Architecture of Neuropsychological Impairment in Epilepsy

Ma Fatima Desacula,

Central Philippine University Philippines

Abstract

This theoretical study aims to provide a conceptual and technical approach to systemic family therapy (SFT). The work starts pointing the postulates of general systems theory, cybernetics second-order theory of human communication practice-oriented representation, the conception of the family, their evolutionary cycle stages of crisis vivencian as you get to form a therapeutic system. It is concluded that the approach is empirical evidence has, holding, and today the SFT is becoming abandoning and / or reinventing some of its original tenets of new constructs. Clinical scale scores increased in all patients over time, most prominently in the SCA1 (SARA) and SCA3 (INAS) groups. New impairments on neuropsychological tests were most commonly observed with executive functions, speed, attention, visual memory and Theory of Mind. Results suggest possible differences in cognitive decline in SCA subtypes, with the most rapid cognitive decline observed in the SCA1 patients, and the least in the SCA6 patients, congruent with observed patterns of motor deterioration. Minimal changes in mood were observed, and MRI measures of atrophy did not correlate with cognitive decline. The spinocerebellar ataxias (SCAs) are a clinically and genetically heterogeneous group of dominantly inherited neurodegenerative disorders, with a prevalence range of 0.9-3 in 100,000. Over thirty genetic subtypes have been described; the most common of these are SCA1, SCA2, SCA3, SCA6 and SCA7. The SCAs are associated with a polyglutamine repeat expansion, the length of which is inversely correlated to the age of onset of symptoms. In SCA1, SCA2, SCA3 and SCA7, symptoms typically develop in the fourth decade, whereas in SCA6 onset is approximately 20 years later. Whilst the cardinal clinical features of the SCAs involve gait and truncal ataxia, dysarthria and limb ataxia, phenotypic differences include diplopia, progressive visual loss, spasticity, dysphagia, parkinsonism, dystonia, peripheral neuropathy and urge incontinence. The progression of cerebellar symptoms in the SCAs has been well characterised. A large two-year follow-up study involving over 400 patients reported the greatest annual increase in ataxia severity amongst SCA1 patients, followed by SCA3 and SCA2 patients. In contrast, SCA6 patients exhibited a slower, non-linear pattern of progression. The findings for non-ataxia signs were similar; the fastest rate of decline was described for SCA1 while SCA2 and SCA3 were slower but similar. Some non-ataxia signs were present in SCA6 but no progression was observed over the two-year period. Polyglutamine repeat length and age of onset were associated with the more rapid clinical progression in SCA1 and SCA2 patients. In SCA3, clinical progression was affected by disease duration. In a second study involving a sub-group of the same sample, clinical progression was assessed in the context of MRI-based volumetry. This study demonstrated a correlation between ataxia symptoms and brainstem atrophy in SCA1 and SCA3. In addition, cerebellar atrophy was also associated with symptoms in SCA3. A comparison of clinical progression in SCA7 patients amongst other SCA groups has yet to be completed, but this phenotype is known to involve retinal degeneration and progressive visual loss.

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