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NEWSLETTER
INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

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This issue contains:

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Best Regards,

Medical Team, Micro Labs Limited.

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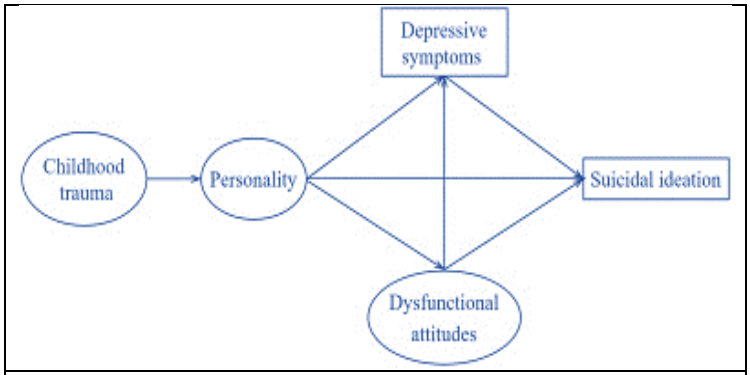
1. A study on the prevalence of comorbid suicide attempts and its clinical correlates in individuals with early-onset and late-onset major depressive disorder

Introduction:

Depression is one of the most common chronic illnesses in the world, affecting an estimated 264 million people.[1] Major depressive disorder (MDD) is a serious psychiatric illness with significant economic consequences worldwide. Suicide is the most serious side effect of MDD, and it happens up to 15% of people with recurring MDD, even after getting pharmacological treatment. Suicide attempts are self-directed harmful behaviours carried out with the intention of dying, and they create a major threat to the public's health.[2] The relationship between depression and suicide attempts has gained a lot of attention in recent years. Suicide in MDD and other mood disorders may have multiple dimensions, several simple treatments, and maybe a different neurological basis. Suicide prevention requires precise and individual psychiatric assessment.[1] The goal of this study was to evaluate the frequency and clinical correlates of suicide attempts among MDD patients with early-onset and late-onset disease.



Suicidal ideation in patient having major depressive disorder

Methods:

A total of 1718 adult patients with MDD were enrolled in this cross-sectional investigation. Each patient's sociodemographic data, including age, gender, marital status, age of MDD onset, length of disease, and level of education, was collected. Clinical data, which were collected by qualified investigators, included body weight and height, as well as systolic and diastolic blood pressure. The Positive and Negative Syndrome Scale (PANSS) positive subscale, the Clinical Global Impression-Severity (CGI-S) Scales, the 17-item Hamilton Rating Scale for Depression (HAMD-17), the Hamilton Anxiety Rating Scale (HAMA), and the severity of the clinical symptoms were all used to evaluate the subjects' depressive, anxiety, and psychotic symptoms, respectively.

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Results:

Late-onset MDD patients had a greater rate of suicide attempts (21.3%) than early-onset MDD patients (15.8%) ($p = 0.023$). (Figure 1) Univariate analysis revealed that the following factors were significantly related to suicide attempts in both early-onset and late-onset groups: HAMA, HAMD, and PANSS positive subscale scores; blood glucose levels; thyroid stimulating hormone (TSH) levels; systolic blood pressure (SBP); and diastolic blood pressure (DBP). Both the early-onset and late-onset groups had significantly higher prevalence rates of serious anxiety disorders and psychotic symptoms than the non-suicide attempt group. Regression analysis revealed that TSH levels, HAMA, and HAMD scores were all independently linked to suicide attempts in the late-onset group, while disease duration, TSH levels, and HAMA scores were all independently linked to suicide attempts in the early-onset group.

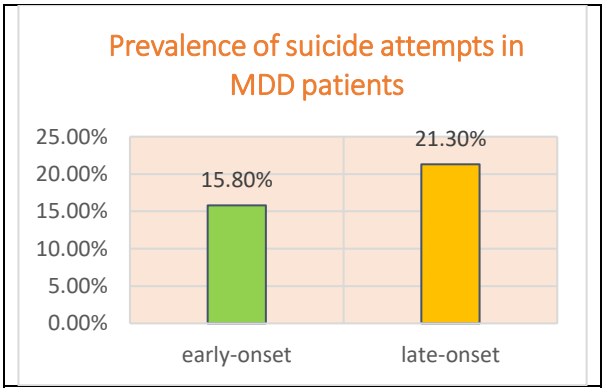

Figure 1: Prevalence of suicide attempts in MDD patients

Discussion:

This study found that longer disease duration, higher TSH levels, and anxiety symptoms were risk factors for comorbid suicide attempts in patients with early-onset MDD, and that higher TSH levels, SBP, HAMA score, and HAMD score were risk factors for suicide attempts in patients with late-onset MDD.[1] According to Fang X et al., a Chinese MDD patient's overall disease duration was a risk factor for suicidal ideation.[3] According to Liang S. et al., patients with MDD have a higher risk of suicide attempts when they experience more depressive episodes.[4]

Conclusion:

This study concludes that suicide attempts are less common in outpatients with early-onset MDD than those with late-onset MDD, and both early- and late-onset MDD exhibit certain clinical correlations with suicide attempts.

Suicide can be prevented with the implementation of a long-term study of the thyroid stimulating hormone (TSH) and Hamilton Anxiety Rating Scale (HAMA) in both early and late-onset MDD.

References:

1. Huang X, et al. Differences in the prevalence and clinical correlates of comorbid suicide attempts in patients with early-and late-onset major depressive disorder. Frontiers in Psychiatry. 2023 Aug 1; 14:1173917.
2. Zhou Y, et al. The association of clinical correlates, metabolic parameters, and thyroid hormones with suicide attempts in first-episode and drug-naïve patients with major depressive disorder comorbid with anxiety: a large-scale cross-sectional study. Translational psychiatry. 2021 Feb 4;11(1):97.
3. Fang X, et al. Prevalence, risk factors and clinical characteristics of suicidal ideation in Chinese patients with depression. J Affect Disorder. 2018 Aug 1; 235:135-14
4. Liang S, et al. Incidence Trends and Risk Prediction Nomogram for Suicidal Attempts in Patients with Major Depressive Disorder. Front Psychiatry. 2021 Jun 23; 12:644038.

2. A study on early antipsychotic non-response as a marker of later non-response in schizophrenia

Introduction:

Schizophrenia is a serious, persistent, and severely disabling mental disorder, and despite extensive and long-term study, the outcomes of best-practise treatment are frequently inadequate.[1] The most effective treatment for schizophrenia is antipsychotic medication. After clinical practise, this treatment is ineffective; after just 4-6 weeks, 19.8–66.9% of schizophrenia patients do not respond to antipsychotics or only partially do so. A clinically significant improvement in the patient's clinical symptoms characterises the response, which is measured by a percentage reduction of the initial total score on symptom rating measures like the Positive and Negative Syndrome Scale (PANSS), which typically ranges from 20 to 50%. A patient's response to antipsychotics is often evaluated by clinicians after a course of treatment.[2] The goal of this study was to determine the best predictive cut-off value for early non-response that would more accurately predict later non-responses to antipsychotics in schizophrenia patients.

Methods:

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A total of 964 patients were included in this randomised control study. All registered subjects were given an 8-week course of either aripiprazole, amisulpride, risperidone, or olanzapine monotherapy. A baseline, week 2, week 4, and week 8 evaluation of the positive and negative syndrome scale (PANSS) were performed. The prediction of nonresponse was the primary outcome. A 20% decrease in the PANSS total scores between the baseline and endpoint is regarded as nonresponse. The baseline PANSS total scores of 58, 75, and 95, respectively, were the severity ratings for mild, moderate, and severe illnesses.

Results:

The patients with severe and mild schizophrenia, as well as those who were treated with the risperidone and amisulpride groups, showed the highest total accuracy at week 2, when a reduction of <5% in the PANSS total score was observed. The moderate schizophrenia group, the olanzapine group, and the aripiprazole group all demonstrated the best overall accuracy for a 10% decrease. The percentages of non-responders for patients with severe schizophrenia in the olanzapine, risperidone, amisulpride, and aripiprazole groups were 35.5%, 27.5%, 29.2%, and 60.0%, respectively. In comparison to the other treatment groups, the aripiprazole group had a significantly larger number of non-responders ($P < 0.001$). The percentages of non-responders in the four groups are the same among patients with mild and moderate schizophrenia. (Figure 1) Regardless of the antipsychotic or the severity of the illness, the best predicted cut-off value at week 4 was < 20% (overall accuracy ranged from 89.8 to 92.1%).

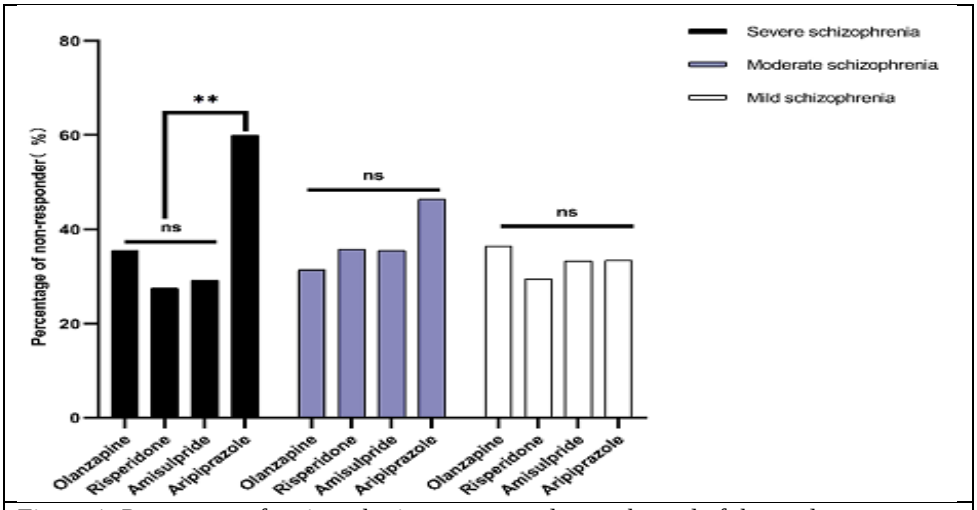

Figure 1: Percentage of antipsychotic non-responders at the end of the study

Discussion:

This study showed that the best prediction thresholds varied depending on the different predicted time points. The two symptom reductions of <20% at week four (early non-response) and <10% at week two (early non-response) showed the strongest predictive value for non-response at week 8.[2] According to the Pagsberg AK et al. study, patients on aripiprazole or quetiapine experienced a <20% reduction in symptoms at week 2 and a <30% reduction at week 4.[3] According to research by Zhang L. et al., 51.05% of respondents were in the early non-response group. The early non-response group had significantly higher PANSS scores than the early response group.[1]

Conclusion:

A more precise predictive cut-off value should be established based on the medication regimen and baseline illness severity. Symptom reduction at week 2 shows adequate discrimination in predicting later non-response to antipsychotics in schizophrenia.

The drug regimen and baseline illness severity should be considered in order to establish a more precise prediction cut-off value for schizophrenia.

References:

1. Zhang L, et al. Incidence and factors associated of early non-response in first-treatment and drug-naïve patients with schizophrenia: a real-world study. Front Psychiatry. 2023 Apr 27; 14:1173263.
2. Long Y, et al. Early non-response as a predictor of later non-response to antipsychotics in schizophrenia: a randomized trial. BMC Med. 2023 Jul 19;21(1):263.
3. Pagsberg AK, et al. Early Antipsychotic Nonresponse as a Predictor of Nonresponse and Nonremission in Adolescents with Psychosis Treated with Aripiprazole or Quetiapine: Results from the TEA Trial. J Am Acad Child Adolescent Psychiatry. 2022 Aug;61(8):997-1009.

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3. A study on evaluation of childhood trauma and anger in adults with and without depressive and anxiety disorders

Introduction:

Childhood trauma (CT) has adverse effects on mental health that can last far into adulthood. The World Health Organisation (WHO) defines CT as "all forms of physical and/or emotional ill-treatment, sexual abuse, commercial or other exploitation, neglect, or negligent treatment, resulting in actual or potential harm to the child's health, survival, development, or dignity in the context of a relationship of responsibility, trust, or power."[1] Childhood trauma is more commonly reported in bipolar disorder (BD) patients than in healthy individuals and raises the severity and complexity of the clinical appearance of BD with symptoms like an earlier age at onset, rapid cycling or mixed episodes, and an increased risk of suicide attempts.[2] Up to one out of every four children in the Netherlands reported experiencing some sort of maltreatment at some point, while the frequency of CT is probably underreported because of fear, concealment, and stigma.[1] The objectives of this study were to (1) determine if childhood trauma predicts adult anger and, if so, (2) determine which types of childhood trauma are more prevalent in the prediction of anger in a cohort of adults with and without present affective disorders.

Methods:

In this prospective study, a total of 2271 patient's data were taken from the Netherlands Study of Depression and Anxiety (NESDA). Childhood trauma was evaluated using the semi-structured Childhood Trauma Interview (CTI) at baseline, and anger was analysed in relation to anger using the Spielberger Trait Anger Subscale (STAS), the Anger Attacks Questionnaire, and cluster B personality traits (i.e., borderline, antisocial) of the Personality Disorder Questionnaire 4 (PDQ-4) at a 4-year follow-up. Cross-sectional regression analyses utilising the Childhood Trauma Questionnaire-Short Form (CTQ-SF), which was also obtained at a 4-year follow-up, were included in the post hoc analysis.

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Results:

All anger dimensions were associated in a dose-dependent manner with childhood trauma. Independent of anxiety or depression, borderline personality traits were found to be highly correlated with all forms of childhood trauma. A higher prevalence of trait anger, rage attacks, and antisocial personality traits in adults was also linked to all types of childhood trauma, with the exception of sexual abuse. (Figure.1) A greater trait anger score was linked to a higher score on childhood trauma ($p < 0.001$). Additionally, participants who had experienced childhood trauma had significantly greater rates of angry outbursts ($p < 0.001$), borderline personality traits ($p < 0.001$), and antisocial personality traits ($p = 0.002$) than participants who had no history of childhood trauma.

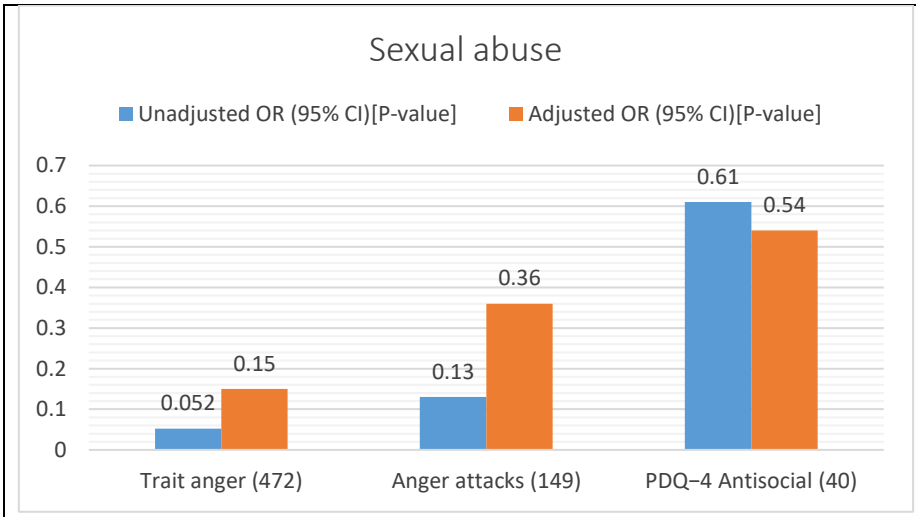

Figure 1: Various anger measurements' (adjusted) odds ratios in relation to sexual abuse

Discussion:

This study shows that a history of CT is linked to higher levels of trait anger as well as a higher prevalence of adult rage attacks, borderline personality traits, and antisocial personality traits.[1] According to a study by De Venter M. et al., childhood trauma is more closely linked to the onset of social phobia and the chronicity of anxiety symptoms than it is to the persistence of panic disorder. These correlations can be explained in part by extraversion and neuroticism, as well as the duration and intensity of depressive and anxious symptoms.[3]

Conclusion:

This study concludes that childhood trauma is associated with adult rage, which is particularly intriguing in the context of psychopathology. Patients with depression and anxiety disorders may benefit from treatment that focuses on childhood trauma and adult rage. When necessary, trauma-focused therapies should be used.

Childhood trauma is linked to adult anger, including a dose-response relationship, and subtypes of childhood trauma show different consequences for adult struggles to control their anger.

References:

1. De Bles N, et al. Childhood trauma and anger in adults with and without depressive and anxiety disorders. European Psychiatry. 2023 Mar;66(S1): S89-90.
2. Aas M, et al. Affective lability mediates the association between childhood trauma and suicide attempts, mixed episodes and co-morbid anxiety disorders in bipolar disorders. Psychological medicine. 2017 Apr;47(5):902-12.
3. De Venter M, et al. Impact of childhood trauma on course of panic disorder: contribution of clinical and personality characteristics. Acta Psychiatrica Scandinavica. 2017 Jun;135(6):554-63.

4. A Case Report of Postictal Psychosis

Introduction:

Patients who have a lengthy history of seizures are more likely to develop serious psychiatric conditions, such as epileptic psychosis. Psychotic syndromes are particularly common in individuals with long-term epilepsy who have a pharmacoresistant profile or in epileptic patients who are nonadherent to therapy [(1)]. Up to 50% of people with epilepsy suffer from psychiatric illnesses; however, the frequency is higher in temporal lobe-specific focal epilepsies than in idiopathic generalised epilepsies. Approximately 6% of epileptics suffer from psychotic illnesses, and their risk of psychosis is nearly eight times higher than that of the general population [(2)]. The psychotic symptoms of ictal psychosis (IP) take place during a seizure or an epileptic state. Interictal psychosis (IIP) is characterised as a psychotic episode without impairment of consciousness that is not temporally connected to ictal events. It can be either transient or chronic, the latter of which lasts for

more than three months. On the other hand, Logsdail and Toone's diagnostic criteria define postictal psychosis (PIP) as psychotic episodes beginning less than a week after an epileptic seizure, particularly clusters of focal seizures accompanied by impaired awareness that may or may not progress to generalised tonic-clonic (GTC) seizures [1]. This case report describes a long-term epileptic female patient with a history of nonadherence to antiepileptic treatment and poorly controlled seizures who had a clinical picture of PIP, characterised by pleomorphic features, without Schneider's first-rank symptoms or negative symptoms of schizophrenia.

Case Presentation:

A 44-year-old female patient was admitted to a psychiatric ward for a psychotic episode that had started three days earlier and was marked by perplexity, self-referent and mystical delusions, irritability, disorganised behaviour, and aggression. The psychotic episode had emerged shortly after a cluster of nocturnal GTC seizures. The patient had no personal or family history of psychiatric disease. Both the positive and negative signs of schizophrenia were absent, including Schneider's first-rank symptoms. During psychiatric hospitalisation, there was also considerable mental confusion with temporal disorientation as well as prolonged, split attention and memory problems. The patient's previous medical history was relevant for epilepsy, which had emerged after a moderate-to-severe traumatic brain injury (TBI) in her early thirties. Seven years prior to the hospitalisation, ancillary tests performed in a neurology ward had revealed a right cortico-subcortical parietooccipital lesion of encephalomalacia (Figure 1) on MRI and a focal slowing activity in the left frontotemporal topography on electroencephalogram (EEG). Haematological and biochemical screening, thyroid function, folic acid, and cyanocobalamin were all normal during the patient's hospitalisation in the psychiatric unit for the index psychotic episode. The serologies for

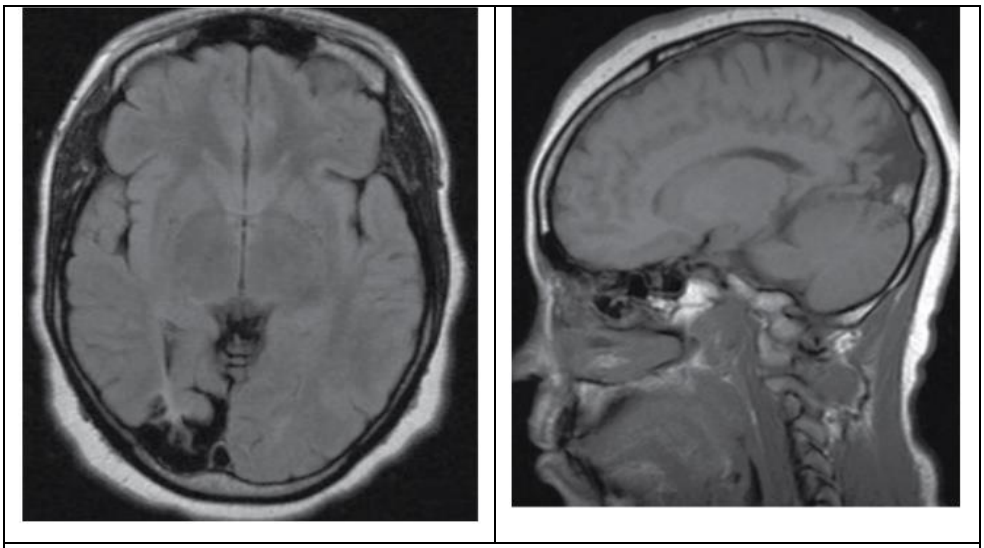

Figure 1: Right cortico-subcortical parietooccipital lesion of encephalomalacia on axial (a) and parasagittal (b) planes of MRI.

VDRL, HIV, and hepatitis B and C were all negative, and the patient's valproic acid levels were below therapeutic ranges. The patient was prescribed aripiprazole 10 mg, valproic acid 1000 mg and diazepam 10 mg. The patient's psychotic symptoms disappeared after 36 hours of admission. Aripiprazole was gradually reduced over the next few months. With the improvement in seizure control, there was no recurrence of psychotic symptoms during the outpatient follow-up consultations with the neurologists.

Discussion:

The presence of at least one of the two nuclear diagnostic criteria, namely a reduction in epileptiform activity on the EEG or the absence of seizures for more than one week, is required for the diagnosis of psychosis secondary to forced normalisation. It is necessary to add another diagnostic criteria, such as a recent modification of antiepileptic medication or prior instances of behavioural problems comparable to those now reported by collateral informants and suggestive of psychotic symptoms [(1)]. Sherer M, et al. revealed that it is important to distinguish between psychotic symptoms and a posttraumatic confusional state, which can present as fluctuating disorientation, cognitive decline, psychomotor restlessness, sleep-wake disturbance, and transient psychotic symptoms while the TBI is being recovered [(3)].

Conclusion:

The extended period between the onset of posttraumatic epilepsy and the emergence of PIP observed in this case provides support to the hypothesis that reduced physiological and structural changes in the brain, including kindling phenomena and secondary brain injury with cerebral volume loss, increased psychosis vulnerability and eventually led to a situation in which a cluster of seizures could initiate a psychotic episode.

Gradual anatomical and physiological alterations in the brain, such as kindling phenomena and secondary brain damage with cerebral volume loss, enhanced the susceptibility to psychosis and finally created a scenario in which a series of seizures may start a psychotic episode.

References:

1. Regala J, et al. Postictal Psychosis: Case Report and Literature Review. Case Reports in Psychiatry. 2023 Mar 16;2023.
2. Braatz V, et al. Postictal psychosis in epilepsy: a Clinicogenetic study. Annals of neurology. 2021 Sep;90(3):464-76.
3. Sherer M,et al. Post-traumatic confusional state: a case definition and diagnostic criteria. Archives of physical medicine and rehabilitation. 2020 Nov 1;101(11):2041-50.