A rare case of dicyclomine abuse

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A B S T R A C T

This report highlights an extremely rare case of dicyclomine abuse for 1.5 years by an 18-year old female resulting in overt features of anticholinergic toxicity. Strict abstinence and rehabilitation measures were employed to revert back the altered physiological state.

1. Introduction

Dicyclomine is an anticholinergic tertiary amine used frequently by oral and parenteral route as an effective anti-spasmodic agent. It is also used in morning and motion sickness, dysmenorrheal, intestinal hypermotility and irritable bowel syndrome. It is a smooth muscle relaxant. It can cause a range of anticholinergic side effects and, at higher doses, deliriant effects. Being a tertiary amine, it can cross blood brain barrier and may result in physical dependence.1–3 Although extremely rare, it has some prominent abusive potentials also.4

2. Case summary

An 18-year old unmarried female patient (body weight 58 kg) from Kolkata, India, studying in 1st year in college, and belonging to a nuclear family with good socio-economic condition, was admitted in a confused state with fever for last 1.5 months associated with severe generalized weakness and palpitations for the same duration. The fever was continuous (100–101 °F), without any diurnal variation, not associated with chill and rigor, and temporarily relieved by oral paracetamol. There was associated progressive blurring of vision, dryness in eyes, chronic constipation and diminished urinary frequency but with maintained urine volume. There were also presence of diminished sweating and dry skin with difficulty in talking and swallowing for the last 6 months.

Repeated enquiry to the family members revealed that for the last 1-year, the patient used to experience occasional euphoria, hallucinations, fatigue, short-term memory loss with some short lasting episodes of altered behaviors including decreased anxiety, mannerisms and agitation. She had no sexual activities and menstrual habit was normal and regular.

A curious interrogation to the family members and later to the patient herself unveiled that the patient was absolutely normal 1.5 years back, just before she had an attack of an acute infective enterocolitis causing fever, diarrhea and severe spasmodic intestinal pain. She was then prescribed oral paracetamol, norfloxacin and intramuscular dicyclomine. Although the symptoms were completely relieved within a week, the patient started to take self injected intramuscular dicyclomine since then on a regular basis, without any medical indications or prescriptions. The reason could not be properly explained by the patients or by the family members, but some familial stress events could have played a role. She used to procure the drug regularly from a local familiar dispensary, by producing the original prescription again and again. The dose varied from 20 to 40 mg once to thrice daily 3–5 times per week throughout this entire period but of late, the frequency increased.

There were no evidences of any other significant medical or surgical history or any other hereditary, traumatic, infective,
muscles with normal responses to light. There was no urinary retention. Respiratory rate of 22 per minute. Pupil was moderately dilated and reactive. Temperature 100.3 °F. Laboratory tests showed normal results except for elevation in liver enzymes and bilirubin. Serum creatinine 0.8 mg/dL, blood urea nitrogen 15.1 mg/dL, serum creatinine 0.5 mg/dL, serum sodium 138 mg/dL, serum potassium 4.1 mg/dL, total bilirubin 0.7 mg/dL, direct bilirubin 0.2 mg/dL, indirect bilirubin 0.5 mg/dL, serum glutamic-oxaloacetic transaminase (SGOT) 21 U/L, serum glutamic-pyruvic transaminase (SGPT) 24 U/L, alkaline phosphatase 66 IU/L, albumin 3.8 g/dL, globulin 3.2 g/dL.

Relevant laboratory results on admission.

<table>
<thead>
<tr>
<th>Parameters detected</th>
<th>Detected values</th>
<th>Normal range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total cholesterol</td>
<td>160 mg/dL</td>
<td>&lt;200 mg/dL</td>
</tr>
<tr>
<td>LDL cholesterol</td>
<td>75.1 mg/dL</td>
<td>&lt;100 mg/dL</td>
</tr>
<tr>
<td>HDL cholesterol</td>
<td>54.4 mg/dL</td>
<td>40–60 mg/dL</td>
</tr>
<tr>
<td>Triglyceride</td>
<td>30.5 mg/dL</td>
<td>6–40 mg/dL</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>0.75 mg/dL</td>
<td>0.3–1.3 mg/dL</td>
</tr>
<tr>
<td>Direct bilirubin</td>
<td>0.2 mg/dL</td>
<td>0.1–0.4 mg/dL</td>
</tr>
<tr>
<td>Indirect bilirubin</td>
<td>0.5 mg/dL</td>
<td>0.2–0.9 mg/dL</td>
</tr>
<tr>
<td>Serum glutamic-oxaloacetic transaminase (SGOT)</td>
<td>21 U/L</td>
<td>12–38 U/L</td>
</tr>
<tr>
<td>Serum glutamic-pyruvic transaminase (SGPT)</td>
<td>24 U/L</td>
<td>7–41 U/L</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>66 IU/L</td>
<td>20–140 IU/L</td>
</tr>
<tr>
<td>Albumin</td>
<td>4.8 g/dL</td>
<td>4.0–5.0 g/dL</td>
</tr>
<tr>
<td>Globulin</td>
<td>3.2 g/dL</td>
<td>2.3–3.5 g/dL</td>
</tr>
</tbody>
</table>

Organic or pathologic diseases and events. 6 months back she took oral azithromycin 500 mg for 3 days for upper respiratory tract infection. There was no history of any other concomitant medications during the entire period.

Physical examination revealed a confused state with altered higher functions manifesting as disorientation, confusion, dysarthria and ataxia. Vitals showed a regular pulse rate of 120 per minute, blood pressure 100/84 mm of Hg, temperature 100.3 °F and respiratory rate of 22 per minute. Pupil was moderately dilated and sluggishly responsive to light. There was no urinary retention. Motor examination showed a diminished power of lower limb muscles with normal reflexes. There were multiple needle puncture marks with erythema and multiple small tender nodules on both arms, more on the left. Other systemic findings were within normal limits.

Routine blood investigation affirmed a normal picture. Septic conditions were ruled out and investigations showed no septic episode. An electrocardiogram revealed sinus tachycardia. Chest X-ray, USG whole abdomen and CT scan of brain were also within normal limits.

She was suffering from anticholinergic toxicities. Immediate treatment was started with slow injections of 2 mg intravenous physostigmine. After repetitive doses higher functions and muscle power gradually reverted to normal. Fever was treated with oral paracetamol and cold sponging. No respiratory resuscitation or urinary catheterization was required. Subsequent symptomatic treatment was continued for the next 5 days, when the general condition got much improved. However, there were some episodes of drug craving, and withdrawal reactions were often present in the forms of nervousness, anxiety, sweating, weakness, depression, anorexia and hypertension.

The patient was then referred to a drug rehabilitation center where a strict abstinence from dicyclomine was followed. Symptomatic therapy was continued. During the initial phases, she showed other withdrawal reactions like insomnia, agitation, occasional tremors, abdominal cramps and blurred vision. At the end of subsequent 6 months of follow up, the patient is quite normal now, with absolutely no drug seeking behavior. Physical and psychological examinations are also well within normal limits.

3. Discussion and conclusion

The above clinical picture corroborated with physical examination and investigation reports could be explained by anticholinergic toxicities attributed to dicyclomine abuse. It essentially appears here that the patient was subjected to dicyclomine abuse chronically for the last 1.5 years. This could have resulted in psychological dependence and habituation. Once that caused therapeutic benefit had been converted into reinforcing and compulsive substance dependence with gradual increasing intake, indicating tolerance, which might have been a ‘conditioned’ one. The altered psychological status as evident by hallucinations, euphoria, diminished anxiety and pleasure feelings were responsible for the continuous irrational intake of the drug by the medical addict. Degree of physical dependence was however questionable. Different physiological anticholinergic effects were clearly manifested systemically.

This is an extremely rare report of drug abuse with parenteral dicyclomine. The abuse liability might have been enhanced by the rapidity of onset of effects after administration that initiated the sequence of events that lead to loss of control over drug taking. Strict rehabilitation measures followed were just appropriate to attempt a slow and justified reversal of the altered physiological state in this young patient. Therapeutic uses of potential addictive drugs should be coupled with strict vigilance and effective counseling of patients and family members to prevent in such an abuse.

Conflicts of interest

All authors have none to declare.

References