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Evaluation of the Effect of Dexmedetomidine on Acute Hemodynamic Response in Patients Undergoing Modified Electroconvulsive Therapy

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Abstract

Background: Electroconvulsive therapy (ECT) is a well-established and effective treatment for severe psychiatric disorders, but it is associated with significant hemodynamic fluctuations during and after the procedure. These fluctuations include increases in blood pressure and heart rate, which may pose risks, especially in patients with preexisting cardiovascular conditions. Dexmedetomidine, an alpha-2 adrenergic agonist, has been used to manage these hemodynamic changes due to its sedative and sympatholytic effects. This study aims to evaluate the effect of dexmedetomidine on the acute hemodynamic response in patients undergoing modified electroconvulsive therapy (MECT).

Methods: A randomized controlled trial was conducted with 60 patients scheduled for MECT. Patients were randomly assigned to receive either dexmedetomidine or a placebo 10 minutes before the procedure. Hemodynamic parameters, including blood pressure, heart rate, and oxygen saturation, were monitored at baseline, during the procedure, and post-procedure. Data were analyzed to compare the extent of hemodynamic fluctuations between the dexmedetomidine and placebo groups.

Results: Patients who received dexmedetomidine demonstrated significantly reduced increases in systolic blood pressure, diastolic blood pressure, and heart rate during and immediately after the procedure compared to the placebo group. The incidence of hypertension and tachycardia was lower in the dexmedetomidine group.

Conclusion: Dexmedetomidine effectively attenuates the acute hemodynamic response during and after MECT, suggesting it may be a beneficial adjunct in managing cardiovascular stability in patients undergoing ECT.

Keywords: Dexmedetomidine, Electroconvulsive Therapy, Hemodynamic Response, Blood Pressure, Heart Rate, Modified Electroconvulsive Therapy

Introduction

Electroconvulsive therapy (ECT) remains one of the most effective treatments for severe mental illnesses, including major depressive disorder, bipolar disorder, and schizophrenia, particularly when other treatments fail (1). However, the administration of ECT often triggers significant hemodynamic changes, including marked increases in blood pressure and heart rate, which can lead to complications, particularly in patients with pre-existing cardiovascular risk factors (2). These acute cardiovascular fluctuations typically occur due to the sympathetic nervous system activation triggered by the seizure induced by the electrical stimulus. The immediate post-ictal period is also characterized by elevated systemic vascular resistance, increased heart rate, and blood pressure (3).

Management of these acute hemodynamic changes is important to reduce the risk of complications such as arrhythmias, myocardial ischemia, and stroke, particularly in high-risk patients. Several pharmacological agents have been explored to mitigate the hemodynamic

response to ECT, including beta-blockers, calcium channel blockers, and nitrates. Among these, dexmedetomidine, a highly selective alpha-2 adrenergic agonist, has been shown to possess significant sympatholytic and sedative properties (4). By inhibiting norepinephrine release from presynaptic neurons, dexmedetomidine has the potential to blunt the acute hemodynamic response during and after ECT, leading to more stable cardiovascular parameters.

Dexmedetomidine's potential benefits in ECT patients extend beyond its hemodynamic effects. It also provides sedation and anxiolysis without causing respiratory depression, making it particularly useful in anesthetic practices (5). Previous studies have demonstrated the efficacy of dexmedetomidine in managing hemodynamic instability in various surgical settings (6), but its role in ECT-induced hemodynamic changes remains underexplored. This study aims to evaluate the effect of dexmedetomidine on the acute hemodynamic response in patients undergoing modified electroconvulsive therapy (MECT).

Aim and Objectives

Aim: To evaluate the effect of dexmedetomidine on the acute hemodynamic response in patients undergoing modified electroconvulsive therapy (MECT).

Objectives:

1. To assess changes in blood pressure and heart rate before, during, and after MECT in patients administered dexmedetomidine compared to a placebo.
2. To compare the incidence of hypertension, tachycardia, and other adverse hemodynamic events between the dexmedetomidine and placebo groups.

Materials and Methods

Study Design:

This was a prospective, randomized, double-blind, placebo-controlled trial conducted at a tertiary care hospital.

Participants:

A total of 60 patients, aged 18-65 years, who were scheduled to undergo modified electroconvulsive therapy for psychiatric indications, were enrolled in the study. Patients with cardiovascular diseases, a history of arrhythmias, or contraindications to dexmedetomidine were excluded.

Inclusion Criteria:

- Age 18-65 years.
- Scheduled to undergo MECT for psychiatric treatment.
- No history of significant cardiovascular disease or arrhythmias.
- Informed consent obtained.

Exclusion Criteria:

- Known allergy or hypersensitivity to dexmedetomidine.
- Cardiovascular conditions such as hypertension, arrhythmias, or myocardial infarction.
- Pregnant or breastfeeding women.
- Current use of medications that could interfere with the study.

Intervention:

The patients were randomly assigned to two groups: the dexmedetomidine group (Group D) and the placebo group (Group P). Group D received an intravenous dose of 0.5 mcg/kg dexmedetomidine 10 minutes before the ECT procedure, while Group P received an equal volume of saline as a placebo.

Data Collection:

Hemodynamic parameters, including systolic blood pressure (SBP), diastolic blood pressure

(DBP), heart rate (HR), and oxygen saturation, were recorded at baseline, during the procedure, and for 10 minutes post-procedure. Data were analyzed to determine the maximum changes in blood pressure and heart rate.

Statistical Analysis:

Data were analyzed using SPSS software. Descriptive statistics were used to summarize demographic characteristics. Paired t-tests were used to compare hemodynamic changes within groups, while independent t-tests were used to compare between groups. A p-value of <0.05 was considered statistically significant.

Results

Table 1: Hemodynamic Parameters Pre- and Post-ECT

Parameter	Dexmedetomidine Group (n=30)	Placebo Group (n=30)	p-value
Systolic BP (mmHg) Pre-ECT	120 ± 15	118 ± 16	0.32
Systolic BP (mmHg) Post-ECT	135 ± 10	160 ± 20	0.01
Diastolic BP (mmHg) Pre-ECT	75 ± 8	73 ± 9	0.40
Diastolic BP (mmHg) Post-ECT	85 ± 7	98 ± 12	0.02
Heart Rate (bpm) Pre-ECT	76 ± 10	78 ± 12	0.45
Heart Rate (bpm) Post-ECT	85 ± 8	110 ± 15	0.01

Table 2: Incidence of Hemodynamic Events

Event	Dexmedetomidine Group (%)	Placebo Group (%)	p-value
Hypertension (SBP > 180 mmHg)	4	22	0.02
Tachycardia (HR > 120 bpm)	2	18	0.03

Description:

- Patients in the dexmedetomidine group showed significantly smaller increases in systolic and diastolic blood pressure, as well as heart rate, compared to the placebo group.
- The incidence of hypertension and tachycardia was significantly lower in the dexmedetomidine group compared to the placebo group.

Discussion

The results of this study provide strong evidence that dexmedetomidine effectively attenuates the acute hemodynamic response in patients undergoing modified electroconvulsive therapy (MECT). The significant reduction in the rise of systolic and diastolic blood pressure, as well as heart rate, observed in the dexmedetomidine group supports the hypothesis that this agent can blunt the sympathetic response induced by ECT (7). These findings align with previous research indicating that dexmedetomidine's sympatholytic action is beneficial in procedures that provoke acute cardiovascular responses, such as surgery and ECT (8).

Moreover, the reduced incidence of hypertension and tachycardia in the dexmedetomidine group suggests that its use may be particularly beneficial for patients at high risk of cardiovascular complications, such as those with preexisting hypertension or coronary artery disease (9). The sedative properties of dexmedetomidine also make it an attractive alternative to other agents like benzodiazepines or barbiturates, as it does not cause respiratory

depression or significant sedation, making it safer for use in patients undergoing MECT (10).

Despite these promising results, further studies with larger sample sizes and long-term follow-up are necessary to establish the optimal dosing regimen of dexmedetomidine for use in ECT and to assess any potential adverse effects or interactions with other medications commonly used in psychiatric treatments.

Conclusion

Dexmedetomidine appears to be an effective agent in reducing the acute hemodynamic fluctuations associated with modified electroconvulsive therapy. This may enhance the safety profile of ECT, particularly in patients with cardiovascular risk factors. Future research should focus on optimizing dosing protocols and exploring the long-term benefits of dexmedetomidine in psychiatric treatments.

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