Case report
Ενδιαφέρουσα περίπτωση

# Psychotic symptoms in normal pressure hydrocephalus

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Normal Pressure Hydrocephalus due to idiopathic aqueductal stenosis is a chronic abnormal accumulation of cerebrospinal fluid in the cerebral ventricles caused by an obstruction in the Sylvian aqueduct. This leads to a dilatation of the ventricular system and to subsequent damage of the adjacent parenchyma. Although NPH typically presents with the progressive 'triad' of cognitive impairment, gait disturbance and urinary incontinence, it has been described that it rarely manifests in the form of predominant psychotic symptoms. It has been suggested that thought and perceptual disorders could develop secondary to the damage caused by NPH. Although precise anatomical correlates have not yet been established, certain cerebral regions –primarily the frontal cortex, mesencephalic and diencephalic structures of the brain– have been implicated in the pathogenesis of hydrocephalic psychosis. Because frontal lobe lesions are traditionally known to facilitate one's inability to integrate and correct perceptual distortions in the face of contradictory evidence, frontal lobe dysfunction may be integral in delineating the etiology of delusions in NPH. We present the case of a 30-year-old female, admitted involuntarily to our acute psychiatric department because she exhibited aggressive behavior while being in an agitated state with delusions of persecution. Her neurological examination disclosed subtle bradykinesia. Neuropsychological batteries and intelligence testing revealed mild cognitive impairment and a CT scan showed considerable dilatation of the ventricular system due to idiopathic aqueductal stenosis. While a conservative approach was chosen for the treatment of NPH, our patient was initiated on 2nd generation antipsychotics showing marked improvement of her psychiatric symptomatology. The atypical presentation of hydrocephalus in the aforementioned case underlines the necessity to thoroughly investigate the possible presence of an underlying organic factor in those patients who present with predominant psychotic symptoms in association with soft non-localising neurological signs and mild cognitive deficits. Furthermore, our patient's marked improvement indicates that, in cases where the primary cause is treated conservatively, hydrocephalic psychosis could respond to 2nd generation antipsychotics. In light of this case report, we reviewed past and present literature on the matter.

**Key words:** Normal pressure hydrocephalus, psychotic symptoms, mild cognitive impairment, frontal lobe dysfunction, atypical antipsychotics, paliperidone

## Introduction

Normal pressure hydrocephalus (NPH) due to idiopathic aqueductal stenosis is a chronic abnormal accumulation of cerebrospinal fluid (CSF) in the cerebral ventricles caused by an obstruction in the Sylvian aqueduct. This leads to a dilatation of the ventricular system and to subsequent damage of the adjacent parenchyma. Although NPH typically presents with the progressive "triad" of cognitive impairment, gait disturbance and urinary incontinence, it has been described that it rarely manifests in the form of predominant psychotic symptoms.

## Case report

### *Chief complaint*

*Ms*. A, a 30-year-old, divorced, unemployed, white woman with some high-school education was referred to and admitted involuntarily in our acute psychiatric department because she exhibited verbal and physical aggressive behavior towards members of her immediate family while being in an agitated state with delusions of persecution.

### *History of present illness*

*Ms*. A's symptoms were first observed by her family approximately five years before her current presentation. Following a period of severe marital and financial difficulties, she became melancholic, irritable and intolerant of social interaction, withdrawn and isolated, negligent of her personal hygiene and constantly involved in delusional ideas of persecutory type.

*Ms*. A unquestionably attributed all her misfortunes to the devious and malevolent acts of certain, living or dead, members of her family. Although the patient was unspecific about the means used by these persons to carry out their plan, it was implied that some kind of magical procedure, like casting of spells or curses, was implemented.

During the aforementioned 5 year period, she remained housebound and showed absolute decline in her social, interpersonal and occupational level of functioning. As the patient was unable of attending to basic daily activities, such as food preparation, house cleaning, washing and bathing, she developed a total dependency upon her mother in whose house she also resided. It should also be noted that during the same 5 year period she exhibited increased appetite and considerable weight gain.

*Ms*. A had a history of mediocre academic performance deciding on her own will to quit high school when she was 15 years old. This decision was greatly influenced by the relocation of her family from a heavily populated modern urban area to a thinly populated conservative rural area where the patient never managed to appropriately adjust. In the following years, subsequently to her father's financial ruin, the patient experienced another traumatic event; this being her somewhat obligatory immigration to Germany in search of employment. This choice was dictated by the presence of close relatives in Germany, especially her maternal grandmother, who could act as a strong support system for her.

While in Germany, she was married to a 2nd generation greek immigrant of significant financial status. However, after a couple of years, certain stressful events, such as the loss of her maternal grandmother and the severe financial and health problems of her husband, put a terrible strain both on her mental health and on their marriage which soon after dissolved.

Immediately after the dissolution of her marriage, Ms. A –already exhibiting the above mentioned signs of serious mental illness and apparently unable of taking care of herself– returned to the safety and support of her parents.

Lacking insight into her mental illness, the patient explicitly rejected her family's persistent suggestions to seek psychiatric help, thus remaining undiagnosed and untreated for several years.

The exacerbation noted in her mental status at the time of referral seemed to have a temporal correlation and might have been triggered by the recent loss of her father who had died of a heart attack 40 days prior to admission.

#### *Diagnostic evaluation*

*Ms*. A's psychiatric examination on admission revealed the following clinical features:

- A guarded and reserved attitude towards the examiner
- Affective flattening with affective non-responsivity, unchanging facial expressions, decreased sponta-

neous movements, paucity of expressive gestures, lack of vocal inflections and poor eye contact
- Poor grooming and hygiene
- Poverty of content of speech
- Persecutory delusions.

Due to her obese physical appearance which raised the question of an underlying endocrine disorder (e.g. Cushing's syndrome, hypothyroidism), along with the fact that this was her first hospitalization, we submitted the patient to an extensive medical work-up.

The following screening tests were performed and proved within normal limits:
- Complete blood count
- Complete blood chemistries (including glucose, electrolytes, B12, folate, tests of hepatic and renal function)
- Thyroid function tests
- Hepatitis markers (HBV, HCV)
- HIV markers and RPR test
- Serum cortisol
- Plasma ACTH
- Pituitary hormone tests
- EKG.

*Ms*. A's slow and cautious gait along with her decreased arm swing gave her a bradykinetic-like appearance and implied the presence of an underlying neurologic disease. However her neurological examination revealed no actual focal deficits and her EEG proved negative for seizure activity and encephalopathy.

She underwent both a CT scan and an MRI which revealed a dilatation of the ventricular system due to idiopathic aqueductal stenosis. This dilatation was slightly asymmetric in favor of the left hemisphere. Mild atrophy of the cerebral parenchyma was also noted.

The patient was also tested with neuropsychological and intelligence batteries which showed borderline intelligence. Her Rorschach Inkblot Test revealed reluctance in processing emotional stimuli, low self esteem, lack of effort and motivation, emotional immaturity and bluntness, repression of aggressive feelings and indicated the possible emergence of a perceptive disorder upon confrontation with unusual situations and stressful life events.

Although she scored 29 out of 30 on her Mini-Mental State Examination, losing only one point for language and praxis, she under-performed in the WAIS-R, receiving a full scale IQ score of 66. Furthermore, a significant discrepancy was noted between her verbal and her performance IQ scores; the patient receiving scores of 74 and 60 points respectively.

Her performance was particularly poor in the Block Design, Object Assembly, Picture Completion and Digit Span subtests, thus indicating a marked impairment of her spatial perception, visual abstract processing, visual details perception, attention and concentration.

*Ms*. A's symptomatology at onset, together with her current psychiatric presentation, initially narrowed down our diagnostic scope to differentiating between a case of Major Depressive Disorder with Psychotic Features and that of a Psychotic Disorder, mainly Paranoid Type Schizophrenia. Although her mental signs and symptoms at disease onset (i.e. 5 years prior to admission) fulfilled criteria of a major depressive episode, her current presentation lacked prominent affective features; the patient being emotionally flat and non-responsive. Thus, Paranoid Type Schizophrenia was chosen as our working diagnosis.

However, the subsequent and unexpected neuroimaging finding of NPH inevitably raised the question if this clinical entity could have influenced, triggered or predisposed the patient to the development of a psychotic disorder; furthermore, if Ms. A could qualify as a case of Psychotic Disorder due to a General Medical Condition.

*Treatment course*

*Ms*. A's lack of insight, along with the long duration of untreated psychosis, weighed in favor of initiating a long acting neuroleptic medication; in this case 50 mg of long-acting risperidone every 2 weeks. Additionally, she received a daily morning oral dose of paliperidone 9 mg.

After the first 6 weeks of treatment, she began to show signs of marked improvement primarily in her thought content disturbance and secondarily in her affective flattening.

The unwavering conviction by which she adhered to her delusional beliefs gradually receded and her persecutory delusions reduced in intensity. This was accompanied by an improvement in her ability for emotional investment and involvement.

A couple of weeks later, she was able to fully distantiate herself from her initially firm persecutory beliefs and recognize them as non-sensical.

Given her marked improvement, she received a weekly leave to visit her hometown. During this stay with her family, she was reported to be calm, sociable and friendly, even towards those relatives whom she used to picture as persecutors in her delusional system.

Although *Ms*. A showed typical neuroimaging findings of NPH, she did not clearly exhibit respective clinical manifestations. Of the classic triad (Hakim and Adams triad) of urinary incontinence, gait disturbance, and dementia only a mild cognitive impairment was substantiated in the neuropsychological and intelligence batteries.

In the ensuing neurosurgical evaluation, a conservative approach was recommended in the form of pharmacotherapy with a daily dose of acetazolamide 250 mg. Based on the neuroimaging and clinical findings, an invasive surgical procedure was deemed unjustified and unnecessary at the time of the examination. Ms. A was scheduled for a neurosurgical follow up and a new MRI after 6 months.

After 2 ½ months of compulsory inpatient care, she was released from our clinic on paliperidone and long acting risperidone. Her mental status examination on discharge revealed emotional responsivity, increased volition at initiating tasks, improved grooming and hygiene, and primarily no active paranoid ideation.

She was scheduled for monthly follow-ups at our outpatient clinic.

## Discussion

A wide range of non-psychiatric medical conditions has the capacity to induce symptoms of psychosis. In this context, it has been suggested that thought and perceptual disorders could develop secondary to the damage caused by hydrocephalus with or without aqueduct stenosis.[1,2]

The cumulative probability of aqueduct stenosis and schizophrenia co-occurring by chance is very low; a finding that is not in accordance with the empirical data. If 0.8–1% of the population develop schizophrenia, congenital hydrocephalus occurs with a frequency of 0.5–1.8 per 1000 births, and between 11% and 43% of hydrocephalics have aqueduct stenosis, then schizophrenia and aqueduct stenosis should occur together in roughly 0.5–8 individuals per million.[3,4]

The relationship of aqueduct stenosis to hydrocephalus is unclear. It is thought that at times it may be involved directly in the pathogenesis of hydrocephalus, or it may develop as a sequel to it, perhaps resulting from compression.[3,5]

Non-tumoral stenosis of the Sylvian aqueduct is a rare developmental anomaly that usually presents in infancy and childhood, but it is well documented as being diagnosed in older patients over 17 years of age.[6]

Normal pressure hydrocephalus results from an obstruction in the subarachnoid space which impedes the normal flow of the cerebrospinal fluid. CSF normally flows from the basal cisterns over the cerebral convexities to the superior sagittal sinus where it is returned to the systemic circulation. The disorder has a variety of different underlying etiologies. These include hypertensive cerebrovascular disease, subarachnoid space occupying lesion or hemorrhage, chronic meningoencephalitis, residual changes following acute bacterial meningitis or spinal anesthesia and carcinomatosis of the meninges; but often there is no obvious cause (idiopathic cases).

Gait disorder is often the initial manifestation, typically in the form of gait apraxia characterized by apprehension or unsteadiness on standing and difficulty in initiating ambulation. Gait dysfunction is often described as broad-based and "magnetic".[7] It is also notable for impaired turning marked by the need for multiple small steps in order to effect a 180° turn.[8] The gait disturbance may be due to a number of factors, including disconnection of basal ganglia from the frontal cortex, paraventricular corticospinal fiber damage and uninhibited antigravity reflexes.[9–11] Extrapyramidal features in the form of hypokinesia,

bradykinesia and cogwheel rigidity have also been documented.[8,12,13]

Urinary incontinence is usually a later development and is thought to be due to damaged periventricular pathways to the sacral bladder center, resulting in reduced inhibition of bladder contraction.[14]

The dementia is characteristic of the subcortical type with prominent frontal lobe features, including psychomotor retardation, amotivation and apathy, resembling a picture of depression. NPH-related cognitive impairment is usually insidious in onset. Initially characterized by generalized slowing and apathy, global cognitive dysfunction ensues if untreated.[9] Based on previous neuropsychological data of apparently greater effect of NPH on executive function, attempts to identify an NPH-specific cognitive impairment profile have utilized tests relatively sensitive to frontal lobe function to examine whether such tasks are particularly susceptible to NPH-induced damage. Results of such studies reveal that there are two distinctive patterns of cognitive change in NPH:[8]

- Predominantly executive (only later becoming more global), frequently not relieved significantly by shunting
- Generalized dementia detectable by gross measures (e.g. MMSE), in which shunting can reverse dementia.[15]

Although there doesn't appear to be a common neuropsychological pattern in all hydrocephalic patients, various research results indicate a tendency to score low on the performance IQ of the WAIS-R, especially in the subtests that require attention and visuoconstructive functions.[16–18]

Normal pressure hydrocephalus presenting solely as a psychiatric disturbance (e.g. paranoid psychosis) without the classic neurological triad of gait ataxia, urinary incontinence and dementia has been documented. Positive psychotic symptoms were not the only handicaps reported. There was also deterioration in social behavior with social withdrawal and loss of concern for personal care, apathy, agitated depression, mania, sudden violent and self-destructive outbursts, aggression, argumentativeness and belligerence.[6,19–26]

As already mentioned above, communicating hydrocephalus is most likely the result of alterations in clearance and/or flow of CSF, causing ventricular enlargement and secondary susceptibility of brain parenchyma to mechanical and/or ischemic injury. Increased ventricular volume can stretch frontocerebellar nerve fibres as they circumnavigate the ventricles, disturbing gait and bladder function. Deranged ventricular CSF flow on adjacent basal ganglia structures might be responsible for the development of parkinsonian features.[8] It has also been suggested that a primarily altered CSF circulation may affect neuronal function in a way that contributes to or directly causes psychotic symptoms.[27]

The mechanism whereby NPH causes symptoms and damage to the brain is not fully understood. Adams et al[28] adapted Pascal's law of enclosed fluids as a possible explanation of the pathophysiology of this condition. They suggested that initially elevated CSF pressure leads to enlargement of the ventricles, but once enlarged they are maintained at that size, with lower and usually normal pressures. They focused on the concept of total CSF force rather than pressure as the critical factor in CNS damage. The total force on the ventricular walls by Pascal's law is the product of the CSF pressure (x) the surface area. Hence, as surface area increases, the total CSF force also increases despite the maintenance of a constant and presumably normal CSF pressure, with resultant ventricular dilatation and central nervous system damage. Furthermore, they argued that the fact that a given pressure results in greater force in the widest part of an enclosed system, provides an explanation for the greater differential enlargement of the lateral ventricles, especially the frontal horns, and hence, the appearance of predominantly frontal signs in NPH.[21]

Precise anatomical correlates accounting for the psychiatric and behavioral symptoms associated with NPH have not yet been established. However, certain anatomical regions have been known to be associated with particular behaviors and symptoms. For instance, ventricular enlargement due to aqueduct stenosis may present as a schizophreniform psychosis, mania has been associated with diencephalic and hypothalamic dysfunction, and temporal lobe abnormalities may cause episodic aggression.[6,9]

Davison & Bagley[29] in their extensive review concluded that schizophrenia-like illnesses with halluci-

nations and delusions were more common among patients with temporal-limbic or diencephalic dysfunction.

Schneiderian first-rank symptoms, involving thought insertion, thought broadcasting and thought blocking, as well as delusions of external influence, have been reported with limbic dysfunction produced by hydrocephalic processes.[30]

The brain dysfunction in idiopathic NPH (iNPH) is mainly in the frontal cortex. Because frontal lobe lesions are traditionally known to facilitate one's inability to integrate and correct perceptual distortions in the face of contradictory evidence, frontal lobe dysfunction may be integral in delineating the etiology of delusions in NPH.[31,32]

Based on anatomical observations concerning delusions, a tentative pathophysiological formulation could be suggested. CNS functions that are relevant to delusion formation include linguistic and verbally-mediated conceptual abilities processed by the left hemisphere, and visuospatial, perceptual and affective functions mediated by the right hemisphere. These functions interact with the limbic system in the assessment of the emotional significance of ongoing experience. The limbic system in turn has dense projections to the basal ganglia creating an integrated limbic-subcortical system that mediates mood, motivation and motion. Any cerebral lesion that disrupts limbic-cortical associations may cause abnormal emotional assessments and produce a delusional syndrome.[30]

Neuroradiological correlates include enlarged ventricles out of proportion to the prominence of cortical sulci, anterior third ventricle enlargement, downward extension of mammillary bodies, bowing of corpus callosum and a flow-void in the fourth ventricle on T2-weighted MRI. Studies using Single Photon Emission Computed Tomography and Positron Emission Tomography reported that patients with iNPH mainly presented hypoperfusion of the frontal lobe. Thus, the cognitive impairment can be attributed to accentuated damage in the frontal lobe.[8,21]

The question naturally arises as to whether these findings are similar to the ones reported in schizophrenic patients.

Neuroimaging studies of brain morphology in acute and chronic schizophrenia have also shown increased ventricular volume with the frontal horns being possibly slightly more enlarged than the rest of the ventricular system, increased ventricular-brain ratio (VBR), larger 3rd ventricle width and decreased thalamus size, a finding that could be consistent with periventricular injury.[33–36]

Furthermore, certain physiological and cognitive studies have implicated a dysfunction of the prefrontal cortex as the possible explanation for the cognitive deficits observed in schizophrenia.[37]

Hence, current knowledge suggests that dysfunction in the frontal lobe and subcortical structures, notably the mesencephalic and diencephalic regions of the brain, may play an important role in the etiology of the schizophrenic psychoses that are found in association with aqueduct stenosis.

In our patient, the barely noticeable typical NPH symptoms (i.e. mild cognitive impairment and bradykinetic appearance) were clearly overshadowed by the florid psychiatric symptoms that prevailed in the clinical picture. The incidence of NPH manifested predominantly with psychiatric features is unknown. Even if the occurrence should be low, efforts to reveal these patients seem well motivated since the condition is usually continuous or progressive. Additionally, it has been shown that in some cases such patients may benefit from neurosurgical procedures.[6,20–22,38]

Even if no direct association can be established between schizophrenia and normal pressure hydrocephalus due to aqueduct stenosis, it is still reasonable to assume that our patient's mild cognitive impairment may have rendered her liable to react in a psychotic manner towards stressful life events.

This case demonstrates the complexity of the psychiatric diagnostic process. Particularly misleading in our patient were the fairly clear-cut psychotic symptoms. Although there were no specific indications of NPH, some clues suggesting a possible organic factor were present; mainly the patient's minor gait disturbances and free family history of psychiatric disorder. The presence of mild extrapyramidal signs in the absence of recent or current neuroleptic medication should raise the possibility of other causes of these symptoms, such as the so called parkinsonian-dementia complex of NPH.[12,13,39–41] In complicated diagnostic problems, such as this patient presented,

where the presence of organic impairment is not clearly established on clinical grounds, the potential value of neuropsychological testing is also evident.[21]

## Conclusions

Patients who present with soft non-localising neurological signs and mild cognitive deficits in association with prominent psychotic symptomatology should raise our index of suspicion and prompt the clinician to explore the existence of an organic factor contributing to a mental or behavioral disorder. Furthermore, our patient's marked improvement indicates that, in cases of NPH where the primary cause is treated conservatively, psychotic symptomatology could respond to 2nd generation antipsychotics.

# Ψυχωσική συμπτωματολογία σε υδροκέφαλο φυσιολογικής τάσης

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Ο Υδροκέφαλος Φυσιολογικής Τάσης λόγω ιδιοπαθούς στένωσης του υδραγωγού του Sylvius αποτελεί μια χρόνια κατάσταση αυξημένης συσσώρευσης εγκεφαλονωτιαίου υγρού εντός των εγκεφαλικών κοιλιών, η οποία οδηγεί σε διάταση του κοιλιακού συστήματος και πίεση του παρακείμενου εγκεφαλικού παρεγχύματος. Εκδηλώνεται τυπικά με την προοδευτική εγκατάσταση γνωστικής έκπτωσης, διαταραχής της βάδισης και ακράτειας ούρων. Έχουν περιγραφεί, ωστόσο, περιπτώσεις της διαταραχής στις οποίες προεξάρχει η ψυχωτική συμπτωματολογία. Έχει προταθεί ότι ο υδροκέφαλος φυσιολογικής τάσης μπορεί, μέσω των δομικών βλαβών που επιφέρει στις γειτνιάζουσες εγκεφαλικές περιοχές, να οδηγήσει δευτερογενώς στην ανάπτυξη διαταραχών της σκέψης και της αντίληψης. Μολονότι δεν έχουν ανευρεθεί ακόμη οι ακριβείς ανατομικές συσχετίσεις, ορισμένες εγκεφαλικές περιοχές –πρωτίστως ο μετωπιαίος φλοιός, διεγκεφαλικές και μεσεγκεφαλικές δομές– έχουν ενοχοποιηθεί για την παθογένεση της υδροκεφαλικής ψύχωσης. Επειδή οι βλάβες του μετωπιαίου λοβού εμποδίζουν το άτομο να ενσωματώσει στοιχεία που διαψεύδουν τις αντιληπτικές του διαστρεβλώσεις και ως εκ τούτου να τις διορθώσει, η δυσλειτουργία αυτής της περιοχής πιθανώς έχει πρωτεύουσα σημασία στην κατανόηση της αιτιολογίας των παραληρητικών ιδεών στους υδροκεφαλικούς ασθενείς. Παρουσιάζουμε την περίπτωση μιας 30χρονης γυναίκας, η οποία νοσηλεύθηκε ακουσίως εξαιτίας διεγερτικής συμπεριφοράς, επιθετικότητας και παραληρητικών ιδεών δίωξης. Η νευρολογική της εξέταση φανέρωσε υποσημαινόμενη βραδυκινησία. Οι νευροψυχολογικές συστοιχίες και οι δοκιμασίες ελέγχου της νοημοσύνης αποκάλυψαν ελαφρά γνωστική έκπτωση και η αξονική τομογραφία εγκεφάλου κατέδειξε διάταση του κοιλιακού συστήματος λόγω ιδιοπαθούς στένωσης του υδραγωγού του Sylvius. Η ασθενής ετέθη σε αγωγή με αντιψυχωτικά 2ης γενιάς, επιδεικνύοντας καλή κλινική ανταπόκριση. Η άτυπη εικόνα του υδροκέφαλου στο παραπάνω περιστατικό υπογραμμίζει την αναγκαιότητα να διερευνάται πλήρως η πιθανότητα ύπαρξης υποκείμενου οργανικού αιτιολογικού παράγοντα σε όσους ασθενείς παρουσιάζουν προεξάρχουσα ψυχωτική συμπτωματολογία σε συνδυασμό με μαλακά, μη εστιακά νευρολογικά σημεία και ήπια γνωστικά ελλείμματα. Περαιτέρω, η καλή κλινική ανταπόκριση της συγκεκριμένης ασθενούς υποδεικνύει ότι, σε περιπτώσεις στις οποίες επιλέγεται η συντηρητική αντιμετώπιση της πρωτογενούς αιτίας, η υδροκεφαλική ψύχωση μπορεί να απαντήσει στη φαρμακευτική αγωγή με αντιψυχωτικά 2ης γενιάς. Με αφορμή αυτό το περιστατικό προχωρήσαμε σε μια σύντομη ανασκόπηση της βιβλιογραφίας.

**Λέξεις ευρετηρίου:** Υδροκέφαλος φυσιολογικής τάσης, ψυχωτικά συμπτώματα, ήπια γνωστική έκπτωση, δυσλειτουργία μετωπιαίου λοβού, άτυπα αντιψυχωτικά, παλιπεριδόνη

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