CASE REPORT

A Case Report of Traumatic Normal Pressure Hydrocephalus

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Abstract

Normal pressure hydrocephalus (NPH) is a condition whereby the ventricles are pathologically enlarged with normal intracranial opening pressure. Here, we highlight a rare case of NPH who first presented atypically to psychiatry team. There were emotional and behavioral changes following a road traffic accident that subsequently presented as acute stress reaction with ataxic gait. CT brain showed NPH. Lumbar puncture was done to relieve intracranial pressure. Patient gradually recovered without any neurological or psychological deficit. In conclusion, a high clinical suspicion is important to identify NPH and prompt treatment should be offered to optimize the clinical outcome.

Keywords: Secondary Normal Pressure Hydrocephalus, Ataxia, Traumatic Brain Injury

Introduction

Normal pressure hydrocephalus (NPH) is a condition whereby the ventricles are pathologically enlarged with normal intracranial opening pressure [1]. It was first described in 1965 [2].

It is a type of communicating hydrocephalus, in which there is no physical blockage within circulating cerebrospinal fluid (CSF) in the ventricular system [3]. NPH is usually diagnosed through computer tomography (CT) brain with ventricular shunting as the only known treatment [4, 5].

Case Report

Mrs K, a 44-year-old married Malay lady, with no history of medical or mental illness, met with a motor vehicle accident with her husband in March 2019. Her husband was admitted in ward, while she was allowed discharge home without any complications. However, over the following two weeks, family noticed she had a sudden change in behaviour. Initially, she appeared quiet and socially withdrawn. She would only respond with one-word answers. She appeared confused and forgotten who her children were. She appeared more tearful than before but could not tell the reason. She also lost interest in house chores and gardening. Her appetite was reduced. She appeared tired and forgetful most of the time.

While patient visited her husband in the orthopaedic ward two weeks after the
accident, she was noted to have psychomotor retardation and appeared depressed by the treating doctor in ward. Hence, a referral to the psychiatry team was made. She revealed that she felt numb and was unable to recall the events during the accident. She complained of intermittent tinnitus of the left ear, but no headaches, syncope, blurring of vision, nausea or vomiting. No history suggestive of post-traumatic stress disorder. There were no other anxiety, depressive, or psychotic symptoms observed. There is no family history of mental illness or substance use. Pre-morbidly, she was a cheerful person.

Mental state examination revealed a middle-aged, medium built and well kempt Malay lady. Eye contact and rapport were poor. She spoke relevantly in Malay, with minimal verbal output. She answered in short phrases and was slow in response. Her mood was euthymic and affect was restricted. She did not have any thought or perceptual disturbances. She was orientated to time, place and person. She appeared alert, pupils were equal and reactive. Her vital signs were stable and afebrile without meningism. Neurological and other systemic examinations were unremarkable, except her ataxic gait. Blood investigations including full blood count, renal profile and liver function test were normal. Thus, an initial diagnosis of acute stress reaction was made, with differential diagnosis of delirium secondary to traumatic brain injury.

An urgent CT brain was performed the next day, which revealed normal pressure hydrocephalus (Figure 1). Then, she was referred to neurosurgery and medical teams. She underwent lumbar puncture, with opening pressure was 21cm H$_2$O, and 30ml of clear and odourless CSF was drained. CSF investigation results were unremarkable. Post procedure, she had marked improvement. She appeared more alert and responsive, with improved verbal output. She no longer complained of tinnitus. She regained her normal gait. She was discharged uneventfully by the medical team. Two months after discharge, she was reviewed in psychiatric clinic. She has returned to her premorbid level of functioning.

**Discussion**

NPH can be classified into idiopathic (50% of NPH) and secondary NPH [6]. The prevalence of NPH varies. Idiopathic NPH (iNPH) is usually found among elderly of age 60 or above whereas secondary NPH (sNPH) can be found in any age [7]. According to a population based study among aged 65 years and older in Swedish county of Jämtland, the prevalence of iNPH was 3.7% [8]. Its prevalence was higher among those aged 80 years and older [8]. There is no gender discrepancy in prevalence [9]. The causes of sNPH includes subarachnoid hemorrhage (46.5%), head trauma (29%), intracranial malignancies (6.2%), meningoencephalitis (5%), and cerebrovascular disease (4.5%) [10]. Hence, in this case report, the etiology of her NPH is most likely due to recent trauma.

NPH is characterized by triad of dementia, ataxia and urinary incontinence [2]. The occurrence of all triad (Hakim triad) is only about 50 to 75% of the patients and usually patients only presented with one or two of the three typical symptoms. The symptoms of gait and cognitive disturbances occurred in 80% to 95%, and urinary incontinence in 50% to 75% of patients [11]. Gait disturbance is generally the first and most common symptom to appear and also the first one to resolve post-operatively [12]. Hence, a gait disturbance plus another
additional symptom from its triad is worth considering a diagnosis of NPH.

In this case, the patient was first identified by other medical personnel and subsequently referred to the psychiatry team. The first symptoms observed were change of personality and cognitive decline, with poor interpersonal interaction, as perceived by others as having depression. This phenomenon may give differentials of acute stress reaction, delirium, depression, grief, post-concussion syndrome, or dementia. However, the ataxic gait should be investigated further.

The progression, severity and the extent of deterioration of sNPH varies from one to another [13]. The initial presentation of sNPH is not necessarily one of the triads of NPH. There is a case of traumatic NPH which was initially treated as post-traumatic stress disorder in psychiatric ward and was not responded to typical psychiatry treatment [14]. Patient’s condition worsened and progressed to seizure after two weeks of admission associated with facial nerve palsy, and progressive weakness of lower limbs. NPH was then diagnosed after CT of the brain. The patient’s condition improved after a ventriculoperitoneal (VP) shunt was done. In comparison to this case, this patient showed improvement after ventricular tapping. Her condition gradually returned to normal after two months.

Conclusion

The occurrence of traumatic NPH is rare, however, we as psychiatrists, should have high suspicion of organic causes to the cases that presented to us whenever there is a traumatic event occurring prior to it. In addition, this case report may serve as an example to increase awareness among primary health care personnel, who are the front-liners to identify it and refer to the respective team for early intervention.

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References


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