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Introduction: It is known the relationship between psychological problems and cardiovascular disease. Psychological alterations can cause cardiovascular diseases, and a cardiovascular event can trigger psychological alterations.

Objectives: The aim was to present a clinical case about a young man with cardiovascular alterations and depressive symptoms and to analyze the role played by cardiovascular drugs, psychoactive drugs, and their interactions.

Methods: We present the clinical case and search the relation between cardiovascular disease and depressive symptoms and treatment at scientific literature of last five years.

Results: A 38-year-old man comes to the emergency room with symptoms of palpitations, fatigue and shortness of breath for 2 weeks. An electrocardiogram is performed showing premature ventricular beats. The patient reports that he is sadder recently due to the loss of work, for which he is prescribed sertraline 50 mg daily and is referred to cardiology. No medical history or consumption of alcohol, tobacco or other toxins. The cardiologist requests ergometry, echocardiography, and Holter monitoring, resulting in all normal tests, with no evidence of ischemia. Bisoprolol 2.5 mg daily is prescribed and sertraline 50 mg daily is maintained. After two months, the patient reports feeling better in spirit. The control electrocardiogram is normal and the patient reports disappearance of palpitations. You are referred to your family doctor.

Conclusions: Elevation of cortisol, platelet hyperactivity, and alteration in heart rate variability were found in depressives. The SSRIs would be the ones of choice. Dual serotonin and noradrenaline reuptake inhibitors should be avoided. Other atypical drugs such as bupropion or trazodone should be considered.

Disclosure: No significant relationships.

Keywords: Depression; Psychotropic drugs; mental health and cardiovascular disease; arrhythmia

EPV0243

They are not real patients

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Introduction: Cognitive depressive disorder (or depressive pseudodementia) is a condition defined by functional impairment, similar to dementias or other neurodegenerative disorders, in the context of psychiatric patients. It is important to consider a differential diagnosis in patients with cognitive impairment.

Objectives: Presentation of a clinical case of a patient with depression with psychotic symptoms who presents cognitive impairment.

Methods: Bibliographic review of the differential diagnosis between cognitive depressive disorder and real dementia by searching for articles in PubMed.

Results: We present a 51-year-old woman, previously diagnosed with adjustment disorder (with mixed anxiety and depressed mood) and unspecific anxiety disorder, who was admitted to the hospital due to delusional ideation of harm and Capgras syndrome, ensuring that her relatives had been replaced and the rest of the patients were not real patients, but actors who conspired against her. The MRI (Magnetic Resonance Imaging) was strictly normal (tumors or acute injuries as stroke or hemorrhage were discarded), and a MoCA (Montreal Cognitive Assessment) test was performed to screen any cognitive impairments (obtaining a score of 19/30, with language fluency and abstraction particularly affected). It would be convenient to repeat the test when this episode and the psychotic symptoms are resolved or improved.

Conclusions: 1. Some patients may have cognitive impairment in the context of a mood disorder. 2. A differential diagnosis and follow-up of these patients should be performed to assess prognosis, reversibility and treatment. 3. Depressive cognitive impairment may precede the development and establishment of a dementia or neurodegenerative picture.

Disclosure: No significant relationships.

Keywords: Depressive pseudodementia; psychotic depression; cognitive impairment; cognitive depressive disorder

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Hyperoxia in depression

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Introduction: Several studies of normobaric hyperoxia in some neurological conditions have demonstrated clinical benefits. Oxygen enriched air may increase oxygen pressure in brain tissue and have biochemical effects such as on brain erythropoietin gene expression, even in patients without lung disease.

Objectives: This pilot, randomized, double-blind study examined the efficacy of normobaric hyperoxia as a treatment for depression.

Methods: Fifty-five consenting patients aged 18-65 years with mild to moderate depression were included in the study. Participants underwent a psychiatric inclusion assessment and a clinical evaluation by a psychiatric nurse at baseline, 2 and 4 weeks after commencement of study intervention. Participants were randomly assigned to normobaric hyperoxia of 35% fraction of inspired oxygen or 21% fraction of inspired oxygen (room air), through a nasal tube, for 4 weeks, during the night. Patients were rated blindly using the Hamilton Rating Scale for Depression (HRSD); Clinical Global Impression (CGI) questionnaire; Sheehan Disability Scale (SDS).

Results: The present study showed a significant improvement in HRSD ($p < 0.0001$), CGI ($p < 0.01$) and in SDS ($p < 0.05$) among patients with depression who were treated with oxygen-enriched air, as compared to patients who were treated with room air. In CGI, 69% of the patients who were treated with oxygen-enriched air improved compared to 23% patients who were treated with room air.

Conclusions: This small pilot study showed a beneficial effect of normobaric hyperoxia on some symptoms of depression.