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Letters to the Editor

Post-stroke depression and cerebral laterality

Sir,

Stroke is the most common serious disorder in the world accounting for half of all the acute hospitalization cases of diseases [3]. Until recently, little attention has been paid to the neuropsychiatric sequelae of stroke. Current observations demonstrate that neuropsychiatric disorders are frequent after stroke and lesions of specific regions or functional systems of the brain result in behavioral syndromes similar to idiopathic conditions [1]. Views have been expressed that the risk of depression is greater with left and anterior hemispheric strokes as compared to posterior and right hemispheric strokes. However, the subject remains controversial. There are few reports in literature in which the symptoms of depression have been reported with right hemispheric stroke.

In a study consisting of 15 patients with stroke, 10 had left sided lesions and depression was seen in seven patients (70%) whereas three (30%) had symptoms resembling generalized anxiety disorder. In rest of the five patients, lesions were found in the right hemisphere and out of these three (60%) had depression and one (20%) had symptoms resembling generalized anxiety disorder whereas one had symptoms resembling obsessive compulsive disorder. In a majority, the onset of symptoms was in the second week followed by those in third and fourth weeks. Hypertension was present in all the patients whereas four also had diabetes mellitus. There was family history of stroke in five patients.

Depression occurs in 30–50% of patients after stroke [1]. Robbinson and Price [4] have found depression and vegetative symptoms in 63% of patients with left hemispheric injury and 1.4% in patients with right hemispheric injury. Carson et al. [2] did a meta-analysis of 143 articles and included 48 articles in the study. Only two reports of original data supported the views of association of post-stroke depression with site or laterality. In the present study also, no association of hemispheric laterality with depression was found in patients with stroke.

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Amisulpride-associated pedal edema

Sir,

A new generation of antipsychotics, such as risperidone [1] and olanzapine [2], has been reported to be associated with pedal edema in patients with schizophrenia. Amisulpride was recently marketed for the treatment of schizophrenia. It is known to have a satisfactory safety profile [3], and there have been no case reports regarding amisulpride-related pedal edema. Here we report a case of bilateral pedal edema after the use of amisulpride.

Mr. Lin is a 51-year-old male who was first admitted to psychiatric ward with the symptoms of delusions of being controlled, persecution and auditory hallucination for one and a half years. He was treated with amisulpride 400 mg per day and lorazepam 2 mg per day. Within two weeks, amisulpride was increased gradually to 800 mg per day, and the psychotic symptoms improved markedly. However, approximately three weeks after amisulpride treatment, Mr. Lin reported significant bilateral pedal edema. Physical examination was unremarkable except 3+ pitting edema on both lower extremities. He had no past history of cardiac, hepatic, vascular, immunologic, or renal diseases. Laboratory examination showed results of urinalysis, blood count, electrolytes, renal, liver and thyroid function were within normal limits. However, immunoelectrophoresis study demonstrated that the IgE value was elevated up to 57.1 IU/ml (normal range <28.6 IU/ml), whereas C3 and C4 were within normal limits.

Diuretic therapy with furosemide 40 mg per day resolved the edema completely within four days. Thereafter, furosemide was continuously administered 40 mg twice per day with amisulpride 800 mg per day. Pedal edema repeatedly

appeared when the diuretic was withheld. The treatment strategy was then changed to taper the dosage of amusulpride from 800 to 400 mg per day and to stop using the diuretic. Within one week with this strategy, the bilateral pedal edema resolved entirely. The patient's psychiatric condition remained stable on this dosage of amisulpride.

In our case, treatment of amisulpride alone or an adverse interaction between amisulpride and lorazepam may be responsible for the patient's bilateral pedal edema. The latter is less likely because amisulpride and lorazepam have not been reported to have significant pharmacokinetic interaction. In addition, amisulpride appeared to be associated with the patient's pedal edema in a dose-related fashion since the pedal edema disappeared after prescribing a lower dosage of amisulpride. The finding of elevated IgE with normal values of C3 and C4 was similar to a previous report regarding risperidone-associated allergic reaction [4]. In that report, the authors suggested that the allergic reaction in their case might be type I or type IV allergic reaction since IgE level was elevated and C3/C4 levels were within normal limits. It suggests that the pathogenesis of pedal edema in both cases might be caused by a similar mechanism. However, the pathophysiological mechanism of amisulpride-associated pedal edema is still not clear as of edema associated with other new antipsychotic agents. It is subject to further clinical investigation and research concerning this issue.

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Antidepressant-associated mania with escitalopram

1. To the editor

Escitalopram, the single isomer of citalopram recently commercially available in Europe, is a potent serotonin transporter (SERT) antagonist and possesses the greatest SERT selectivity [4]. Although the treatment with the SSRI citalopram has a reduced emergence of antidepressant associated mania (AAM) in patients with unipolar depression we describe the case of AAM in a patient undergoing escitalopram therapy [1,2].

Mr. H. a 33-year-old Caucasian male inpatient, heavy smoker (>20 cigarettes/d), with a body mass index of 24.36, was diagnosed for the first time in 1992 as suffering from unipolar depressive disorder in comorbidity with a narcissistic personality disorder according to ICD-10. He was rehospitalized for the 3rd time after experiencing his 5th severe depressive episode manifested by sleep disturbances, depressed mood, decreased interest and by suicidal ideations. Based on previous experiences of treatment resistance to citalopram, the new compound escitalopram was started tapering the dosage from 20 to 40 mg/d reaching a steady state plasma concentration of 60 ng/ml (corresponding to 40–80 mg/d of citalopram) [5]. For sleep induction lorazepam 2 mg was given. Ten days following this treatment regimen within 2 days the patient suddenly developed severe insomnia, racing thoughts, talkativeness, elevated mood, psychomotor restlessness, aggressive and uncontrolled behaviour with sexual desinhibition toward other inpatients. Carbamazepine at 600 mg/d combined with risperidone 2 mg/d was prescribed. Escitalopram was tapered off within 10 days and just 15 days after its discontinuation the manic episode resolved. It is noteworthy that in a previous treatment trial with ECT during his 4th depressive episode (diagnosed as therapy refractory) the patient manifested a delirium with euphoria accompanied by confusion and memory disturbances and thus discernable from a switch to hypomania [3]. This presumed delirious state subsided after discontinuation ECT and prescribing the mood stabilizer valproic acid the patient recovered as described in the index episode.

This case suggests that (1) escitalopram has the potency to induce a AAM in patients affected by unipolar depression rather than citalopram, (2) the previously observed delirium with euphoria retrospectively was an ECT emergent mania indicating the difficulty to distinguish between these two conditions and (3) the prescription of escitalopram may require particular monitoring in patients suffering from bipolar disorders since these patients are most likely to exhibit hypomanic or manic reactions.

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