Ke d : Cerebrospinal uid; Alzheimer disease; Normal pressure hydrocephalus; Amyloid; Tau proteins; Diagnosis

I d c

Alzheimer's disease (AD) is characterized by a progressive dementia, the presence of neuritic plaques, and nerve cell degeneration [1]. Idiopathic normal pressure hydrocephalus (iNPH) was rst described as a combination of gait disturbance, urinary incontinence, and cognitive impairment [2]. It is estimated that AD a ects about 11% of individuals aged 65 years or older [3], whereas iNPH only a ects 1.4% of the same population [4]. Of all patients clinically diagnosed with AD, post-mortem examinations reveal that 10%–20% of these patients have died with conditions other than AD [5]. Neuropathological examinations reveal that about 56% of patients clinically diagnosed with NPH also su ered AD [6].

Studies show that amyloid-β (Aβ42), total-tau (t-tau), and phospho-tau181 (p-tau) are e ective biomarkers for AD patients [7]. e sensitivity and speci city for Aβ42 to discriminate AD from non-demented individuals is 86% and 89%, respectively; for t-tau, 81% and 91%, respectively; regarding p-tau, 81% and 91%, respectively; and for the combination of t-tau and Aβ42, 89% and 90% respectively [8]. However, these same biomarkers are much less understood when applied to iNPH, as results from studies are contradictory [9,10]. Biomarkers are not routinely drawn for iNPH in clinical practice; incorporating these indicators may improve accuracy in the diagnosis of the dementia. Current methods for the diagnosis of iNPH include MRI scans, high volume cerebrospinal uid (CSF) removal, nuclear medicine studies (cisternography), and evidence-based guidelines

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