

The Incidence and Predictors of Headache and Myalgia in Patients After Electroconvulsive Therapy (ECT)

Mohammad Haghighi,¹ Abbas Sedighinejad,¹ Bahram Naderi Nabi,^{1,3} Cyrus Emiralavi,¹ Gelareh Biazar,¹ Kaveh Mirmozaffari,¹ Cyrus Zahedan,² and Mehdi Jafari¹

¹Anesthesiology Research Center, Guilan University of Medical Sciences (GUMS), Rasht, Iran

²Shafa Hospital, Rasht, Iran

*Corresponding author: Bahram Naderi Nabi, Anesthesiology Research Center, Guilan University of Medical Sciences (GUMS), Rasht, Iran. Tel/Fax: +98-2122297895, E-mail: Naderi_bahram@yahoo.com

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Abstract

Background: Electroconvulsive therapy (ECT) is a safe and effective mode of therapy for a wide variety of psychiatric disorders. However, it is associated with some disturbing side effects, such as nausea and vomiting, dental and tongue injury, confusion, dizziness, headache, and myalgia.

Objectives: The present study focused on the evaluation of myalgia and headache and their predictors after ECT.

Patients and Methods: A prospective analytical descriptive study was conducted from October 2014 to January 2015, in an academic hospital in northern Iran. Before sampling, the study was approved by the ethics committee of Guilan University of Medical Sciences. 621 patients with psychiatric disorders who were referred to Shafa hospital enrolled in the study. They were evaluated based on a verbal rating scale (4 point scales) 6 hours after ECT, regarding headache and myalgia side effects.

Results: 6 hours after ECT, 126 patients (21.9%) reported headaches, and 56 patients (9%) reported myalgia. The presence of headache or myalgia 6 hours after ECT was not correlated to the duration of convulsion, treatment sessions, sex, or age. But myalgia at 2 hours after treatment was correlated with sex (0.04). Sex, age, duration of seizure, and treatment sessions were not predictors of headache and myalgia 6 hours after ECT (log regression, enter mode). The intensity and frequency of headaches decreased during 6 hours after ECT ($P = 0.0001$ and $P = 0.0001$, respectively), and myalgia frequency decreased ($P = 0.062$) but the intensity increased ($P = 0.87$).

Conclusions: The results of the present study demonstrate that headache after ECT procedures was more common than myalgia, but it was mild, tolerable, and decreased within 6 hours of the treatment. It is also notable that we did not find any predictors for post-ECT headache and myalgia.

Keywords: Myalgia, Headache, Electroconvulsive Therapy (ECT), Predictors

1. Background

Electroconvulsive therapy (ECT) is the oldest surviving physical treatment for psychiatric disorders. Studies have noted that major depression is the most common indication for an ECT procedure. However, it is also safe and effective in a wide variety of conditions, such as mania, schizophrenia, and acute catatonia, and also in patients with severe and drug resistant psychiatric diseases or those whose treatment is limited by unacceptable side effects (1-6). Although studies have indicated ECT's efficacy and low mortality and morbidity rate, a few adverse events have been reported: oral cavity damage to tongue, teeth, and gums; damage to implants or intraosseous denture supports; confusion; dizziness; nausea and vomiting; headache, and myalgia (1, 3, 7).

In the United States about 100,000 patients receive electroconvulsive therapy (ECT) per year, and more than 45% of them report remarkable headaches after treatment

(7). Past studies indicate that ECT induces headaches and exacerbates previous headaches such as migraines. These headaches have been reported to be mild, moderate, or severe and in some cases are refractory and resistant to available treatments (8, 9). The precise cause of headache after ECT is not clearly known. However, studies have hypothesized that contracture of the temporalis and masseter muscles and vasodilation may play an important role (10, 11).

The other well-known side effect after ECT is myalgia. The prevalence of post-ECT myalgia is not clear worldwide, due to the lack of evidence and correlation of myalgia to the technique, drugs and expertise of the psychiatrist. It is suggested that post-ECT myalgia is related to the use of muscle relaxant agents such as succinylcholine and to muscle injury during the procedure, due to fasciculation and seizure. It is also claimed that the biophysical effects of succinylcholine independent of fasciculation are capable of inducing myalgia, and applying non-depolarizing

agents may prevent myalgia in these patients (10-13). Considering that myalgia and headache are two side effects of ECT that in some cases cause the patients to stop the course of treatment, and given the lack of similar studies in this area, this research seeks to clarify the causes and prevalence of post-ECT headache and myalgia (8, 10, 14).

2. Objectives

We evaluated myalgia and headache after ECT in patients referred to Shafa hospital in Rasht, Iran, to find the approximate prevalence of these two complications, and their predictors. The results of this study call for future studies to establish effective modalities to prevent or reduce these unacceptable adverse effects.

3. Patients and Methods

This prospective analytical descriptive study took place in an academic hospital in northern Iran affiliated with Guilan University of Medical Sciences (GUMS), from October 2014 to January 2015. Before sampling, the study proposal was approved by the GUMS Ethics committee, and written informed consent was acquired from all patients.

3.1. Participation Criteria

Reliable patients with major depression disorder, bipolar disorder, schizophrenia, or suicidal thoughts and delusions, based on DSM-IV definitions, were eligible to participate. Participants also had to be under treatment with risperidone (1.25 mg, adjusted based on the patient's needs, produced by Abu Reihan company), be between 18 - 60 years old, and be voluntarily receiving ECT. Patients with pregnancy, need for emergency ECT, ASA class III or IV, EF < 50%, uncontrolled HTN, mental retardation, addiction to any drug or substance, history of headaches, history of neuromuscular disease, or lack of written consent were excluded. Based on these criteria a total of 621 eligible patients were enrolled in the study. The demographic data such as sex, age, and weight were recorded for all participants.

3.2. ECT Procedure

First, each patient was fully examined and his or her fasting status was checked by an anesthetist. On arrival in the operating room, an 18 gauge intravenous catheter was inserted into the forearm vein and standard monitoring was applied, including electrocardiography with both leads II, V5 with automated ST-segment analysis to detect ischemia, pulse oximetry, and non-invasive blood pressure (NIBP) with an interval of 3 minutes. In order to decrease

oral and upper respiratory tract secretion, 0.5 mg of atropine was administered 30 minutes before the procedure. Pre-oxygenation was achieved with continuous oxygen flow (O₂ 100%) via a face mask, then anesthesia was induced with 3 mg/kg of sodium thiopental (Chandra Bhagat Pharma Pvt. Ltd.) and succinylcholine 0.5 mg/kg. A bite block was inserted prior to treatment to prevent tongue bite and to protect the patient's teeth.

Next, two ECT electrodes (somatic thymatron IV, Class I, type BF, constant current 0.9 amps, pulse width 0.25 to 1.5 MS, duration 0.14 to 7.99 seconds, 4 recording channels, maximum output 220 ohms, and impedance 504 Mc) were placed bitemporally. The level of energy output was adjusted based on 25% of total power and increased to 80% during lack of peripheral convulsion for at least 20 seconds or until a spike wave on EEG. Manual ventilation was continued during the clonic phase to avoid oxygen desaturation, and was maintained until adequate spontaneous ventilation resumed. The bite block was removed when seizure activity had stopped.

At the end of the procedure, the patient was transferred to the recovery room and monitored via ECG and pulse oximetry, and received 100% O₂ (5-6 L/min) by a nasal cannula. Close monitoring was continued until the patient was awake and maintained oxygen saturation on room air. Patients were evaluated by a trained nurse regarding the presence and severity of headache and myalgia immediately after the ECT, 2 hours later, and 6 hours later. Evaluations used a 4-point verbal rating scale, a tool to measure severity of pain where 0 = no pain, 1 = mild pain, 2 = moderate pain, and 3 = severe pain. Previous studies have indicated the validity and reliability of this instrument (12-16).

To evaluate the correlation between the number of treatment sessions and the severity of myalgia, our patients were allocated into three groups: 1-2 sessions, 2-4 sessions, and 5-6 sessions. The convulsion duration was measured based on EEG, and the patients were again divided into 3 groups (\leq 19 seconds, 20 - 39 seconds, and $>$ 40 seconds). The correlation of presence and severity of headache and myalgia was recorded with the convulsion duration. In patients with tonic-clonic seizures lasting more than 1 minute, IV midazolam (3 mg) was administered, and if the patient complained of a headache with moderate or severe pain, the patient was treated with IV ketorolac (30 mg).

3.3. Statistical Analyses

Data were analyzed using SPSS version 16. Categorical data are presented as numbers (%), and continuous data as mean \pm SD. We used the chi-square, Friedman and Cochran's Q tests to compare categorical variables, Correlations between headache and myalgia with ECT variables

were calculated by a logistic regression test. $P < 0.05$ was considered as significant.

4. Results

6 hours after ECT, the frequency of headache was 126 (21.9%) and that of myalgia was 56 (9%). Regarding the presence of headaches, Cochran's Q test indicated a significant difference between 0 to 6 hours after ECT (CI = 99%, $P = 0.0001$), but for myalgia the difference between 0 to 6 hours after ECT (CI = 99%) was not significant ($P = 0.062$). The presence of headache and myalgia 6 hours after ECT was not correlated to the duration of convulsion ($P = 0.98$ and $P = 0.84$, respectively). Moreover, the correlation between presence of headache 6 hours after ECT and number of treatment sessions was not significant ($P = 0.48$).

The correlation between presence of myalgia 0 ($P = 0.92$) and 2 hours ($P = 0.56$) after ECT with number of treatment sessions was not significant, but the occurrence of myalgia at 6 hours after ECT was correlated with number of treatment sessions ($P = 0.017$). Specifically, patients reported significantly less myalgia after 3 - 6 sessions than after their first or second sessions ($P = 0.017$). Headache 6 hours after ECT was not correlated with sex ($P = 0.14$), and myalgia at 0 and 6 hours after ECT was also not correlated with sex ($P = 0.11$ and $P = 0.058$, respectively), but myalgia at 2 hours after treatment was correlated with sex ($P = 0.04$): women complained of myalgia in the recovery room (2 hours after treatment) more than they did immediately after the ECT (0 hour) or when they were in the ward (6 hours post ECT). The presence of headache and myalgia 6 hours after ECT was not correlated with age ($P = 0.70$ and $P = 0.70$, respectively).

Fewer patients complained of a headache 6 hours after ECT ($P = 0.0001$) and moreover the number of patients with mild headaches was reduced significantly ($P = 0.0001$). Among the patients with headaches the percentage of patients with moderate and severe headaches at 6 hours after ECT was higher than at 0 and 2 hours after ECT (34% for 6 hours after ECT, vs. 24% at 0 and 18% at 2 hours) but no statistical analysis was performed. The correlation of myalgia with post-ECT duration was not significant ($P = 0.06$) (Table 1).

At 0 and 2 hours after ECT, the presence of severe headaches was more common in patients over 40 than in other patients: 8 of 11 the patients that showed headache at 0 hour after ECT were over 40, as were 6 of the 9 patients with severe headaches at 2 hours after ECT. The correlation between age and presence of myalgia 6 hours after ECT was not significant ($P = 0.70$). Logistic regression (enter mode) indicated that sex ($P = 0.057$), age ($P = 0.50$), duration of convulsion ($P = 0.74$), and number of treatment sessions

($P = 0.51$) were not predictors for headache and myalgia 6 hours after ECT ($P = 0.13$, $P = 0.35$, $P = 0.88$, and $P = 0.46$, respectively).

5. Discussion

ECT is an effective mode of therapy in the field of psychiatry. In the current study we evaluated the post-ECT frequency of myalgia and headache, and investigated the probable predictors. We used thiopental because this drug has been considered as the first line anesthetic for ECT procedures for a long time. As this treatment is generally not recommended for those less than 16 years old, our selected population was at least 18 years old (1, 14, 15). Our results indicated that headache was a more frequent adverse effect than myalgia within 6 hours after ECT (22% vs 9%). However, this type of headache was mild and tolerable. Considering that these patients did not have a history of headache or myalgia, it is likely that their complications were related to the ECT procedure.

Comparing the results of the present study to similar previous studies, we observe that the incidence of headache was lower in our study. Notably, Wang et al. reported that 46% of their patients experienced headache after ECT treatments (16). Saricicek et al. reported 62.5% headache and 37.5% myalgia incidence 6 hours after the procedure, and 30% headache and 12.5% myalgia after 12 hours (17). In the present study the frequency of headache and myalgia decreased from just immediately after ECT to 6 hours after treatment (from 18.8% to 7.1% and 6.1% to 4%, respectively), but the headache reduction was significant while the reduction of myalgia was not. The presence of headache and myalgia in our research was not correlated to sex, age, duration of convulsion, or the number of treatment sessions. In contrast to our findings, Rasmussen et al. showed that the number of ECT sessions was correlated with myalgia one day after treatment (18).

Our results found no correlation between the intensity of headache and myalgia and other variables, in contrast to Dinwiddie et al. (10), who indicated that the intensity of headache 2 hours after ECT was correlated to the duration of seizure. Moreover, they observed that the presence of myalgia was correlated with age and was worse in patients younger than 45 years old. They emphasized that headaches were mild and more frequent than myalgia, which is in agreement to our findings. We found that the severity of headache and myalgia decreased within 6 hours after ECT, and similarly Ferreira-Valente et al. pointed out that headache severity peaked 2 hours after ECT and returned to baseline one day after treatment (19). Although our study and a few previous studies (10, 14, 17) indicated

Table 1. The Frequency and Severity of Headache and Myalgia

Variation	After ECT, h			P Value
	0	2	6	
Headache				0.0001
No	504 (81.2)	504 (81.2)	577 (92.9)	
Yes	117 (18.8)	117 (18.8)	44 (7.1)	
Myalgia				0.062
No	583 (93.9)	586 (94.4)	596 (96)	
Yes	38 (6.1)	35 (5.6)	25 (4)	
Severity of headache				0.0001
0 (no)	504 (81.2)	504 (81.2)	577 (92.9)	
1 (mild)	89 (14.3)	96 (15.5)	29 (4.7)	
2 (moderate)	17 (2.7)	12 (1.9)	10 (1.6)	
3 (severe)	11 (1.8)	9 (1.4)	5 (0.8)	
Total	621 (100)	621 (100)	621 (100)	
Severity of myalgia				0.087
0 (no)	583 (93.8)	586 (94.4)	596 (96)	
1 (mild)	31 (5)	30 (4.8)	14 (2.3)	
2 (moderate)	6 (1)	4 (0.6)	6 (1)	
3 (severe)	1 (0.2)	1 (0.2)	5 (0.8)	
Total	621 (100)	621 (100)	621 (100)	

that headache after ECT was mild, the results are not consistent, and a review by Markowitz showed that headache after ECT was severe in 46.2% of patients and moderate in 53.8% (20). The reason for the differences among the results might be multifactorial, and partly due to the different structures regarding population selection, study duration, the number of treatment sessions, and the intervals. For example, unavoidable variability among patients affects seizure duration (4).

In summary, the results of our study and similar previous reports (8, 10, 16, 17, 21) emphasized that although ECT is generally a safe and effective therapy, some patient subgroups, such as those with certain cardiac conditions, a history of headache or chronic pain, and patients taking analgesics may require further evaluation. We applied a four-point verbal rating scale with a confirmed reliability, but other scales could also be used to evaluate ECT-treated patients (19, 22). Further comparative studies (comparing pre- and post-ECT) with larger sample sizes and longer duration of follow up (at least 24 hours) are required to validate the results of the present study.

Some logistical factors limited this study, such as the short post-ECT follow up duration (6 hours) that restricted us from evaluating the possible long-term incidence of

headache and myalgia. If we followed up with our patients after three months, the results might be different. In addition, our subjects were not evaluated with the 4-point scale before ECT, so their pre- and post-treatment conditions could not be compared. However, we evaluated a large sample size in this cohort, the main strength of this study. This study revealed that headache after ECT was more common than myalgia in our ECT-treated patients. Also, it was shown that this type of headache is mild and decreases within 6 hours after ECT.

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Footnotes

Authors' Contribution: Mohammad Haghighi: study design, and conduct, data analysis, and manuscript prepara-

Table 2. Characteristics of Patients

Variables	Values ^a
Sex	
Male	385 (62)
Female	236 (38)
Age, y	
< 30	215 (34.6)
30 - 40	191 (30.8)
40 - 50	150 (24.2)
> 50	65 (10.5)
Mean \pm SD	36.77 \pm 10.93
Range	18 - 60
Weight, kg	
Mean \pm SD	71.47 \pm 15.29
Range	37 - 150
Treatment course number	
1 - 2	231 (37.2)
3 - 4	205 (33)
5 - 6	185 (30)
Convulsion duration, EEG	
0 - 19	38 (6.1)
20 - 39	299 (48.1)
> 40	284 (45.7)
Mean \pm SD	37.03 \pm 10.52
Range	10 - 85
Myalgia	
No	565 (91)
Yes	56 (9)
Headache	
No	495 (78.1)
Yes	126 (21.9)

^aValues are expressed as No. (%).

tion; Bahram Naderi Nabi: study design, data collection, manuscript preparation, and data analysis; Abbas Sedighinejad: study design, data analysis, and manuscript preparation; Cyrus Emiralavi: study design, data collection, manuscript preparation, and data analysis; Gelareh Biazar: data analysis manuscript preparation; Kaveh Mirzozafari: manuscript preparation and data analysis; Cyrus Zahedan: manuscript preparation; Mahdi Jafari: manuscript preparation.

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