.

## Clinical manifestations and semiology

Temporal lobe epilepsy with HS is traditionally characterised by a latency period between the initial damaging incident and the onset of seizures, although the primary damage is often subtle and undetectable. Seizures may initially



Figure 2. Typical semiology of a temporal lobe seizure in a patient with right-sided hippocampal sclerosis:
A – onset of motor manifestations with ipsilateral automatism in the right hand. Mild ipsilateral rotation of the head to the right;
B – complication of automatism, development of typical "masturbatory" automatism;
C – automatism in the right hand is joined by contralateral dystonia in the left hand;
D – the end of the seizure with the development of ambulatory automatism (the patient's attempts to escape)

respond well to treatment before becoming drug-resistant. Nevertheless, not all patients with temporal lobe epilepsy and HS develop refractory seizures, and people without a typical history are not uncommon, especially in familial cases [9].

The first typical seizures usually occur in late childhood or early adolescence. The first seizure event may be a generalised seizure or a focal seizure with impaired consciousness. Focal seizures are often preceded by an aura, which usually includes a "rising sensation" in the epigastrium associated with emotional distress, such as fear. Other mental (e.g., déjà vu) and autonomic symptoms (e.g., flushing, pallor, tachycardia) are also observed, and some patients may experience olfactory or gustatory sensations (it should be noted that gustatory and olfactory aura is more common in insular epilepsy than in temporal lobe epilepsy). Auras often occur alone (previously referred to as simple partial seizures), as well as in combination with focal seizures with impaired consciousness [3].

Focal seizures usually begin with fixed gaze and oral automatisms, such as lip smacking or chewing, which may be accompanied by impaired consciousness. Gesticulations and repetitive movements (automatisms) are also typical and may occur in the ictal or postictal period. Dystonic conditions of one limb are usually opposite to the side of the seizure. Hand automatisms are frequent and mostly correspond to the side of hippocampal sclerosis, especially when accompanied by dystonia on the opposite side. Verbal automatisms can be observed in seizures in the non-dominant hemisphere. Postictal disorientation is temporary, and speech impairment is possible in seizures in the linguistically dominant hemisphere. Postictal nasal wiping is often performed with the hand on the side of the seizure onset. Patients are usually unable to recall the ictal period, although they can partially respond to stimuli during this time. The aura, however, remains in the memory. Often, after the seizure, ambulatory automatism occurs — the patient tries to go somewhere, looks for something, runs away (Fig. 2).

The attacks usually last between one and two minutes and are relatively constant and stereotypical in a given patient. Patients may have episodic auras many years before they have their first focal seizure with impaired awareness. Triggering factors include stress, lack of sleep, and hormonal changes associated with the menstrual cycle in women. Secondary generalisation, as well as status epilepticus, are rare but can occur [6].

There are no clear features that distinguish focal seizures in temporal lobe epilepsy with HS from focal seizures generated in the anterior temporal lobe. The classic presentation, as described above, may be similar to ictal symptoms reported by patients with mesial temporal lesions other than HS or without any detectable MRI abnormalities. Thus, an



Figure 3. Typical EEG changes in HS: A — acute wave-slow wave complexes over the anterior temporal leads of the left hemisphere (electrodes T1 and T3, monopolar installation, use of an additional T2 sensor); B — rhythmic theta and delta activity over the anterior parts of the left hemisphere of the brain at the beginning of the attack (maximally expressed under electrode F7)

#### Огляд / Review



Figure 4. Typical changes on MRI of the brain in a patient with left-sided hippocampal sclerosis. Reduced size of the left hippocampus and increased signal intensity from it: A — in T2 mode; B — in FLAIR mode; C — in T1 mode

accurate diagnosis is based on a combination of clinical signs and diagnostic findings [5].

Seizures that begin with primary visual, auditory, or focal somatosensory auras, focal or violent motor behaviour, and the presence of extratemporal electroencephalography (EEG) spikes do not meet the clinical criteria for temporal lobe epilepsy with HS [5, 6].

## Diagnostic criteria and additional examinations

The diagnosis of temporal lobe epilepsy with HS requires a combination of signs and symptoms, among which the most important is the presence of a characteristic seizure semiology [14–16]. The diagnosis of hippocampal sclerosis requires concordance of MRI, EEG, video EEG, and neuropsychological testing, and for research purposes, positron emission tomography (PET) and single-photon emission computed tomography SPECT (Fig. 3).

The neurological examination is usually normal, except for isolated cases of facial asymmetry or memory deficits, which can differ significantly depending on whether the dominant hemisphere is affected or not [33].

The interictal EEG usually shows unilateral or bilateral independent mesial temporal discharges, which are best visualised with basal leads, such as sphenoidal and inferior temporal electrodes. Temporal intermittent rhythmic delta activity has been found to be of localising importance for the epileptogenic zone in mesial temporal lobe epilepsy, which distinguishes it from intermittent rhythmic delta activity in other brain regions [5].

Seizure EEG recordings usually demonstrate a characteristic pattern of regular, well-lateralised rhythmic activity (most commonly theta) in the anterior temporal and inferior temporal regions. This pattern occurs before the first clinical manifestations or within the first 30 seconds of the seizure, with or without contralateral spread [9].

In some cases, when the side of the mesial temporal seizure onset cannot be determined or superficial cortical foci are suspected, long-term monitoring with intracranial electrodes is recommended. This is often achieved through the insertion of deep electrodes, although in some cases subdural tapes, meshes or oval electrodes may be used. Currently, there are no biochemical markers that would allow the diagnosis of HS. Despite the active study of immunological factors of temporal lobe epilepsy, no specific immunological tests for the detection of HS have been developed yet.

High-resolution MRI proves to be a reliable non-invasive tool for the diagnosis of HS, especially when using coronal slices oriented perpendicular to the hippocampal axis. Thin slices (up to 3 mm) improve visualisation of hippocampal anatomy. T1-weighted inversion-recovery images are important for analysing the size, shape and structure of the hippocampus, while T2-weighted or FLAIR images help assess signal intensity.

The characteristic MRI features of HS include atrophy of one hippocampus with increased signal on T2-weighted images and FLAIR sequences, along with a normal appearance of the opposite hippocampus. Alterations in the amygdala, temporal neocortex, white matter, fornix, insula, thalamus, or basal frontal cortex may be detected, as well as enlargement of the temporal horns of the lateral ventricles (Fig. 4).

Studies have shown that MRI analysis performed by a neuroradiologist with experience in diagnosing epileptogenic brain lesions has a higher sensitivity than analysis by a general radiologist, probably due to the radiologist's inability to assess changes in signal or hippocampal T2 architecture [10].

Proton magnetic resonance imaging (1H-MRI) has revealed decreased N-acetyl aspartate indicative of neural dysfunction in patients with temporal lobe epilepsy and hippocampal sclerosis, including those whose conventional MRI showed no abnormalities. This decrease in N-acetyl aspartate, which is reflected in both singleand multi-voxel analyses, correlates with EEG abnormalities and cell loss, offering a more sensitive diagnostic method than standard MRI. Fluorodeoxyglucose PET scans often reveal hypometabolism in the damaged temporal region, highlighting its value in identifying target areas for surgical intervention. However, the use of PET and SPECT scanning is limited due to high costs and logistical difficulties, although SPECT may be useful for identifying epileptogenic foci in the early postictal period in patients with temporal lobe epilepsy.

Neuropsychological testing reveals that memory impairment in patients with hippocampal sclerosis correlates with the hemisphere involved, with verbal memory being affected in the left hippocampus and visuospatial memory in the right. Cognitive impairment in patients with temporal lobe epilepsy is often associated with neurodegenerative processes, in particular with HS. Various aspects of the course of cognitive impairment in HS are considered, including the relationship between seizure frequency, hippocampal atrophy, and the potential role of tau pathologies. The mechanisms underlying cognitive impairment are not fully understood, despite studies of clinical factors such as seizure frequency and cellular mechanisms of excitotoxicity. Early onset of seizures leads to poor verbal memory development and impedes cognitive potential.

# **Principles of treatment**

The management of HS in patients with epilepsy is based on therapeutic principles and may include medical and surgical intervention. Patients with hippocampal sclerosis have a variety of clinical responses to treatment with anticonvulsants. This variability in response to AEDs may be due to several factors, such as the duration and severity of the disease, structural changes in the brain, network disorders, and genetic aspects. An important role in this process is played by the etiology of seizures, in particular unilateral hippocampal sclerosis, which is often associated with drug resistance, although in some cases success can be achieved [34].

For treatment, we use those drugs that have the fewest side effects. Each drug is selected individually, initially as monotherapy at the lowest therapeutic dose and then increasing. If seizures recur, higher doses or combinations of AEDs can be considered, in which case careful monitoring of interactions and side effects is essential. Carbamazepine, levetiracetam or phenytoin as monotherapy are appropriate choices for the treatment of HS, and higher serum levels may be required compared to generalised seizures. Other broad-spectrum AEDs, such as valproate, topiramate, and lamotrigine, may be useful if initial medications fail [35].

A study [32] demonstrated that both old and new anticonvulsants have low efficacy in the medical treatment of seizures in hippocampal sclerosis. In this analysis, levetiracetam and carbamazepine were assessed as the most effective in HS; the proportion of patients free of seizures was only 11.0 % in the carbamazepine group and 9.2 % in the levetiracetam group. These results are supported by the findings of a group of researchers from Texas who compared old and new anticonvulsants in the treatment of mesial temporal lobe epilepsy. But they did not find any advantage of the latter in terms of effectiveness. In this study, the highest proportion of patients free from seizures was in the group taking phenytoin, levetiracetam and lamotrigine - 28.6, 24.6 and 23.8 %, respectively [32].

However, when seizures are medically refractory and cannot be treated with AEDs, surgery becomes a treatment option. Surgery is considered when patients meet the criteria for medical refractoriness, which usually includes the inability to achieve seizure control with two or more AEDs in adequate doses and properly prescribed regimens. Referral to an epilepsy centre should be made at an early stage when it becomes apparent that seizure control cannot be achieved with first-line drugs. Patients with temporal lobe epilepsy and unilateral HS are excellent candidates for surgical treatment, with a 60–80% chance of being free of disabling seizures. However, long-term seizure-free rates decrease over time.

The preoperative work-up for HS includes clinical factors, EEG, MRI, neuropsychological assessment, and sometimes functional imaging [9]. The choice of surgical approach, such as an anterior temporal lobectomy or a more selective approach, may depend on the preferences of the surgical team [9]. Temporal lobectomy is most commonly associated with postoperative seizure control, but can lead to impaired verbal memory, especially in the language-dominant hemisphere. Measurement of hippocampal volume can help predict postoperative memory impairment, with bilaterally symmetrical severe hippocampal atrophy posing the greatest risk.

The treatment of HS requires a comprehensive approach that includes AEDs as initial treatment and consideration of surgery in cases of drug resistance. The success of surgery varies from patient to patient, and ongoing research is needed to improve treatment outcomes through a better understanding of neuropathological patterns and the underlying pathogenesis of HS [35].

# Conclusions

The diagnosis of mesial temporal lobe epilepsy with hippocampal sclerosis is based on a combination of clinical signs and symptoms, including the characteristic seizure semiology, as well as ancillary diagnostic tests such as MRI, EEG, video EEG, and neuropsychological assessment. HS is the most common pathological substrate in pharmacoresistant temporal lobe epilepsy, accounting for 60-70 % of patients undergoing surgical treatment.

Generalised seizures are a complex condition with numerous potential etiological factors contributing to its development. The various underlying causes may explain the different responses to pharmacotherapy, ranging from patients who have good seizure control with AEDs to those who remain pharmacoresistant and require surgery. The success rate of surgery for HS is not uniform, with approximately 60-65% of patients experiencing significant improvement or absence of seizures, while 25-30% have limited improvement. It should be noted that some patients who are initially seizure-free after surgery may experience relapses 5-10 years after it, while other patients remain seizure-free for a long time.

To improve treatment outcomes, further research is needed to better stratify neuropathological patterns and gain a deeper understanding of the pathogenesis of HD. These advances will contribute to more rational and targeted therapeutic approaches.

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### Типовий перебіг гіпокампального склерозу: огляд літератури

Резюме. У статті проведено огляд проблеми гіпокампального склерозу як основної причини фармакорезистентної скроневої епілепсії. Наведено клінічні прояви, діагностичні критерії, рекомендовані методи обстеження та лікування гіпокампального склерозу. Проведено аналіз ефективності хірургічного й медикаментозного лікування. Актуальність цієї статті зумовлена загальним зростанням виявлення даного захворювання, у першу чергу за рахунок поліпшення методів нейровізуалізації. Зважаючи на те, що дане захворювання є відносно рідкісним, і те, що хірургічне лікування часто проводиться із затримкою в багато років, огляд цієї теми є корисним для ранньої діагностики й поліпшення результатів лікування. Були досліджені сучасні літературні джерела на тему гіпокампального склерозу. Вивчений матеріал узагальнений і поданий в цій статті у формі огляду літератури. Пошук літературних джерел здійснювався у двох основних наукових базах даних: Scopus і PubMed. Огляд включав оригінальні статті, дослідження й офіційні рекомендації медичних асоціацій.

**Ключові слова:** епілепсія; гіпокампальний склероз; фармакорезистентна епілепсія; скронева епілепсія; огляд