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Pro-/antiinflammatory dysregulation in early psychosis: results from a 1-year follow-up study.

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Abstract

BACKGROUND:

Previous studies indicated a systemic deregulation of the pro-/antiinflammatory balance in subjects after 6 months of a **first psychotic episode**. This disruption was reexamined 12 months after diagnosis to identify potential risk/protective factors and associations with symptom severity.

METHODS: Eighty-five subjects were followed during 12 months and the determination of the same pro-/antiinflammatory mediators was carried out in plasma and peripheral blood mononuclear cells. Multivariate logistic regression analyses were used to identify risk/protective factors. Multiple linear regression models were performed to detect the change of each biological marker during follow-up in relation to clinical characteristics and confounding factors.

RESULTS: This study suggests a more severe systemic pro-/antiinflammatory deregulation than in earlier pathological stages in **first psychotic episode**, because not only were intracellular components of the **inflammatory** response increased but also the majority of soluble elements. Nitrite plasma levels and cyclooxygenase-2 expression in peripheral blood mononuclear cells are reliable potential risk factors and 15d-prostaglandin-J2 plasma levels a protection biomarker. An interesting relationship exists between antipsychotic dose and the levels of prostaglandin-E2 (inverse) and 15d-prostaglandin-J2 (direct). An inverse relationship between the Global Assessment of Functioning scale and lipid peroxidation is also present.

CONCLUSIONS: Summing up, pro-/antiinflammatory mediators can be used as risk/protection biomarkers. The inverse association between oxidative/nitrosative damage and the Global Assessment of Functioning scale, and the possibility that one of the targets of antipsychotics could be the restoration of the pro-/antiinflammatory balance support the use of antiinflammatory drugs as coadjuvant to antipsychotics.

KEYWORDS: antipsychotics; biomarker; **first-episode psychosis**; inflammation; oxidative damage; risk/protective factors

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