



POSTOPERATIVE OCCULT OTOGENIC CEREBROSPINAL FLUID LEAKAGE IN TEMPORAL-BASAL BRAIN TUMOR SURGERY: PATHOPHYSIOLOGY, DIAGNOSIS, AND MANAGEMENT STRATEGIES

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Abstract. Postoperative cerebrospinal fluid (CSF) leakage remains one of the most severe complications in skull base neurosurgery and is associated with a high risk of intracranial infectious complications. Occult otogenic CSF leakage is a rare but clinically significant condition that often mimics nasal liquorrhea and may remain undiagnosed in the early postoperative period. The present study analyzes the etiopathogenetic mechanisms, diagnostic approaches, and therapeutic strategies for postoperative occult otogenic CSF leakage in patients undergoing temporal-basal brain tumor surgery. Clinical, biochemical, and radiological data from patients with postoperative CSF leakage were analyzed, emphasizing the role of temporal bone fractures and dural defects as primary etiological factors. Conservative treatment combined with lumbar drainage and local otorhinolaryngological therapy demonstrated high efficacy in eliminating CSF leakage and preventing infectious complications. Early diagnosis and targeted preventive neurosurgical strategies are crucial to improving postoperative outcomes in patients with cranial base tumors.

Keywords: cerebrospinal fluid leakage, skull base tumors, temporal bone fracture, otogenic liquorrhea, neurosurgical complications, lumbar drainage.

ПОСЛЕОПЕРАЦИОННАЯ СКРЫТАЯ ОТОГЕННАЯ ЛИКВОРЕЯ ПРИ ХИРУРГИЧЕСКОМ ЛЕЧЕНИИ ВИСОЧНО-БАЗАЛЬНЫХ ОПУХОЛЕЙ ГОЛОВНОГО МОЗГА: ПАТОФИЗИОЛОГИЯ, ДИАГНОСТИКА И ТАКТИКА ВЕДЕНИЯ

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Аннотация. Послеоперационная ликворея является одним из наиболее серьезных осложнений в хирургии основания черепа и сопровождается высоким риском внутричерепных инфекционных осложнений. Скрытая отогенная ликворея представляет собой редкое, но клинически значимое состояние, которое часто имитирует назальную ликворею и может оставаться недиагностированным в раннем послеоперационном периоде. В данной работе анализируются этиопатогенетические механизмы, диагностические подходы и терапевтические стратегии лечения скрытой отогенной ликвореи у пациентов, перенесших хирургическое лечение височно-базальных опухолей головного мозга. Проанализированы клинические, биохимические и радиологические данные, с акцентом на роль переломов пирамиды височной кости и дефектов твердой мозговой оболочки как основных этиологических факторов. Консервативная терапия в сочетании с люмбальным дренированием и местным оториноларингологическим лечением продемонстрировала высокую эффективность в устранении ликвореи и профилактике инфекционных осложнений. Ранняя диагностика и профилактические нейрохирургические стратегии являются ключевыми факторами улучшения послеоперационных исходов.

Ключевые слова: ликворея, опухоли основания черепа, перелом височной кости, отогенная ликворея, нейрохирургические осложнения, люмбальный дренаж.

VAQTINCHALIK-BAZAL MIYA O'SMALARINI JARROHLIK DAVOLASHDAN SO'NG YASHIRIN OTOGEN LIKVOREYA: PATOFIZIOLOGIYA, DIAGNOSTIKA VA DAVOLASH STRATEGIYALARI

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Annotatsiya. Operatsiyadan keyingi likvoreya bosh suyagi asosini jarrohlik davolashda eng og'ir asoratlardan biri bo'lib, intrakranial infeksiya asoratlari xavfi bilan bog'liqdir. Yashirin otogen likvoreya kam uchraydigan, ammo klinik jihatdan muhim holat bo'lib, ko'pincha nazal likvoreyani taqlid qiladi va erta operatsiyadan keyingi davrda aniqlanmay qolishi mumkin. Ushbu tadqiqotda vaqtinchalik-bazal miya o'smalarini jarrohlik davolashdan keyin yuzaga keladigan yashirin otogen likvoreyaning etiopatogenetik mexanizmlari, diagnostik yondashuvlari va davolash strategiyalari tahlil qilindi. Klinik, biokimyoviy va radiologik ma'lumotlar asosida chakka suyagi piramidasining sinishi va dura mater nuqsonlari asosiy etiologik omillar sifatida baholandi. Konservativ terapiya lumbal drenaj va mahalliy otorinolaringologik davolash bilan birgalikda likvoreyani bartaraf etishda yuqori samaradorlik ko'rsatdi. Erta diagnostika va profilaktik neyroxirurgik strategiyalar operatsiyadan keyingi natijalarni yaxshilash uchun muhim hisoblanadi.

Kalit so'zlar: likvoreya, bosh suyagi asosi o'smalari, chakka suyagi sinishi, otogen likvoreya, neyroxirurgik asoratlari, lumbal drenaj

Introduction. Cerebrospinal fluid (CSF) leakage represents one of the most clinically significant postoperative complications in neurosurgical oncology, particularly following cranial base tumor resections. Basal brain tumors, including meningiomas, gliomas, and other skull base neoplasms, often require complex cranio-basal surgical approaches that inevitably increase the risk of dural defects, osseous fractures, and iatrogenic injury to the skull base structures. Postoperative liquorrhea remains a major concern because of its association with intracranial infections, including meningitis, ventriculitis, and brain abscess formation, which significantly increase morbidity and mortality [1,2].

The cranial base is a complex anatomical region composed of multiple osseous and neurovascular structures. Surgical interventions in this region often involve extensive bone drilling, dural dissection, and manipulation of pneumatized temporal bone structures. Such procedures can disrupt the integrity of the temporal bone pyramid and middle cranial fossa, leading to the development of occult otogenic cerebrospinal fluid leakage. This phenomenon is particularly challenging to diagnose because CSF may drain through the Eustachian tube into the nasopharynx, mimicking nasal liquorrhea and thereby masking its otogenic origin. The clinical significance of postoperative CSF leakage is underscored by its reported incidence ranging from 2.5% to 4% following cranial base trauma or surgery, with an associated risk of meningitis reaching 13–25% [3]. Despite advancements in microsurgical techniques, neuronavigation, and skull base reconstruction, occult CSF leakage remains underdiagnosed due to subtle clinical manifestations and insufficient postoperative surveillance protocols. The present study aims to analyze the etiological mechanisms underlying iatrogenic occult otogenic CSF leakage following cranio-basal tumor surgery, to refine diagnostic algorithms, and to propose optimized therapeutic and preventive strategies in neurosurgical practice. The study is based on clinical observations and diagnostic findings in patients who developed postoperative occult otogenic liquorrhea following temporal-basal tumor resection.

Materials and Methods. A retrospective analysis was conducted on three male patients (mean age 45 years) who underwent surgical treatment for temporal-basal brain tumors and subsequently developed postoperative occult otogenic cerebrospinal fluid leakage. All patients underwent comprehensive preoperative neuroimaging in accordance with contemporary neurosurgical standards, including contrast-enhanced computed tomography (CT), magnetic resonance imaging (MRI), MRI angiography, and single-photon emission computed tomography (SPECT) with tumorotropic radiopharmaceuticals in cases of suspected malignancy. Tumors were

localized predominantly in the mediobasal temporal lobe, with extension into adjacent lobes. In two cases, the neoplasms extended into the parietal lobe, while in one case extension into the posterior frontal lobe was observed. Surgical access was achieved using bone-plastic craniotomy techniques with basal bone flap formation. Postoperatively, patients were monitored for neurological deficits, otological symptoms, and signs of CSF leakage. Clinical symptoms included ipsilateral hearing impairment, ear fullness, and persistent nasal moisture sensation. Otoloscopic examination was performed to evaluate tympanic membrane integrity and detect hemotympanum. Audiological assessments included tonal audiometry to differentiate sensorineural and conductive hearing loss.

Biochemical analysis of nasal secretions was conducted using rapid glucose detection strips ("Glucotest"), inserted into the inferior nasal meatus to identify CSF glucose content. Radiological evaluation included temporal bone radiography in Schüller and Stenvers projections and axial CT scans to identify longitudinal fractures of the temporal bone pyramid, pneumatization changes, and pneumocephalus. Therapeutic management included conservative and surgical interventions. Conservative measures consisted of strict bed rest, fluid restriction, dehydration therapy, antibacterial prophylaxis, and local otorhinolaryngological treatment. Surgical interventions included lumbar puncture with subarachnoid air injection and lumbar drainage in selected cases. Audiological monitoring was performed throughout hospitalization and during follow-up examinations at 3 months.

Results. All patients developed clinical symptoms within 2–3 days after surgery, including ipsilateral conductive hearing loss, ear fullness, and subjective nasal moisture. Otoloscopic evaluation revealed bluish discoloration of the tympanic membrane, hemotympanum, membrane thickening, absence of the light reflex, and poorly defined anatomical landmarks. Soft tissue hemorrhage in the mastoid region (Battle's sign) was observed. Audiological testing demonstrated bilateral sensorineural hearing impairment associated with tumor-related neurophysiological damage and additional ipsilateral conductive hearing loss caused by middle ear pathology secondary to CSF accumulation. The endonasal glucose test was positive in all patients, confirming the presence of CSF in nasal secretions. Radiological imaging demonstrated longitudinal fractures of the temporal bone pyramid at the level of the auditory canal, decreased pneumatization of temporal bone cells, and pneumocephalus in one patient.

Therapeutic Outcomes. Conservative therapy was initiated in all cases. Lumbar puncture with subarachnoid air injection was performed in two patients, while lumbar drainage for up to 10 days was used in one patient. Treatment protocols included fluid restriction (1–1.5 L/day), antibacterial therapy, dehydration therapy, and symptomatic management. Local therapy included vasoconstrictive and antibacterial ear and nasal drops, nasal antibiotic inhalation, endaural phonophoresis, and electrophoresis of medications. Audiological monitoring demonstrated objective improvement in conductive hearing loss and subjective reduction in tinnitus. Follow-up endonasal glucose tests on days 5 and 10 were negative in all patients, and long-term follow-up at 3 months confirmed complete resolution of CSF leakage.

Discussion. Occult otogenic CSF leakage is primarily caused by iatrogenic injury to the temporal bone pyramid during basal craniotomy. Basal bone flap formation may result in longitudinal fractures of the temporal bone, leading to dural defects and CSF communication with pneumatized mastoid cells and the middle ear cavity. The CSF subsequently drains through the Eustachian tube into the nasopharynx, mimicking nasal liquorrhea. This mechanism highlights the anatomical vulnerability of the cranial base during neurosurgical interventions, especially in elderly patients with osteoporosis, hyperostosis associated with meningiomas, and skull base anatomical variations. The absence of tympanic membrane perforation complicates clinical diagnosis and may delay therapeutic intervention. The detection of occult otogenic CSF leakage remains challenging due to its nonspecific clinical presentation. Patients may report only nasal moisture, mild hearing loss, or ear fullness. Standard nasal CSF leakage diagnostic protocols may fail to identify the otogenic origin, resulting in misclassification as rhinogenic liquorrhea. Endonasal glucose testing using indicator strips provides a rapid and cost-effective diagnostic tool, especially in cases where CSF volume is insufficient for laboratory analysis. Radiological imaging remains essential for

identifying skull base fractures and pneumatization changes. High-resolution CT of the temporal bone is considered the gold standard for detecting temporal bone fractures and CSF fistulas [4].

Conservative management remains the first-line therapy for postoperative CSF leakage, including bed rest, fluid restriction, dehydration therapy, and antibiotic prophylaxis. Lumbar drainage reduces intracranial pressure and promotes spontaneous closure of dural defects. Surgical reconstruction is indicated in persistent leaks or recurrent meningitis cases. Local otorhinolaryngological therapy plays a crucial role in preventing secondary infections. Combined systemic and local antibacterial therapy reduces the risk of meningitis, which remains the most severe complication of postoperative CSF leakage. Preventive strategies include meticulous basal bone flap formation, careful inspection of the cranial base during surgery, and immediate reconstruction of skull base defects using adhesive compositions or autologous grafts. Patients with technically difficult craniotomies should undergo postoperative otoneurological monitoring to detect early signs of occult CSF leakage. Early diagnosis and timely intervention significantly reduce the risk of intracranial infectious complications and improve functional outcomes.

Conclusion. Postoperative occult otogenic cerebrospinal fluid leakage represents a clinically significant and underdiagnosed complication following temporal-basal brain tumor surgery. Iatrogenic temporal bone fractures during basal craniotomy constitute the primary etiological mechanism. Early clinical recognition, biochemical confirmation, and radiological assessment are essential for accurate diagnosis. Conservative therapy combined with lumbar drainage and local otorhinolaryngological treatment demonstrates high efficacy in resolving postoperative CSF leakage. Preventive neurosurgical strategies, including careful cranial base reconstruction and postoperative otoneurological surveillance, are critical for reducing the incidence of this complication. Timely elimination of CSF leakage prior to hospital discharge and long-term follow-up verification are mandatory to prevent delayed infectious complications and improve neurosurgical outcomes.

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