Changes in the Subarachnoid Space Precede Ventriculomegaly in Idiopathic Normal Pressure Hydrocephalus (iNPH)

Chifumi Iseki, Yoshimi Takahashi, Manabu Wada, Shigeki Arawaka, Toru Kawanami and Takeo Kato

Abstract

We report on an 80-year-old woman who had shunt-responsive idiopathic normal pressure hydrocephalus (iNPH). Her brain MRI showed an initial change in the subarachnoid space (a disproportional narrowing of the subarachnoid space at the cerebral high convexities) at the age of 70 years, followed by enlargement of the ventricles (Evans index: 0.31) at the age of 78. The observation suggests that changes in the subarachnoid space may precede ventricular enlargement in iNPH.

Key words: idiopathic normal pressure hydrocephalus (iNPH), cerebrospinal fluid (CSF), magnetic resonance imaging (MRI), asymptomatic ventriculomegaly, subarachnoid space

(Intern Med 51: 1751-1753, 2012)  
(DOI: 10.2169/internalmedicine.51.7254)

Introduction

Idiopathic normal pressure hydrocephalus (iNPH) occurs in the elderly and is characterized by a clinical triad of gait disturbance, cognitive impairment, and urinary incontinence. Although the ventricles of the brain are enlarged, the cerebrospinal fluid (CSF) pressure is within normal limits (1). At present, neuropathological changes common to iNPH cases have not been established, and the etiology of iNPH remains unknown, although genetic factors are suggested to be involved in the pathogenesis (2, 3). The clinical significance of iNPH is that neurological symptoms are treatable by ventricular shunt placement. The diagnosis of iNPH has progressed with the use of brain magnetic resonance imaging (MRI). A report by Kitagaki et al. revealed that, in addition to ventriculomegaly of the brain, a disproportional narrowing of the subarachnoid space and cortical sulci at the high convexity of the cerebrum, as compared with widening of Sylvian fissures, is a hallmark for iNPH on a brain MRI (4). These MRI features have proved useful in the diagnosis of iNPH and are now included in the “Guidelines for Management of iNPH” by the Japanese Society of NPH (5). However, it remains undetermined which MRI feature occurs first: ventricular enlargement or the subarachnoid changes described above. This detail is important because it may be related to the pathogenesis of iNPH.

Since 2000, we have conducted a community-based, prospective study of elderly people using brain MRI scans as well as various medical and neurological examinations (6-8). In the study, we found an individual who showed an initial change in the subarachnoid space, followed by enlargement of the ventricles.

Case Report

We report a 70-year-old asymptomatic woman whose brain MRI in 2000 showed a disproportional narrowing of the subarachnoid space at the cerebral high convexities, as compared with widening of the Sylvian fissures; at that time, she was diagnosed as having no apparent ventriculomegaly because the ventricular size was similar to that of many other elderly participants with no neurological symptoms and the Evans index was 0.28 (FigA, B). At an examination in 2008, the woman (now 78 years old) had no neurological symptoms, including gait disturbance and cognitive decline. However, her brain MRI showed an apparent ventriculomegaly (Evans index: 0.31) (FigB). The observation suggests that changes in the subarachnoid space may precede ventricular enlargement in iNPH.
Her mental irritability also improved, although the MMSE scores remained unchanged. Based on the Guidelines for NPH in Japan (9), she was diagnosed as having “definite iNPH”.

**Discussion**

The present observation demonstrated that changes in the subarachnoid space preceded ventricular enlargement in this subject. In iNPH, ventriculomegaly is a prominent feature of a brain MRI; however, the present observation suggests that the primary morphological change of the brain in iNPH may occur in and around the subarachnoid space, followed by enlargement of the ventricles. It has been speculated that a significant amount of CSF is absorbed by the capillaries and veins of the subarachnoid space, leptomeninges, and their underlying brain parenchyma (10). A possible explanation for the MRI changes in the present case is that a disturbance of the ability of vessels to absorb CSF in and around the Sylvian fissures may lead to CSF accumulation in the Sylvian fissures, which may cause the cerebral high convexities to be mechanically compressed upward, resulting in the tight high convexity, followed by dilatation of the ventricles. However, the exact pathomechanism of iNPH with convincing evidence remains undetermined. To further clarify the pathogenesis of iNPH, more attention should be paid to the pathological changes of the walls of the vessels, including venules and capillaries, in the subarachnoid space and the underlying parenchyma in the iNPH brain.

**The authors state that they have no Conflict of Interest (COI).**

**Acknowledgement**

The study was supported in part by a Grant-in-Aid from the Global COE Program (F03) of the Japan Society for the Promotion of Science and in part by the Research Committee of Normal Pressure Hydrocephalus, Studies on the Epidemiology, Pathogenesis and Therapy from the Ministry of Health, Labor and Welfare of Japan.

**References**


