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Disturbo della marcia, parkinsonismo e funzionalità dopaminergica striatale in pazienti con idrocefalo normoteso idiopatico: uno studio prospettico longitudinale

Gait disorder, parkinsonism, and striatal dopaminergic function in patients with idiopathic normal pressure hydrocephalus: a prospective longitudinal study

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Introduction: Motor phenomenology in idiopathic normal pressure hydrocephalus (iNPH) can include parkinsonism. Striatal dopamine reuptake transporter (DAT) density was found to be reduced and to correlate with the severity of parkinsonism in several iNPH patients. However, pathophysiology of gait disturbances and parkinsonism is still controversial, and large longitudinal evaluations are lacking.

Objective: We aimed to longitudinally assess higher-level gait disorder (HLGD), parkinsonism, and striatal DAT binding in iNPH patients who did or did not undergo shunt surgery.

Methods: At baseline, we evaluated iNPH patients through clinical rating scales, response to levodopa treatment, brain MRI, and [^{123}I]-FP-CIT SPECT. We followed up patients who did or did not undergo lumboperitoneal shunt surgery, and [^{123}I]-FP-CIT SPECT was repeated after two years.

Results: Among 115 iNPH patients, 102 subjects did not show either a significant improvement with levodopa or signs supporting an atypical parkinsonism. In this subgroup, 59 patients underwent shunt surgery. In particular, [^{123}I]-FP-CIT SPECT was performed by 92 subjects at baseline and by 58 subjects also at follow-up. DAT density of caudate nucleus correlated with gait impairment both in patients with a disequilibrium subtype of HLGD and in patients with a locomotor subtype of HLGD. In this latter group, DAT density of putamen and caudate nucleus also correlated with parkinsonism. In patients with a disequilibrium subtype of HLGD, gait and DAT density of caudate nucleus improved after surgery, while worsened without surgery. In patients with a locomotor subtype of HLGD, gait, parkinsonism, and DAT binding of putamen and caudate nucleus improved after surgery, while worsened without surgery.

Conclusions: Parkinsonism in iNPH is featured by lack of levodopa response, but is shunt-responsive in several patients. Gait disturbances and parkinsonism in iNPH could be due to striatal dopaminergic dysfunction, which can be reversible after shunt treatment, while worsens in patients who decline surgery.