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Short Communication

The Prognosis in Idiopathic Standard Pressure Hydrocephalus

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DESCRIPTION

Normal pressure hydrocephalus can occur asymptomatically or as a result of a specific pathology such as subarachnoid hemorrhage or brain trauma. Idiopathic NPH (iNPH) is a kind of communicating hydrocephalus for which the pathophysiology is unknown. According to some research, prolonged hypertension and white matter disorders can cause periventricular ischemia, which raises ventricular wall resistance and promotes gradual ventricular enlargement. Conversely, periventricular ischemia can cause localized increased vascular resistance, which can result in decreased Cerebro Spinal Fluid (CSF) consumption and ventricular hypertrophy. One ultrasonography research in patients with iNPH found indications of retrograde flow in the internal jugular veins, indicating fundamental carotid valve insufficiency [1,2].

The CSF in individuals with iNPH represents one of the grounds of its pathogenesis, with impaired periventricular metabolism and axonal degeneration without severe cortical damage. The altered CSF dynamics were found to be one of the main causes of iNPH, with decreased amplitude of cardiac-related intracranial pressure pulsations in patients who benefited from shunt operation. Idiopathic NPH is more frequent in persons over the age of 60. The diagnosis is problematic because the presenting symptoms and ventriculomegaly are frequently attributed to ageing or neurodegenerative disorders. Shunt insertion is the most commonly used therapy approach, with the gait problems improving the most. Yet, there are fewer guarantees that such a surgery will be successful, and it has its own share of serious complications [3-5].

Although the clinical signs and symptoms of iNPH can be confused with the natural ageing process or other neurodegenerative diseases such as Alzheimer's Disease (AD) or Parkinson's Disease (PD) or even vascular diseases such as Vascular Dementia (VD), key characteristics in their presentations distinguish them. The age of onset for "probable iNPH" was retained at more than 40 years, with symptoms lasting at least 3 to 6 months; for possible iNPH, onset could begin at any age after childhood, with symptoms lasting less than 3 months. A clinical diagnosis of "probable" needs a gait or balance issue as well as impaired in cognitive or bladder control, or both [6,7].

Individuals who appear with incontinence or cognitive impairment but no discernible gait or balance problem are classified as probable. Changes in gait are the most noticeable clinical features in the early stages of iNPH, and they are also thought to be the most sensitive to shunting. Dementia without gait disturbance is safe to rule out. The gait of iNPH has been described as "fastened to the floor," magnetized gait, gait apraxia, or a frontal ataxia, with short steps, decreased stride in both height and length, externally turned feet, reduced cadence, and a broadened base as opposed to a narrow base in Parkinson's disease [8].

Individuals turn slowly, their positional stability is reduced, and they may have a history of falls. Patients occasionally complained of nonspecific discomfort in their legs after walking a moderate distance. A computerized gait analysis demonstrates an aberrant tendency towards contraction in antagonist muscle groups, as well as diminished pelvic rotation and counter rotation of the torso, demonstrating a loss in subcortical motor coordination [9,10].

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