The characteristic of cognitive dysfunction in remitted late life depression and amnestic mild cognitive impairment

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A B S T R A C T

Remitted late life depression exhibits persistent cognitive impairments and enhances the risk of dementia. This study aimed to examine the characteristics of cognitive dysfunction in remitted late life depression and amnestic mild cognitive impairment (MCI). Remitted late life depression (n=61), amnestic MCI (n=61) and age-education-matched controls (n=65) were evaluated with a battery of neuropsychological tests grouped into executive function, memory, processing speed, attention and visuospatial domains. Compared with control subjects, amnestic MCI individuals showed more severe cognitive impairments in all domains, while remitted late life depression individuals performed worse in executive function and memory. The pattern of cognitive profiles significantly differed between remitted late life depression and amnestic MCI groups, which might be mainly attributed to worse impairments in memory and executive function in amnestic MCI individuals. Executive function was the core impaired cognitive domain mediating the influence of predictors on other cognitions in both remitted late life depression and amnestic MCI groups, which indicated a possible etiopathogenic mechanism underlying the conversion to dementia.

1. Introduction

Late life depression, characterized by a pervasive and persistent low mood in elderly people (60 years or older), is invariably accompanied by multiple cognitive impairments, including executive function, memory, processing speed, attention and visuospatial skill domains (Butters et al., 2004; Koenig et al., 2015; Kohler et al., 2010; Yeh et al., 2011). In recent years, late life depression has been considered a part of the preclinical course of Alzheimer’s disease (AD) (Mahgoub and Alexopoulos, 2016; Orsolo et al., 2014) and was associated with a 2–5-fold increased risk of developing AD compared with healthy aging individuals (Szczyinski et al., 2010; Wilson et al., 2002). Amnestic mild cognitive impairment (amnestic MCI), a MCI subtype characterized by memory deficit, is a transitional stage between normal aging and AD. Patients with amnestic MCI have been reported an 8.6-fold higher conversion risk to AD compared with patients reporting memory problems without objective impairments on neuropsychological testing (Lehrer et al., 2005). The classification and pattern of the cognitive deficits in remitted late life depression and amnestic MCI may shed light on the pathogenesis of their conversion to dementia.

To date, the majority of studies have demonstrated that processing speed deficit is the core cognitive impairment followed by executive function in the acute phase of late life depression, meaning that changes in these two cognitive domains were sufficient to mediate changes in other cognitive domains (Butters et al., 2004; Delaloye et al., 2008; Kohler et al., 2010; Nebes et al., 2000; Sheline et al., 2006). However, a 5-year follow-up study only partially agreed with the previous findings, as it showed that executive function and memory deficits, but not processing speed deficit, were potential predictors of the late life depression conversion to dementia during acute depression (Potter et al., 2013). Moreover, the symptom spectrum of cognitive impairments varied substantially from the acute to the remitted stage of patients with late life depression. Most cognitive dysfunction in the acute depressive phase might improve, although not return to normal levels, after depressive symptoms are reduced (Herrera-Guzman et al., 2010). For instance, the processing speed deficits observed at baseline in late life depression patients had largely recovered at the 18-month follow-up (Kohler et al., 2010). Executive function and memory impairment were reported to persist in 28.5% patients with amnestic MCI and 23.8% with non-amnestic MCI in remitted late life depression.
The present study. Subjects with remitted late life depression were categorized as major depression and were determined from personal testimony and medical records. The 17-item Hamilton Rating Scale for Depression (HAMD-17) represents current depressive symptom severity (Hamilton, 1967). The Hachinski Ischemic Score is a clinical tool to evaluate vascular factors and is helpful to differentiate major types of dementia (Kim and Kwon, 2014).

2.3. MRI acquisition and analysis

All participants underwent a MRI scan using a 3.0 T Trio Siemens scanner with a 12-channel head-coil (Siemens, USA) at Affiliated ZhongDa Hospital, Southeast University. High-resolution T1-weighted axial images covering the whole brain were acquired using a T1-weighted 3D magnetization-prepared rapid gradient echo (3D-MPRAGE) sequence (TR = 1900 ms, TE = 2.48 ms, TI = 900 ms, FA = 9°, number of slices = 176, thickness = 1 mm, gap = 0 mm, imaging matrix = 256×256, FOV = 250 mm × 250 mm, acquisition duration: 4 min and 18 s). T2-weighted axial images were acquired using T2 fluid-attenuated inversion recovery (T2-FLAIR) sequences (TR = 8400 ms, TE = 94 ms, TI = 2439 ms, FA = 150°, number of slices = 20, thickness = 5 mm, gap = 0 mm, imaging matrix = 256×256, FOV = 230 mm × 230 mm, acquisition duration: 1 min and 59 s). The periventricular hyperintensity (PVH) and deep white matter hyper-intensity (DWMH), which represented white matter changes in the T2-weighted axial images, were rated using the modified Fazekas scale (Fazekas et al., 1987). Brain atrophy on the T1-weighted axial images were analyzed by optimized voxel-based morphometry (VBMA) combined with Statistical Parametric Mapping (SPM8) software, obtaining grey matter volume (GM), white matter volume (WM) and cerebrospinal fluid volume (CSF).
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