Case report

Parkinsonism in alcohol withdrawal: case report and review of the literature


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Abstract

A case of severe, acute parkinsonism occurring in a 60-year-old man after cessation of chronic alcohol consumption, is reported. He recovered completely in 3 months without specific therapy. The literature on alcohol withdrawal parkinsonism including nine other cases, is reviewed.

Keywords: Alcohol withdrawal; Parkinsonism; Apomorphine test

1. Introduction

A variety of neurological syndromes, e.g. Wernicke's encephalopathy, dementia, polyneuropathy, delirium tremens, may arise as a complication of alcoholism and alcohol withdrawal [1–3]. One of the rare consequences of alcohol withdrawal is parkinsonism. Until now only 9 cases of acute, transient parkinsonism after alcohol withdrawal have been reported [4–8]. We report a 10th case of alcohol withdrawal parkinsonism in a 60-year-old man with a history of chronic alcohol abuse. The literature on withdrawal parkinsonism is reviewed.

2. Case report

A 60-year-old man with a 10-year history of excessive alcohol consumption (one bottle of 'jenever' (Dutch gin) and several beers a day) was given disulfiram (Antabus 200 mg per day) by his family physician. He stopped intake of alcohol. According to his spouse he became apathic within 4 days and experienced increasing difficulty in walking. After being bedridden (his spouse could not remember whether rigidity was already present) for 3 days, during which time he did not eat or drink because of nausea, he became fully disoriented and somnolent. On inquiry both his wife and his family physician denied any use of neuroleptic drugs. Former episodes of parkinsonism during alcohol withdrawal were also denied. The patient's family history was negative for Parkinson's disease. He was admitted to the department of internal medicine at our hospital because of suspected hepatic coma. A general medical examination did not show any abnormalities. The liver was not palpable. The patient had no fever. Blood analyses gave mildly elevated liver enzymes (alkalic phosphatase 144 U/l, gamma-GT 211 U/l), normal electrolytes, renal function, and ammonia. One day after admission we were asked to evaluate the patient neurologically.

A confused man with an impaired consciousness was seen (Glasgow Coma Score: E2M4V3). Neurological examination showed no meningeal irritation. Pupils were normal. The patient had an expressionless face, second degree horizontal nystagmus in both directions, and lateral rectus palsies. Positive glabella tap, palmo-mental and snout reflexes were found as well as an intermittent resting tremor of both arms. No muscle weakness or
atrophy was present. The patient showed an extreme 'lead pipe' type rigidity of the neck, trunk and limbs. The upper extremity reflexes were symmetrically normal, knee and ankle tendon reflexes were absent, plantar reflexes were indifferent. The patient was not able to walk.

A probable diagnosis of Wernicke's encephalopathy was reached and intravenous therapy of thiamine (100 mg/day) was started. Computer tomographic (CT) scanning of the brain showed generalized atrophy (Fig. 1). In a few days his consciousness improved and eye movement abnormalities disappeared. It now appeared that there was a short-term loss of memory with confabulation. Positive primitive reflexes, mask-like face, resting tremor of the arms, and extreme rigidity of trunk and limbs persisted. Magnetic resonance imaging (MRI) of the brain showed moderate generalized cerebral atrophy without focal lesions (Fig. 2). Electroencephalography showed diffuse slowing without evident focal abnormalities. Lumbar puncture revealed increased protein (0.82 g/l) without any other abnormalities. Electromyography showed a moderately severe axonal polyneuropathy. An apomorphine test was done as described [9]. The initial dose was 0.1 ml apomorphin-HCl 1% (= 10 mg/ml). Sequential subcutaneous injections were given at hourly intervals increasing the dose by 0.1 ml at each step. When 0.8 ml did not result in any clinical response, a final injection of 10 ml was given. With this dose the patient did not show any improvement on motor performances, so levodopa treatment was not started.

A diagnosis of alcohol withdrawal parkinsonism was reached.

During the next 12 weeks the parkinsonian features disappeared completely without specific treatment. The patient was able to walk without any support. A new electromyogram did not show any improvement of the axonal polyneuropathy. Mental functions improved only partially.
3. Discussion

We describe here a 60-year-old chronic alcoholic, who developed severe parkinsonism after he stopped drinking. The parkinsonian syndrome consisted of expressionless face, positive primitive reflexes, tremor of the extremities and severe 'lead-pipe' rigidity of the trunk and extremities. No family history of Parkinson's disease was present, nor use of neuroleptic drugs or intoxications. Parkinsonism associated with disulfiram was ruled out since the total amount of disulfiram taken was not high enough to cause parkinsonian symptoms: this syndrome has been reported in patients taking disulfiram for several months at dosages of 1–2 g/day and in patients with acute autointoxication involving more than 25 g of disulfiram [10–12]. Marchiava-Bignami disease and central pontine myelinolysis [1] were ruled out by negative brain imaging. Therefore alcohol withdrawal parkinsonism was diagnosed.

Carlen et al. [4] were the first to describe 4 patients, alcoholics, in whom parkinsonian features developed within two days after cessation of drinking. Table 1 summarizes the described cases. Withdrawal parkinsonism can include tremors, bradykinesia or dyskinesias. All patients showed cortical atrophy on CT scan, reflecting long-lasting alcohol abuse. As described in the other cases our patient also recovered completely without specific therapy. The unchanged electromyogram demonstrated that the axonal polyneuropathy did not contribute to his gait disturbance.

The cause of alcohol withdrawal parkinsonism remains obscure. Striatal dopamine release is reduced during the first few days of alcohol withdrawal [13], although this does not explain the long duration of the syndrome. In mice ethanol withdrawal is associated with diminished responsiveness of striatal dopamine-sensitive adenyl cyclase activity [14]. As most patients have had prior episodes of parkinsonism during alcohol withdrawal, Carlen et al. [4] proposed that the described patients had underlying parkinsonism that was intensified by alcohol withdrawal. However, long term follow-up of three of these patients did not show development of parkinsonism, which argues against this hypothesis [15–16]. Testing the same hypothesis, Lang et al. [5] surveyed the drinking habits of 125 idiopathic Parkinson's disease patients and found no difference from a control population concerning the amount of alcohol consumed. Effects of alcohol on parkinsonian symptoms were only mild in a small percentage (16%) of patients.

The negative results of the apomorphine test in our patient favor the hypothesis of a disturbance in the postsynaptic dopaminergic transmission pathway. This accords with the observation by Tabakoff et al. [17] who demonstrated effects of chronic ethanol on the lipid microenvironment containing the postsynaptic dopamine receptor. Future molecular dopamine receptor studies will give more insight in the complex interaction between chronic alcohol and dopaminergic neurotransmission [18].

Acknowledgements

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References


Table 1
Described cases of alcohol withdrawal parkinsonism

<table>
<thead>
<tr>
<th>Reference</th>
<th>Sex</th>
<th>Age (yrs)</th>
<th>Recovery</th>
<th>Comments</th>
</tr>
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<tr>
<td>Carlen et al. [4]</td>
<td>M</td>
<td>53</td>
<td>5 days</td>
<td>2 prior episodes</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>62</td>
<td>6 weeks</td>
<td>tremor for 1 year</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>64</td>
<td>1 week</td>
<td>2 prior episodes</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>70</td>
<td>7 weeks</td>
<td>2 years shuffling gait</td>
</tr>
<tr>
<td>Lang et al. [5]</td>
<td>M</td>
<td>54</td>
<td>4 months</td>
<td>4–5 prior episodes</td>
</tr>
<tr>
<td>Shen [6]</td>
<td>M</td>
<td>73</td>
<td>2 months</td>
<td>2 prior episodes</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>53</td>
<td>7 days</td>
<td>1 prior episode</td>
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<tr>
<td></td>
<td>M</td>
<td>50</td>
<td>1 week</td>
<td>2 prior episodes</td>
</tr>
<tr>
<td>Neiman et al. [7]</td>
<td>M</td>
<td>55</td>
<td>4 months</td>
<td>several prior episodes</td>
</tr>
<tr>
<td>Luyckx et al.</td>
<td>M</td>
<td>60</td>
<td>3 months</td>
<td>no prior episode</td>
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