SERUM FOLATE AND HOMOCYSTEINE IN CHRONIC SCHIZOPHRENIC PATIENTS

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Introduction

Homocysteine is an amino-acidic product of methionine demethylation. Hyperhomocysteinemia (which is frequently found in schizophrenia (Nishi et al. 2014)) increases cardiovascular risk (Marti-Carvajal et al. 2009) and may trigger neuronal apoptosis (Mattson & Shea 2003). It may result from low folate intake (Selhub 2006), while folate deficiency is common in schizophrenia and may be associated with severity of negative symptoms (Goff et al. 2004) or cognitive deficits. Below, a new report on folate and homocysteine levels in schizophrenia is provided.

Material and methods

We studied 30 Caucasian in-patients with stable, chronic paranoid schizophrenia (SHZ) and thirty age -(38.8±12.6 vs. 36.8±12.3 years), gender - (17 men in both groups) and weight - (78.8±12.9 vs. 74.4±15.4 kg; body mass index 26.9±3.5 vs. 25.1±3.9 kg/m2) matched healthy controls. All schizophrenic patients were on stable (for \geq 2 months) treatment with clozapine in monotherapy (duration of treatment 54.0±74.5 months; dose 359.6±147.7 mg/day, serum level 385.6±412.3 ng/mL; there was no correlation between dose and concentration). Excluded were subjects with malabsorption of folate or taking folate supplementation. Fasting serum folate and homocysteine were measured with a chemiluminescence method, serum clozapine level was determined using ELISA method.

Results

There were no differences for folate (schizophrenia 8.59 ± 7.26 , control 7.62 ± 4.87 ng/mL, p=0.89) and homocysteine (schizophrenia: 14.45 ± 4.19 , control: 12.88 ± 4.74 µmol/L, p=0.18) levels. Men and women had comparable levels of folate. Women had lower homocysteine levels in the schizophrenia group (p=0.008) and in the whole study sample (p=0.001). Smoking did not affect folate or homocysteine levels.

Folate deficiency (<4 ng/mL) was infrequent (schizophrenia: 2 (6.7%), control: 1 (3.3%)). Hyperhomocysteinemia (>12 μ mol/L) was found in the majority of

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subjects (schizophrenia: 21 (70.0%), control: 17 (56.7%), p=0.42); all three subjects with folate deficiency had hyperhomocysteinemia. Only in the schizophrenia group a correlation between folate and homocysteine levels was significant (r=-0.42, p=0.02). There were no correlations between age, duration of schizophrenia, clozapine treatment (duration, dose, concentration) and folate or homocysteine levels.

Discussion

We found no previously reported differences in folate or homocysteine levels between schizophrenic and healthy subjects. Hyperhomocysteinemia may be more closely associated with obesity or other metabolic abnormalities than with schizophrenia itself. Study limitations include low number of subjects and the lack of behavioral data (i.e. food intake logs). Due to the cross-sectional study design causal relationships cannot be established and the effect of previous treatment cannot be excluded.

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