

A Case Study: Idiopathic Dyskinesia Treated With Choline and Lecithin

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Synopsis

The author reviews the use of choline and lecithin in the treatment of tardive dyskinesia. To the author's knowledge this is the first documentation of the use of choline and lecithin in the treatment of idiopathic dyskinesia. The major improvement was in the area of the dyskinetic movements, although there was some brief improvement in mentation. The encouraging results in terms of the reduction in mouth movements is worthy of note and is the reason for this report.

The phenomenon of tardive dyskinesia has generated considerable concern and interest. There have been reports on the administration of choline and lecithin in tardive dyskinesia treatment (Klawans; Growdon et al.; Gelenberg et al.; Jackson et al.). As well, the APA Task Force addresses the issue rather bluntly and not too optimistically (APA Task Force). The working hypothesis is that tardive dyskinesia is related to an imbalance in the relationship

between dopaminergic and cholinergic neurons in the basal ganglia. Dopamine transmission may be excessive relative to a deficit of choline, and efforts to increase cholinergic activity, such as the use of choline or lecithin, might prove to be beneficial (Kobayashi; Casey and Denney). Most of the literature has dealt with patients who have tardive dyskinesia. A report appeared in the American Journal of Psychiatry (Newhouse and Bridenbaugh) in which a patient was treated who had presumably a non-tardive dyskinetic picture. However, in that patient there was some concern for the dyskinetic movements and it was reported that the patient had been on anti-histamines as well. In addition, only lecithin was used in her treatment. To my knowledge this is the first documentation of the use of choline and lecithin in the treatment of idiopathic dyskinesia. The major improvement was in the area of dyskinetic movements, although there was some brief improvement in mentation. Unfortunately, there has been deterioration, but the encouraging results in terms of the reduction in mouth movements is worthy of note and provides the main thrust for submission of this report.

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Case Report

Mrs. P. was a 74 year old widow who had been in reasonably good health until eight months before her consultation with a neurologist in March, 1977. At that time she developed involuntary movements of tongue and jaw, blinking of her eyes, and bobbing of her head. There was also slurred speech. Movements were described as both chewing and protrusion of the tongue. Two weeks prior to consultation the symptoms became more severe and hospitalization was recommended. Significant past history was cerebral hemorrhage 20 years previously, with nausea and loss of consciousness. According to the daughter she was unconscious for some time following admission, but recovered well with only headaches and numbness on the left side of her face which subsequently cleared. In recent years patient had been living alone, but there had been some impairment in memory. She was able to do light housekeeping with assistance. At the time of consultation, examination revealed continual dyskinesia with movements of the jaw, lip smacking, and protrusion of the tongue, associated with vigorous eye closure and slight nodding of the head. Speech was slurred, but normal in volume. She was able to eat and swallow without difficulty and there was no brady-kinesia. Additionally, the patient was moderately demented, not able to remember any of three test words after two minutes and moderate disorientation for time. Current events testing revealed presumed deficits with forgetfulness. General neurological examination was unremarkable without lateralization except for mild left upper extremity weakness, spasticity, and slowness of rapid movements. The main diagnostic problem was the oral-facial-lingual-mandibular dyskinesia, which was obvious and severe. All studies were unremarkable except CAT scan which showed "moderately enlarged cerebral ventricles. There is no evidence of ventricular obstruction. There also does appear to be some slight increase in the density of the white matter, particularly opposite the frontal horn." The evaluation in hospital led to the conclusion that this was indeed a

primary movement disorder and she was discharged on haloperidol, 1 mg, t.i.d.

Patient came under psychiatric care in May, 1977, when she was brought for an evaluation of an inability to sleep and the family was concerned that she might be depressed. Admission mental status revealed a woman who had some striking remembrances of events in the recent past and distant past, but difficulties with recent recall. She had some impairment of time sense, but was oriented to month and year, and gave enough time duration in terms of recent hospitalizations to suggest a fair and possibly retrievable orientation. There were no gross disorders of thought process or thought content, but the suggestion was there of a change in her intellectual performance. Her mood was despondent and she appeared dysphoric. The differential included pseudementia with depression and senile dementia. Hospitalization work-up included a follow up visit by the neurologist who said the oramandibular dyskinesia was fairly well controlled with the haloperidol. Routine lab findings were unremarkable. She was treated symptomatically for anxiety and depression, but mentation changed very little. By the time of discharge, dementia was a possibility with mild secondary depression. She was discharged on haloperidol, 1 mg, t.i.d., Cefol® , t.i.d., and amitriptyline, 10 mg, b.i.d., and 50 mg at h.s., and she had been put on aldactazide by her internist. The next psychiatric admission came two years later when she was admitted for evaluation of nervousness. The family felt that they could not control her as an outpatient. She had fair orientation to time and place. Speech was impaired because of the profound oral facial dyskinesia, but she denied sadness. However, much of the speech was perseverative, in that she would constantly be repeating "yes, yes, yes," or "no, no, no." She admitted to poor sleep, but denied any other complaints. Mental status revealed her sitting uncomfortably in a wheel chair, with marked oral facial dyskinesia, particularly rapid movements of the mouth, with her dentures clattering and considerable blep-horospasm. Speech was monosyllabic and

mood was flat. Biochemical studies were all thought to be within normal range. Her CAT scan showed prominence of the ventricular system representing involitional change with age or mild cerebral atrophy. Because of the small size of the sulci, hydrocephalus could not be entirely excluded, and there was evidence of old ischemic disease in the deep white matter and basal ganglionic area of the right cerebral hemisphere. EEG was mildly abnormal because of the diffuse 4-5 hertz slow activity consistent with diffuse neuronal disease as occurs in a degenerative process or diffuse neuronal dysfunction as may occur with toxic metabolic encephalopathy. No focal paroxysmal or lateralizing discharges were noted.

Our conclusion was that this was an organic brain syndrome, senile dementia, secondary to non specific factors, possibly metabolic, possibly related to the old stroke. The major therapeutic challenge was the dyskinesia and restlessness. The restlessness was possibly from akathisia and although there was some dysphoria, the mouth movements and tremulousness were the most distressing features. Around this time the reports of choline and lecithin treatment were appearing for tardive dyskinesia (Klawans; Growdon et al.; Gelenberg et al.) and we felt it appropriate to try this on our patient, even though the mechanism of the dyskinesia may be altogether different. Treatment included apresoline, 25 mg, q.i.d., inderol, 10 mg, q.i.d. for control of blood pressure. She was also maintained on Stresstabs® with iron b.i.d., haloperidol, 1 mg, at h.s., and doxepin, 25 mg, b.i.d. and choline, 650 mg, 5 tablets, t.i.d. (Willner Chemists). She was started on the choline and lecithin on 10/3/79 and at the time of discharge on 10/29/79 we were seeing a marked improvement. Mouth movements had diminished considerably. The head shaking and bobbing had been reduced, and possibly related to the benefits from the medications, her mood was improved and family reported a sense of humor returning. Follow up visit in July, 1980. revealed her to be in good spirits, somewhat slower, but quiet and happy. In November, 1980. she was reported

doing better. Movements were considerably less. She was more talkative and seemed to be more aware of social surroundings, and the family reported more spontaneous smiling. By early March, 1981, she had shown more improvement and was laughing at television shows and following along appropriately. Mouth movements had virtually ceased. She was swallowing without difficulty, and there was no head bobbing or lip smacking. However, by late April, 1981, she had shown deterioration in mental functioning. Her mouth was now staying open a great deal, her tongue was protruding; however, there were no rabbit movements and minimal head bobbing. She appeared anergic and mildly restless. This was the first time the daughter reported a disagreeable odor, and felt that her mother had shown considerable deterioration in mental state. It was my impression that she had become more demented, but the family was satisfied that there had been a period of tremendous improvement and recognized that this could be a downhill malignant course in terms of her global functioning. The only therapeutic change made was to increase the lecithin to 500 mg, 2 tablets, b.i.d. gradually up to a total of 6 times daily. From the standpoint of purely clinical practice, I felt it entirely appropriate to try this woman on something that could offer her relief, something that the APA Task Force itself in some respects encouraged (APA Task Force). In retrospect, I suspect that we should have used rating scales (Simpson et al.) to document more adequately her degree of dyskinetic movement, but the clinical impression from family and this physician was clear cut and obvious that there had been a profound reduction in the mouth movements and the patient had improved considerably. The other possibilities for additional treatment would be deanol instead of doxepin, and some reports have appeared with reference to the use of manganese, niacin, and peri-doxine in addition (Kunin).

The most striking finding was the dramatic improvement in the dyskinetic movements to the extent that the patient was no longer disabled and immobilized because of the

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exhausting head and mouth movements. The family reported a mild improvement in her mental functioning and social interactions, although that may be more of a reflection of their wished for perception. Clinically she didn't change as much from the standpoint of the dementia over the course of treatment, but the motor components were brought under control rather readily.

In conclusion, this report is an attempt to alert others to the beneficial effects of choline and lecithin in the treatment of an idiopathic dyskinesia. One can only speculate as to the relationship this has to the tardive phenomenon, but if the pathophysiology is in any way similar, this would lend support to the treatment possibilities proposed by others.

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