Movement disorders: what the psychiatrist needs to know

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We move by voluntary (intentional), semi-voluntary (eg. stretching, compulsive touching), involuntary (eg. myoclonus, tremor) or automatic movements (eg. walking, speaking). Movement disorders are neurological syndromes in which there is either an excess of movement (hyperkinesia, dyskinesia or abnormal involuntary movement) or paucity of voluntary and automatic movements (hypokinesia, bradykinesia, akinesia) unrelated to weakness or spasticity. They commonly occur due to basal ganglia dysfunction and are often found in neuropsychiatric disease. Movement disorders can present as an initial manifestation of psychiatric disease (eg. chorea in Huntington's disease), or they can occur as a consequence of treatment of psychiatric disorder (eg, tardive dyskinesia) or they can occur as a somatoform symptom (eg. psychogenic). The psychiatrist should have knowledge of movement disorders to aid in the diagnosis and management of neuropsychiatric disease and in the management of side effects of psychotropic medication.

Initial assessment

To recognise a movement disorder it is best to see the patient in person or review the abnormal movement on videotape. Some basic principles should be kept in mind when first approaching a patient with a movement disorder. Take time to observe the patient, describe what you see and do not label the abnormal movement immediately, classify the movement as hyperkinesia or hypokinesia, give the movement a name (eg. tremor, chorea), diagnose a specific disease after naming the movement and finally think about treatment. Patients can have either a hyperkinetic (tremor, chorea, dystonia, tics, myoclonus, akathesia, ataxia, ballism) movement disorder or hypokinetic movement disorder (bradykinesia, akinesia, freezing, apraxia, catatonia, stiff muscles, and rigidity).

For example when a patient develops a tremor on valproate, lithium or a neuroleptic the psychiatrist should firstly observe the patient at rest (best seen when the arms are hanging down while the patient walks). If a rest tremor is present it is likely the neuroleptic is responsible or perhaps the patient is developing an alternative form of parkinsonism.

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Now look for rigidity at the wrist, elbow, trunk or neck and hypokinesia (decreased amplitude of movement) and bradykinesia (slowness of movement), stooped posture and poor postural stability to confirm parkinsonism. If a postural tremor is present (tremor present with the patients' arms outstretched), without rest tremor, then lithium, valproate or essential tremor (ET) may be responsible. ET and medication-induced tremor may both be worsened by action (finger-nose-finger manoeuvre). Thus the examination alone may not distinguish ET from medication-induced tremor. However, in ET there is usually a family history of tremor and the tremor is often eased by alcohol or by holding objects with two hands.

Tardive dyskinesia syndromes

The tardive dyskinesia syndromes are abnormal movements that occur within six months of exposure to a dopamine blocking agent and persist for at least one month after cessation of the offending drug. They include classical tardive dyskinesia (repetitive rhythmical oral-buccal-lingual chewing movements, and occasionally rhythmical movements of the hands, feet, trunk, or diaphragm), tardive dystonia, (retrocollis, opisthotonus), tardive akathisia (inner restlessness), and tardive parkinsonism' (symmetrical bradykinesia, rigidity, postural instability, occasional rest tremor). Symmetrical bradykinesia and rigidity and lack of response to L-dopa help to distinguish tardive parkinsonism from idiopathic Parkinson's disease (IPD). IPD is usually asymmetrical and responds well to L-dopa. Rest tremor is less common in tardive parkinsonism compared to IPD.

The tardive dyskinesia syndromes are the commonest movement disorder seen in psychiatry with a wide range of prevalence reported in the literature. The prevalence on the Island of Curacao, with only one psychiatric facility, is 39.7% for classical tardive dyskinesia, 36.1% for parkinsonism, 13.4% for tardive dystonia, and 9.3% for akathisia.[2,3] They can occur as a side effect of commonly used antiemetics (eg. metoclopramide). Atypical neuroleptics rarely cause tardive dyskinesia, which is more common in the elderly. Tardive akathesia can be extremely uncomfortable. The patient may complain of an oral or vaginal burning sensation and may rock or moan constantly. Tardive dystonia can mimic idiopathic focal or segmental dystonia. However, it causes a characteristic retrocollis (arching of head backwards) and opisthotonic posturing rather than rotation or lateral of the neck flexion seen in idiopathic torsion dystonia. Moreover the pattern of internal rotation of the shoulders, elbow extension and wrist flexion is characteristic of tardive dystonia.

The tardive dyskinesia syndromes tend to persist and can

be permanent. Hence the decision to start neuroleptics must be made cautiously and if neuroleptics are required it is preferable to use atypical neuroleptics. The sooner the offending drug is withdrawn the more likely the syndrome will fade with time. One third of classical tardive dyskinesia patients get remission when off neuroleptics for two years, remission rates are lower for tardive dystonia and tardive akathisia.[4] The neuroleptic should be tapered slowly as sudden cessation of the medication can aggravate the movement disorder. If possible the physician should wait for spontaneous remission unless the symptoms are disabling.

If the symptoms require treatment use dopamine depleting agents (reserpine or tetrabenazine) first. These agents result in improvement of most classical tardive dyskinesia and tardive akathisia patients, but improvement is seen in only 50% of patients with tardive dystonia. They act probably by decreasing the dopaminergic synaptic activity. Tetrabenazine is better tolerated and has a quicker onset. Neuroleptics can suppress some tardive syndromes. Antimuscarinics are effective in tardive dystonia, but they may worsen classical tardive dyskinesia.[1] Benzodiazepines and botulinum toxin can be useful adjuncts in combination with dopamine depleters or anticholinergics. ECT can be of help for tardive akathisia.[1] Increase dosage of typical neuroleptics can be used to suppress the dyskinesias when the above approaches fail. Thalamotomy, pallidotomy and pallidal deep brain stimulation can be effective for tardive dystonia and classical tardive dyskinesia.

Hereditary disorders

Many hereditary disorders can be associated with psychiatric symptoms and movement disorders. Some, such as Wilson's disease (WD), must be diagnosed early as it is curable if treated early. WD is a rare autosomal recessive disorder of copper accumulation due to mutations in the copper-transporting P-type ATPase gene on 13q14.3.[5] WD can present with hepatic (40%), neurologic (40%) or psychiatric disease (20%). It presents in so many guises that any patient with a movement disorder under the age of 50 years should be considered to possibly have WD. The movement disorders include tremor, orobuccal dystonia (risus sardonicus), chorea and parkinsonism. Teenagers can present with insidious deterioration of school performance, personality or mood change.[6] Psychosis is usually a late complication. A low ceruloplasmin level in the serum and the presence of Kayser-Fleischer (KF) ring on slit-lamp examination aid in the diagnosis of WD. Huntington's disease (HD) can be associated with depression, obsessive compulsive disorder, psychosis, aggression or apathy in addition to the well-known chorea or parkinsonism when young onset. Genetic counselling, psychological support, dietary advice to maintain weight and antidepressants are the methods of treatment. Tetrabenazine or neuroleptics can ease the chorea and behavioural problems.

Tourette's syndrome

Tourette's syndrome (TS) is characterised by motor and vocal tics commencing during childhood. For a definite diagnosis of TS, the following criteria have to be present as suggested by Tourette Syndrome Classification Study Group in 1993:[7]

- Multiple motor and one or more vocal tics have to be present at some time during the illness
- Tics must occur many times a day, nearly every day, or intermittently throughout a period of more than one year
- Anatomic location, number, frequency, type, complexity, or severity of tic must change over time
- Onset must be before age 21
- Involuntary movements and noises cannot be explained by other medical conditions
- Motor and/or vocal tics must be witnessed by a reliable examiner or be recorded by videotape at some stage during the illness.

Tics are often preceded by a build of tension and can be suppressed for a period of time. This suppression is associated with increased 'tension' that is relieved by the tic. Stress, fatigue, idleness, exposure to heat and drugs like steroids, dopaminergic drugs, can aggravate tics. They usually diminish with mental or physical activities or with alcohol consumption. TS is associated with Attention Deficit/Hyperactivity Disorder (impulsivity, inattention, restlessness, fidgeting, poor concentration, poor school or social work performance and learning impairment) in 50% and obsessive-compulsive disorder (repetitive, stereotyped, involuntary, senseless thoughts or behaviour that intrude into the patient's consciousness or actions) in 30%-50%. Genetic factors have a major role in the aetiology of TS, however environment influences risk, severity and course of the disorder. Education of the patient and family is often sufficient to allay anxiety about the disorder. Behavioural and cognitive treatment approaches can be tried in the treatment of Tourette's syndrome. Medication may not be required if the tics do not interfere with the activities of daily living. Dopamine receptor blockers, noradrenergic drugs (eg. clonidine) and dopamine depletors (eg. tetrabenazine) can be of benefit.[8] Functional neurosurgery, cingulotomy, limbic leucotomy or thalamic deep brain stimulation, are still experimental.

CNS infections and endocrine disorders

The psychiatrist needs to be aware of some CNS infections that can present with neuropsychiatric symptoms and movement disorders. Creutzfeldt-Jacob Disease, a prion disease, can present with diffuse myoclonic jerks, ataxia, a rapidly progressive dementia, and abnormalities of vision. Agitation, delusions and hallucinations are present in the early phase of the disease. Myoclonic jerks are best demonstrated by flicking the outstretched hands and fingers of the patient (ie. stimulus sensitive myoclonus). Repeated EEGs are often required to show the characteristic grossly disorganised background activity interrupted by repetitive 'pseudoperiodic' discharges consisting of large, sharp waves about once per second.

Some endocrine disorders can have abnormal movement and neuropsychiatric manifestations. Hashimoto's encephalopathy, an autoimmune encephalopathy, can present with myoclonus and dementia or encephalopathy in addition to seizures, alternating hemiparesis, and high serum titres of antithyroid antibodies. Patients can be euthyroid, hypothyroid or hyperthyroid. The CSF protein is usually raised. The diagnosis must be considered in a patient with

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* trade mark.
† Meta-analysis of randomised, placebo-controlled trials.
§ Healthy volunteer studies.
¶ Data from Efexor Tablets.

seizures, dementia and encephalopathy as there can be a dramatic response to steroids or plasma exchange. Finally, thyrotoxicosis can present with tremor, chorea, mental confusion, seizures, manic or depressive attacks and delusions.[9]

Neurodegenerative disorders

Neurodegenerative disorders, such as Alzheimer's disease, multisystem atrophy (striatonigral degeneration, olivopontocerebellar degeneration, Shy-Drager syndrome), the tauopathies (frontotemporal dementia, progressive supranuclear palsy, corticobasal degeneration), or diffuse Lewy body disease (DLBD), are commonly associated with parkinsonism, myoclonus, occasionally dystonia and neuropsychiatric symptoms. For example, DLBD is characterised by parkinsonism, fluctuating level of consciousness, cognitive impairment (short term memory and visuospatial impairment), florid visual hallucinations, and myoclonus. It is important to avoid the typical neuroleptics because of heightened sensitivity to D2 blockers and atypical neuroleptics can effectively resolve the visual hallucinations. Visual hallucinations can precede the slowness, stiffness, rest tremor, stooped posture, and postural imbalance by years, thus making the diagnosis difficult. Improvement in cognition and behaviour has been shown with the use of cholinesterase inhibitors like rivastigmine, donepezil and galantamine.[10]

Psychogenic movement disorders

Psychiatrists need to be aware of the array of strange movements that can occur as part of psychogenic movement disorders. They present as psychogenic tremor, psychogenic dystonia, psychogenic myoclonus, psychogenic parkinsonism or psychogenic gait ataxia. Psychogenic disease can by very disabling and requires a great deal of attention and effort by the both the psychiatrist and neurologist. The diagnosis should be made by a neurologist experienced and familiar with the varied and often bizarre nature of organic movement disorders.

Historical clues suggesting a psychogenic movement disorder include an abrupt onset, history of minor trauma, a static course, paroxysmal worsening, spontaneous remissions, multiple somatisations and associated other psychiatric illnesses. Clinical clues suggesting a psychogenic movement disorder include movements incongruous with organic movement disorders (bizarre movement disorders), paroxysmal attacks, variability over time, improvement with distraction, and increases with attention. The movement disorder may temporarily correct itself with voluntary movement on the side opposite to the affected one. Other clinical clues are the presence of extreme slowness, false weakness, false sensory disturbances, atypical pain and abnormal affect. The prognosis is quite varied and can be poor especially in those with longstanding symptoms. Management should include a multidisciplinary team approach involving a neurologist, psychiatrist and a specialist in rehabilitation medicine. A good working relationship between the neurologist and the psychiatrist is vital so that a consensus approach is offered and explained to the patient.

Other disorders with abnormal movements and psychiatric manifestations include stiffness associated with the neuroleptic malignant syndrome and the serotonin syndrome, dystonia and psychosis associated with Niemann Pick type C, chorea in systemic lupus and erythematosis and the antiphosphlipid antibody syndrome.

In summary knowledge of movement disorders can be of great benefit in the diagnosis and management of neuropsychiatric disease. The psychiatrist should take time to observe the patient, describe what he/she sees and avoid initial labelling, classify the movement as hyperkinesia or hypokinesia, give the movement a name (eg. tremor, chorea), diagnose a specific disease after naming the movement and finally think about treatment.

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