

Comorbidity of Primary Torsion Dystonia with Psychiatric Disorders

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Abstract

The authors investigate and describe comorbidity of PTD with psychiatric diseases reviewing the medical records of all hospitalized patients, diagnosed with PTD for a period of four years. 75 patients with confirmed diagnose of PTD gave their informed consent and were clinically investigated including an examination by a specialist psychiatrist. The majority of the patients with the psychiatric comorbidity experience depressive symptoms, but more importantly a high percent of the patients (4%) suffer from schizophrenia. Therefore one can assume that there is an abnormality in the common cerebellar pathways in the brain in PTD and schizophrenia.

Keywords: Schizophrenia; Dystonia; Cerebellum

Introduction

Primary torsion dystonia (PTD) is a rare neurological disease, characterized by sustained or intermittent involuntary muscle contractions, causing repetitive twisting movements and postures, which could affect any body region. PTD is manifested as an isolated movement disorder without clinical evidence of cognitive, sensory or cerebellar deficit or any other symptoms except tremor [1,2]. As it is with most movement disorders, dystonic symptoms intensify with overstraining and emotional stress [2]. Mental influence over the clinically expression of the condition let us to the hypothesis that there might be a deeper connection between a "pure" movement disorder such as PTD and some psychiatric pathology, moreover that some interference between psychiatric diseases and movement disorders, such as dystonia, were discussed even at the dawn of modern psychiatry by Kahlbaum in the original description of catatonia [3] and in a substantial minority of neuroleptic naïve schizophrenia patients, as described quite specifically far ago by Kraepelin [4].

Aim

The aim of the current study is to investigate and describe comorbidity of PTD with psychiatric diseases and eventually to be established some common pathological mechanisms.

Materials and Methods

Period prevalence of PTD in the Bulgarian population is established to be about 7.5/1 000 000. Annually, about 1/3 of patients with PTD are hospitalized in one University hospital with a Movement disorder department in the capital [5]. A medical records research of all the hospitalized patients, diagnosed with PTD for a period of four years was done. For the period mentioned a total of 103 PTD patients were hospitalized, but in 19 cases diagnose of PTD was uncertain and these patients are excluded from the analysis. 75 from the rest 84 patients with confirmed diagnose of PTD gave their informed consent and were clinically investigated. Additionally a detailed history of psychiatric disorders available was investigated and patients were examined by a specialist psychiatrist.

Results

All the clinically verified 75 PTD patients are at the age from 23 to 82 years old, average 52.8 (SD=16.0) years old. 52 (69.3%) of them are females at an average age of 53.7 (SD=16.5), and 23 (30.7%) are males, at an average age of 50.6 (SD=15.2). A total of 17 (22.7%) patients at an average age of 52.3 (SD=13.1) are diagnosed to suffer from a psychiatric

disorder. 13 (76.5%) of them are females, at an average age of 50.5 (SD=3.5), and 4 (23.5%) of them are males, at an average age of 53.3 (SD=15.4). In all 17 patients any drug provocation of psychiatric or neurological disorder is excluded. Depressive symptoms are observed most frequently- in a total of 11 (14.6%) patients, 7 (9.3%) of them suffer from major depression, and 4 of them (5.3%) suffer from mixed anxiety and depressive disorder. Another three patients (4%) suffer also from anxiety disorders. One of them was diagnosed with a social phobia, one of them- with neurasthenia, and the last - with adjustment reaction (Table 1). One of them experience also alcohol abuse. Three patients (4%) were diagnosed with schizophrenia, but none of them has an exacerbation. One of them experience also alcohol abuse.

Discussion

The examined sample may looks a little bit small, but we must not forget that PTD is very rare disease worldwide (including Bulgaria) and we have examined around 1/3 of the patients that are hospitalized yearly for the country [5]. That is why we think that we can accept our findings as representative. Our results suggest that majority of the patients with the psychiatric comorbidity experience depressive

Psychiatric comorbidities	N	% patients PTD	% total psychiatric pathology
Major depression	7	9,3	41,2
Mixed anxiety and depressive disorder	4	5,3	23,5
Social phobia	1	1,3	5,9
Schizophrenia	3	4,0	21,4
Adjustment reaction	1	1,3	5,9
Neurasthenia	1	1,3	5,9
Total psychiatric pathology	17	22,7	100,0
Without psychiatric disorder	58	78,7	
Total	75	100,0	

Table 1: Psychiatric comorbidity.

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symptoms. These symptoms are developed after the onset of the PTD, which can be explained with the chronic and progressive course of the disease, high level of disability, and accompanying, usually severe pain, so we can assume these symptoms as “reactive” to the PTD. Probably we can speculate that this is supported by the fact that anxiety disorders also are related to the symptomatology and social maladaptation-adjustment disorder, social phobia in patient that has difficulties in locomotion connected with torticollis and neurasthenia with headache and irritability, anxiety and tearfulness due to worsened social environment. We could not observe significantly higher difference in the prevalence for depression and anxiety in the sample compared with the general population (10-20%), so we can hardly say that dystonia is provoking those symptoms. It is quite obvious that there is a high percent of patient suffering from schizophrenia (4%), having in mind that in this sample females are more than males and the average age is around 50 years of age (i.e. before the well-known second peak of the prevalence). And this percent is significantly higher than evaluated for the general population (0.5-1%). Besides nowadays there are at least 10 studies that have investigated the presence of spontaneous dyskinesia in schizophrenic neuroleptic naive individuals, seven of which observed some spontaneous dyskinesia in these patients, including tremor, choreoathetosis, dystonia, and akathisia. Thus, it appears that spontaneous dyskinesia may be associated with the diagnosis of schizophrenia and may not be simply an artefact of medication, although further study is required given the methodological constraints of these studies and the inconsistent results [6].

The pathophysiology of PTD is connected with the disturbances in the system basal ganglia-thalamus-cortex, but still not understood completely. There is no discovered significant structural brain anomaly to this moment, neither inherited metabolic disturbances [1]. There are findings of different changes of the normal dopamine level - increase but also decrease in the patient with PTD. Neychev et al. succeed to induce clinical symptoms dystonia by pharmacological intervention in the cerebellar cortex using mice in the experimental model. In the same time they observed significant reduction in the release of the strial dopamine. They argue that the clinical manifestation of the dystonic movements is controlled not only by the basal ganglia and also by the cerebellum [7]. This suggested that dysfunction in dystonia may originate from other regions, particularly the cerebellum and that dystonia is a network disorder that involves multiple brain regions [8]. Many experts following Bleuler views are thinking that the core features of schizophrenia are the abnormalities in cognitive fluency or volition, often referred to as “negative symptoms”, and that psychotic (“positive”) symptoms are secondary. This can be explained by abnormalities in the “executive” portions of the brain, the frontal lobes and also possible impairment in excitatory/ inhibitory tone in the cerebellum regulating cognition. More recent papers hypothesized that the symptoms in schizophrenia may be tied to a pathophysiology involving misconnections within cortico-cerebellar-thalamic-cortical circuits, resulting in “cognitive dysmetria,” or incoordination of mental activity [9]. Independently, Schmahmann proposed a “dysmetria of thought” model, in which various neuropsychiatric conditions, may reflect abnormal modulation of cognitive and affective processes by the cerebellum [10]. Some lesion studies support the role of cerebellum in associative learning and symptoms close to those in the psychosis such as mutism. Cerebellar lesions do not, however, normally produce psychotic symptoms. Many authors are reporting its abnormal size in schizophrenia. Picard et al. recently conducted a review of the evidence for cerebellar abnormalities in schizophrenia from multiple perspectives as symptoms, neurological signs, eye movements, non-

declarative learning, and cognition [1]. Therefore one can assume that there is an abnormality in the common cerebellar pathways in the brain in PTD and schizophrenia, because of the fact that these two disorders are more common than expected together and not only by chance alone, but because some commonalities in their biological mechanisms. The observed dopaminergic dysregulation in both diseases also supports this hypothesis, despite the lack of absolute evidences for the classical theories for disturbances in the dopaminergic neurotransmission in schizophrenia and dystonia.

Of course, there is an obvious need from more extensive research in this area on bigger sample to prove such hypothesis.

Conclusion

The comorbidity between PTD and psychiatric disorders cannot be denied. There is higher than expected prevalence of schizophrenia in our sample, which can lead to the conclusion that there is a common pathological substrate. Further investigation on larger sample is needed to prove or deny a proposed hypothesis.

Limitations of the Study

The main limitation of the current study is connected with the small sample of investigated patients, due to the rarity of the PTD as a disease. Despite of certain limitations, this study was meant to draw attention to some possible interference between neurologic and psychiatric disorders, which have nothing in common at first sight.

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