The Management of a Completely Edentulous Patient with Tardive Dyskinesia

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Tardive dyskinesia (TD) is an involuntary movement disorder of neurologic origin caused by the use of neuroleptic drugs known as dopamine receptor antagonists. The condition may adversely affect prosthodontic interventions and their possible merits. In this case history description, the rigor of scrupulous history-taking and clinical evaluation of patients with TD or neurologic disorders is emphasized. The successful prosthodontic management of a 74-year-old completely edentulous TD patient is presented, with the conclusion that a prosthodontic intervention may aid symptom reduction and contribute to improvement in a TD patient's quality of life. *Int J Prosthodont 2010;23:217–220.*

Tardive dyskinesia (TD) is derived from the French word *tardif*, meaning tardy, and was first reported in 1956.[1] It is an involuntary movement disorder of neurologic origin caused by the use of neuroleptic drugs known as dopamine receptor antagonists, or DRAs.[2–4] TD differs from Parkinson disease in that tremors appear to be absent[5] and it is actually iatrogenic in nature. TD is caused by medications prescribed for the management of psychoses.[3]

TD is usually a late side effect of prescribing so-called typical and atypical neuroleptics (Table 1).[2] It has also been reported that dyskinetic movements can occur in patients who were never medicated.[6]

A lower incidence of TD has been reported in association with atypical antipsychotics when compared with the differently acting typical ones,[7] although there is little consensus for a definition of the term "atypical antipsychotics." However, there is evidence that atypical antipsychotics have less affinity for D2 receptors and more of an affinity for D4 ones.[1] There seems to be an agreement that *(1)* they are relatively free of extrapyramidal side effects, *(2)* they provide improved treatment for schizophrenia compared with the typical or first-generation antipsychotics,[3–5,7,8] and *(3)* they have a relatively higher serotonin-to-dopamine receptor-blocking ratio.[3,7] The terms "atypical" and "second generation" have been used to describe every antipsychotic drug introduced in the United States since clozapine.[3] Their use suggested that with the exception of clozapine, there was no proof that newer antipsychotices caused less TD. In fact, TD still occurred, especially with risperidone or olanzapine.[1]

Early signs of TD can be mild movements of the tongue accompanied by chewing, licking, or smacking movements.[5,6] Some patients will also develop choreiform (involuntary jerky displacements of short duration) of the hands, fingers, and arms. They are said to resemble purposeful incomplete actions with varying severity. These movements can take the form of foot tapping, squirming of the feet and toes, and movements of the fingers, as if playing an invisible guitar.[6,9] The differential diagnoses of TD include Parkinson disease, Huntington disease, spontaneous orofacial dyskinesias, stroke-induced chorea, lithium toxicity, and dilantin toxicity.[5]

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Table 1 Antipsychotics and Antidepressants Known to Cause TD

Generic	Brand name	Manufacturer
Antipsychotics		
Typical		
Chlorpromazine	Thorazine (discontinued)	SKB Pharmaceuticals
Fluphenazine	Prolixin	Bristol-Meyers Squibb
Haloperidol	Haldol	McNeil Labs
Loxapine	Loxitane	Lederle Pharmaceuticals
Metoclopramide	Reglan	A.H. Robins
Molindone	Moban	Gate Pharmaceuticals
Perphenazine	Trilafon	Schering-Plough Welcome
Pimozide	Orap	Gate Pharmaceuticals
Prochlorperazine	Compazine	SmithKlineBeecham
Thioridazine	Mellaril	Novartis
Thiothixene	Navane	Roerig
Trifluoperazine	Stelazine	SmithKlineBeecham
Zuclopenthixol	Clopixol	Lundbeck Pharmaceuticals
Atypical		
Aripiprazole	Abilify	Otsuka Pharm and Bristol Myers Squib
Clozapine	Clozaril	Novartis
Olanzapine	Prolixin	Eli Lilly
Paliperidone	Invega	Janssen Pharmaceutica
Quetiapine	Seraquel	AstraZeneca
Risperidone	Risperdal	Janssen Pharmaceutica
Ziprasidone	Geodon	Pfizer
Antidepressants		
Alprazolam	Xanax	Upjohn Pharmaceuticals
Trazodone	Desyrel	Bristol Myers Squib

TD is different from Parkinson disease in that it manifests with a wide variety of involuntary, repetitive, persistent, and stereotypic movements of the mandible, consisting of vertical and horizontal components forming an elliptical movement pattern.[10] TD is rarely reversible since the symptoms of this condition may remain long after discontinuation of neuroleptic drugs.[11] Schooler and Kane[12] developed the research diagnosis for TD, which is used for classifying TD type, and the Abnormal Involuntary Movement Scale, which gives prerequisites to exclude other movement disorders and is used to diagnose TD and its severity.[8,9]

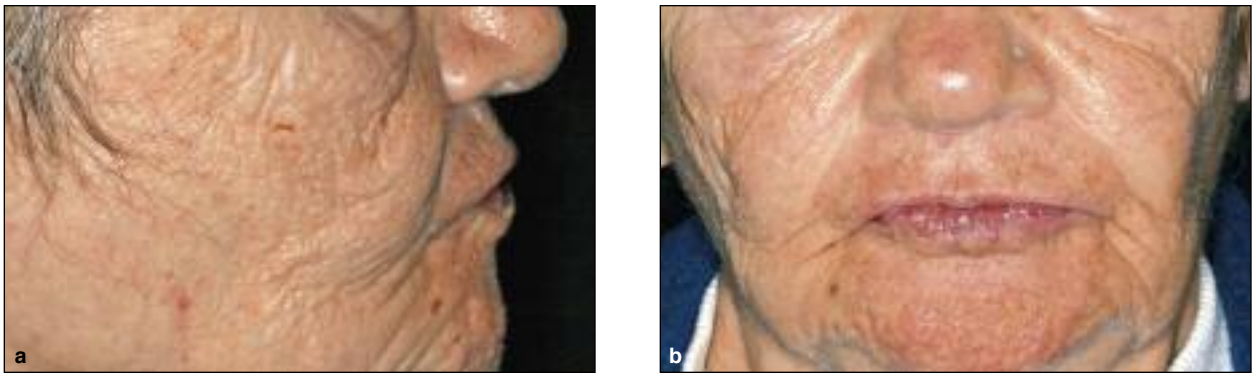
It has been suggested that elderly women have an increased risk for developing TD.[10,13] The duration of treatment with antipsychotic drugs, cumulative amounts of the drugs, and early signs of extrapyramidal symptoms are all potential contributing factors.[4] There is a wide variation in reported prevalence, which can vary from 1% to 54% of those taking antipsychotics.[3,7,14,15] The reasons differ from author to author. Swartz et al[15] felt that the variations in prevalence were reflected by the methodology, population, and assessment. Gharabawi et al[3] reported that the prevalence rate can be affected by the settings in which the patients are treated, ie, private versus state hospitals. Chou and Friedman[5] reported that some experts are finding large numbers of TD patients who had no history of being psychotic but had been on prolonged regimes of prochlorperazine (Compazine) or metoclopramide (Reglan) for gastroenterologic symptoms. There is also controversy over the terminology and diagnosis used. An example of this is that some authors are now referring to many cases as spontaneous oral dyskinesia, which is defined as an oral dyskinesia with no history of antipsychotic drugs.[13]

While Myers et al[16] reported that edentulism is a risk factor for developing TD, Blanchet et al[14] observed no association between edentulism and oral dyskinesia. However, they did report an association with ill-fitting dentures. TD makes the fabrication of removable prostheses extremely difficult. Once made, TD-associated parafunction can cause increased occlusal wear and fracturing of the prostheses.[16]

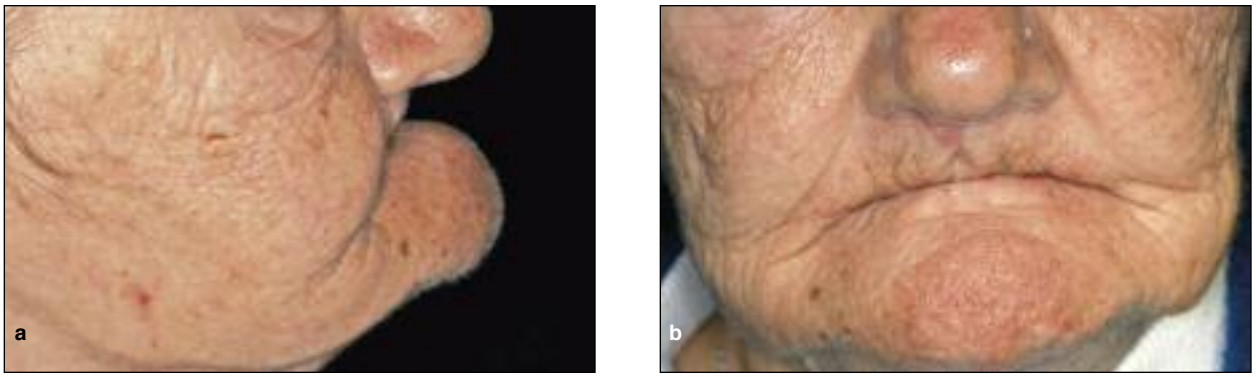
This report seeks to provide a concise synthesis of those considerations that will inform the clinician of the challenges associated with the prosthodontic management of patients with symptoms of TD.

Clinical Report

A 74-year-old woman presented with a chief complaint of ill-fitting, painful complete dentures, which she was unable to wear. Her medical history revealed hypertension, mitral valve prolapse (followed by replacement of the valves in 1981 and 1991), type 2 diabetes, anemia, osteoarthritis, and depression. Her depression was controlled by 4 mg of Xanax (alprozolam) and 100 mg of Desyrel (trazodone) at night for the past 5 years.



Figs 1a and 1b **(a)** Lateral and **(b)** frontal views of the patient at the beginning of her tracking movement.



Figs 2a and 2b **(a)** Lateral and **(b)** frontal views of the patient at the end of her tracking movement.

Extraoral examination revealed loss of occlusal vertical dimension, a sagging appearance of the lower third of her face, a complete disappearance of the lips (cheiloptosis), and constant stereotypical orofacial movements (Figs 1 and 2). A radiographic survey of the mandibular bone height showed areas of less than 10 mm vertical height of the mandible (mandibular bone height: type 4). Intraoral examination of the maxilla revealed poorly defined tuberosites and hamular notches (residual ridge morphology: type B). Mandibular muscle attachments were at the mucogingival junction except for the labial vestibule, where the mentalis muscle attachment was near the crest of the alveolar ridge (muscle attachment: type B). Maxillomandibular incoordination and hyperactivity of the tongue posed difficulty in reproducing maxillomandibular records (maxillomandibular relationship: Class III).

Surgical management with dental implants was not an option due to the compromised medical condition of the patient. Complete maxillary and mandibular dentures were fabricated for the patient by means of traditional clinical techniques. The challenge with this patient was the establishment, recording, and verification of her maxillomandibular relationship records. While anatomical landmarks such as paralleling of the residual alveolar ridges, measurements of the face height, phonetics, and esthetics were helpful in establishing an estimated occlusal vertical dimension, her excessive elliptical mandibular movement patterns made jaw verification records quite tenuous. While the needed verification was possible at an individual appointment, it was rarely reproducible at a subsequent one. Considerable patience, time, and empathy were necessary before a final decision could be made regarding what was judged to be the optimal jaw records for the patient. Ultimately, a lingualized occlusal scheme was designed in an effort to permit a definitive and maximal intercuspal position with a bilateral balance in excursive movements.

Video recordings were made of the patient's mandibular movements without dentures with a Sony Handicam DCR-TRV 310NTSC, and employed as a teaching and demonstration tool for both the patient's and her family's benefit. The recordings showed tracking ranges of 18 mm vertically and 15 mm horizontally in the frontal view.

With the dentures in the mouth, the video recordings were repeated and demonstrated a movement reduction, namely 5-mm vertical movements and 4-mm horizontal frontal ones. Removal of the dentures was accompanied by a return to excessive movements, which suggested that in this patient's case, properly designed and constructed complete dentures contributed to a reduction in her involuntary movements. It cannot of course be claimed that the diminution in this patient's uncontrolled movements was due to any one of the determinants of optimal complete denture treatment. But, the net result was one that both she and her family were extremely pleased with. This particular patient's significant and discernible improvement in what was an extremely embarrassing situation enabled her and her family to subjectively acknowledge a profound improvement in her quality of life.

It is unlikely that an inference of the causal relationship between removable prostheses and TD can be drawn from the available literature.[15,16] Nonetheless, while excessive tongue and mandibular movements are bound to compromise the denture-wearing experience, it may be argued that stable prostheses may offer a scope for spatial orientation that may in turn modify the TD's manifestations beneficially. The mechanism of this hoped-for response is certainly not understood, nor is it possible to predict whether the management of this patient can be replicated in a predictable manner in other patients with similar TD signs and symptoms.

The effects of the combination of edentulism and TD can be devastating. Speech can be virtually unintelligible and mandibular movement patterns embarrassing. Therefore, in the absence of robust evidence regarding the possible benefits of routine prosthodontic management, a case should be made for the inclusion of preventive dental programs and routine dental care as an integral part of the regimen of all patients at risk for TD development. The condition is medically mandated, but the dental clinician may very well be the first health professional to identify the uncontrolled movements that may suggest TD, especially those involving the orofacial complex. A frank discussion with the patient and referral to his or her physician can lead to an early diagnosis and effective management of this movement disorder. Above all, edentulism should be prevented whenever possible, while research into the validity of implant-supported/retained prostheses for such patients may prove to be an important new direction in the discipline.

Dental treatment for these patients is highly individualized and no standard treatment is available. The knowledge and empathy of the clinician are critical elements in the management of TD patients.

Conclusion

Proper diagnosis of the challenging predicament of TD combined with proper, albeit palliative, prosthodontic management may be of inestimable help to such a patient. This particular case history endorses such an approach.

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