

## CASE REPORT

**“Gas leak at home”****Martin VALIŠ<sup>1</sup>, Jiří MASOPUST<sup>2</sup>, Ladislav HOSÁK<sup>2</sup>, Aleš URBAN<sup>2</sup>**

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**Abstract**

In the present case history, we describe a male patient who experienced his first mental changes at the age of 57. Dominant among these were symptoms of paranoia and persecution, and auditory and olfactory hallucinations. The presence of blunt euphoria, reduced ability to understand social situations and contexts, personality changes, cognitive disorders (particularly of executive functions) and attention disorders corresponded best to prefrontal cortex damage. CT scan of the brain revealed a picture of normotensive communicating hydrocephalus. Quetiapin 200 mg daily was introduced for psychotic disorders. Delusional experiences gradually went away and no hallucinations were observed. The patient received a ventriculoperitoneal shunt implant with a programmable valve set at the pressure of 130 mm H<sub>2</sub>O. This case provides proof of the necessity of somatic and neurological examination and brain imaging in patients with newly developed mental disorders.

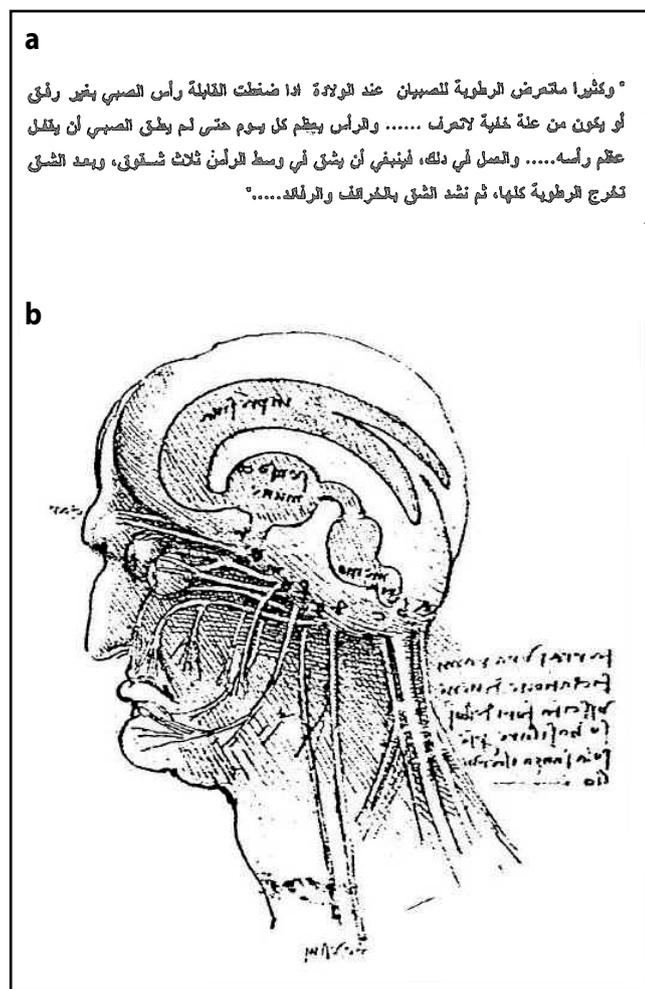
**INTRODUCTION**

The word hydrocephalus originates from the Greek hydro (water) and kefalè (head). It refers to a presence of water inside the intracranial cavity. The first discoveries of skulls apparently belonging to people with hydrocephalus were made in Egypt, and they were people from the First Dynasty around year 3000 B.C. Hippocrates (466–377 B.C.) believed hydrocephalus resulted from the transformation of brain tissue into water during epileptic seizures. Abul Qasim Al-Zahrawi (936–1013) described clinical symptoms

of hydrocephalus and proposed surgical solutions\* (**Fig. 1a**). Leonardo da Vinci (1452–1519) made anatomical drawings (sketches) containing the ventricular system (**Fig. 1b**). He also created a wax model of oxen brain ventricles. Vesalius (1514–1564) had an idea about the accumulation of the cerebrospinal fluid in the brain ventricles in individuals suffering from hydrocephalus (Rocca 1997).

Hydrocephalus forms due to an excess generation of cerebrospinal fluid in the brain ventricles or in the subarachnoid spaces of the brain at the expense of brain parenchyma. The internal and external CSF

\* Precious documents in the Arabic language are stored in the library of the Indian city of Patna (Aschoff *et al* 1999). Al Zahrawi reports that “...dropsy of the brain occurs more frequently in small boys, which is caused by uncautious squeezing of the head by the midwife or when a child has a hidden congenital disorder ... the head increases day by day, and the skull bones do not close...it is necessary to make an incision in the middle of the head with three openings so that the fluid can evaporate, then we close the wound with a bandage – in two layers, to be placed on the wound and tighten the skull.”



**Fig. 1. a:** Description of the clinical symptoms of hydrocephalus and proposed surgical treatment in the Arabic version .  
**b:** Leonardo's drawing of brain ventricles from the year 1510.

space is enlarged in people with hydrocephalus. Acute hydrocephalus develops quickly, for example due to brain hemorrhage or infarction, colloid cysts of the third ventricle, craniocerebral trauma, exudative meningitis or viral encephalitis. On the other hand, chronic hydrocephalus develops gradually; this can be caused by stenosis of the Sylvian aqueduct, granulomatous meningitis, malformation of the posterior cranial fossa, meningeal carcinomatosis, brain tumor, or spinal cord tumor (Ambler *et al* 2004).

In hydrodynamic terms, there are two types of hydrocephalus: active or progressive, where the CSF causes increased pressure on the brain tissue, and requires brain surgery; and passive – where the CSF fills the enlarged brain spaces only passively and thus does not require a surgical procedure.

The main symptoms reported for acute hydrocephalus in adults are sudden headache, nausea and vomiting, edema of the optic nerve papilla, double vision, partial epileptic seizures, endocrine dysfunction (amenorrhea,

polydipsia, polyuria) and gait disorders (atactic, wide-based gait). Concerning psychopathology, psychomotor slowing or hypersomnia can be manifested. The condition leads quickly to a consciousness disorder and can be fatal when left untreated. Chronic (normotensive) hydrocephalus in adults was first described by Adams in the year 1965. Normotensive hydrocephalus is the most frequent form of hydrocephalus in adults. The name does not fully match the real condition, as the liquor pressure is elevated and causes widening of the ventricular system. The brain parenchyma is oppressed. The mechanism of development is related to a disorder of the equilibrium between the three intracranial compartments – liquor, vascular and parenchymal. The blockage is present in the basal cisterns of the brain or in the subarachnoid space and the cerebrospinal fluid is inadequately absorbed into the superior sagittal sinus. Inside the brain ventricles, there is no blockage and the liquor pressure can be normal. Causes can include brain injury, subarachnoid bleeding, encephalitis or meningitis with subsequent adhesions in the basal cisterns of the brain. Hypertensive disease is a known risk factor. Often, no cause can be found, and the condition is idiopathic. Victims are usually patients in the sixth or seventh decade of life but the same disorder is also found in middle-aged or younger individuals. The classic triad of symptoms is dementia, gait disorder, and incontinence. This diagnostic triad should not be overestimated. The leading symptom of normotensive hydrocephalus is atactic gait. Symptoms develop over several weeks or months (Bret *et al* 2002). Mental disorders usually appear first and are prominent later. The most frequently reported symptoms are cognitive function disorders, dementia, bradypsychia, apathy, reduced emotional reactions or swinging mood changes, mania, aggressiveness, and anxiety disorders. Paranoid psychosis is not excluded. Patients often have no or little perception of the disease. When the disorder progresses, dyscalculia, dysphasia, dysgraphia, severe memory disorder (such as in Korsakoff's syndrome), or disorientation can occur. Rarely depression, hallucination, delusions, or outbursts of aggressiveness are reported (Adler *et al* 1992; Cummings 1994; Pinner *et al* 1997). In some cases, we can observe episodes of mutism and akinesia, similar to catatonia. The following gait disorders are reported: uncertainty, slowing, wide-based gait, instability, paraspasticity, staggering, tottering, and apraxia (inability to raise legs above the base, while having preserved muscle strength). Patients often have problems to turn or start a movement and can easily fall. In extreme cases, the patient is not able to stand or get up. As concerns incontinence, urine incontinence is typical while fecal incontinence is reported rarely. Incontinence is present mostly in patients in an advanced stage of the disease. Other symptoms may also be present, such as the slowing of upper extremity movements, hand tremor, ataxia and nystagmus. Headache, on the other hand, is reported rarely, and there is

no edema of the optic nerve papilla, no vertigo, central vomiting or epileptic seizures. If the treatment is successful, the symptoms can be reversible. When left untreated, the symptoms gradually progress. Fluctuations in symptoms from day to day or week to week are typical. After many months of worsening, the condition can temporarily stabilize at a certain level. Further worsening may result in unconsciousness or death (Mitchell 2004; Jiráček & Koukolík 2004; Lishman 2005; Yudofsky & Hales 2007; Doležil 2009; Kito *et al* 2009).

The examination techniques are lumbar puncture (pressure and composition of the liquor, with a rapid transient improvement of the clinical status after the puncture), CT scan and MRI scan of the brain (marked enlargement of the brain ventricles with a narrow subarachnoid space, lucent tissues around the front corners of the lateral ventricles, and in some cases a minor obstruction in the posterior cranial fossa on the MRI scan), liquor pressure monitoring for at least 24 hours (spikes of increased pressure).

The basic treatment procedure is surgery with shunt placement, which reduces the cerebrospinal fluid pressure in the brain ventricles. The objective of the treatment is optimization of the central nervous system function rather than normalization of the ventricle size. The most frequently used types of drainage (95% of cases) are ventriculoperitoneal (from the lateral brain ventricle into the abdominal cavity), ventriculojugular (into the jugular vein) and ventriculoatrial (into the heart atrium) shunting, or a ventriculocisternostoma can be created from the lateral ventricle into the cerebellomedullary cistern (cisterna magna).

## CASE HISTORY

A 61 year-old male patient was referred to psychiatry from ENT department, where he had received treatment for face and larynx contusion. He injured himself when he attempted strangulation in the bathroom. However, the clothesline he used broke and the patient fell to the floor.

The patient's niece is being treated for schizophrenic disease. The patient had been treated only for high blood pressure. Two years ago he quit smoking. He has not drunk alcohol for three years, before he drank 5 to 6 beers a day. He is married, lives with his wife, and they have two adult daughters. He is a former mason. During the last years, he worked only as a helper and he retired early six months ago.

The patient's daughter told doctors that her father started changing gradually approximately 4 years ago. *"Before Christmas, he had a feeling that someone was recording him on a camera. First he suspected the daughter and then their neighbors. He closed the Venetian blinds in all windows. Family members told him "it was nonsense". He decided not to talk about his feelings anymore and he continued that for 2 whole years. Then he confided that someone wanted to poison him*

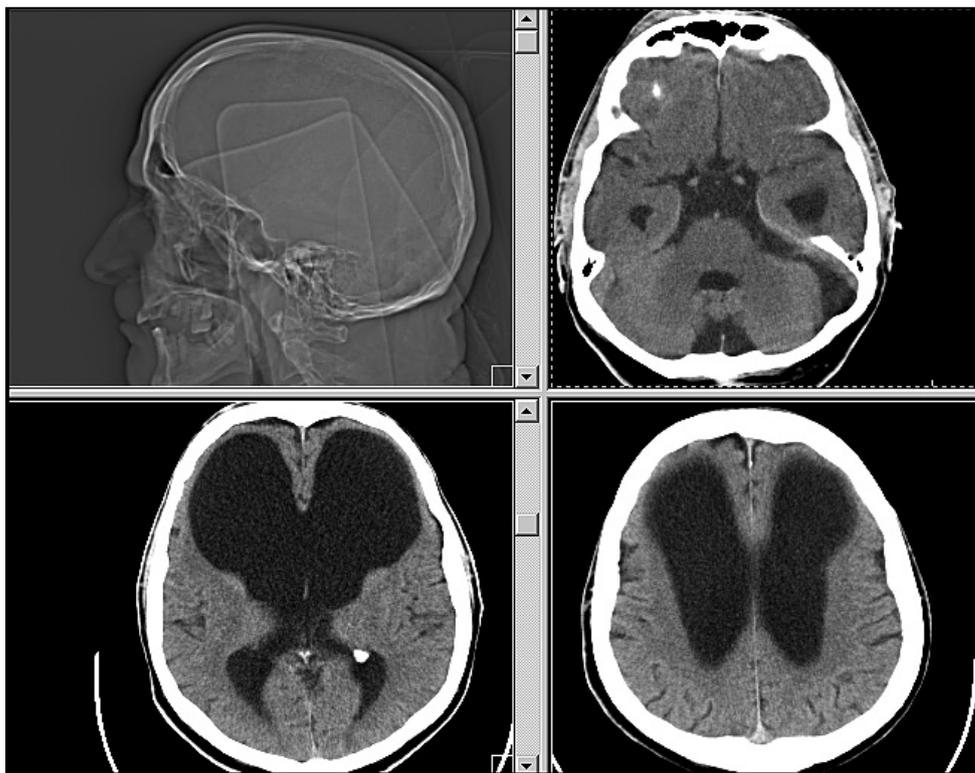
*– with drugs or poison. Based on his fear, he emptied out kitchen detergent and shampoo bottles, he threw out laundry detergent. He saw danger everywhere. He examined what his wife put in food. He quit smoking all of a sudden. Everything had a strange taste and he smelled acid everywhere. He checked the roof to see if it was collapsing. He smelled gas in the house and he turned off the gas supply completely. His memory worsened. He felt pressure behind the eyes. He did not communicate with the family for about two weeks, and then they found him in the bathroom after he had made an unsuccessful hanging attempt.*

*The patient himself told us that he smelled gas everywhere. He also heard rumbling in the house, like if the roof was collapsing. He was worried they were going to be buried alive in the house. He had "burns" under his beard and he was not able to get rid of them. They were caused by the gas. He didn't want to live there waiting until the entire house collapsed. So he wanted to hang himself.*

On admission to a locked hospital department, the patient looked unkempt, he was badly groomed. He responded with latency and briefly. He was oriented. He had altered emotions, not adequate to the situation. He seemed to have a mildly elated mood. The thinking was continuous and rigid with perseverations. Paranoid and persecution moods and poisoning delusion were apparent. The patient described experiences corresponding to smell hallucinations and perhaps also to auditory verbal hallucinations. The patient was not critical when talking about the events.

Neuropsychological examination revealed a markedly damaged focus of attention. Visual analysis and synthesis capacity, visual motor coordination, and verbal fluency were impaired. The patient had reduced resistance against perception burden. His overall performance in memory tests ranged within the borderline zone between the lower normal and defects. Examinations and tests revealed impaired executive functions (some improvement was observed when the patient had sufficient time and carried out more trials and errors; he completely failed in the Wisconsin card sorting test, and he had a better result in the London Tower test). He had preserved long-term memory, general knowledge and computing and deductive abilities. In general, the reduction of cognitive abilities vs. premorbid level was evaluated as mild. He had reduced ability to understand rules in social reality.

Signs of hydrocephalus were obvious on the patient's head at the first look. The dominant forehead was strikingly large compared to the facial area, the occipital part of the head was flattened. His upper extremities showed signs of hypermetria. The gait was wide-based, uncertain, but relatively fast. Otherwise Neurostat findings were normal. Laboratory tests provided no pathological results. CT scan of the brain revealed a picture of communicating normotensive hydrocephalus. The brain tissue of the frontal lobes was so atrophied that it remained only in a narrow strip of up to 4 mm wide (**Fig. 2**).



**Fig. 2.** CT scan of the brain. Dilatation of the ventricular system under the picture of inactive fourth ventricle hydrocephalus. Mild predilection of the ventricular dilation in the frontal corners of the lateral ventricle. Brain atrophy is seen with residual tissue of the frontal lobes of up to 4 mm wide.

The EEG record was mildly abnormal with overall reduction of the basic activity in the subalpha range and enhancement of beta activity frontally. Ophthalmic fundus examination provided physiological findings. Lumbar puncture was carried out by the neurologist who found normal CSF pressure.

Quetiapin 200 mg daily was introduced to treat psychotic symptoms. Delusional experiences gradually went away and we recorded no hallucinations. The emotions were mostly of euphoric type. The patient was diagnosed with organic delusional disorder. After a one-month hospitalization, the patient was referred to the Department of Neurosurgery for surgical treatment of the hydrocephalus. At the said department, pressure of up to 225 mm H<sub>2</sub>O was measured during lumbar puncture. The patient felt better after the puncture. Gross examination using MMSE test revealed that patient's cognitive performance score after the puncture improved from 26 to 30. The patient received a ventriculoperitoneal shunt implant with a programmable valve set at a pressure of 130 mm H<sub>2</sub>O. After the intervention, the patient generally feels more lively. Logorrhea was reported in the medical records. Psychological follow-up showed similar results to the previous examination. The overall memory capacity was at the lower average. The patient was discharged home one month after the surgery. After several months, he was oper-

ated on for ileus. Later he experienced a consciousness disorder with confusion and subsequent vomiting. No disorder of the shunt or valve performance was found at the Department of Neurosurgery. Control CT scan of the brain revealed persistent ventriculomegaly and no changes were visible.

## DISCUSSION

In the presented case, the picture of dilated ventricular system was atypical. It cannot be ruled out that congenital atrophy (hypoplasia) of the frontal lobes was also present. However, it is not possible to determine exactly how long the hydrocephalus was present in the patient. Mental changes occurred at the age of 57. Symptoms of paranoia and persecution, and auditory and olfactory hallucinations were dominant among the symptoms. The presence of blunt euphoria, reduced ability to understand social situations and contexts, personality changes, cognitive disorders (particularly of executive functions) and attention disorders corresponded best to prefrontal cortex damage. A change from being an active mason to a helper and early retirement could be partially attributed to apathy-abulia syndrome and impairment of the ability to manage complex problems requiring the planning of goals and their implementation. Prefrontal syndrome is classi-

fied into three types according to the manifestation of mental disorders. Dorsolateral prefrontal syndrome (with the involvement of the lateral convexity of the lobe) includes impairment of executive functions and motor programming, verbal fluency, perseveration and rigidity while solving problems. The orbitofrontal type (with the involvement of the pole and base of the frontal lobe) manifests as a typical picture of uninhibited behavior and emotional instability. Anterior cingulate (medial frontal) syndrome manifests as apathy and kinetic mutism (Alao & Naprawa 2001; Lishman 2005). Our patient had diffuse involvement of the frontal region rather than localized lesion of a certain region. Therefore the final picture is a combination of all three syndromes. This case provides proof of the necessity of somatic and neurological examination and brain imaging in patients with newly developed mental disorders.

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