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EDITORIAL

Differential diagnosis of major depression and bipolar disorder

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This issue is dedicated to the differential diagnosis of affective disorders with focus on clinical and neuroimaging markers. From a first view, psychosocial dysfunction is mainly associated with schizophrenia. However, Bottlender et al. [1] analyzed the association between psychosocial function and psychopathology in schizophrenia, schizoaffective and affective disorder. For all diagnostic groups, they found higher levels of psychopathology to be associated with higher levels of psychosocial dysfunction in various domains, highlighting the importance of symptom remission in major psychiatric disorders. Major depression and bipolar disorder are known to have a high heritability. Serretti et al. [2] investigated the impact of a family history on clinical and socio-demographic variables. They found a positive family history more common in bipolar patients than in major depression plus an association with anxiety disorders and age of onset in both diseases. However, hypomania is frequent in patients with major depressive episodes, and the DSM-IV concept of hypomanic episodes should be revised [3].

The serotonergic system, including the serotonin transporter [4] and serotonin 1A receptor genes, plays a major role in the pathophysiology of mood disorders. In a meta-analysis, Kishi et al. [5] are reporting two SNPs of the receptor gene to be associated with major depression and overlapping with bipolar disorder. Therefore, the neurobiological classification of subtypes of mood disorders is an unresolved challenge and alterations in GABA synthesis have been proposed to discriminate unipolar from bipolar I depression [6]. Discrimination of unipolar and bipolar depression at early stages of the illness could help improving specific treatment regimes. Based on a functional MRI paradigm with pattern classification, Grotegerd et al. [7] found feature weights in medial prefrontal and orbitofrontal regions specific for unipolar depression, whereas the dorsolateral prefrontal cortex contributes to classification as bipolar. For negative faces condition, the amygdala showed strong feature weights in unipolar depression, while during positive face presentation, higher amygdala feature weights were detected, thus discriminating between the two diseases. However, the results have to be replicated in a larger group of unmedicated patients. With respect to brain volumes, antipsychotic treatment has been shown to be associated with structural alterations [8]. Keeping this in mind, McFarland et al. [9] report symptom misattribution to be related to increased gray matter volumes of the caudate nucleus, thalamus, insula, putamen and cerebellum in first-episode schizophrenia patients, while in chronic patients under antipsychotic treatment, no volume alterations have been found to be associated with insight.

Although affective disorders are frequent in the post-partum period, knowledge about psychosocial factors influencing maternal health is still sparse. Escribà-Agüir et al. [10] investigated effects of psychological violence, social support and alcohol or drug abuse of the intimate partner on psychological well-being at 5 and 12 months post-partum. While intimate partner violence increased the risk of deficits in well-being, having an unstable social network, alcohol and drug abuse of a family member are predictors of post-partum depression, highlighting the impact of environmental factors. Another risk factor for depression is higher age and decreased functional capacity as a common problem among elderly. In a group of 2,072 individuals living in a low-income area of Sao Paulo and

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aged 65 years or older, Almeida da Silva et al. [11] showed depression to be strongly associated with high functional disability. However, with appearance of depressive symptoms in the elderly, differential diagnosis is early dementia as a major cause for functional deficits.

Unraveling plasma biomarkers pose one of the challenges in psychiatric research. Bipolar disorder has been shown to be associated with a proinflammatory status involving altered plasma cytokine levels [12]. The same group now reports altered levels of chemokines in bipolar disorder [13], contributing to the hypothesis of disturbed immune function. Those findings may lead to better understand disease mechanisms and treatment strategies. Electroconvulsive therapy (ECT) is commonly used in treatment-resistant depression and catatonia. In schizophrenia, ECT may lead to recovery from disruptive behavior [14]. In major depression, synergistic effects of ECT and ketamine anesthesia have been reported to improve cognitive side effects [15]. Estimation of the seizure threshold is used in weighing effectiveness against the risk of side effects. In a prospective study, van Waarde et al. [16] found higher age and bifrontotemporal electrode placement to be predictors for higher seizure threshold levels at different time points, which should be taken into account when selecting ECT dosage. With respect to treatment of affective disorders, new treatment strategies based on mechanisms of neuroplastic processes should be developed and lead to regeneration of disturbed brain function.

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