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## **CATAMNESIS OF ORGANIC HALLUCINOSIS CASE AT PITUITARY MACROADENOMA**

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**Ключевые слова:** *катамнез, органический галлюциноз, гипофиз, макроаденома*

**Abstract.** *Catamnesis of organic hallucinosis case at pituitary macroadenoma. Yur'yeva L.N., Shusterman T.Y., Leonov S.F., Varshavskij Ya.S. The article describes a one-year follow-up study of the clinical case of organic hallucinosis that we have described in the previous article in the patient with pituitary macroadenoma. At primary admission to the hospital (one year earlier) the likelihood of permanent or recurrent nature of hallucinosis and absolutely poor prognosis for recovery and life without removal of the tumor was pointed out to the patient and his family. However, the patient and his relatives flatly refused to undergo neurosurgical intervention. The article describes the dynamics of psychopathological and somatic statuses of the patient during his readmission to a psychiatric hospital. The exciting cause of death on the 25th day of hospital stay was phenomenon of cerebral edema. By results of post-mortem studies the underlying cause of death was small cell chromophobe pituitary adenoma. There was a complete accuracy between clinical and post-mortem diagnosis. Absolutely unfavorable prognosis made before has been confirmed. The conclusion about the need of psychoeducational interventions with this contingent of patients and their relatives for prevention of an adverse disease outcome is made.*

**Реферат.** *Катамнез случая органического галлюциноза при макроаденоме гипофиза. Юрьева Л.Н., Шустерман Т.И., Леонов С.Ф., Варшавский Я.С. В статье приведено годовичное катамнестическое исследование описанного нами в предыдущей статье клинического случая органического галлюциноза у пациента с макроаденомой гипофиза. При первичном поступлении в стационар (годом ранее) пациенту и его семье было указано на вероятность постоянного или рецидивирующего характера галлюцинирования и абсолютно неблагоприятный прогноз для выздоровления и жизни без удаления опухоли. Однако пациент и его родственники категорически отказались от проведения нейрохирургического вмешательства. В статье описана динамика психопатологического и соматического статусов пациента при его повторной госпитализации в психиатрический стационар. Непосредственной причиной смерти пациента на 25-е сутки пребывания в стационаре послужили явления отека головного мозга. Основной причиной смерти по результатам патологоанатомического исследования явилась мелкоклеточная хромофобная аденома гипофиза. Имелось полное совпадение клинического и патологоанатомического диагнозов. Сделанный ранее абсолютно неблагоприятный прогноз подтвердился. Делается вывод о необходимости проведения психообразовательных мероприятий с данным контингентом пациентов и их родственниками для предотвращения неблагоприятного исхода заболевания.*

Organic hallucinosis is characterized by stable or constant hallucinations in the waking state that may be due to influence of a specific organic factor, such

as brain tumor [2]. Pituitary adenomas occupy the third place among intracranial neoplasms, ranging from 7,3% to 18% of all verified brain tumors [1, 6].

Macroadenomas make up 9,8% of all pituitary tumors [5]. As the first stage of treatment of macroadenomas of pituitary gland, in most cases, surgical intervention is used [3,6].

In the previous article the clinical case of organic hallucinosis was described, the onset of which has been determined to be linked with pituitary macroadenoma. Due to patient's refusal from neurosurgical intervention for neoplasm removal it was not possible to reliably determine disappearance of complex hallucinations, the presence of atherosclerotic changes in cerebral vessels, concomitant essential hypertension (EH) increased the likelihood of permanent or recurrent nature of hallucinosis and made prognosis for the recovery and life as unfavourable [4].

The aim of this article was the description of follow-up aspects of the clinical case of organic hallucinosis in patient with pituitary macroadenoma to confirm the correctness of made unfavourable prognosis in the absence of neurosurgical intervention.

**Patient**, K.Yu.A., 62 years old, pensioner, resident of Dnipropetrovsk. He had in-patient treatment in CI «Dnipropetrovsk clinical psychiatric hospital of Dnipropetrovsk regional council» from 28.07.2015 till 28.08.2015. He died 28.08.2015, at 5:55 p.m.

**Catamnesis**: for the first time the patient was hospitalized to CI «Dnipropetrovsk clinical psychiatric hospital of Dnipropetrovsk regional council» from 26.02.2014 till 11.04.2014 with diagnosis: organic hallucinosis (according to International Classification of Diseases, 10th revision, the code F06.0). Secondary diagnosis: pituitary macroadenoma with disseminated organic microsymptomatology. Cerebral atherosclerosis, second degree of discirculatory encephalopathy (DE), second degree of EH. Complex hallucinations (visual, auditory, olfactory, tactile) with zoological theme (rats) dominated in the clinical picture. He was discharged with improvement, intake of supporting psychotropic medication and carrying out of routine neurosurgery for removing of brain neoplasm were recommended. The patient and his relatives flatly refused from surgery, likelihood of permanent or recurrent nature of hallucinosis and absolutely unfavourable prognosis for recovery and life without removal of the tumour were explained to him and his.

After discharge from the hospital, despite receiving the recommended treatment of antipsychotic drugs, the patient continued «to see the rats» periodically, refused from neurosurgical intervention as before.

The mental state has deteriorated sharply a week before re-hospitalization to a psychiatric hospital

when he became restless, angry, jumped from the apartment at night with a stick, trying to «protect» family from «the rats». The patient intended to burn the bed «the rats lived in». In the kitchen, he closed the window and turned on the gas, trying to «poison the rats». He began to declare that his mother was «alive», though she had died in the year 2005, that «cars were driving for him in order to kill». On the insistence of his wife he has been examined by a local psychiatrist and directed at in-patient psychiatric treatment.

**On admission**: complaints of headache, poor sleep, anxiety. Contact was formal; he answered questions reluctantly, with irritation. The mood was low, the patient was emotionally labile. He said that he «saw huge rats», «they were black and white», «they gnawed through holes in the walls and climbed into the apartment». He was completely sure in the reality of his experiences. The patient was oriented to person, place and time correctly. He agreed to examination and treatment.

Somatically: skin was of normal colour, clean. Vesicular breathing in the lungs, no rales. Heart sounds were muffled, rhythmic, blood pressure (BP) – 130/80 mm Hg, heart rate (HR) – 70 beats per minute. The abdomen was soft, painless on palpation. Bowel and bladder habits were normal.

Neurologically: no meningeal signs. Pupils were D=S, photoreactions – sluggish. Tendon reflexes were equally reduced, no pathological ones. Grogginess in Romberg's position. He performed finger-to-nose test with miss.

**In the department**: the patient was enrolled in an acute psychiatric department #31. At the beginning he spoke about his experience reluctantly. Hereafter he said that «saw» a huge amount of «rats» at home that «climbed» into his apartment through the windows, «gnawed holes in the walls». He tried to defend himself against them with a stick and pipe, put mesh on the windows. He saw as «the rats sit and look at people». The patient did not deny that he intended to burn the sofa, because there were «lots of rats». The patient periodically «saw a white woman», «she was my mother». He said that someone wanted to «kill» him and even «killed» his dog. He noticed that «hooligans» were sitting on the benches under his windows and filming by the «video camera». On the third day of stay the patient became drowsy, flassid, spent time in bed. On the fourth day the condition deteriorated rapidly, speech became slurred; state of mind was in the form of light clouding of consciousness. He carried out simple instructions with the help of medical staff. The skin was of pale colour; fluctuations in BP were marked from 120/100 to 180/120 mm Hg, HR – 100

beats per minute. The patient was examined by a doctor of the intensive care unit, according to severity of the condition he was transferred to resuscitation unit.

The patient's general condition was severe, speech contact – formal. He was trying to answer simple questions, disoriented to place. Hallucinatory symptoms persisted. He carried out elementary instructions after repeating, selectively. Enteral nutrition was via nasogastric tube. Insight of disease was absent. The skin was of pale colour. There was harsh breathing in the lungs, weakened in the mid-lower divisions, rales were not noted. BP was 150/90 - 160/100 mm Hg.

On the 10th day of in hospital stay at 1:05 p.m. the patient's condition deteriorated sharply, mottling of the skin was marked, tachypnoea to 24 per minute, hemodynamics dropped to 90/60 mm Hg, followed by cessation of breathing and circulation. There was loss of consciousness. Resuscitation was conducted: closed-chest cardiac massage, intubation, artificial pulmonary ventilation, intravenous adrenaline and defibrillation. On the 11th minute, cardiac activity recovered spontaneously. Over the next 2 days artificial pulmonary ventilation continued, consciousness flickered from soporous to recovery. Phenomena of exsiccosis with intestinal paresis were observed in the patient.

Over the next two weeks, against a background of ongoing intensive therapy, the patient's condition improved slightly, night sleeping was physiological, he remained conscious, but was inhibited. The contact was available; he answered the questions more often in one word. Psychotic symptoms continued to be actual. The patient oriented to person correctly, roughly – to place and time. He performed instructions. Turns in bed and enteral nutrition were with the help of medical personnel, although he became more active in bed.

On the 25th day of hospital stay the patient was transferred to psychosomatic department #5 for further treatment. He remained conscious, but was not readily available to contact, passive, negativistic. Inhibition was marked; he was in sharp asthenic state. He said periodically that «saw the rats». He was fully serviced by medical personnel. On the 32nd day of hospital stay the patient's condition deteriorated dramatically at 11:30 a.m.: consciousness – stupor, no speech contact, the patient did not carry out the instructions, hyperthermia to 39,0°C, asymmetry of nasolabial folds, the reflexes of oral automatism and increase in the overall muscle tone were present. Despite intensive care, condition deteriorated progressively, at 5:50 p.m. a state of mind – coma, the pupils were wide and did

not react to light, muscle atony was present, facial features were sharpened, breathing was rare and pathological, BP – 60/20 mm Hg, there were involuntary defecation and urination. Resuscitation was being conducted. At 5:55 p.m. cardiac and respiratory arrest occurred, biological death has been pronounced.

**Analyzes:** complete blood count (CBC) from 28.07.15 – normal. CBC (05.08.15, 06.08.15, 07.08.15, 08.08.15, 09.08.15, 10.08.15) – leukocytosis, erythrocyte sedimentation rate (ESR) – 35 mm/hour. CBC (25.08.15): ESR – 26 mm/hour. Clinical urinalysis (28.07.15, 07.08.15, 17.08.15) – normal. Feces analysis on helminth eggs and threadworm (03.08.15) – negative. Pharyngeal swab on diphtheria (28.07.15) – negative. Blood glucose test (31.07.15-19.08.15, 21.08.15) – normal. Wasserman reaction (03.08.15) – negative. Antigen p24 and/or antibodies to human immunodeficiency virus (04.08.15) have not been identified. Enzyme immunoassay for markers of viral hepatitis (07.08.15) – negative. Biochemical blood assay (liver panel) from 29.07.15: aspartate transaminase – 0,54; from 31.07.15 – 0,7; from 05.08.15 – 0,6. Total bilirubin (29.07.15, 03.08.15) – normal. Renal panel (29.07.15, 31.07.15, 05.08.15, 10.08.15, 20.08.15) – normal. Biochemical blood assay on electrolytes (31.07.15-17.08.15) – normal. Blood coagulation and anticoagulation system test (31.07.15, 07.08.15, 12.08.15, 14.08.15) – normal.

Plain chest radiograph (31.07.15, 10.08.15, 28.08.15) – lung fields with out pathological shadowing.

Abdominal radiograph (06.08.15) – no sings of obstruction. The colon is overstretched by gases. 10.08.15 – no pathological changes.

Electrocardiogram (06.08.15) – sinus tachycardia, left anterior hemiblock, left ventricular hypertrophy with left ventricular systolic overload.

Electroencephalogram (03.08.15): not rough changes with predominance of low amplitude slow wave potentials in frontotemporal leads without clear lateralization. Involvement of cortical structures in pathological process.

Echoencephalogram (EchoEG from 03.08.15): no midline brain shifts. Third ventricle of cerebrum is up to 7 mm. Signs of superficial liquor discirculation in frontotemporal area of mild degree.

EchoEG (07.08.15): no midline brain shifts. Ventricular system of the brain is enlarged. Third ventricle of cerebrum is up to 8,6 mm. Signs of moderate liquor hypertension.

Therapeutist is consultation (31.07.15): ischemic heart disease (IHD). Diffuse cardiosclerosis, first degree heart failure (HF). First degree EH.

Follow up from 03.08.15: IHD. Diffuse cardio-sclerosis, second degree HF, function class 2. Second degree EH, stage 2, risk 3 (high), hypertensive heart. Hypotonic colon dyskinesia.

Follow up from 07.08.15: chronic obstructive pulmonary disease in the exacerbation phase, first degree respiratory failure. Bilateral hypostatic pneumonia.

Neurologist's consultation (31.07.15, 03.08.15): cerebral atherosclerosis, second degree DE on the background of EH, dismetabolia, pituitary adenoma. Follow up from 28.08.15 – cerebral edema on the background of chronic circulatory brain insufficiency and pituitary adenoma.

Ophthalmologist's consultation (04.08.15): arteriosclerotic retinopathy of both eyes.

Surgeon's consultation (05.08.15): functional intestinal disorder.

**Treatment:** haloperidol 5 mg once a day, seduxen 10 mg once a day, quetiron 200 mg bis in day, cyclodol 2 mg once a day, antibiotic therapy, vitamin therapy, systemic medication, symptomatic treatment, basic life support.

### Diagnoses:

**1. Predominant psychiatric:** organic hallucinosis F06.6

**2. Somatic:** brain neoplasm (adenoma).

**3. Secondary diagnosis:** IHD, diffuse cardio-sclerosis. Left anterior hemiblock. Second degree EH, stage 2, risk 3 (high), second degree HF, function class 2.

**4. Complications:** hypostatic pneumonia. Cerebral edema on the background of chronic circulatory brain insufficiency. Acute cardiovascular insufficiency.

According to the extract from the protocol of post-mortem examination #228 (29.08.15) **paragnosis:**

**1. Predominant:** small cell chromophobe pituitary adenoma;

**2. Complications:** congestion, parenchymatous degeneration of internal organs. Hypostatic pneumonia. Pulmonary edema. Cerebral edema with foramen magnum cerebellar tonsillar herniation;

**3. Secondary:** IHD, diffuse cardio-sclerosis due to coronary artery atherosclerosis (40%). EH (left ventricular wall thickness 2,1 cm, heart mass 410 grams).

The cause of death: a) cerebral edema, b) pituitary adenoma.

### CONCLUSION

Based on the clinical data and results of post-mortem examination small cell chromophobe pituitary adenoma should be considered as underlying disease of the deceased. The course of the disease proceeded with compression of brain structures, the exciting cause of death was phenomenon of cerebral edema. The clinical picture was marked by an increase of true hallucinations with zoological theme, onset of other visual perceptual deceptions, joining secondary delusional inclusions on the background of progressive cerebral symptoms. There was complete accuracy between clinical and post-mortem diagnoses. Thus, our previous absolutely unfavourable prognosis for recovery and life of the patient without removal of pituitary tumor, indicating the likelihood of permanent or recurrent nature of the hallucinosis, has been confirmed. It is necessary to conduct psychoeducational interventions with this contingent of patients and their relatives for prevention of an adverse disease outcome.

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## ЭФФЕКТИВНОСТЬ ДРЕНИРОВАНИЯ СЛЕЗНОГО МЕШКА ПРИ ДАКРИОЦИСТОЦЕЛЕ У НОВОРОЖДЕННЫХ

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**Ключевые слова:** новорожденные, дакриоцистоцеле, флегмона слезного мешка

**Key words:** newborns, dacryocystocele, lachrymal sac phlegmon

**Реферат.** Ефективність дренування слізного міхура при дакриоцистоцеле у новонароджених. Сакович В.М., Сердюк В.М., Клопоцька Н.Г., Тарнопольська І.М. У статті наводяться результати лікування 14 новонароджених з одностороннім дакриоцистоцеле. Середній вік дітей – 7,2±0,5 дня. У 9 (64,3%) дакриоцистоцеле ускладнилося флегмоною слізного міхура, у 5 (35,7%) перебіг був без ускладнень. Всім дітям проводили дренування слізного міхура крізь нижній слізний каналець, інстиляції антибіотиків, а у випадку флегмони – системну антибактеріальну терапію. Промивання проводили розчином антибіотика офлоксацин до евакуації прозорої рідини. За необхідності дренування повторювали через 10 днів. У всіх пацієнтів дренування міхура виявилось вдалим. Зондування носослізного каналу через 2-3 тижні після дренування потребували 4 дітей (28,6%), в тому числі 3 дітей з флегмоною слізного міхура. У 71,4% регрес дакриоцистоцеле відбувався самостійно. Ускладнень та сторонніх ефектів від лікування не спостерігалось. Позитивний ефект дренування, на думку авторів, пояснюється тим, що розширення слізних каналців перед дренуванням дозволяє усунути стеноз клапана Розенмюллера, а евакуація рідини, слизу та гною, які є поживним середовищем для мікроорганізмів, зі слізного міхура сприяє стиханню запального процесу. Автори також рекомендують звернути увагу на пренатальну діагностику дакриоцистоцеле при ультразвуковому дослідженні в 3-му триместрі вагітності.