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Short Communication

Serum lipid profile changes after successful treatment with electroconvulsive therapy in major depression: A prospective pilot trial

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ABSTRACT

Background: Cholesterol is reduced in depressed patients, however, these patients have a higher risk for cardiovascular diseases. Electroconvulsive therapy (ECT) is a highly effective treatment option for specific forms of depression. Like for other non-pharmacological therapies targeting depression such as psychotherapy or sleep deprivation, there is a lack of evidence about the effects on peripheral lipid parameters. Our objective was to study the impact of ECT as a non-pharmacological treatment on the peripheral lipid pattern in depressive patients.

Method: Peripheral lipid profile composition before and after a course of ECT was analysed in 27 non-fasting inpatients at a university psychiatric hospital with DSM-IV major depressive episode. For the impact of ECT treatment on each lipid parameter a multivariate repeated measurement regression analysis was performed and computed separately for every dependent variable.

Results: Total Cholesterol and the cholesterol subtypes HDL and LDL were increased after the treatment compared to baseline. Apolipoprotein A1 was also increased after ECT, whereas apolipoprotein B was not. Indices for the prediction of cardiovascular diseases were unchanged after successful treatment by ECT. The reduction of depressive psychopathology negatively correlated with increases of HDL cholesterol and apolipoprotein A1.

Limitations: Subjects received several antidepressants and other psychotropic medication before and during the ECT.

Conclusions: In our preliminary pilot study ECT as a non-pharmacological, effective treatment of depression led to distinct effects on the peripheral lipid pattern.

1. Introduction

Major depression episodes (MDE) in uni- or bipolar affective disorders are primarily diseases of the brain, but are affecting the body as a whole in many different ways. In this article we focus on the peripheral lipid profile in depression and its changes due to non-pharmacological antidepressant treatment. It is well known, but not yet fully understood, that total serum cholesterol and cholesterol subtypes are lowered in depressed patients compared to healthy controls (Glueck et al., 1994; Olusi and Fido, 1996; Partonen et al., 1999). Patients with MDE, however, have a higher risk for cardiovascular diseases (CVD) such as heart failure, myocardial infarction or stroke (Lesperance et al., 2002; Penninx et al., 2001). Although there are several other factors which might explain the higher likelihood for cardiovascular morbidity and mortality in depression such as disturbed platelet function (Lederbogen et al., 2001), hypercortisolism (Deuschle and Lederbogen, 2002), endothelial dysfunction (Rajagopalan et al., 2001), inflammation (Appels et al., 2000) or reduced flexibility of the autonomic nervous system (Carney et al., 2003), the relevance of alterations of lipid metabolism in depression has not yet been unravelled.

Effective antidepressant pharmacotherapy modulates some of the dyslipidemic patterns observed in depressive patients, especially total cholesterol, HDL and LDL cholesterol (Hummel et al., 2011; Maes et al., 1997; Rabe-Jablonska and Poprawska, 2000). This may be a primary effect of the pharmacotherapy itself or may follow the amelioration of depressive symptoms. Successful treatment of depression in patients with or without prior cardiovascular diseases may lead to a reduced risk for future CVD (Davidson et al., 2010; Glassman et al., 2002), however data are not

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unanimous on this issue (Berkman et al., 2003; van Melle et al., 2007).

Electroconvulsive therapy (ECT) is a highly effective treatment option for specific forms of depression, mania and schizophrenia. Like for other non-pharmacological therapies targeting depression such as psychotherapy or sleep deprivation, there is a lack of evidence about the effects of ECT in the treatment of depression on peripheral lipid parameters. One previous study evaluated the effects of ECT on total cholesterol and reported a treatment-associated increase (Ramamurthy et al., 2013).

In the present study, pre- and post-ECT peripheral lipid profiles were analysed in order to determine the impact of ECT as a non-pharmacological treatment on the peripheral lipid pattern in depression. Our hypothesis was that ECT positively influences the peripheral lipid pattern of the patients.

2. Methods

2.1. Patients

Our prospective study has been approved by the appropriate ethics committee and was performed in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki). Written informed consent was obtained from all participants before inclusion in the study. The study took place from 2011 to 2013 at the Department of Psychiatry at the Central Institute of Mental Health in Mannheim, Germany. Inclusion criteria were a present depressive episode within the context of a diagnosis of either major depressive disorder or bipolar disorder according to DSM-IV, age above 18 years and the clinical decision for an ECT treatment. Exclusion criteria were substance-related disorders and lifetime diagnosis of schizophrenia and the intake of lipid lowering medication. The following data were documented for each patient: (a) before the treatment: Age and sex, (b) before and after the ECT: Hamilton Depression Rating Scale (HDRS; 21 items version), body mass index (BMI) and (c) after the treatment: numbers of ECT sessions needed and changes of the psychopharmacological medication prior or during ECT treatment.

2.2. ECT treatment

Right unilateral brief pulse ECT was performed with a Thymatron IV device (Somatics, LLC. Lake Bluff, IL, USA), anaesthetics were chosen according to clinical experience or preferences. Seizure threshold in all patients was titrated and energy was subsequently increased if patients did not respond clinically or if seizures were insufficient during the ECT course.

2.3. Sampling

Before the first ECT sessions and between one and seven days after the last ECT session, blood was drawn from an antecubital vein at 8:30 am. The patients were in a non-fasting status, but had a light breakfast before 8:00 am, because unlike in other departments the ECT treatment in our department usually does not start before noon. Each sample was centrifuged after the adequate clotting time and then stored at −80 °C until analysis.

2.4. Laboratory measurements

All laboratory measurements were performed at the Institute for Clinical Chemistry and Pathophysiology, Otto-von-Guericke-University, Magdeburg, Germany. Cholesterol was quantified by commercial enzymatic methods using a random-access analyser (Modular, Roche Diagnostics, Mannheim, Germany) as described by Hummel et al. (2011). For quantification of HDL and LDL cholesterol, lipoprotein fractions were separated by ultracentrifugation over a time period of 18 h at 100.000 g in a 50.4 Ti rotor with a Beckman L-60 ultracentrifuge (Beckman Coulter, Munich, Germany). After removal of the VLDL and chylomicron rich supernatant, the LDL fraction was precipitated with a phosphotungsten acid/magnesium chloride reagent to obtain the HDL fraction. Cholesterol was measured in VLDL fractions, HDL fractions, and the infranatant. LDL cholesterol was calculated from the bottom fractions. Total apolipoproteins A1 and B were analysed by immunoturbidimetric assays using goat anti-human polyclonal antisera (Greiner Bio-One, Bad Homburg, Germany).

2.5. Statistical analyses

Statistics were performed using STATA® (StataCorp, Texas 77845, USA, version 11) at a significance level ≤ 0.05 (two-tailed). For the impact of ECT treatment on each lipid parameter a multivariate repeated measurement regression analysis, as described previously (Bundy et al., 2010; Sartorius et al., 2006a; Sartorius et al., 2006b), was performed (i.e. multilevel mixed-effects linear regression using STATA 11 “xtregar” [Statacorp LP, College Station, Texas]) and computed separately for every dependent variable (BMI, total cholesterol, LDL cholesterol, HDL cholesterol, apolipoprotein A1 and B, the ratios of LDL to HDL cholesterol and apolipoprotein B to A1 including independent variables (number of ECT sessions needed) and the two covariates “sex” and “age”, because both might be possible confounders. No corrections for multiple testing were made. The influence of changes in the lipid parameters on the HRDS reduction was calculated by a linear regression analysis.

3. Results

Altogether, 35 patients were screened and 27 patients were included into the study. We were able to receive complete data sets from all 27 participants. Demographic and clinical data of the patients are shown in Table 1.

Total Cholesterol ($p=0.050$) and the two subtypes of cholesterol, HDL ($p=0.009$) and LDL ($p=0.032$) were increased after the ECT treatment compared to the baseline before the intervention of depression (Table 2). Apolipoprotein A1, a major component of the high-density lipoprotein complex, was also increased after ECT ($p=0.040$). No change of serum concentration could be detected for apolipoprotein B, the primary apolipoprotein of chylomicrons, VLDL, IDL, and LDL particles. The HDL/LDL ratio and the ratio of the two apolipoproteins B and A1, which are important indices for the prediction of cardiovascular disease, were unchanged after successful depression treatment by ECT. Additionally the treatment did not influence the BMI of the patients.

Table 1
Demographic and clinical features of the patients.

Number of included subjects	27
Age (mean) ± SE in years (min–max)	64.8 ± 3.8 (22–83)
Sex female/male *n*/*n* (in %)	14/13 (52.4/47.6)
Baseline Body Mass Index, mean ± SE (min–max)	25.5 ± 0.9 (19.1–30.1)
Duration of the episode in month (min, max)	6 ± 1.8 (0.5–36)
HDRS, sum score	
Baseline HDRS, mean ± SE (min–max)	28.6 ± 1.3 (20–42)
Final HDRS, mean ± SE (min–max)	9.8 ± 1.2 (1–21)
Mean change (min–max)	−18.8 (−4–36)
Numbers of ECT sessions, mean ± SE (min–max)	10.5 ± 3.6 (4–19)
Remitters/Responders/Nonresponders *n*/*n*/*n* (in %)	10/12/5 (37.0/44.4/18.6)