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Dysphagia

Case Report

Dysphagia in Drug-Induced Parkinsonism: A Case Report

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Abstract. Dysphagia complicates both idiopathic Parkinson's disease (IPD) and drug-induced parkinsonism (DIP). Although parkinsonism of DIP and IPD are often clinically indistinguishable, there is no assurance that their abnormalities of swallowing will be similar. We evaluated a patient with DIP who complained of difficulty chewing and swallow initiation. The dysphagia evaluation demonstrated abnormalities during all stages of ingestion. However, the prepharyngeal stages were disproportionately affected when compared with patients with IPD and similar levels of parkinsonian functional disability. This case gives additional support for a significant basal ganglia influence on motor deglutitive functions.

Key words: Dysphagia — Drug-Induced parkinsonism — Dopamine — Deglutition — Deglutition disorders.

Drug-induced parkinsonism (DIP) is an iatrogenic extrapyramidal syndrome usually caused by exposure to a variety of neuroleptics, antiemetics, and antihypertensive drugs [1,2]. In addition to replicating the cardinal parkinsonian features of idiopathic Parkinson's disease (IPD), DIP patients may become dysphagic, like their counterparts with IPD. However, clinical similarities between DIP and IPD do not ensure similar dysphagic clinical or radiologic characteristics.

Recent reports have increased our awareness of dysphagia in IPD [3,4]. To our knowledge, there are no detailed accounts of aberrant swallowing in DIP, only comments acknowledging its presence [5]. A patient with DIP and dysphagia recently provided an opportunity to compare dysphagia in DIP with that reported previously in patients with IPD.

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Case Report

A 38-year-old woman with no history of significant past medical illnesses was admitted to the Psychiatry Unit of Crozer-Chester Medical Center (CCMC) for treatment of depression. After 12 days, she was discharged on trifluoperazine, 5 mg/day, and paroxetine, 20 mg/day. She was hospitalized again 11 days later after complaining of tremor, sialorrhea, and difficulty chewing. She denied coughing or choking during meals, nausea, vomiting, odynophagia, pharyngoesophageal food retention, or reflux. All psychotropic medications were withdrawn on admission. Within 24 hours, slight improvement of mastication was noted.

A neurologic consultation was completed on the day of her second admission. The general medical examination was normal. The neurologic examination showed an alert, attentive woman with slowed, hypophonic, monotonic, dysarthric speech. The motor examination demonstrated obvious parkinsonian features including moderately severe bradykinesia, akinesia, rigidity of all limbs, and slight postural instability. Her gait was slow and shuffling but without festination. A fine 5–6 Hz resting tremor was observed, primarily in the right hand. The patient functioned at stage III on the Hoehn and Yahr Disability Scale (H&Y) [6] and scored 33 (maximum = 108) on the motor portion of the Unified Parkinson Disease Rating Scale (UPDRS) [7]. The remainder of the neurologic examination was intact. Benztropine, 1 mg. PO and diphenhydramine, 75 mg IM administration provided no relief of either DIP or dysphagic symptoms.

A dynamic videofluoroscopic swallowing function study (DVSFS) using liquids and foods of varying consistencies was completed by the CCMC Dysphagia Service 3 days after all drugs were stopped. Abnormalities observed during the pre-esophageal stages of ingestion included a severe reduction of lip and tongue movements, minimal chewing of solids, segmented lingual transfer with moderate posterior lingual leakage for all test substances, decreased velar retraction which delayed bolus transfer, and slow vocal cord adduction with no aspiration or penetration. Tongue movements were so limited that solid food had to be removed manually from the mouth. Esophageal

peristalsis was mildly slow with tertiary waves. Esophageal bolus redirection with reverse aspiration risk was also noted.

The patient reported a daily rapid relief of both parkinsonism and dysphagia, particularly mastication and transfer functions. Recovery began prior to 4 days of dysphagia therapy. No antiparkinsonian drugs were prescribed. On the day of discharge, one week after her admission, the neurologic examination showed only mild upper extremity rigidity, and slight hypophonia, hypomimia, and bradykinesia (H&Y = Stage II; UPDRS = 8). A repeat DVSFS 4 days after the initial study demonstrated marked improvement. Residual abnormalities included a mild impairment of mastication. Lingual movements were now only minimally slow. Pharyngeal transport and esophageal bolus transport and emptying were normal. Three months after discharge from the hospital no parkinsonism was observed. At that time she refused a third DVSFS.

Discussion

IPD and DIP are the two most common causes of a bradykinetic-rigid extrapyramidal syndrome [8]. A deficiency of dopamine in the basal ganglia induces both clinical syndromes [9]. In IPD, dopamine (DA) deficiency is secondary to a loss of dopaminergic neurons in the substantia nigra. In DIP, the responsible drugs reduce central dopaminergic activity by either blocking the postsynaptic DA-2 receptor (neuroleptics and antiemetics) or impairing the formation and storage of DA at the presynaptic terminal (antihypertensives, e.g., reserpine, alpha methyldopa). These drugs provoke not only typical parkinsonian features, but also other complications of IPD including dysphagia. Indeed, in the absence of a history of drug exposure, parkinsonism of IPD and DIP may appear clinically identical.

Dysphagia is a frequent complication in IPD, particularly in patients with more advanced disease [3]. Its prevalence in DIP is unknown. All stages of ingestion are affected in IPD, but patient complaints most frequently arise from pharyngoesophageal stage dysfunction [3]. The clinical dysphagia examination demonstrates multiple abnormalities including slowed, labored feeding, inefficient mastication, and impaired lingual motility [3,4,10]. The DVSFS often demonstrates multiple abnormalities of bolus transfer and transport (3,4,11–13).

In the present patient, symptomatic dysphagia coincided with DIP. She manifested many dysphagic features observed in patients with moderately advanced IPD. However, pharyngoesophageal stage dysfunction was relatively minor. In our experience with IPD, the discrepancy between orolingual and pharyngoesophageal swallowing functions witnessed in the present patient is exceptional. Whether this clinical presentation is typical DIP-related dysphagia is unknown. Hoffman et al. [14] described a somewhat similar patient rendered parkinsonian after an accidental haloperidol overdose. Without mentioning dysphagia, Delay and Deniker [15] acknowledged mouth, jaw, and perioral muscle involvement in DIP.

Although our patient received 4 days of dysphagia therapy, her rapidly improving dysphagia and parkinsonism coincided with discontinuing a dopamine (D-2) receptor antagonist. This presentation suggests that dysphagia in DIP has neurotransmitter similarities to IPD and, by inference, that dysphagia in both forms of parkinsonism are significantly dependent on defective DA-dependent basal ganglia influences on motor deglutitive functions.

The role of DA in the ingestive process is unknown. Most reviews of the neurophysiologic and neurochemical controls of swallowing emphasize results from animal experimentation; basal ganglia and DA influences are given little consideration [16–19]. Recent clinical studies of dysphagic PD patients comment that preesophageal dysphagia severity follows progression of parkinsonian dopamine-dependent bradykinesia and rigidity [3,4]. Dysphagia also occurs in parkinsonism due to 1-methyl-4-phenyl-1,2,3,6 tetrahydropyridine (MPTP), a synthetic heroin toxic to nigral dopaminergic neurons. In the MPTP nonhuman primate model, impaired prepharyngeal deglutition includes severely impaired swallow initiation of poorly chewed foods (personal communication). *In humans,* limited descriptions of dysphagia mention impaired tongue movements and sialorrhea [20]. Descriptions of DVSFS in victims of this form of DIP have not yet been published. In addition, esophageal bolus movement may be disturbed further by central or peripheral dopaminergic activity [21,22]. Finally, several anecdotal reports document that indirect (levodopa) and direct (apomorphine) DA agonists ameliorate dysphagia in some IPD patients [4,21,23].

In addition to parkinsonism, patients treated with neuroleptics also experience anticholinergic side effects, either from the neuroleptic [24] or from anticholinergic drugs administered to block or blunt DIP. In some patients, reduced peripheral cholinergic activity affects deglutition by either impairing esophageal motility or, *because of dry mouth,* by interfering with intraoral bolus transit [25,26]. Lowering central cholinergic activity in other patients may improve bradykinesia and rigidity and, by implication, preesophageal *dysphagia. Anticholinergics* provided the only effective symptomatic therapy for IPD prior to the introduction of levodopa.

Dysphagia may also be a somatization. As a symptom of an underlying psychiatric disease [27], difficulty initiating the swallow or "food getting stuck" after swallowing may represent delusions. The current trend to initiate treatment of psychotic patients with atypical neuroleptics *without* significant extrapyramidal side effects will lessen both the need for adjuvant anticholinergics and the diagnostic confusion between those few patients with psychogenic dysphagia [28] and those with iatrogenic dysphagia. During this transition period, we

urge a greater awareness of this form of iatrogenic dysphagia.

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References

1. Ayd FJ: A survey of drug-induced extrapyramidal reactions. *JAMA 175:*1054–1060, 1961
2. Marsden CD, Tarsy D, Baldessarini RJ: Spontaneous and drug-induced movement disorders in psychotic patients. In: Benson DF, Blumer D (eds.): *Psychiatric Aspects of Neurologic Disease.* New York: Grune and Stratton, pp 219–266
3. Leopold NA, Kagel MC: Prepharyngeal dysphagia in Parkinson's disease. *Dysphagia 11:*14–22, 1996
4. Bushmann M, Dobmeyer SM, Leeker L, Perlmutter JS: Swallowing abnormalities and their response to treatment in Parkinson's disease. *Neurology* (Minneap) *39:*1309–1314, 1989
5. Stoschus B, Allescher H-D: Drug-induced dysphagia. *Dysphagia 8:*154–159, 1993
6. Hoehn MM, Yahr M: Parkinsonism: onset, progression, and mortality. *Neurology* (Minneap) *17:*427–442, 1967
7. Fahn S, Elton RL, the UPDRS Development Committee: Unified Parkinson Disease Rating Scale. In: Fahn S, Marsden CD, Goldstein M, Calne D (eds.): *Recent Developments in Parkinson's Disease*, vol 2. Floral Park, NJ: Macmillan, 1987; pp 293–304
8. Rajput AH, Offord KP, Beard CD, Kurland LT: Epidemiology of parkinsonism: incidence, classification, and mortality. *Ann Neurol 16:*278–282, 1984
9. Marsden CD, Jenner P: The pathophysiology of extrapyramidal side effects of neuroleptic drugs. *Psychol Med 10:*55–72, 1980
10. Atlin E, Norberg A, Axelsson K, Moller A, Nordstrom G: Aberrant eating behavior in elderly parkinsonian patients with and without dementia: analysis of videorecorded meals. *Res Nur Health 12:*41–51, 1989
11. Eadie MJ, Tyrer JH: Alimentary disorder in parkinsonism. *Aust Ann Med 14:*13–22, 1965
12. Robbins JA, Logemann JA, K, Kirshner HS: Swallowing and speech production in Parkinson's disease. *Ann Neurol 19:*283–287, 1986
13. Stroudley J, Walsh M: Radiologic assessment of dysphagia. *Br J Radiol 64:*890–893, 1991
14. Hoffman AS, Schwartz HI, Novick RM: Catatonic reaction to accidental haloperidol overdose: an unrecognized drug abuse risk. *J Nerv Mental Dis 174:*428–430, 1986
15. Delay J, Deniker P: Drug-induced extrapyramidal syndromes. In: Vinken PJ, Bruyn GW (eds.): *Handbook of Clinical Neurology,* vol 6, Amsterdam: North-Holland Publishing Co. 1968, pp 248–260
16. Doty RW: Neural organization of deglutition. In: Code CF (ed.): *Handbook of Physiology, Alimentary Canal*, section 6, vol. 4. Washington, DC: American Physiology Society, 1968, pp 1861–1902
17. Sessle BJ, Henry JL: Neural mechanisms of swallowing: neurophysiological and neurochemical studies on brain stem neurons in the solitary tract region. *Dysphagia 4:*61–75, 1989
18. Miller AJ: The search for the central swallowing pathway: the quest for clarity. *Dysphagia 8:*185–194, 1993
19. Bieger D: Central nervous system control mechanisms of swallowing: a neuropharmacological perspective. *Dysphagia 8:*308–310, 1993
20. Ballard PA, Tetrud JW, Langston JW: Permanent human parkinsonism due to 1-methyl-4-phenyl-1,2,3,6- tetrahydropyridine (MPTP); seven cases. *Neurol* (Minneap) *35:*949–956, 1985
21. Kempster PA, Lees AJ, Crichton P, Frankel JP, Shorvon P: Off-period belching due to a reversible disturbance of eosophageal motility in Parkinson's disease and its treatment with apomorphine. *Movement Dis 4:*47–52, 1989
22. Mumhopudhyay AK, Weisbrodt N: Effect of dopamine on oesophageal motor function. *Am J Physiol 232:*E19–24, 1977
23. Calne DB, Shaw DG, Spiers ASD, Stern GM: Swallowing in parkinsonism. *Br J Radiol 43:*456–457, 1970
24. Kane J, Honigfeld G, Singer J, Meltzer H: Clozapine for the treatment-resistent schizophrenic, a double-blind comparison with chlorpromazine. *Arch Gen Psychiatry 45:*789–796, 1988
25. Bramble MG, Cunliffe J, Delipiani AW: Evidence for a change in neurotransmitter affecting oesophageal motility in Parkinson's disease. *J Neurol Neurosurg Psychiatry 41:*709–712, 197
26. Liedbereg B, Owall B: Masticatory ability in experimentally induced xerostomia. *Dysphagia 6:*211–213, 1991
27. Solyom L, Sookman D: Fear of choking and its treatment. *Can J Psychiatry 25:*30–34, 1980
28. Ravich WJ, Wilson RS, Jones B, Donner MW: Psychogenic dysphagia and globus: reevaluation of 23 patients. *Dysphagia 4:*35–38, 1989