

Physical deformity associated with psychotic symptoms in two patients with schizophrenia



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SUMMARY

Movement disorders and related physical deformities may sometimes be observed in patients with chronic psychotic disorders. In this article, we present the case of two patients with physical deformities associated with chronic psychotic disorders. In the first case, the patient had never sought psychiatric care despite her long-standing psychiatric disorder. The patient, diagnosed with disorganized schizophrenia, developed cervical kyphosis, due to her constant neck flexion posture. The other patient had been undergoing treatment for a long period under the diagnosis of paranoid schizophrenia. In the latter case, peroneal nerve injury and dropped foot had developed due to a constant crossing of the legs. Physical deformity may also develop as a result of physical inactivity-hypokinesia, a fixed body posture, and postural disorders in chronic psychotic patients. Due attention should be given to physical symptoms in this group of patients and physical deformities should be treated alongside the patient's psychotic symptoms.

Key Words: schizophrenia, posture, deformity, motor activity

INTRODUCTION

Knowledge about the development of physical deformity in chronic psychotic disorders is limited (Hamera et al. 2010). Apart from the side-effects of treatment for schizophrenia and other psychotic disorders, movement disorders may also occur independently (Chen et al. 2000, Pappa and Dazzan 2008). Among the movement disorders which emerge as a clear diagnostic indicator of the illness, hypokinetic movements are the most frequently encountered (Pappa and Dazzan 2008).

Fixed body posture, as a hypokinetic movement, leads to postural disorders (Dickson, 1988). In healthy people, physiological feedback systems alert the nervous system to change the body posture to prevent tissue damage related to maintaining a fixed position (Bear et al. 2001). In patients with chronic psychotic disorders however, it was reported that joint contractures may develop as a result of long-term catatonic

manifestations (Mashimo et al. 1995). Muscular atrophy may develop due to extreme flexion of the wrist, and joint deformities may develop due to degeneration of the joints and damage to connective tissues (Frykman et al. 1983). There is, however, a lack of data on the development of physical deformities in this patient group.

This article presents the cases of two patients diagnosed with schizophrenia and afflicted with severe physical deformities resulting from fixed posture and immobility developing over the course of their illness, with the attention of drawing attention to the risk of physical deformity emerging in connection with psychotic disorders.

Patient 1

A 39 year-old, widowed female, housewife and secondary-school graduate living with her children. She was admitted by

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her daughter to our emergency psychiatric unit and throughout the previous months had been manifesting behaviors such as increasing social withdrawal, sitting in the dark, not cutting her nails, not bathing, and soliloquy month. Her family revealed that her initial symptoms such as social withdrawal, lack of communication with other family members and always sitting in the dark had begun five years previously and additional behaviors such as allowing garbage to accumulate, not bathing, not cutting her nails and soliloquy had been recently added to these pre-existing symptoms. The patient had not previously been admitted to a psychiatric clinic. She would sometimes cook, however, due to mixing salt and sugar together in the food, her meals were generally inedible.

During her psychiatric examination, she was able to communicate and her orientation was intact. She looked older than she actually was. Her self-care was markedly reduced. She was respectful to the interviewer, but unwilling to engage; her emotions were blunt and her associations were poor. She gave only partial responses to the questions. Her thoughts consisted of persecutory delusions and irritability; she experienced auditory and visual hallucinations, but did not have psychomotor agitation or emotional overreactions. She displayed lack of judgment, impaired abstract thinking, insufficient behavioral planning and an inadequate level of knowledge. She had no capacity for introspection. During the interview, she kept her neck bent forward and her daughter revealed that she had been in this posture for two years. Her total PANSS (Positive and Negative Symptoms Scale) score was 59. The neurological examination was unremarkable. Her medical history did not reveal any previous diseases. She had no history of smoking, or use of alcohol, nor any other psychoactive substance. Her family history was unremarkable except for a suicide attempt by her brother.

Due to the bending position of the neck, cervical X-rays were taken and a physical therapy and rehabilitation consultation were requested. The evaluations revealed cervical kyphosis (severe) and degenerative cervical changes (C5-6, C6-7 level), which were considered to be a result of the continuous flexion posture of the neck, rather than any other organic reason.

The patient was diagnosed with disorganized schizophrenia according to the DSM IV-TR diagnostic criteria (American Psychiatric Association, 2000). Her treatment regime was planned with haloperidol: 20 mg / day and biperiden: 4 mg / day. An anti-inflammatory-analgesic was added to the treatment. She was referred for physiotherapy. Following a course of physiotherapy, it was reported that the degenerative changes were severe and permanent, and that no treatment, other than the administration of anti-inflammatory drugs, was possible. As the patient failed to show an adequate response to the treatment applied, she was treated with 500mg/day of clozapine .

Patient 2

A 32 year-old, single, unemployed, high-school graduate female living with her parents. She was hospitalized after manifesting behaviors such as talking to herself, refusal to take her medication, aggression and suspicions of a self-harming nature such as the belief that someone had put chemical substances in her food, leading to a refusal to eat and throwing her food into the garbage.

The patient's symptoms had first appeared 13 years previously, she was diagnosed with schizophrenia and hospitalized seven times during this period, and, according to our hospital's medical records, she had been monitored through the outpatient clinic. She had been intermittently using several antipsychotic treatments such as haloperidol: 20 mg / day, clozapine: 400 mg / day, paliperidone: 9 mg/day, amisulpride: 800mg/day and risperidone: 6mg/day.

During her psychiatric evaluation she was alert, able to communicate, and her orientation was intact. Her appearance was in line with her age, her self-care was appropriate with her socioeconomic status and she maintained eye-contact although she was unwilling to participate in the interview. She sat in a cross-legged position, answering the questions, sometimes by mumbling and sometimes in a normal tone of voice. When she was asked for the reason for her mumbling, she claimed that she was speaking with the lights. She displayed reduced speech, limited emotion and her associations tended to be loose. She described hallucinations of auditory, visual and sensory duality (synesthesia). She had no capacity for introspection. Her total PANSS score was 67. It was observed that she had difficulties walking on her toes. She had no previous diseases in her medical history.

The patient was assessed by a neurologist in connection to her difficulty in walking and, as a result of this evaluation, it was determined that the cranial area was intact, the upper and lower deep tendon reflexes were normo-active, the bilateral top and lower proximal muscle strength was complete and there was a +4 / 5 bilateral foot dorsiflexion. An EEG, EMG and MRI were performed, as the patient was unable to step on her toes and she had bilateral foot drop. The patient's EEG and MRI results were within normal limits. According to the EMG evaluation, the combined muscle action potentials of the bilateral peroneal nerve could not be obtained from the extensor digitorum brevis muscles, however the tibialis anterior muscles were normal. The motor unit potential of other nerves, bilaterally examined in the lower and upper-right extremities, and the sensory functions of the examined nerves, were normal. In the needle EMG, deep bilateral nerve innervations and advanced thinning in the muscles with long-term, polyphasic motor unit potential were observed. The other muscle-thinning was within normal parameters. This data was consistent with a diagnosis of a subacute (process

improvement) lesion, which had resulted in severe (complete) axonal damage in the deep peroneal nerves.

In the differential diagnosis, previous treatments undergone by the patient together with other possible factors were considered. At the second evaluation of the patient, involving findings from her examination and the test results, it was concluded that her clinical condition was due to the patient sitting constantly in a bent-knee or cross-legged position. As the patient had been diagnosed with paranoid schizophrenia according to the DSM IV-TR diagnostic criteria (American Psychiatric Association, 2000), a treatment regimen of paliperidone: 9 mg / day was prescribed at our clinic. The patient and her family were informed about the causes of the walking difficulties. Results of the control EMG evaluation, three months after informing the patient and applying psychiatric treatments, a partial healing of the peroneal nerves was observed.

DISCUSSION

There have been case reports of patients with psychotic symptoms, having deformities due to excessive flexion of the wrist over extended periods (Frykman et al. 1983, Srivastava et al. 2008). Mashimo et. al (1995) reported a catatonic schizophrenic patient, who had developed joint contractures due to excessive standing in the same position. In a study conducted by Hamera et al. (2010), 30% of patients with severe mental disorders, evaluated prior to exercise and weight loss programs, had musculoskeletal problems. Similarly, joint deformities have been reported in patients with late-stage Alzheimer's disease, due to immobility (Souren et al. 1995). Apart from these case reports, there is insufficient data available on the development of physical deformity in patients with schizophrenia, who, according to clinical indicators and findings, may be considered a high-risk group.

Postural disorders, and physical deformities associated with postural disorders, are well known in the general population (Dickson, 1988). In particular, this problem is a significant issue for some occupational groups (Mahbup et al. 2006). In both the cases presented in this report, the postural disorder and related physical deformity was a sign of an underlying illness. In schizophrenic patients, social withdrawal, whether or not they are on medication, as well as adopting a fixed posture, may be accepted as clear clinical indicators of the illness (Ozturk and Ulusahin 2008). In the light of these facts, it may be assumed that at least some schizophrenic patients are at risk of developing physical deformities related to the presence of postural disorders. However, it is noteworthy that there is a lack of studies on this issue.

The first patient had been in a fixed, forward-bending neck position, almost since the beginning of the illness and the sec-

ond patient always sat cross-legged. The consultation notes for both patients stated that the deformities had developed as a result of their postural disorders. Although patient 1 had remained untreated, patient 2 had been on long-term antipsychotic treatment, either hospitalized or as an outpatient. The physical deformity of the treated patient was monitored during the treatment period. It is significant to note that this postural disorder developed while the patient was under regular clinical observation.

With regard to the development of the deformities themselves, it was confirmed, both by the medical records and the information given by the relatives of the patients, that the postural disorders in the patients occurred subsequent to the onset of symptoms of the illness. The differential diagnosis of both patients call to mind Friedreich's ataxia, in which schizophrenia-like psychotic symptoms may occasionally observed. This disease usually appears in the patient prior to the age of 25, and generally, multi-system related disorders such as skeletal system abnormalities and abnormal signal transmission of the heart are observed, while the dominant indicators are ataxia, difficulties in walking, dysarthria, proprioceptive sensory loss, loss of deep tendon reflexes and signs of upper motor neuron disorders (Gunduz and Apaydin 2007). Shepherd (1955) reported on a family, in which separate individuals had peroneal nerve injury, schizophrenia, and Friedreich's ataxia. Kyphoscoliosis may also be observed in this disease (Gunduz and Apaydin 2007). Neither of the two patients, however, met the diagnostic criteria for Friedreich's ataxia.

Impaired neurological symptoms and physical movement disorders have been shown to occur at much higher frequencies in schizophrenic patients when compared with the healthy control subjects (Chen et al. 2000). It has been determined that dyskinetic movements may occur and frequent instability is seen in schizophrenic patients even if no medication is used (Gupta et al. 1995, Pappa and Dazzan 2008). In addition, it is known that structural and functional impairment in skeletal muscles is not connected to the use of drugs (Borg et al. 1987). Walther et al. (2009) reported in their study investigating motor activity differences between paranoid and catatonic schizophrenia subtypes, that the incidence of motor activity disorders in catatonic schizophrenic patients was higher. In the diagnostic view, our first patient was diagnosed with disorganized schizophrenia while the second patient was diagnosed with paranoid schizophrenia. Together with this study, the results of the work of Walther et al. (2009) are significant, in that they emphasise motor activity problems in patients with schizophrenia.

The deformities observed in both cases arose as a result of fixed body posture. In physiological conditions, excessive maintenance of a single posture over a long-period of time causes tissue damage. In healthy people, tissue damage stimulates the pain signals through nociceptors and ensures a change of

posture prevention of tissue damage (Bear et al. 2001). These receptors possess no characteristics of adaptation.

The development of physical deformities in patients with schizophrenic disorders may result from pain-related sensory perception or sensory- system disorders as well as catatonic hypokinetic movement disorder. Impairment of proprioception-like sensory perception disorders are reported in these patients (Korn 2000). Decreased response to painful stimulants has been reported in schizophrenic patients (Singh et al. 2006, Bonnot et al. 2009). This is also emphasized in Kraepelin and Bleuler's reports on defining the illness (Bonnot et al. 2009). Merskey et al (1962) reported that patients with schizophrenia had decreased perception of pressure pain.

Health care for chronic psychiatric patients is more accessible today compared with in the past. However, it is significant that both of the patients developed physical deformities as a result of their illnesses. There have been very few reports on the onset of physical deformities in patients with schizophrenia and this may lead the physicians monitoring the patient to underestimate the possibility of physical deformities developing.

Consequently, motor activity problems in schizophrenic patients may arise regardless of the treatment and hypokinetic movements may be observed in most of the schizophrenia subtypes (Chen et al. 2000, Gupta et al. 1995, Pappa and Dazzan 2008). A decrease in daily activities, deterioration in the quality of life and reduced social functionality related to postural disorders have been observed in patients without psychiatric disorders (Takahashi et al. 2005). The development of physical deformities results in more severe deterioration of quality of life in patients with schizophrenia who already have reduced social functionality. It must be kept in mind that physical deformities may arise in patients with physical inactivity-hypokinesia, and their physical examination and evaluation of symptoms should be performed with caution. Structured exercise programs should be recommended for both inpatients and outpatients (Hamera et al. 2010). Psychotic patients under the risk of physical deformity should be referred to the relevant specialist. Rehabilitation programs should not only treat psychiatric complaints, but also incorporate physical therapies to prevent deformities which may arise due to immobility and fixed posture.

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