Does Electroconvulsive therapy aggravate the rise in potassium and creatine kinase following suxamethonium administration?

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Abstract

Background: Potassium and creatine kinase levels increase after the administration of suxamethonium. This rise may be exaggerated by the combination of suxamethonium fasciculation and the modified tonic/clonic convulsion induced by electroconvulsive therapy. This study compared the magnitude of increase in potassium and creatine kinase levels after electroconvulsive therapy and surgery using suxamethonium.

Methods: A total of 40 patients were studied; electroconvulsive therapy (ECT), n=20 and surgery (Control), n=20. Intravenous sodium thiopentone (5mg/kg) and suxamethonium (1.5mg/kg) were administered. The changes in potassium and creatine kinase levels were assessed at presuxamethonium, 1 and 3 minutes after fasciculation in Control group and ECT-induced seizure activity in the ECT group. Our hypothesis was that a significant increase occurs in the mean potassium and creatine kinase levels after suxamethonium administration during electroconvulsive therapy.

Results: Both groups exhibited a rise in potassium concentration after administration of suxamethonium. The mean increase was significantly higher in the ECT group than in the Control group; at 1 minute; ECT (0.71 ±SEM 0.24) versus control (0.28 ±SEM 0.19) mmol/L, p =0.003, and at 3 minutes; ECT (0.35 ±SEM 0.23) versus control (0.20 ±SEM 0.15), p =0.044. The mean increase in the creatine kinase concentration was significantly higher in the ECT group (34.11 ±SEM 10.76) than in the Control group (19.71 ±SEM 6.32) IU/L, p = 0.023, at 3 minutes.

Conclusion: The creatine and potassium concentrations following suxamethonium administration were significantly higher in the electroconvulsive therapy group than in the control group.

Key words: ECT, surgery, creatine kinase, potassium

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Introduction

Musculoskeletal injury, which may present as asymptomatic spinal compression fracture was a common complication in electroconvulsive therapy (ECT) before the use of muscle relaxants.[1] Suxamethonium is the preferred muscle relaxant of choice for ECT because of its rapid onset, fast recovery profile and intense muscle relaxation.[2] Nevertheless, suxamethonium like ECT is associated with muscular injury, which has been attributed to the initial depolarization of the muscles prior to paralysis.[2]

Following the administration of suxamethonium during ECT; a significant increase in potassium of 0.25 to 2.90 mmol/L,[3-4] and serum creatine kinase level up to values of 110 to 3,000 IU/L have been reported.[5-7] The higher values were reported in patients with neuroleptic malignant syndrome, and catatonic Schizophrenia.[4,7] In recent times, ECT under anaesthesia was commenced in the psychiatry ward of our institution. There is a need to evaluate the practice and investigate the occurrence of any untoward side effects, with the intention of improving patients' outcome. We hypothesized that ECT will increase the release of potassium and creatine kinase levels in psychiatry patients after suxamethonium administration. Whether this rise is potentially injurious to our patients, needs to be investigated.

Materials and Methods

The approval of the Human and Research Ethic Committee of our institution, and informed consent were obtained. This was a prospective non-randomised open label trial conducted from April 2011 to March 2012 in 40 consecutive patients assigned to either ECT group (n = 20) or surgery (control) group (n = 20). This included males or non-pregnant female patients aged 18 years and older, with American Society of Anesthesiology (ASA) I and II physical status scheduled for electroconvulsive therapy (ECT) or surgery under general anaesthesia.

The patients with a major depressive episode as part of a diagnosis of either major depressive disorder or bipolar disorder,[1] were included in the study. Patients were excluded from the study if they fulfilled the criteria for DSM-IV 8 (Diagnostic and Statistical Manual of

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Mental Disorders) substance abuse disorder in the last 12 months; had received ECT within the previous 6 months, or were ASA III or IV. In addition, pregnant or nursing patients, those with a known allergy to suxamethonium or who refused to participate were excluded. Individuals who were receiving intramuscular injections were changed to oral or intravenous routes 48 hours before intervention. Haemolysed blood samples were excluded from analysis.

ECT was administered using a brief-pulse, square-wave, constant-current ECT device (120 mC, 70 Hz/ 0.1 sec). Bilateral ECT was given using the standard bifrontotemporal placement. During the course of ECT, each patient had their maintenance, anti-depressant medication to reduce the risk of relapse.

Anaesthesia technique

All patients fasted to solid food for six hours, and to clear fluid for two hours before their procedure, patients for electroconvulsive therapy were allowed to have their oral antipsychotic medication. In all patients, a portable Multiparameter Cardiocap 7100 monitor was attached for continuous monitoring of the heart rate (HR), non-invasive blood pressure (NIBP), electrocardiogram, respiratory rate (RR), and oxygen saturation (SPO_2). Intravenous access was secured with an 18G cannula.

In the ECT group; the attending psychiatrist placed the bitemporal ECT electrodes on the patient's forehead. All patients were preoxygenated with 100% oxygen via a bag mask valve device for 3-5 minutes. Anaesthesia was induced with intravenous sodium thiopentone (5mg/kg), and IV suxamethonium (1.5mg/kg) was administered for neuromuscular relaxation.[8-9] Continuous, intermittent positive pressure ventilation (IPPV) was continued with the bag mask valve device during the period of apnoea. When the fasciculation had subsided and adequate neuromuscular relaxation was obtained, an appropriate sized Guedel's airway was inserted to prevent a tongue bite, and IPPV was continued. The electrical stimulation of the ECT was administered 15 to 20 seconds after suxamethonium fasciculation using a brief pulse stimulus (90-120 volts MECT) given for about 2 msec to produce ECT-induced seizure activity. Subsequently, all the patients were ventilated with 100% oxygen at the rate of 12 breaths per minute until spontaneous breathing returned, and patients were fully recovered. Intravenous fluid was administered according to standard protocol.

In the control group; all patients were preoxygenated with 100% oxygen for 3-5 minutes. General anaesthesia was induced with IV sodium thiopentone 3-5 mg/kg until loss of eyelid reflex. IV suxamethonium 1.5 mg/kg was then administered to facilitate intubation with an appropriate sized endotracheal tube. The maintenance of anaesthesia was continued at the discretion of the attending anaesthetist, depending on the patient's needs.

Blood sample

Venous blood (4mls) was withdrawn in all patients for potassium and creatine kinase estimation; before suxamethonium (pre-suxamethonium), 1 and 3 minutes after fasciculation or ECT-induced seizure activity. Potassium was analysed using the ion selective electrode technique, while creatine kinase was analysed using a Semi-automated Chemistry Analyser Microlab 300™ with the International Federation of Clinical Chemistry and laboratory16 (IFCC) recommended method, (Techno Diagnostics Kit, USA). Creatine phosphate and ADP were converted to Creatine and ATP by SCK catalyses. The glucose and ATP were converted to ADP and glucose-6-phosphate Hexokinase (HK). Glucose-6-phosphate dehydrogenase (G-6-PDH) oxidised the D-glucose-6-phosphate and reduced the Nicotinamide Adenine Dinucleotide (NAD). The rate of NADH formation, measured at 340 ηm is directly proportional to CK activity in the serum. Normal reference range considered for females and males was 34-145 IU/L and 46-171 IU/L at 37 °C respectively. The linearity of the assay was 1,200 IU/L. Two level controls were run with each batch. Results which were outside the linearity range were re-run after appropriate dilution according to the recommendations of the manufacturer of the kit and re-analysis/interpretation of results were done accordingly (Creatine Kinase-MB, 2012). The laboratory scientist who performed the potassium and creatine kinase assays was unaware of the patient data.

Statistical Analysis

Data collated included the duration of fasciculation or ECT-induced seizure activity, the heart rate, blood pressure and oxygen saturation, as well as potassium, and creatine kinase concentrations. Data were presented mean ±SD (SEM) or frequency, and the difference between the groups was determined with an independent t test or chi square. A p value <0.05 was considered significant. The All data were analysed using the Statistical Package for Social Sciences (SPSS) version 17 for windows computer programme.

The primary outcome determined and compared the changes in creatine kinase and potassium concentrations after suxamethonium administration during ECT and surgery. The secondary outcome determined and compared the changes in heart rate (HR), systolic blood pressure (SBP, and oxygen saturation (SPO_2) after suxamethonium administration during ECT and surgery.

Results

Forty patients were studied; ECT (n = 20) and Control (n = 20). The mean age, height, weight and body mass index

were comparable between the groups. In both groups, the mean pre-suxamethonium potassium concentrations were comparable, p =0.23. The mean potassium level was significantly higher in the ECT group than in the Control group at 1 minute, p =0.02. A significantly greater increase in the mean potassium level occurred at 1 minute after ECT-induced seizure activity in the ECT group (0.71 mmol/L) than in the Control group (0.18mmol/L), p= 0.01, Table 1.

Table 1. The Demographic and Clinical Parameters

Variables	ECT Mean ±SD	Control Mean ±SD	p value
Demographics			
Age (years)	36.28±12.44	37.20±12.25	0.90
Sex (M:F)	4:3	2:1	0.26
Weight (Kg)	61.58±10.42	65.45±10.62	0.27
Height (m)	1.61±0.08	1.65±0.08	0.15
Body mass Index (kg/m2)	23.70±3.31	24.21±2.69	0.61
Creatine Kinase (IU/L)			
Pre-suxamethonium	103.93 ±77.21	67.87 ±27.38	0.06
1 minute post-seizure	124.99 ±88.94	79.60 ±25.71	0.04
3 minutes post-seizure	137.41 ±92.72	83.48 ±30.51	0.02
Change in CK from Pre-suxamethonium value			
1 minute post-seizure (±SEM)	+23.83 ±4.79	+15.47 ±4.74	0.22
3 minutes post seizure (±SEM)	+34.11±10.76	+19.71±6.25	0.02
Potassium (mmol/L)			
Pre-suxamethonium	4.31 ±0.64	4.07 ±0.62	0.23
1 minute post-fasciculation	4.92 ±0.93	4.25 ±0.74	0.02
3 minutes post-fasciculation	4.50 ±0.96	4.32 ±0.83	0.52
Change in K from Pre-suxamethonium value			
1 minute post-fasciculation (±SEM)	+0.71 ±0.24	+0.18 ±0.14	0.01
3 minutes post-fasciculation (±SEM)	+0.25 ±0.17	+0.20 ±0.15	0.60
Creatine Kinase > 171(IU/L)			
1 minute (percent)	3(15%)	0	0.23*
3 minutes (percent)	7(35%)	1(5%)	0.04*
Potassium >5.5 (mmol/L)			
1 minute (percent)	5(25%)	2(10%)	0.40*
3 minutes (percent)	4(20%)	2(10%)	0.66*

Values are mean ±SD (SEM), frequency ad percentile, p =0.05 was significant.
* Indicate Fischer Exact Test SEM (standard error of means), ECT (electro convulsive therapy)

A rise in potassium occurred in 25(62.5%) patients, range (+0.09 to +2.27 mmol/L), no change in 2(5%) patients, and a reduction in 13(32.5%) patients, range (-0.05 to -0.9 mmol/L). The maximum rise in potassium was 2.27 mmol/ in a patient with a pre-suxamethonium potassium of 4.8mmol/L. At 1 minute the proportions of patients with potassium concentration above upper limit in ECT and control groups were 5(25%) and 2(10%) respectively, (p = 0.40) and at 3 minutes, 4(20%) and 2(10%) respectively, p = 0.66. The maximum level of potassium concentration (7.15 mmol/L) was observed in the ECT group at 1 minute after fasciculation. There was no significant association between the duration of fasciculation or ECT-induced seizure activity and changes in potassium and CPK concentration, Table 2

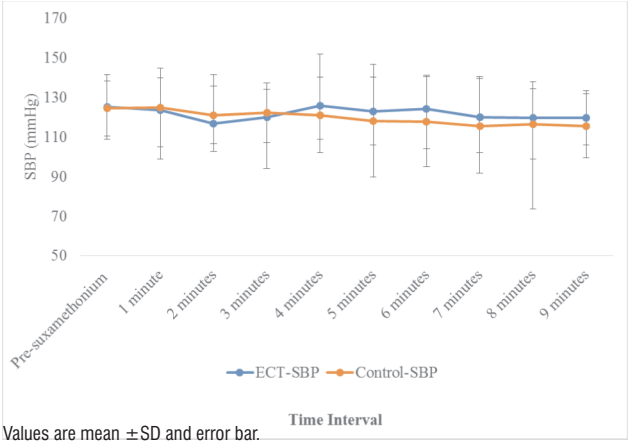
Table 2: The Association between duration of fasciculation/seizure and increase in Creatine Kinase and Potassium Concentration

Time of Sampling	Rho	p value
Creatine Kinase		
1 minute	0.167	0.30
3 minutes	0.171	0.29
Potassium		
1 minute	0.199	0.22
3 minutes	0.089	0.59

Values are Spearman Correlation (rho), p=0.05 was significant.

In both groups, the mean pre-suxamethonium creatine kinase (CPK) concentrations were comparable, p =0.06. At 1 and 3 minutes, the mean CPK level was significantly higher in the ECT group than in the Control group, p=0.04 and p =0.02 respectively. The mean increase in the CPK level at 3 minutes in the ECT group was significantly higher than in the Control group, p= 0.02.

A rise in CPK occurred in 31(77.5%) patients, (range +3 to +163 IU/L), no change in 4(10%) patients, and a reduction in 5(12.5%) patients, (range -0.96 to - 33 IU/L). The maximum rise in CPK was 72 IU/L in a patient with pre-suxamethonium CPK of 50 IU/L. At 1 minute, patients with CPK concentration about upper limit in ECT and Control groups were 3(15%) and 0 respectively, (p = 0.23) and at 3 minutes, 7(35%) and 1(5%) respectively, p = 0.04 (Table 1).



Values are mean ±SD and error bar.

Figure 1. The mean Systolic Blood Pressure during ECT versus Control

The maximum level of CPK concentration (421.3 IU/L) was observed in the ECT group at 3 minutes after ECT-induced seizure activity.

The mean heart rate was comparable at preinduction, 1 and 3 minutes and throughout the study period; Control (95.47±22.09) versus ECT (93.35±10.48) bpm, p = 0.71, Control (95.65±8.07) versus ECT (97.36±19.22), p = 0.66, and Control (99.58 ±18.28) versus ECT (91.95±21.69), p = 0.24 respectively, Figure 1

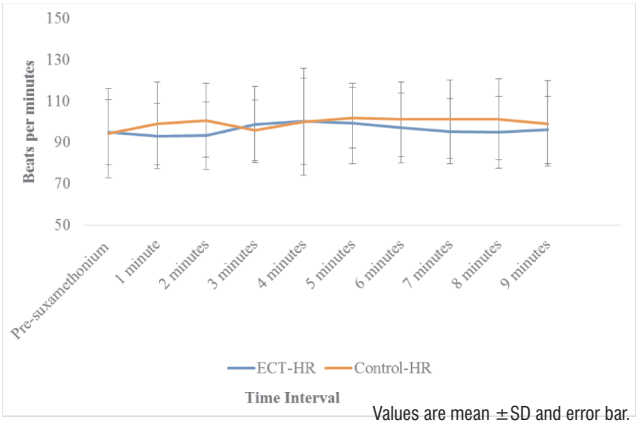


Figure 2. The mean Heart Rate during ECT versus Control

The mean systolic blood pressure was comparable at preinduction, 1 and 3 minutes and throughout the study period; Control (124.39 ±14.26) versus ECT (114.66 ±15.24) bpm, p = 0.13, Control (134.99±31.06) versus ECT (126 ±6.28), p = 0.71 and Control (139.99 ±13.5) versus ECT (123.87 ±12.69), p = 0.17 respectively, Figure 2.

The preinduction SPO2 was comparable; Control (98%) versus ECT (98%), p = 0.16. Similarly, the mean SPO2 was comparable in both groups at 1 and 3 minutes; Control (98%) versus ECT (98%), p = 0.36 and Control (99%) versus ECT (99%), p =0.27 respectively. However at 5 minutes post induction there was a significant difference between the groups; Control (98%) versus ECT (99%), p = 0.02, however at no time was the SPO2 lower than 95%.

Discussion

Suxamethonium is the preferred muscle relaxant for electroconvulsive therapy, however it may result in a surge in potassium, [12] creatine kinase and myoglobin concentrations.[13,14] Our findings showed that electroconvulsive therapy enhanced the elevation of plasma potassium normally seen after suxamethonium. A rise in potassium occurred in 25(62.5%) patients, a range of (+0.09 to 2.27mmol/L), this is close to the range of 0.25-2.90 in previous reports.[3,13-14,16] Such an increase can result in severe hyperkalemia following ECT, which may induce arrhythmias.[13]

The Royal College of Psychiatrists recommended a dose of 0.5 mg/kg suxamethonium for electroconvulsive therapy, however, larger dosages (0.75-1.5 mg/kg) are often used in clinical practice.[8-9,15] (The same suxamethonium and sodium thiopentone protocol was used for our entire cohort in order to avoid the variable effect of suxamethonium dosage on potassium concentrations. This is because the wide variation in the mean increase in potassium concentration following suxamethonium administration in previous studies has been attributed to different confounding factors, which include the dose of suxamethonium administered.[3,12,15-16]

In addition, the presence of clinical situations such as muscular dystrophy, upper motor neuron lesions, renal failure, burns and prolonged immobility, which potentiate the release of potassium from perijunctional or extrajunctional neurons.[2,14] High levels of serum potassium could be detrimental as level =7 mmol/L has been implicated in triggering morbid arrthymias.[14] Therefore the use of a higher dose of suxamethonium (> 0.5mg/kg) should be discouraged in psychiatry patients at risk of hyperkalemia and arrthymias such as those with a previous history of bradyarrhythmias,[7,10] (individuals susceptible to organophosphate poisoning, malignant hyperthermia, neuroleptic malignant syndrome, and catatonic schizophrenia.[5,14,17] (Another undesirable effect of a large dose of suxamethonium is prolonged apnoea,[5,15,18] which may increase the risk of aspiration of gastric content in the ECT setting since patients were routinely not intubated.

We observed that the mean potassium concentration 1 minute after ECT-induced seizure activity in the ECT group was significantly higher than 1 minute after fasciculation in control group. Similarly, the proportion of patients with potassium values above the normal reference range and the maximum level of potassium concentration was more in the former; however, the difference was insignificant. One patient in the ECT group had a potassium level of 7.15 mmol/L, unfortunately we were unable to do 12 lead ECG, to detect any significant changes. However, it has been previously documented that an increase in the amplitude of T waves occurs with potassium values = 7 mmol/L, which is described as narrow and tented with widened QRS.[14] Though, in our study we did not observe such ECG changes. This may be because there was no 12 lead ECG available during our study, as these changes are difficult to identify in the rolling oscilloscope used in anaesthesia monitors. Nevertheless, the ECG rhythm was sinus, the heart rate and blood pressure were within normal limit, and the plethysmograph was regular. We therefore concluded that the observed hyperkalemia was most likely uneventful. We also did not attribute this to haemolysis because lysed blood samples were excluded

from the analysis.

We observed no significant association between the duration of fasciculation or ECT-induced seizure activity and changes in potassium concentration. However, there were conflicting reports on the relationship between the duration of fasciculation or ECT-induced seizure activity and changes in potassium concentration.[3-4,19-20] In studies with the mean duration of fasciculation less than one minutes, there was no association observed,[3,4,19] which is in agreement with our observation. However, a significant association was reported when the mean duration of fasciculation was greater than one minute. There is a need for further investigation on possible association between duration of fasciculation/seizure and potassium release during ECT. Pilditch et al.[17] also observed that the grade of ECT-induced seizure using an arbitrary grading system produced no significant differences in serum potassium levels.

The mean CPK levels at 1 and 3 minutes after ECT-induced seizure activity was significantly higher than after suxamethonium induced fasciculation in our study. The surge in CPK during surgery has been attributed to muscle injury.[21]The administration of halothane or enflurane anaesthesia, and the dissection of muscle also contribute to the surge in CPK during surgical procedures, with more CPK been produced in major surgical procedures such as exploratory laparotomy, hysterectomy than minor procedures like herniorraphy and tonsillectomy.[21-22]

To avoid conflicting confounding factors such as influence of volatile anaesthetic agent or muscle injury from surgical incision or tissue dissection on CPK release in our review, venous specimen was withdrawn well before serum CPK reaches its peak, (9-24 hours after suxamethonium administration.[22] Likewise intra-muscular injection was discontinued 48 hours prior to intervention, and all medication was administered via the oral or intravenous route. We considered it unethical to intubate patients who were not scheduled for a surgical procedure. Therefore, volunteers were not recruited for the control group, hence the use of a surgical population. Endotracheal intubation, however, was unlikely to result in the rise in CPK level observed by us, as intubation has not been reported to induce muscle injury. Therefore the rise in CPK in our study is probably due to the effects of the suxamethonium induced fasciculation and ECT-induced seizure activity.

Serum creatine kinase has been described as the best marker for detection and monitoring of skeletal muscle diseases and damage.[21] Creatine kinase is found in myocardium, skeletal muscle, brain, and the gastrointestinal tract hence its use as a marker for brain injury (CPKBB) or myocardial infarction (CPK-MB,).[23] Elevated CPK has also been reported in convulsive disorders and after ECT.[6] It is the total CPK that is elevated during ECT, while the Brain-type CPK (CPK BB) remain within normal range.[23]

The influence of suxamethonium (0.5 mg/kg) and (1.0 mg/kg) on motor seizure modification, CPK, and potassium was investigated in a randomized cross over trial.[10] The authors observed that suxamethonium (1mg/kg) resulted in optimal motor seizure modification; there was a significant increase in the levels of serum potassium.[11] The increase in potassium was, however, within the normal range and did not cause any cardiac side effects.[11] However, there was no change in creatine kinase level. This is contrary to our observation of a significant increase in CPK after suxamethonium with a dose of 1.5mg/kg. The difference in observations we attributed to variation in dose of suxamethonium administered.

In our review, a rise in CPK occurred in 31(77.5%) of patients, (range +3 to +163 IU/L), the maximum level of CPK was 421.3 IU/L in the ECT group. In none of our patients was the CPK = 1,000 IU/L, values which are suggestive of the possibility of malignant hyperthermia, anaesthesia-induced rhabdomyolysis and underlying muscle disease.[5,7,14,21] It has been suggested that any surge of creatine kinase concentrations above what is expected should prompt further investigation.[20]

The duration of ECT-induced seizure activity in the ECT group was significantly longer than the duration of fasciculation in the Control group. The mean duration of ECT-induced seizure activity (0.35±0.08) seconds is, however, close to the predicted value of 30 seconds recommended for achieving an adequate seizure for the ECT to be effective.[24]

The mean systolic blood pressure, heart rate and peripheral oxygen saturation between the groups were comparable in our study, which may be because we recorded our vital signs every minute. This time interval has been shown to be prolonged for any rapid change in blood pressure, and heart to be noticed.[25-26] This is because the use of an automated noninvasive blood pressure monitoring takes more than 30 seconds before measurement, and cardiac action may not remain constant during this period.[26] The typical cardiovascular response to ECT consists of generalized autonomic nervous system stimulation, with an initial parasympathetic-induced bradycardia lasting 10 to 15 seconds followed immediately by a more prominent sympathetic response that results in transient tachycardia and hypertension lasting 5 min or longer. Systolic blood pressure is transiently increased by 30-40%, and heart rate (HR) is increased by 20%.[26] It has been suggested that for any rapid change in the blood pressure and heart rate to be identified, the use of

an arterial catheterization, or a special beat-to-beat monitoring device, or recording of electrocardiogram (ECG) and pulse oximeter curves should be imbibed.[26]

A possible limitation to this study was the collection of venous blood sample at 1 and 3 minutes well before serum CPK reaches its peak, at 9-24 hours after suxamethonium administration.[24] This was done by us to avoid the potential influence of volatile anaesthetic agent and surgical manipulation on CPK release. We were also unable to blind the investigator who collected the blood sample; however, the laboratory scientist was blinded for the analysis. The use of arterial catheterization might have ensured the detection of rapid changes in heart rate and blood pressure.

In conclusion, ECT-induced seizure activity is associated with an exaggerated increase in potassium and creatine kinase. There is the need to exercise caution with a larger dose of suxamethonium in at risk patients.

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