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Some particularities of depression in diabetic patients

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The presence of diabetes doubles the odds of comorbid depression. In patients with preexisting diabetes, depression is an independent risk factor for coronary disease and appears to accelerate the presentation of coronary heart disease. Concurrent depression is associated with a decrease in metabolic control of diabetes mellitus, poor adherence to medication and diet regimens, a reduction in quality of life and an increase in health care expenditures.

Objective: To diagnose and treat the depression illness in diabetic patients.

Methods: A sample of 30 diabetic patients (15 women, 15 men), mean age 59,6 years was assessed for depression - ICD 10 criteria. HAM-D (Hamilton for depression scale), CGI-S (Clinical global impression –severity) and CGI-I (Clinical global impression –improvement) were performed at baseline, 7, 14, 21, 28 and 42 days. Patients received antidepressive medication: tianeptine 37,5 mg/day or venlafaxine 75-150 mg/day.

Results: Mean score HAM D at baseline was 21.4. The reassessment after 7, 14, 21, 28 and 42 days revealed significant decrease of depressive symptomathology after 4 weeks of medication (HAM D was 15.4). After 42 days the mean score HAM-D was 9,5. CGI-S at baseline was 4.5 and on 42 day 1.8. Mean blood glucose was evaluated from 215,5 mg/dl at inclusion and 142,3mg/dl on day 42.

Conclusions: 1) Successful treatment of depression is associated with improvements in glycemic control. 2) Improvements in mood increase the functioning and quality of life. 3) Further studies are important to demonstrate the role of maintenance antidepressant treatment for the prevention of recurrence.

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Effect of once-daily extended release Quetiapine Fumarate (Quetiapine XR) as add-on to antidepressants in major depressive disorder (MDD)

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Objective: To evaluate the efficacy and tolerability of once-daily quetiapine XR adjunctive to antidepressant therapy versus antidepressant alone in patients with MDD showing an inadequate response to antidepressant treatment (mainly SSRIs/SNRIs).

Methods: 6-week, multicentre, double-blind, parallel-group study (D1448C00007). Patients were randomised to receive quetiapine XR 150mg/day (n=167), 300mg/day (n=163) or placebo (n=163) as add-on to maintained antidepressant treatment. Primary endpoint: baseline to Week 6 change in MADRS total score. Secondary variables included: baseline to Week 1 change in MADRS total score; baseline to Week 6 change in HAM-A total and psychic anxiety subscale scores. Safety assessments included AE reporting.

Results: Mean change in MADRS total score (overall baseline mean, 28.4) from baseline to Week 6 was significant ($p<0.01$) for quetiapine XR 150mg/day (-15.26) and 300mg/day (-14.94) versus placebo (-12.21). Separation from placebo in MADRS total score was apparent from Week 1 for both quetiapine doses ($p<0.001$).

At Week 6, mean change from baseline in HAM-A total score (overall baseline mean, 20.8) was significant for quetiapine XR 150mg/day (-10.27, $p<0.01$) and 300mg/day (-9.70, $p<0.05$) versus placebo (-7.92). Mean change from baseline in HAM-A psychic anxiety subscale score (overall baseline mean, 12.83) was significant with quetiapine XR 150mg/day (-6.82, $p<0.001$) and 300mg/day (-6.47, $p<0.01$) versus placebo (-5.11).

Most common AEs (>10%) were dry mouth, somnolence, fatigue, sedation, constipation and dizziness with quetiapine XR.

Conclusion: In patients with MDD with an inadequate response to antidepressant treatment, adjunctive quetiapine XR 150mg/day and 300mg/day was well tolerated and effective at reducing depressive and anxiety symptoms.

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Mindfulness-based cognitive therapy reduces depression symptoms in people with a traumatic brain injury: Results from a pilot study

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Background and Aims: Major depression is a significant problem for people with a traumatic brain injury (TBI) and its treatment remains difficult. A promising approach to treat depression is Mindfulness-based cognitive therapy (MBCT), a relatively new therapeutic approach rooted in mindfulness based stress-reduction (MBSR) and cognitive behavioral therapy (CBT). We conducted this study to examine the effectiveness of MBCT in reducing depression symptoms among people who have a TBI.

Methods: Twenty individuals diagnosed with major depression were recruited from a rehabilitation clinic and completed the 8-week MBCT intervention. Instruments used to measure depression symptoms included: BDI-II, PHQ-9, HADS, SF-36 (Mental Health subscale), and SCL-90 (Depression subscale). They were completed at baseline and post-intervention.

Results: All instruments indicated a statistically significant reduction in depression symptoms post-intervention ($p < .05$). For example, the total mean score on the BDI-II decreased from 25.2 (9.8) at baseline to 18.2 (11.7) post-intervention ($p=.001$). Using a PHQ threshold of 10, the proportion of participants with a diagnosis of major depression was reduced by 59% at follow-up ($p=.012$).

Conclusions: Most participants reported reductions in depression symptoms after the intervention such that many would not meet the criteria for a diagnosis of major depression. This intervention may provide an opportunity to address a debilitating aspect of TBI and could be implemented concurrently with more traditional forms of treatment, possibly enhancing their success. The next step will involve the execution of multi-site, randomized controlled trials to fully demonstrate the value of the intervention.

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Homogeneous expression of candidate genes in patients with major depression is followed by heterogeneous return to normal levels after treatment

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Major depression (MD) is a major public health problem worldwide. Nevertheless, its pathophysiology remains unclear and no specific biological marker has been associated to the disease so far.

To investigate whether such marker(s) exist(s), we collected peripheral blood mononuclear cells (PBMC) from a restricted cohort of MD inpatients at two different time points: at the time of major depressive episode with melancolic features and 8 weeks later (median score on the Hamilton Depression Rating Scale were 38 and 14.5 ($p<0.05$), respectively). We also collected PBMC from age and sex-matched control individuals. Total RNAs were extracted and we studied the mRNA level alterations of 83 candidate genes by qRT-PCR using the TaqMan Low Density Array technology.

When compared to control samples, a significant down- and up-regulation of mRNA level was observed for numerous genes involved in MD. Remarkably, while the transcription level of these genes was heterogeneous within both controls and patients, 8 weeks after the major depressive episode, it was very homogeneous during the acute phase of the disease. Furthermore, some mRNA level variations were statistically correlated to the clinical severity of the symptomatology during the acute phase.

Thus, we conclude that some mRNA level alterations provide a good signature of the MD state.

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Major depression is very frequent in poorly regulated diabetes

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Background and Aims: It is often reported that patients with diabetes have increased risk of suffering from major depression (1). We wanted to study the frequency of depression in an special unit for diabetes at the University Hospital.

Methods: Fiftythree patients were recuited at this outpatient clinic. They were diagnosed using the structured clinical intervju MINI (2).

Results: Of the 53 patients with diabetes, 12 (23%)had an ongoing depressive episode. In addition 8 patients had suffered from previous episodes of depression. Thus 20(38%) had a lifetime history of major depression. Of the 12 patients with an ongoing depression, 58% had a first degree relative with psychiatric disorder, in contrast to 33% in those with no history of depression.

Conclutions: The propotion of depressive disorders in patients with poorly regulated diabetes, is very high. An astonishing finding is the very high frequency of first degree relatives with affective disorders.

It may be speculated that diabetes and depression have some pathophysiological features in common (3).

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Depression, sensitization and chaos in autonomic response: Implications for anticonvulsant treatment

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Background and Aims: According to recent findings stress experiences represent significant condition in pathophysiology of depression and influence abnormal development in the brain. Repeated stress and cognitive conflict also may determine limbic irritability and temporal-limbic epileptic-like activity. Because recent findings indicate that epilepsy and epileptiform processes are related to increased neural chaos, in the distinct contrast to normal brain activity, aim of this study is to find relationship between neural chaos in autonomic responses reflecting brain activity during stress activation and limbic irritability.

Method: For empirical examination of suggested hypothesis Stroop word-colour test, ECG recording, calculation of chaos indices i.e. largest Lyapunov exponents (LLEs) in nonlinear data analysis and psychometric measures of limbic irritability (LSCL-33) and depression (BDI-II) in 35 patients with unipolar depression and 35 healthy controls were used.

Result: Significant correlation $r=0.68$ ($p<0.01$) between LLEs and LSCL-33 found in this study indicate that degree of chaos in autonomic responses during conflicting Stroop task reflected by LLEs is closely related to limbic irritability. Significant correlation $r=0.47$ ($p<0.01$) also has been found between LLEs and symptoms of depression assessed by BDI-II. In the control group similar correlations have not been found.

Conclusion: The results are in agreement with findings that epileptiform activity represents typical form of chaotic organization. Because limbic irritability is linked to seizure-like processes in the temporo-limbic structures, the correlation between LSCL-33 and LLEs might represent useful finding for understanding of neurobiological mechanisms underlying stress-related sensitization and could be useful for future research regarding anticonvulsant treatment of depression.

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How to FACE© polydrug use: Pathways toward an integrative structured care model to facilitate adjustment of cognitions and emotions

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Background: Recent developments in the field of polydrug use along with alcoholism provide growing insights into how cognitive, affective, motivational and neurobiological pathways are altered in addictive persons. Few of these insights have as yet been implemented in everyday care.

Aim and Method: Framed within the multi-site FACE© program (Facilitating Adjustment of Cognitions and Emotions), this paper presents an integrative scientist-practitioner model that aims to translate the above insights into systematized multidisciplinary practice. The pathways from model to structured care are specified using a mixed-method design of bottom-up and top-down approach. Their

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