Acute dystonia due to amitriptyline

Acute dystonic reactions are most frequently seen in patients receiving neuroleptic medication or metoclopramide but have also been observed in association with phenytion and carbamazepine. Although it is generally believed that the anticholinergic and dopamine-blocking properties of these drugs cause such reactions the precise mechanism is unclear although it has been suggested that they may be due to enhanced dopaminergic compensatory release on supersensitive post-synaptic receptors. However, most explanations cannot fully explain why only a small proportion of patients develop acute dystonia or why it may occur durin chronic therapy. A 20 year old man was admitted with severe muscular spasm. He had first been aware of stiffness in his lower limbs whilst jogging on the day of admission. He then developed spontaneous arching of his back and involuntary tongue protrusion. His past medical history was unremarkable but because of a depressive illness he had been taking amitriptyline 50 mg daily for three months prior to his admission. He denied taking any other medication. A subsequent examination of his tablets confirmed they were amitriptyline. Examination revealed marked opisthotonus, reticulosis and orofacial contrortion with spontaneous tongue protrusion. Intravenous procyclidine (10 mg) terminated the attack. Routine haematological and biochemical indices were normal.

Amitriptyline and other tricyclic antidepressants only rarely cause extrapyramidal side-effects although tremor, dystonia and akathisia have been observed. There are two previous reports of acute dystonia due to amitriptyline 4 and we believe this patient is a further case. Whilst tricyclic antidepressants have anti-cholinergic properties and potentiate the actions of biological amines in the central nervous system the mechanism of this reaction seems unclear. However, the drugs are widely used and it is a side effect that prescribers should be aware of.

D ORNADEL
EA BARNES
DJ DICK
Norfolk and Norwich Hospital,
Norwich, Norfolk, UK

Paradoxical akinesic response to apomorphine in Parkinsonism

Apomorphine is a direct D1 and D2 dopaminergic receptor agonist, and its efficacy in Parkinson’s disease (PD) depends on intact post-synaptic receptors. Administration by subcutaneous injection and intranasally, has been beneficial in Parkinsonian patients with declining motor response, intractable on/off fluctuations which are commonly accompanied by dyskinesia, and other non-motor chiasitc symptoms. We describe studies in a patient who showed a hitherto unreported profound akinesic response to the drug. A 60 year old man presented in 1988 with three months of lethargy, slowness of movement and slurred speech. Examination showed an extrapyramidal type of dystarhria, facialis hypomimia, reduced spontaneous and automatic movements, asymmetrical bradykinesia of both upper limbs, micrographia and a shuffling short stepped gait. There was no tremor, no supranuclear palsy and no signs of autonomic desenervation; rigidity was minimal in axial muscle groups. Disability was minimal and treatment was withheld.

Over the next year he deteriorated with increasing gait disorder, difficulty with stairs and reduced arm-swing, but no tremor. In July 1989 he was given Sinemet plus, three times daily, without improvement. He was admitted for further investigation and treatment. Examination confirmed the previous signs except there was diminished arm-swing, slight postural flexure and masked facies; tremor was absent, rigidity minimal.

Routine haematological, biochemical, intravenous edrophonium tests and CT head scan were normal. EMG showed no myasthenic reaction.

All drugs were withdrawn for 24 hours. An oral dose of 2 tablets of Madopar (levodopa 400 mg, benzbenzide 100 mg) given at 9.00 am produced no significant change in the Webster rating measured 1 hour for 3 hours (table). On a separate day, on domperidone 20 mg, 8 hours and apomorphine 2 mg, 4 mg and 6 mg were administered subcutaneously at 8 hours intervals. Serial Webster scores recorded over 2 hours.

Apomorphine 4 mg produced no change in score at 5 and 10 minutes. At 15 minutes he became totally immobile and mute, lying on his bed, conscious but apparently drowsy and sweating. There was no voluntary movement to commands, muscle tone was not obviously altered from his pre-treatment state. Eyes were closed, mouth slightly open, no abnormal movements were seen. Webster scores are shown in the table. This state continued until 90 minutes when he walked to the office door and his Webster score had returned to basal values. Identical episodes, with profound akinesia, resembling a very severe “off” period occurred with both 2 mg and 6 mg doses. On the latter dose there was a short period of pre-syncope, BP 90/60 mm Hz, pulse 52/min.

The batch of apomorphine was assayed by the manufacturer’s and its potency and freedom from contaminlants were confirmed.

The diagnosis of idiopathic PD is excluded by bilateral signs at presentation, lack of tremor and lack of response to levodopa and other drugs. 3 The probable diagnosis is striatogniral degeneration, with no current evidence of nigral degeneration, dopa-responsive dystonia or symptomatic levodopa-resistant parkinsonism.

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>0</th>
<th>5</th>
<th>10</th>
<th>15</th>
<th>30</th>
<th>60</th>
<th>90</th>
<th>120</th>
<th>150</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oral Madopar × 2 (400 mgms Levodopa)</td>
<td>6</td>
<td>4</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>Apomorphine (Subcutaneous)</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>6</td>
</tr>
<tr>
<td>2 mg</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>20</td>
<td>23</td>
<td>22</td>
<td>15</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>4 mg</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>19</td>
<td>19</td>
<td>12</td>
<td>3</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>6 mg</td>
<td>3</td>
<td>3</td>
<td>19</td>
<td>21</td>
<td>21</td>
<td>20</td>
<td>19</td>
<td>5</td>
<td></td>
</tr>
</tbody>
</table>

Correspondence to: Dr Dick.

Acute dystonia due to amitriptyline.

D Ornadel, E A Barnes and D J Dick

*J Neurol Neurosurg Psychiatry* 1992 55: 414
doi: 10.1136/jnnp.55.5.414

Updated information and services can be found at:
http://jnnp.bmj.com/content/55/5/414.1.citation

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
http://group.bmj.com/group/rights-licensing/permissions

To order reprints go to:
http://journals.bmj.com/cgi/reprintform

To subscribe to BMJ go to:
http://group.bmj.com/subscribe/