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Management of subarachnoid hemorrhage in a neurological department (clinical case)

Subarachnoid hemorrhage (SAH) is a life-threatening form of stroke (5—10 % of strokes) caused predominantly by aneurysm rupture. In 10 % to 15 % of patients with spontaneous subarachnoid hemorrhage, no aneurysm is detected on the first angiogram. In two thirds of these patients the CT scan shows a perimesencephalic pattern of hemorrhage. Patients typically present with sudden thunderclap headache, neck stiffness, vomiting, and neurological deficits. Early mortality is 25—50 %, and complications such as rebleeding, vasospasm, and hydrocephalus drive outcomes. Non-aneurysmal SAH generally has a better prognosis than aneurysmal SAH, with many patients experiencing full recovery. Modern management requires rapid CT-based diagnosis (high sensitivity if within 6 h), prompt angiography, and neurocritical unit management. Key measures include meticulous blood pressure (BP) control (e.g. systolic BP <160 mmHg), oral nimodipine for 21 days, intracranial pressure reduction, seizure prophylaxis if needed, and deep vein thrombosis prophylaxis. Remaining controversies (e.g. optimal BP targets, nimodipine dosing) are noted. Complications include rebleeding mainly in first 24—72 h, cerebral vasospasm in 60—70 % angiographically, hydrocephalus, seizures which present in 5—15 %, cardiac and pulmonary disorders, hyponatremia, ventriculitis.

Aseptic meningitis is a well-recognized sterile inflammatory reaction occurring 3—10 days after SAH, caused by the breakdown of blood products in the subarachnoid space. In non-aneurysmal SAH — including perimesencephalic cases — this inflammation can mimic meningitis with headaches, fever, and neck stiffness. In SAH, the cytosis in the cerebrospinal fluid (CSF) undergoes significant changes, reflecting the inflammatory response to blood degradation products. A predominance of neutrophilic pleocytosis in the first 1—3 days after the hemorrhage is observed. This is a reaction to inflammation caused by blood breakdown products. Over time, usually from 3—4 days onwards, lymphocytes may become more prominent in the cytosis structure, although general pleocytosis may persist for quite a long time (up to 2—3 weeks). In addition, CSF findings may also include erythrocytes and macrophages, which appear later to engulf erythrocytes. We present a recent case of CT-confirmed SAH managed conservatively, illustrating these principles. The classic presentation (sudden headache, stiff neck) and CT findings prompted immediate treatment. The absence of an aneurysm on angiography suggested perimesencephalic-type SAH, which carried a better prognosis. The management followed guideline principles: admission to a neurocritical setting, controlled BP, prophylactic administration of nimodipine, and vigilant monitoring.

The presented case is notable because of the dynamic changes observed in the CSF profile. In our case, the results of the CSF examination raised diagnostic questions, so several of CSF studies were conducted. In the first study, lymphocytic pleocytosis (82 %) prevailed, neutrophils were 18 %, and elevated protein levels (2.28 g/L) were detected. When re-analyzed 2 days later, the protein decreased by 3 times, mixed pleocytosis was detected: lymphocytes (68 %) and increased proporsion of neutrophils (32 %). This raised questions, since the expected increase in the number of neutrophils was not at the beginning of the hemorrhage, but occurred during treatment simultaneously with an increase in the neck stiffness as a meningeal symptom. It was important not to miss the concomitant infectious lesion. Rebleeding was excluded, chlorides and glucose in the CSF were within normal limits. When examining the cerebrospinal fluid after 3 days, the protein increased slightly, cytosis increased to $58.5 \cdot 10^2$/L, lymphocytic pleocytosis prevailed (75.5 %), neutrophils were 24.5 %. In the control study, after another day, the protein decreased by half, lymphocytic pleocytosis prevailed (75 %), neutrophils were 25 %. Objectively, the headache and neck stiffness muscles decreased, the general condition and mobility improved. Thus, the observed clinical and CSF dynamics was regarded as aseptic (reactive) meningitis with subarachnoid hemorrhage.

Keywords: non-aneurismal subarachnoid hemorrhage (NASAH), examination of cerebrospinal fluid, brain CT, CT angiography of the head and neck.

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Subarachnoid hemorrhage (SAH) is defined as bleeding into the subarachnoid space and is most often due to a ruptured cerebral aneurysm [10]. Its incidence is near 10—14 per 100,000 per year. SAH patients are younger than typical stroke patients, with peak incidence in the 50s—60s [2]. Risk factors include hypertension, smoking, family history, and polycystic kidney disease. Prognosis is poor: without treatment, one-third die on first day, and up to 50 % die within weeks [12, 15, 19]. Even survivors often have cognitive or neurological deficits [14]. It is thus critical that suspected SAH is rapidly identified and managed per current guidelines (AHA/ASA 2012 and 2023, ESO 2013, NICE 2022, etc.), which emphasize specialized multidisciplinary care. In Ukraine, a Unified clinical protocol of emergency, primary, secondary (specialized), tertiary (highly specialized) medical care and medical rehabilitation for hemorrhagic stroke (intracerebral hematoma, aneurysmal subarachnoid hemorrhage) is used. According to the Protocol, modern principles of patient management are established: specialist consultations, diagnostic procedures, treatment methods and their results, as well as time standards [1].

In 10 % to 15 % of patients with spontaneous SAH, no aneurism is found on the first angiogram. In two thirds of these patients the CT scan shows a perimesencephalic pattern of hemorrhage; may be shown diffuse, sulcal, and primary intraventricular location also [6, 16]. Non-aneurismal SAH (NASAH) generally has a better prognosis than aneurysmal SAH, with many patients experiencing full recovery.

According to pathophysiology, an acute rise in intracranial pressure (ICP), global cerebral ischemia, and irritation of meninges are registered. Blood breakdown products cause vasospasm, peaking at 3—14 days [3]. Symptomatic delayed cerebral ischemia (DCI) occurs in near 20—40 % if not prevented. Other consequences include acute hydrocephalus (30 % of cases) from arachnoid granulation blockage, and neurogenic myocardial dysfunction due to catecholamine surge [13]. The Hunt—Hess scale and World Federation of Neurosurgical Societies scale (WFNS) grade initial severity. The Fisher scale classifies the CT blood load and correlates with vasospasm risk.

SAH typically presents as a sudden, severe headache often described as «thunderclap» or «worst headache of life». Nausea, vomiting, photophobia, and neck stiffness (meningismus) are common [6]. Transient loss of consciousness occurs in near 40 %. Focal deficits (e.g. third nerve palsy) occur if specific regions are affected. Seizures occur in about 5—15 % of patients; when present, they may indicate extensive cortical irritation. On exam, neck stiffness and photophobia suggest meningeal irritation, and a positive Kernig/Brudzinski sign often appears. A high Hunt—Hess grade (e.g. sopor or coma) signals severe hemorrhage. These findings should prompt immediate neuroimaging.

Conservative SAH management in a neurology setting involves neuroprotective critical care: rapid stabilization, BP control, nimodipine, vigilant ICP/fluid/electrolyte management, and prevention of complications [4, 16]. This bridges the patient safely through the high-risk period until definitive aneurysm treatment (if any) is achieved. Immediate priorities in treatment follow the ABCs: secure airway if Glasgow Coma Scale (GCS) ≤ 8 or if vomiting is persistent; ensure adequate oxygenation and ventilation. Establish intravenous access and place arterial line for close blood pressure (BP) monitoring. To reduce rebleeding risk, maintain systolic BP < 150—160 mmHg. Intravenous antihypertensives are used to keep BP within this range. Necessary to avoid hypotension, as cerebral perfusion must be preserved. Analgesics and sedation (e.g. fentanyl, propofol) can help lower BP and improve comfort [17]. Short-acting sedatives should be used cautiously to allow frequent neuro exams. Oral nimodipine 60 mg every 4 hours (or 30 mg per day if hypotension) for 21 days is mandatory. This calcium-channel blocker is the only therapy proven to improve outcome (reduce DCI) in SAH [8]. It is started as soon as possible (even before angiography). Even though its exact mechanism is unclear, it is given prophylactically in all aneurysmal SAH patients (level I evidence). Recent studies underscore that skipping or under-dosing nimodipine may worsen outcomes, so adherence is emphasized. Controversies remain, such as the optimal BP target (some advocate lower systolic BP than 160 mmHg) and the utility of hyperdynamic therapy. The balance between preventing rebleeding and maintaining cerebral perfusion is delicate. Also, while nimodipine is standard, questions persist about intravenous vs oral administration and dose adjustments [18]. Necessary is maintaining of normovolemia; neither prophylactic hypervolemia nor hemodilution are recommended in the absence of symptomatic vasospasm. Using of isotonic fluids (e.g. normal saline) to avoid hypovolemia is recommended. Hyponatremia is corrected to keep sodium > 135 mEq/L, as low Na can exacerbate cerebral oedema and DCI. Keep head-of-bed elevated at 30°. Osmotic therapy (mannitol or hypertonic saline) is used for acute ICP spikes. Clinical seizures should be treated promptly (e.g. with levetiracetam or phenytoin). Prophylactic anticonvulsants are not universally recommended unless seizures occur on EEG. Deep vein thrombosis prophylaxis is used immediately with the help of compression stockings or pneumatic devices. Low-dose subcutaneous heparin or enoxaparin is used usually after 24—48 h, when bleeding is controlled, to prevent pulmonary embolism, unless contraindicated.

Complications include rebleeding mainly in first 24—72 h, cerebral vasospasm in 60—70 % angiographically, hydrocephalus, seizures which present in 5—15 %, cardiac and pulmonary disorders, hyponatremia, vasospasm, ventriculitis [5].

Aseptic meningitis is a well-recognized sterile inflammatory reaction occurring 3—10 days after SAH, caused by the breakdown of blood products in the subarachnoid space. In NASAH — including perimesencephalic cases — this inflammation can mimic meningitis with headaches, fever, and neck stiffness [3, 8].

Results of 101 valid cerebrospinal fluid (CSF) specimen data indicated that in 95 % of patients after spontaneous SAH, the leukocyte count in CSF was less than $880 \cdot 10^6$/L. Additionally, the proportion of neutrophils, lymphocytes, and monocytes did not exceed 75 %, 75 %, and 15 %, respectively, in 95 % of the population. Furthermore, in 95 % of the specimens, the concentration of chloride, glucose, and protein was > 115 mmol/L, > 2.2 mmol/L, and < 2.3 g/L, respectively. Compared to the normal reference values, the CSF indexes after spontaneous SAH showed significant changes, especially in the centration. Using «white blood cell count < 880/mm^3, glucose > 2.2 mmol/L, chloride > 115 mmol/L» as the reference values for SAH pathological status is more meaningful for reference purposes [8].

Case history

The presented case is interesting in terms of the dynamics of CSF.

Woman, 65 years old, retired, resident of Kyiv, was treated in the neurological department within 15 days.

Diagnosis: Cerebrovascular disease. Cerebrovascular atherosclerosis. Spontaneous SAH with right-sided pyramidal insufficiency, central paresis of the VII pair of cranial nerves on the right, pronounced cephalgia syndrome. Arterial hypertension III stage, stage 2, risk 4. Asthenic-neurotic syndrome.

Complaints of the patient: severe headache, more in the occipital region; pain in the cervical spine.

Medical history: According to the patient and relatives, in the morning at about 9 am, while bending down, she felt a sharp headache and neck pain, so emergency medical care (EMC) team was called, but she refused hospitalization. Due to the fact that her condition did not improve, she was taken to the emergency department of the hospital, accompanied by her relatives, examined by a neurologist on duty, CT + CT-angiography of the brain was performed, and she hospitalized according to the act of self-referral to the stroke department.

Life history (from the patient's words): Allergological, epidemiological, oncological anamnesis were not burdened. Tuberculosis, hepatitis, venereal diseases, diabetes mellitus — denied. Had chronic diseases — cataract, glaucoma. Did not take medications on a regular basis, used only eye drops. Had surgical interventions — appendectomy 52 years ago.

Objectively: General condition of the patient was severe. The skin and visible mucous membranes were pale, clean. Peripheral lymph nodes were palpable, not enlarged, not painful on palpation. There was hard breathing in the lungs. Cor tones were muffled, rhythmic. BP 180/90 mm Hg, pulse 84 beats/min, rhythmic. The abdomen was soft, not enlarged, did not respond to palpation.

Neurological status: Consciousness was clear (GCS 15 points). She followed the instructions, was available for productive contact. Palpebral fissures D = S, pupils D = S, following the malleus, small-sweeping horizontal nystagmus bilaterally were registered. The face was asymmetrical, due to the smoothing of the right nasolabial fold. The tongue was in the midline. The dysphagia test was negative. Deep reflexes from the extremities D > S, were lively. No paresis was detected at the time of examination. Sensitivity at the time of examination was not impaired. Right-sided hemihypesthesia. Babinski's sign was positive on the right. Stiffness of the neck — on 2 fingers.

The distribution by severity of hemorrhage on the Hunt—Hess scale was grade III.

Examinations

General blood test: Hb 135 g/L, Er 4.13 g/L, L 7.4 g/L, Tr 247 g/L, Lymph 19.2 %, Mon 6.4 %, Gran 74 %, Glucose 5.9 mmol/L.

Biochemical blood test: total protein 73.95 g/L, creatinine 85.88 μmol/L, total bilirubin 12.58 μmol/L, indirect bilirubin 10.44 μmol/L, direct bilirubin 2.14 μmol/L, total cholesterol 6.73 mmol/L, ALT 12.26 U/L AST 22.47 U/L, C-reactive protein (CRP) 2.72 mg/L, Sodium 142 mmol/L, Potassium 4.20 mmol/L, Chlorine 109 mmol/L.

Coagulogram: prothrombin index (PTI) 73.4 %, prothrombin time (PT) 11.2 sec, fibrinogen 2.88 g/L, activated partial thromboplastin time (APTT) 54.2 sec.

General urine test: light yellow, transparent, pH 7, specific gravity (SG) < 1005, blood 0.3 mg/L, L 0—1 in field of vision, squamosus epithelium — in insignificant quantity.

First cerebral fluid analysis: 2.5 ml, color before centrifugation: light red, color after centrifugation: xantochromic, transparency before centrifugation: cloudy, transparency after centrifugation: transparent, protein 2.28 g/L, chlorides 119.9 mmol/L, glucose 2.99 mmol/L, Pandy's reaction +++, cytosis $20.4 \cdot 10^2$/L, lymphocytes 82 % (14 lymphocytes), neutrophils 18 % (3 neutrophils), of which 180—190 are unchanged in the field of vision.

Second cerebral fluid analysis: 2.8 ml, color before centrifugation: light red, color after centrifugation: xantochromic, transparency before centrifugation: cloudy, transparency after centrifugation: transparent, protein 0.99 g/L, chlorides 121.56 mmol/L, glucose 3.20 mmol/L, Pandy's reaction ++, cytosis $22.8 \cdot 10^2$/L, lymphocytes 68 % (13 lymphocytes), neutrophils 32 % (6 neutrophils), of which 190—200 are unchanged in the field of vision.

Third cerebral fluid analysis: 3.0 ml, color before centrifugation: light pink, color after centrifugation:

colorless, transparency before centrifugation: slightly cloudy, transparency after centrifugation: transparent, protein 1.14 g/L, chlorides 121.3 mmol/L, glucose 2.6 mmol/L, Pandy's reaction +++, cytosis 58.8 · 10^2/L, lymphocytes 75.5 % (37 lymphocytes), neutrophils 24.5 % (12 neutrophils), of which 30—35 are unchanged in the field of vision.

Fourth cerebral fluid analysis: 2.6 ml, color before centrifugation: slightly yellowish, color after centrifugation: slightly yellowish, transparency before centrifugation: transparent, transparency after centrifugation: transparent, protein 0.65 g/L, chlorides 119.5 mmol/L, glucose 2.87 mmol/L, Pandy's reaction ++, cytosis 24 · 102/L, lymphocytes 75 % (15 lymphocytes), neutrophils 25 % (5 neutrophils), of which 8—10 are unchanged in the field of vision.

Electrocardiogram (ECG): sinus rhythm, regular. electrical axis of the heart (EAH) — horizontal position, HR 73 beats per minute.

CT + CT angiography of the head and neck: CT signs of SAH in the projection of the frontotemporal regions of both hemispheres of the brain, without signs of aneurysms, arteriovenous malformations (AVMs) of the cerebral vessels were not detected.

CT of the brain after treatment: at the time of examination, no data for acute cerebral circulation disorders were detected.

Ultrasound examination of the brachiocephalic vessels: ultrasound signs of stenosis atherosclerotic lesion of the brachiocephalic arteries (BCA), S-hemodynamically marked tortuosity of the right posterior communicating artery (PCA).

Initial examination of the multidisciplinary rehabilitation team: Barthel scale 65 points, Rankin scale 2 points.

Speech therapist: No speech and language disorders.

Psychologist: Emotional-volitional instability of the excitable type. Exogenous-organic register syndrome. Mild degree of cognitive decline.

Neurosurgeon: Conservative treatment is recommended.

Cardiologist: Stenosis atherosclerosis of the BCV. Subarachnoid hemorrhage. Secondary arterial hypertension (against the background of hemorrhage).

Treatment

Mannitol, Influgan, Magnesium sulfate 25 %, Potassium chloride 4.5 %, Omeprazole, Diclofenac, Ringer's solution, Olsapres H. Zanidip, Termidol, Cereglia, Nemotan, Tizalud, Baclofen, Contryven, Analgin, Lidocaine, Magnicum-adaptogen, Diacarb, Detralex, Moxogamma.

At discharge: The distribution by severity of hemorrhage on the Hunt—Hess scale was grade I, Barthel scale 85 points, Rankin scale 1 point.

Were recommended:

1. Observation of a family doctor, neurologist at the place of residence.
2. Control of BP, heart rate.
3. Consultation of a neurosurgeon/vascular surgeon with subsequent cerebral angiography if necessary.
4. Lifestyle modification: adherence to the Mediterranean diet; exclude alcohol and smoking, observe a drinking regimen, normalize sleep; perform aerobic exercises (duration of each workout at least 10 minutes) 4—7 days a week, total duration 150 minutes of physical activity per week in addition to routine activities in everyday life; target low-density lipoproteins (LDL) cholesterol level is < 1.8 mmol/L (preferably < 1.5 mmol/l).
5. Continue taking: Nimodipine (Nemotan) 30 mg 6 times a day 7 days; Zanidip 10 mg in the evening constantly, under BP control; Moxogamma 0.3 mg with increased BP > 140/90 mmHg; Clopidogrel 75 mg in the evening constantly; Epadol Neo 1000 mg 2 times a day 3 months with subsequent lipid profile control; Ibuprofen 400 mg (or Paracetamol 500 mg) situationally for headache; Detralex 500 mg 2 times a day 2 months; Magnicum-adaptogen 2 capsules in the evening 2 months; Theralene (Alimemazine) 5 mg 30 minutes before bedtime 1 month.

Discussion

This case typifies modern SAH management. The classic presentation (sudden headache, stiff neck) and CT findings prompted immediate treatment. The absence of an aneurysm on angiography suggests perimesencephalic-type SAH, which carries a better prognosis. The management followed guideline principles: admission to a neurocritical setting, controlled BP, prophylactic nimodipine, and vigilant monitoring [2, 4, 14, 16, 18].

In SAH, the cytosis in the CSF undergoes significant changes, reflecting the inflammatory reaction to the blood. A predominance of neutrophilic pleocytosis in the first 1—3 days after the hemorrhage is observed. This is a reaction to inflammation caused by blood breakdown products. Over time, usually from 3—4 days onwards, lymphocytes may become more prominent in the cytosis structure, although general pleocytosis may persist for quite a long time (up to 2—3 weeks). In addition, cytosis contains erythrocytes and macrophages, which appear later to engulf erythrocytes.

In our case, the results of the CSF examination raised diagnostic questions, so several of CSF studies were conducted. In the first study, lymphocytic pleocytosis (82 %) prevailed, neutrophils were 18 %, and increased protein (2.28 g/L) were registered. When re-analyzed 2 days later, the protein decreased by 3 times, mixed pleocytosis was detected: lymphocytes (68 %) and increased number of neutrophils (32 %). This raised questions, since the expected increase in the number of neutrophils was not at the beginning of the hemorrhage, but occurred during treatment simultaneously with an increase in the stiffness of neck as a meningeal symptom. It was necessary to

no miss the concomitant infectious lesion. Rebleeding was excluded, chlorides and glucose in the CSF were within normal limits. When examining the CSF after 3 days, the protein increased slightly, cytosis increased to 58.5 · 10^2/L, lymphocytic pleocytosis prevailed (75.5 %), neutrophils were 24.5 %. In the control study, after another day, the protein decreased by half, lymphocytic pleocytosis prevailed (75 %), neutrophils were 25 %. Objectively, the headache and stiffness of neck decreased, the general condition and mobility improved. Thus, the current dynamics of clinical symptoms and changes in the CSF was regarded as aseptic (reactive) meningitis with SAH.

Conclusions

The absence of an aneurysm on angiography suggests mainly perimesencephalic-type subarachnoid hemorrhage, which carries a better prognosis than in case of an aneurism rapture. The classic presentation (sudden headache, stiff neck) and computed tomography findings prompted immediate treatment even in neurological department. The management followed guideline principles: admission to a neurocritical setting, controlled blood pressure, nimodipine, and vigilant monitoring. Severe headache, increased manifestations of meningeal syndrome, and the appearance of additional symptoms require monitoring of cerebrospinal fluid to identify possible ongoing bleeding or an accompanying inflammatory process. Cellular composition, protein and glucose levels, and the type of dissociation over time help rule out complications. Aseptic meningitis, as a reaction of the meningeal membranes to blood irritation, is characterized by neutrophilic pleocytosis in the first days of hemorrhage, which later changes to lymphocytic pleocytosis. In this case, protein-cell dissociation is more characteristic. In complex cases, repeated cerebrospinal fluid analysis is necessary to assess its composition over time.

There is no conflict of interest.

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Ведення хворих з субарахноїдальним крововиливом в умовах неврологічного відділення (клінічний випадок)

Субарахноїдальний крововилив (САК) — це форма інсульту (5—10 %), що загрожує життю, спричинена переважно розривом аневризми. У 10—15 % пацієнтів зі спонтанним САК на першій ангіограмі аневризму не виявляють. У двох третинах цих пацієнтів комп'ютерна томографія (КТ) показує перимезенцефальну картину кровотечі. Пацієнти зазвичай скаржаться на раптовий головний біль, схожий на удар блискавки, ригідність потилиці, блювання та неврологічний дефіцит. Рання смертність становить 25—50 %, її головними причинами є такі ускладнення, як повторна кровотеча, вазоспазм і гідроцефалія. Неаневризматичний САК (НАСАК) має кращий прогноз, ніж аневризматичний, багато пацієнтів повністю одужують. Сучасна допомога потребує швидкої діагностики (висока чутливість, якщо кровотеча виявлена протягом 6 год), негайної ангіографії та ведення в нейрореанімаційному відділенні. Ключовими заходами є ретельний контроль артеріального тиску (АТ) (зокрема систолічного АТ < 160 мм рт. ст.), пероральний прийом німодипіну протягом 21 дня, зменшення внутрішньочерепного тиску, профілактика судом за потреби та тромбозу глибоких вен. Не вирішено питання щодо оптимальних цільових показників АТ і дози німодипіну. Ускладненнями САК є рецидив кровотечі переважно в перші 24—72 год, виявлений при ангіографії церебральний вазоспазм у 60—70 %, гідроцефалія, судоми, які виникають у 5—15 % пацієнтів, серцеві та легеневі розлади, гіпонатріємія та вентрикуліт.

Асептичний менінгіт — це добре відома стерильна запальна реакція, що виникає через 3—10 днів після САК, спричинена розпадом продуктів крові в субарахноїдальному просторі. При НАСАК, зокрема в перимезенцефалічних випадках, це запалення може імітувати менінгіт із головним болем, лихоманкою та ригідністю потиличних м'язів. При САК цитоз у спинномозковій рідині (СМР) зазнає суттєвих змін, що відображує запальну реакцію крові. Спостерігається переважання нейтрофільного плеоцитозу в перших 1—3 дні після крововиливу. Це реакція на запалення, спричинене продуктами розпаду крові. З часом, зазвичай з 3—4-го дня, відсоток лімфоцитів збільшується у структурі цитозу, хоча загальний плеоцитоз може зберігатися тривало (до 2—3 тиж). Також при субарахноїдальному крововиливі у ліквор наявні і макрофаги, які пізніше поглинають еритроцити.

Представлено випадок консервативного лікування САК, підтвердженого за допомогою КТ, що ілюструє ці принципи. Класична клінічна картина та дані КТ спонукали до негайного лікування. Відсутність аневризми на ангіографії свідчила про перимезенцефалічний тип САК, що має кращий прогноз. Лікування проводили згідно з рекомендаціями: госпіталізація до ургентного неврологічного відділення, контроль АТ, профілактичний прийом ніподипіну та моніторинг. Наведений клінічний випадок є цікавим щодо динаміки СМР. Результати дослідження СМР викликали діагностичні питання, тому було проведено кілька досліджень СМР. За даними першого дослідження переважав лімфоцитарний плеоцитоз (82 %), нейтрофіли становили 18 %, також зареєстровано підвищений вміст білка (2,28 г/л). При повторному аналізі через 2 дні вміст білка зменшився втричі, виявлено змішаний плеоцитоз: лімфоцити (68 %) та підвищену кількість нейтрофілів (32 %). Це викликало питання, оскільки очікуване збільшення кількості нейтрофілів зареєстровано не на початку крововиливу, а під час лікування одночасно з підвищенням ригідності потиличних м'язів, як менінгеального симптому. Важливо було не пропустити супутнє інфекційне ураження. Рецидив кровотечі був заперечений, вміст хлоридів і глюкози в СМР були в межах норми. При дослідженні СМР через 3 дні вміст білка дещо підвищився, цитоз збільшився до 58,5 · 10^2 л, переважав лімфоцитарний плеоцитоз (75 %), нейтрофіли становили 25 %. Об'єктивно зменшилися головний біль і ригідність потиличних м'язів, поліпшилися загальний стан та рухливість. Динаміку клінічних симптомів і змін у СМР було розцінено як асептичний (реактивний) менінгіт, асоційований із НАСАК.

Ключові слова: неаневризматичний субарахноїдальний крововилив (НАСАК), дослідження спинномозкової рідини, КТ головного мозку, КТ-ангіографія голови та шиї.

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