



Psychological Profile of 50 Patients with Hereditary Ataxia

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Introduction

Patients with Functional Cognitive Disorder (FCD) have severe subjective cognitive symptoms that are inconsistent with their observed level of cognitive functioning and are not explained by an identifiable neurodegenerative, psychiatric, or systemic main cause. To diagnose FCD, there must be a significant disparity between self-reported cognitive symptomatology and observed or reported cognitive performance. When symptom severity is compared to neuropsychological testing or ordinary “real world” cognitive abilities, this inherent contradiction in symptoms becomes apparent. Above-average success on list learning tasks, for example, or on-going work in a skilled job without trouble, are incompatible with a self-report of complete inability to recall any new material in a short period of time.

The cause is unknown; however it is believed to be linked to underlying psychological problems. FCD is a relatively new clinical category that falls under an ever-expanding spectrum of cognitive disorders that range in severity from Mild Cognitive Impairment (MCI) to severe dementia. Significant disagreement between subjective and objectively observed cognitive performance, a greater severity of self-reported symptomatology, and resistance to reassurance that observed cognitive functioning is intact separate FCD from SCD. Currently, diagnosing FCD requires an expert opinion from a cognitive disorders specialist. There is on-going dispute over the best diagnosis criteria and therapeutic tech-

niques for FCD, and longitudinal studies of prognosis and rate of diagnostic change are missing.

The risks of misdiagnosis of early-stage neurodegeneration or multifactorial cognitive impairment are two topics of particular attention. The prevalence of FCD is currently being researched, and it will differ depending on the demographic analysed. A third of patients aged 60 or less who attended a tertiary referral cognitive disorders clinic had a functional diagnosis, according to a study. SCD is quite frequent, especially among the elderly. According to a German LIFE study, 53% of persons aged 40-79 years have subjective memory difficulties, whereas 11.1% of adults over 45 years in the United States have cognitive concerns. Variable nomenclature and definitions have plagued the SCD literature, making it impossible to estimate true frequency and incidence.

Some people may experience cognitive symptomatology as a result of a combination of etiological factors, similar to the concept of “functional overlay” seen in systematic and functional neurological disorders, in which core symptoms caused by an underlying structural disease process are complicated by additional functional features. It’s fairly rare to have a dual diagnosis of epilepsy and psychogenic nonepileptic seizures, and those with chronic illnesses like multiple sclerosis may have additional functional symptoms that are misdiagnosed. There are considerable obstacles in verifying a diagnosis in the context of cognitive symptoms, as well as the possibility of diagnostic evolution through time and overlapping illnesses.

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Rapid access to extensive neuropsychological testing (including effort testing) in a clinical context, as well as the use of CSF biomarkers and advanced imaging techniques such as amyloid PET, is sometimes limited. As a result, determining whether cognitive symptoms are caused by neurodegenerative or functional or alternative reasons typically relies on a combination of self-report, collateral history, and professional judgement. Memory complaints are prevalent among healthy older persons, and psychiatric symptoms may be an early sign of neurodegenerative illness, further complicating the issue.

When considering a diagnosis of FCD, a thorough examination of symptom severity, evaluation of discrepancies between self-reported and observed cognitive ability, consideration of potential contributing psychological factors, beliefs, or mental health symptoms, and search for evidence of neurodegenerative, toxic, or metabolic causes of cognitive decline are all required. Furthermore, a correct diagnosis of FCD does not guarantee that an individual will not develop neurodegeneration in the future.

Functional Cognitive Disorder

In this case study of patients with FCD and neurodegenerative MCI, we discovered that those with FCD reported the same level of cognitive symptomatology as those with MCI. Total PRMQ T scores in the FCD group were 33.3 and 35.1 in the MCI group, respectively, about 1.6 SD lower than the normative population mean. This finding is consistent with prior research on functional neurological disorders, which found that individuals with functional symptoms self-rate their suffering as bad as or worse than those with organic neurological disease. On the MoCA, there was no difference in performance between FCD and MCI, since both groups had significantly lower scores than the controls.

When compared to the controls and the MCI group, the FCD group performed worse on the ToPF. The ToPF is intended to assess premorbid cognitive ability, however it is known to be susceptible to the effects of neurodegeneration and nonorganic underperformance. Martin et al. investigated the relationship between performance validity test results and the ToPF, finding that individuals who failed validity tests had a lower ToPF score than demographically predicted. On neuropsychological testing, people with FCD

frequently display an incorrect pattern of performance, and it's probable that the ToPF results in this FCD group don't adequately reflect their genuine premorbid baseline.

The lack of "gold standard" diagnostic criteria for FCD is one of the study's limitations. Diagnostic criteria are continually being refined, and clinician judgement is still the most important factor in making a diagnosis. Because the ReMemBr Group cognitive clinic receives difficult referrals from general and secondary care, there is likely to be referral bias toward less clear diagnostic circumstances, which may have influenced the participants in this study. A recurrent issue in cognitive research is that research participants have higher levels of education and socioeconomic position than the general population.

As a result, we wouldn't consider our study group to be genuinely representative of the local community, and considerable cultural variations could have an impact on how FCD presents and is diagnosed. To learn more about FCD, we need to do larger research with more diverse populations in different parts of the world. Traditionally, cognitive illnesses have been assumed to be a one-way, binary process. They might be present or absent, develop in severity with time, and do not remit, with the exception of rare, curable causes of cognitive decline. Large studies of populations with MCI show that a considerable percentage of affected individuals revert to normal or near-normal cognition over time, contradicting this fairly linear view of cognition. Those who progress have a higher chance of MCI or dementia in the future than those who have never been diagnosed with MCI, therefore they may switch diagnostic categories multiple times along their cognitive journey. Individuals with SCD are also extremely diverse.

People with SCD were separated into three groups in the SCIENCE cohort study: those with preclinical Alzheimer's disease, those with very mild psychiatric symptoms, and those with neither. Preclinical Alzheimer's disease was linked to older age and Apolipoprotein E4 status. Those with psychiatric symptoms had more cognitive symptomatology than those with preclinical Alzheimer's disease. Other research into the long-term prognosis of SCD has identified a number of different clinical trajectories, including symptom remission.