

good clinical descriptions

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Tardive Dyskinesia

Haag WHO TD excerpts

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Table 1. Classic tardive dyskinesia.

General characteristics

- often resembling normal (chewing, licking, grasping) or repetitive choreic movements
- monotonously repeated with a low frequency of about 10–40 min⁻¹
- involuntary, can often only temporarily be suppressed by volition; patients often unaware of dyskinesias.
- activation by voluntary movements of non-affected muscle groups
- lack of a subjective feeling of inner restlessness
- reduced with drowsiness or sedation, absent during sleep

*Individual symptoms**Face*

- tongue: protrusion, “bonbon sign”, vermicular contraction
- jaw: vertical or horizontal chewing movements
- perioral: pouting, “bridling”
- others: distortion of the eyebrows, frowning, eye-blinking

Extremities

- fingers: “piano-playing” movements, grasping
- wrists: rotatory and flexion/extension movements
- toes: flexion/extension movements
- ankles: rotatory and flexion/extension movements
- legs: stamping movements

Trunk

- hip-rocking
- irregular contractions of diaphragm (grunting, respiratory difficulties)
- others: head nodding, shrugging of shoulders, rocking movements of the upper torso

tients perform these movements involuntarily, and – with more severe forms of the disorder – they are at best temporarily capable of suppressing these involuntary movements. There is often a striking discrepancy between the objective motor restlessness and the lack of a subjective feeling of inner restlessness. Many patients with tardive dyskinesia do not even notice their involuntary movements unless their attention is drawn to them by other persons. Sixty percent of the 138 tardive dyskinesia patients in our own epidemiological survey (HAAG et al., 1985) maintained they had not yet noticed their abnormal movements, and only 33% experienced mild or moderate distress.

Table 2. Tardive dystonia.

General characteristics

- resembling dystonia musculorum deformans
- sustained dystonic contractions that may lead to muscular hypertrophy or fixed postures
- involuntary, can often only temporarily be suppressed by volitional effort
- in some patients brought out by volitional action of affected muscle groups ("action dystonia")
- lack of a subjective feeling of inner restlessness
- decreased with drowsiness or sedation, absent during sleep
- patients usually aware of, high subjective discomfort

*Individual symptoms**Face*

- tongue: sustained tonic protrusion
- jaw: sustained tonic opening of the mouth
- eyes: blepharospasm

Extremities

- legs: inversion, plantar flexion of the feet, "spastic" gait
- arms: abduction of the shoulder, flexion of the elbow, hyperextension of wrist and fingers

Trunk

- torticollis, retrocollis
 - shoulder and pelvic girdle: twisting and undulating movements
 - axial dystonia, lateral flexion of the spine
-

neck. Tardive dystonia does not exhibit such a clear-cut periodicity as classic tardive dyskinesia; thus, frequency counts are not useful as an indicator of severity.

In some patients dystonic contractions are activated only when voluntary movements of the respective muscle groups are performed. This phenomenon has been referred to as *action dystonia* by some authors.

Interestingly, dystonias may be brought about only by slow movements or other specific movement patterns. For example, the attempt to walk slowly or at normal speed may result in intensive dystonic contractions, making walking virtually impossible. Yet the same patient may have surprisingly little difficulty running, climbing up chairs or riding a bike.

Some patients with idiopathic torticollis spasmodicus manage to control their dystonic contractions by slightly pressing their hand against the cheek ("geste antagoniste"). Similarly, some patients with tardive dys-

Table 8. Psychosocial and physical impairment by tardive dyskinesia.

Psychosocial

- feeling of embarrassment and guilt; shame; depression; social withdrawal
- handicapped in personal relationships, stigmatization by strikingly abnormal movements
- difficulty in professional activities

Physical

- denture problems, ulceration of the tongue, difficulty in swallowing
- dysarthria, spastic dysphonia
- respiratory disturbances
- gastrointestinal disturbances
- difficulty in motor function (fine motor skills, walking etc.); falls and injuries
- fixed postures
- increased mortality?

reported statistical association of tardive dyskinesia with increased mortality would be a direct causal connection (e. g. from an increased incidence of deaths by choking). Another possibility – and probably the more important one – is the existence of a common variable predisposing to both, tardive dyskinesia and increased mortality.

THE NATURAL COURSE OF TARDIVE DYSKINESIA

Initially, it was believed that tardive dyskinesia develops only after a minimum period of two or more years of continued neuroleptic exposure. But there is only little empirical evidence supporting the idea of such a minimum threshold for tardive dyskinesia development. In a prospective study, KANE and colleagues (1984) showed that the yearly incidence of tardive dyskinesia stayed fairly constant for the first seven years of treatment. There are case reports of an occurrence of tardive dyskinesia after as little as three months and sometimes after even shorter courses of treatment (CHOUINARD & JONES, 1979).

The study by KANE et al. cited above suggests that there may be important differences between cases with an early onset (less than two years of neuroleptic treatment) and cases with a later onset (after more than two years), since early-onset tardive dyskinesia was found to be associated with significantly lower maximum neuroleptic doses than late-onset tardive dyskinesia. This might indicate that early onset of

Table 9. Signs preceding the onset of tardive dyskinesia.

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- tongue fibrillation; minimal choreoathetoid movements of the tongue when extended by the patient
 - minimal perioral choreoathetoid movements
 - increased frequency of eye-blinking
 - eyelid and tongue tremor
 - "marching syndrome": walking in place like soldiers marking time (with or without a feeling of subjective restlessness)?
 - tardive akathisia?
 - muscular hypotonia?
-

tardive dyskinesia is more closely related to an increased individual vulnerability rather than to the intensity of neuroleptic therapy.

Tardive dyskinesia usually has an insidious onset, and the date of first appearance can often not be determined precisely in retrospect. For an early diagnosis of tardive dyskinesia it would be of great interest to know the very first clinical symptoms. Some (listed in Table 9) have been suggested as early signs of tardive dyskinesia. However, these are only tentative and need to be corroborated in prospective studies (GARDOS et al., 1983 a).

The clinical manifestation of tardive dyskinesia is often precipitated by a dose reduction or withdrawal of neuroleptics, or by concomitant administration of anticholinergic drugs (antiparkinsonian drugs, some antidepressants).

Once the symptomatology of tardive dyskinesia has fully developed, a rapidly deteriorating course is very uncommon, and – if present – should raise doubts whether the diagnosis is in fact correct.

If neuroleptic drugs can be withdrawn completely, tardive dyskinesia remains either stable or slowly improves with time (except for an initial transient rebound aggravation caused by the "unmasking" effect of neuroleptic withdrawal). In general, the rate of improvement appears to be highest during the first months after discontinuation of neuroleptics. However, it should be pointed out that full remissions may occur as late as two to five years after withdrawal.

Table 10 shows several studies investigating the reversibility of tardive dyskinesia after neuroleptic withdrawal. Most of these studies were performed on older, chronically institutionalized populations, so that the unfavorable results obtained cannot be generalized. Studies involving younger patient samples (YAGI et al., 1976, QUITKIN et al., 1977) have reported considerably higher rates of remission (53% and 92%, respectively).