Atropine Premedication in Electroconvulsive Therapy

Mehrad Mahdian, Samad Noorizad, Goudarz Akasheh, Gholamabbas Mousavi, Abdollah Omidi

Abstract- Atropine has been used in ECT to eliminate parasympathetically mediated dysrhythmias. However, it may increase heart rate and myocardial workload. The aim of this study was to investigate the effect of atropine premedication on the heart rate and blood pressure. Study was performed in 80 patients referred for a course of ECT. They enrolled randomly in one of the two equal groups to receive atropine premedication or placebo. ECT protocol was the same for both groups except for atropine 0.5 mg which injected intravenously in the case group. There was no significant difference between heart rate and MAP in atropine 0.5 mg group compared to placebo at the most of the time points. We concluded the use of intravenous atropine 0.5 mg prior to induction of ECT anesthesia has no significant effect on heart rate and MAP while it may has some protective effect against bradyarrhythmias.

Keywords—Blood pressure, Electroconvulsive therapy, Heart rate.

I. INTRODUCTION

MAJOR depression is a recurrent, syndromal illness that involves both psychological and physiologic components. It can be treated with the combination of psychopharmacologic agents and psychotherapy. Patients whose severe depression has not responded to pharmacologic therapy are candidate for electroconvulsive therapy (ECT). Although use of this therapy declined through 1970s because of negative publicity, acceptance and use of ECT have again increased. Current acceptance of this procedure is in part due to use of general anesthesia to reduce the physical and psychological trauma associated with ECT. [1]

Cardiovascular changes occur consistently during ECT [2]. Cardiovascular responses to ECT include significant hemodynamic changes that may result in complications, even in patients without preexisting cardiovascular conditions [3]. Seizure activity causes an initial parasympathetic discharge manifested by bradycardia, occasional asystole, premature atrial and ventricular contractions, or a combination of these abnormalities. The parasympathetic discharge is followed immediately by sympathetic discharge associated with tachycardia, hypertension, premature ventricular contraction and rarely ventricular tachycardia. The tachycardia peaks at 2 minutes after the stimulus and is normally self-limited [1]-[4]. Acute increases, but albeit transient, occur in heart rate and arterial blood pressure, and hence rate pressure product (RPP), an index of myocardial oxygen consumption. The increase in RPP during ECT can create an imbalance between myocardial oxygen supply and demand [2]. In contemporary ECT practice, subconvulsive stimuli used in stimulus titration during ECT, increases a risk of bradyarrhythmias [5]. Atropine has been used in ECT to eliminate parasympathetically mediated dysrhythmias. However, it may increase heart rate and myocardial workload and may increase risk of cardiac adverse events [6]. Therefore, some psychiatrists avoid atropine during ECT [2]. This study was designed to examine the effect of atropine on heart rate and blood pressure in a randomized, controlled trial during the third ECT session.

II. METHODS

After obtaining approval from our local IRB 80 patients referred for a course of ECT (mean age 34.17(range 20 – 50) yr; 62 males) enrolled the study. All patients signed written consent. The inclusion criterion was age > 20 years. Exclusion criterion was presence of cardiologic condition. Patients continued to receive concurrent psychotropic drugs before the ECT session; these were neuroleptics (n=61), selective serotonin reuptake inhibitors (n=19), tricyclic antidepressants (n=16). All patients were ASA grade I. During the third session of ECT patients allocated randomly in one of two equal groups to receive atropine premedication or placebo. ECT procedure was achieved using atropine 0.5 mg (if given), thiopental 3 mg/kg and succinylcholine 1 mg/kg I.V, in that order. In control group and in cases who did not receive atropine 1 ml distilled water injected as a placebo. The psychiatrist and anesthesiologist were blinded to the treatment group. Intermittent positive pressure ventilation with 100% oxygen was provided until resumption of spontaneous and regular breathing. ECT was administered using a constant current bidirectional brief pulse ECT device (mean stimulus dose 136(SD 38.6) mC). In third ECT session, patients received threshold bilateral ECT and developed a seizure in
response to the first stimulus in the session. Cardiovascular monitoring was performed using automated cardiac monitor (pulse oximetry, non-invasive arterial pressure and heart rate). Cardiovascular recording were made just before anesthesia (baseline values), 1 minute after giving drugs (induction) and 1, 3 and 5 minutes after the stimulus.

The software package SPSS (v.13) was used for statistical analysis. Mean arterial pressure (MAP) and pulse rate in the two groups after induction of anesthesia and stimulus were compared with baseline values. Mann-Whitney test and t-test were used for analysis. All analyses were performed using α=0.05.

### III. RESULTS

The two groups were comparable in patient's age, sex and stimulus dose. There was no significant difference between heart rate and MAP in atropine 0.5 mg group compared to placebo at the most of the time points. Among different time points only a significant higher heart rate was observed in atropine group in minute 5 after stimulus (Table I). Also the only significant increase in MAP was in minute 3 after stimulus in atropine group (Table II).

#### TABLE I

<table>
<thead>
<tr>
<th></th>
<th>HR0 Mean(SD)</th>
<th>HRI Mean(SD)</th>
<th>HRS1 Mean(SD)</th>
<th>HRS3 Mean(SD)</th>
<th>HRS5 Mean(SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Atropine (bpm)</strong></td>
<td>88.32(19.01)</td>
<td>100.90(21.10)</td>
<td>102.77(28.55)</td>
<td>104.27(21.76)</td>
<td>105.75(21.34)</td>
</tr>
<tr>
<td><strong>Placebo (bpm)</strong></td>
<td>85.65(15.88)</td>
<td>96.47(15.82)</td>
<td>99.27(19.44)</td>
<td>96.27(17.22)</td>
<td>96.37(14.95)</td>
</tr>
<tr>
<td><strong>P.V</strong></td>
<td>0.316</td>
<td>0.085</td>
<td>0.524</td>
<td>0.072</td>
<td>0.026</td>
</tr>
</tbody>
</table>

HR0= heart rate before induction of anesthesia (baseline), HRI= heart rate 1 min after induction of anesthesia, HRS1, HRS3, HRS5= heart rate 1, 3, and 5 min after stimulus respectively, bpm=beat per minute.

#### TABLE II

<table>
<thead>
<tr>
<th></th>
<th>MAP0 Mean(SD)</th>
<th>MAPI Mean(SD)</th>
<th>MAPS1 Mean(SD)</th>
<th>MAPS3 Mean(SD)</th>
<th>MAPS5 Mean(SD)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Atropine (mmHg)</strong></td>
<td>8.76(1.47)</td>
<td>10.03(2.0)</td>
<td>10.85(2.38)</td>
<td>10.40(2.27)</td>
<td>9.63(1.87)</td>
</tr>
<tr>
<td><strong>Placebo (mmHg)</strong></td>
<td>8.75(1.09)</td>
<td>9.75(1.50)</td>
<td>10.18(1.81)</td>
<td>9.46(1.76)</td>
<td>9.02(1.57)</td>
</tr>
<tr>
<td><strong>P.V</strong></td>
<td>0.973</td>
<td>0.479</td>
<td>0.164</td>
<td>0.043</td>
<td>0.116</td>
</tr>
</tbody>
</table>

MAP0= Mean arterial pressure before induction of anesthesia (baseline), MAPI= Mean arterial pressure 1 min after induction of anesthesia, MAPS1, MAPS3, MAPS5= Mean arterial pressure 1, 3, and 5 min after stimulus respectively.

### IV. DISCUSSION

This Study showed that use of atropine 0.5 mg as premedication for ECT maybe effective to prevent of bradycardia with not significant raise in heart rate and MAP.

Several controlled trials evaluated cardiovascular parameters in ECT in which seizures modified by atropine were compared with seizures without any anticholinergic drug or placebo. Wynat and MacDonald (1980) administered either atropine (1.0 or 0.5 mg) or placebo before ECT anesthesia. They did not find significant difference in heart rate at any time point in their study groups. They finally suggested there is no need for routine use of anticholinergic agents [7]. Rasmussen et al. During a randomized trial, used low-dose atropine in ECT. They administered 0.006 mg/kg intravenous atropine (typical atropine dose=0.3-0.6mg).
before ECT anesthesia. They concluded low-dose atropine effectively blocked vagal tone with a small and probably not clinically significant rise in myocardial workload [6]. Our study results appear in agreement with findings of two mentioned studies. On the other hand, Mayur et al. (1998) used atropine premedication and considered its effect on rate pressure product. They found that mean RPP was attenuated when atropine premedication was withheld [2]. Their results are against our findings. This discrepancy between their results and ours seem to be due to use of different dosage of atropine in two studies. We used 0.5 mg atropine, whereas they administered 0.15mg/kg. The uses of higher dose of atropine in Mayur’s study maybe the reason of this difference.

Finally, we concluded the use of intravenous atropine 0.5mg prior to induction of ECT anesthesia has no significant effect on heart rate and MAP while it may has some protective effect to prevention of bradyarrhythmias.

ACKNOWLEDGMENT

The authors gratefully acknowledge Deputy of Research of Kashan University of Medical Sciences for its supports. We also thank Mr. Amir Motamed nejad for his help in type of the manuscript.

REFERENCES