Transient Choreiform Dyskinesias During Alcohol Withdrawal

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SUMMARY: *Three chronic alcoholics developed choreiform dyskinesias involving the face, lips, tongue and, in one case, all limbs; 2 patients for the first time, 9 to 10 days after alcohol withdrawal. These abnormalities improved spontaneously with maintained abstinence from alcohol for 2 to 7 weeks. None had a family history of movement disorder, there was no history of other psychoactive drug use or abuse, and there was no evidence of portal-systemic encephalopathy.*

RÉSUMÉ: *Trois patients alcooliques chroniques ont développé des dyskinésies du visage, des lèvres, de la langue et, dans un cas, de tous les membres, 9 à 10 jours après sevrage de l'alcool. Ces anomalies se sont améliorées spontanément avec une abstinence de 2 à 7 semaines. Il n'existait aucune histoire familiale de troubles du mouvement; il n'y avait aucune évidence d'emploi régulier ou abusif de drogues psychoactives et il ne fut démontré aucune encéphalopathie portale systémique.*

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INTRODUCTION

Acute and chronic alcohol ingestion has significant effects on central neurotransmitter activity including dopamine metabolism (Tabakoff et al, 1979). We examined three chronic alcoholics who, during a period of alcohol withdrawal, demonstrated transient choreiform dyskinesias which resolved with abstinence from alcohol.

CASE REPORTS

Patient 1

A 50 year old woman with a history of heavy drinking for more than 20 years (approximately 9 ounces of liquor a day) was admitted following her first alcohol withdrawal seizure. Other than some recent memory deficit, the initial neurological examination was normal. The patient has spider nevi and hepatomegaly but no evidence of portal-systemic encephalopathy. She had not received any psychoactive medication. Forty-eight hours following admission and 9 days after her last drink, she developed facial grimacing, buccolingual dyskinesia and rapid choreiform neck movements to the right side. These movements rapidly disappeared over the next week, and there were no neurologic sequelae during a 6 month follow-up period with maintained alcohol abstinence. Laboratory investigations showed transiently abnormal liver function tests. A CT scan revealed diffuse cerebral atrophy.

Patient 2

A 50 year old man who had been a heavy consumer of liquor and beer for 15 years, was admitted 3 days after his last drink because of hallucinations, paranoid ideation and bizarre behavior. Neurological examination on admission was otherwise normal. Thiamine, 50 mg p.o., was the only drug given initially. The patient improved until the tenth day after the last drink when he developed increasingly bizarre behavior, paranoid ideation, facial grimacing, buccolingual dyskinesia and choreiform movements in all limbs. Haloperidol 5 mg p.o. b.i.d. was then administered for nine days. Within 3 weeks he had recovered and follow-up 3 and 6 months later, with maintained abstinence, revealed no abnormal involuntary movements. CT Scan showed severe generalized cerebral atrophy. There was no clinical or biochemical evidence of liver disease.

Patient 3

A 66 year old female with a 30 year history of chronic alcoholism had also had benign essential tremor for many years. Two years prior to admission she noted a pill-rolling tremor of the hands and six months' prior to admission she developed abnormal involuntary movements of the tongue and lips. On admission, 2 days after the last drink, the patient demonstrated Parkinsonian features previously reported by Carlen et al, 1981: Her gait was ataxic, she had constant lip smacking and repeated protrusions of her tongue. She was disoriented to place and date and recent memory was impaired. Sixteen days after her last drink the patient was given Sinemet, which was discontinued after 1 week following the disappearance of the Parkinsonian features. Four weeks later she had greatly reduced dyskinesia. There was no clinical or biochemical evidence of liver disease. Marked generalized cerebral atrophy was evident on CT scan.

DISCUSSION

Acute reversible dyskinesias affecting chronic alcoholics during withdrawal or related to maintained alcohol ingestion have been, to our knowledge, reported only once (Mullin et al, 1970). This report did not clearly

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delineate the relationship between the time of development of the dyskinesia and the time of alcohol withdrawal. Dyskinesias are associated with a wide variety of neurological disease and may be provoked by drugs (Marsden, 1976). Neuroleptic drug-induced tardive choreiform dyskinesias are related to chronic dopamine receptor blockade (Curzon, 1976; Baldessarini et al, 1980), and may be a form of denervation supersensitivity (Baldesarini et al, 1980; Klawans et al 1980). Acute alcohol intoxication in younger patients (ages 20-30) on chronic phenothiazines or butyrophenones can cause short-lived akathisia and dystonia (Lutz, 1976), whereas chronic alcohol intoxication or early alcohol withdrawal can provoke Parkinsonism in older patients (ages 53-70) (Carlen et al, 1981). Chorea has been described in patients with acquired hepatocerebral degeneration (Victor et al, 1965) but none of our patients had the findings or history of portal-systemic encephalopathy and 2 patients had no clinical or biochemical evidence of liver disease.

During the first few days of alcohol withdrawal, when we previously noted Parkinsonism (Carlen et al, 1981), we would not expect an excessive dopaminergic clinical response. Animal studies have shown decreased striatal dopamine levels (Lai et al, 1979) and diminished responsiveness of striatal dopamine-sensitive adenylate cyclase activity (Lai et al, 1980) during early alcohol withdrawal. To explain the dyskinesias in our patients, we propose that dopamine receptor supersensitivity can develop in association with chronic alcoholism. Functional dopamine receptor supersensitivity has been demonstrated in the nucleus accumbens of rats for approximately 4 weeks following 5 weeks of ethanol intake (Engel and Liljequist, 1976; Liljequist, 1978). It has also been shown both behaviorally and biochemically in chronically ethanol-fed rats that a dopamine antagonist can rapidly increase the responsiveness of the cerebral dopamine system to dopamine agonists (Lai et al, 1980) and this effect persists for at least 10 days after withdrawal.

These animal data may explain the development 9 to 10 days after alcohol withdrawal and the later recovery of the dyskinesias in patients 1 and 2 with maintained abstinence. Patient 3 demonstrated both Parkinsonism and dyskinesia, which is often seen in Parkinsonian patients treated with L-Dopa (Klawans et al 1980). The Parkinsonian features resolved more quickly than the dyskinesia. Clearly the dyskinesias in all patients were related to chronic excessive alcohol intake since these symptoms improved remarkably with abstinence. It is unclear if alcoholism unmasked, provoked, or caused the dyskinesia syndrome. Further studies measuring CNS transmitter levels and metabolism are necessary to delineate this syndrome more fully.

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