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Vol 2
ISSUE 12
Petril Times
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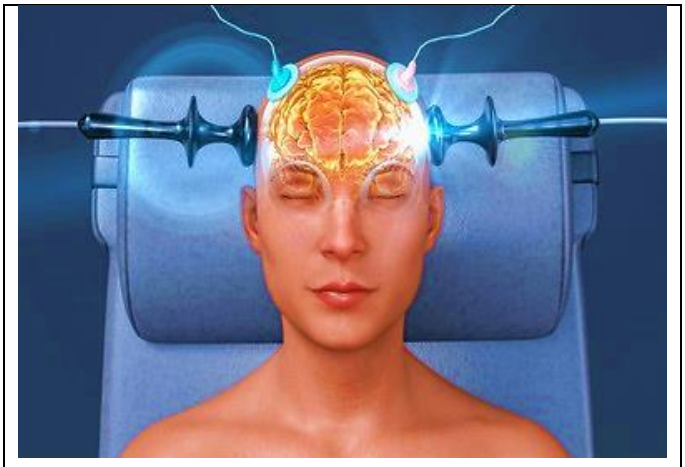
1. Comparison of antidepressant efficacy between Electroconvulsive Therapy and Magnetic Seizure Therapy for Major Depressive Episodes

Introduction

Major depressive disorder (MDD), a primary cause of global disability, was associated with severe morbidity and mortality. Electroconvulsive therapy (ECT) has a risk of negative neurocognitive effects, even if it was quite effective, especially for individuals with acute suicide risk, treatment resistance, or psychosis.[1] Since remission rates for major depressive illnesses can range from 40% to 70%, electroconvulsive therapy was typically regarded as one of the most effective antidepressant treatments. ECT has been shown to affect patients' cognitive abilities, particularly their memory, according to earlier research. Therefore, the broad range of clinical use of ECT was limited because of fear and worries regarding cognitive impairment caused by ECT. Recently, magnetic seizure therapy (MST) has become a popular physical therapy approach for the treatment of depression. It has been demonstrated through studies that MST treatment for MDD can not only maintain high cognitive function and greatly reduce symptoms of depression, but its antidepressant effects may also be connected to local metabolic alterations in the bilateral frontal cortex linked to MDD. Using transcranial magnetic stimulation (TMS), high frequency, strongly pulsed magnetic fields were continuously used in MST technology to stimulate the cerebral cortex. By stimulating the local cortex, it causes convulsive seizures. It combines the low-side effect of TMS with the antidepressant effect of ECT.[2] So, this study aimed to assess the antidepressant efficacy of MST vs ultrabrief pulse right unilateral (RUL) ECT.



Electroconvulsive Therapy for Major Depressive Episodes

Methods

In this randomized controlled trial, 73 patients with a severe depressive episode in the context of major depressive disorder or bipolar disorder with a baseline 24-item Hamilton Depression Rating

Vol 2
ISSUE 12
Petril Times
NEWSLETTER
INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

Scale (HDRS-24) total score of 18 or above were enrolled. Patients were randomized into two groups: one for ultrabrief pulse RUL ECT and another for MST treatment. Following the course of treatment, patients were monitored for up to six months in order to assess the long-term clinical consequences. The major outcome was a change in the HDRS-24 total score from the baseline, and patients were monitored for a maximum of six months. A total score of 8 or less and a reduction of at least 50% in the HDRS-24 score showed remission, and at least a 60% decrease in the HDRS-24 score indicated response.

Results

Both MST and ECT showed antidepressant effects that were clinically significant. Thirteen (37.1%) of the MST group and ten (26.3%) of the ECT group fulfilled the remission criteria in the intent-to-treat sample, while 18 individuals (51.4%) in the MST group and 16 (42.1%) in the ECT group satisfied the response criteria. The MST group had 17 of 29 completers (58.6%), and the ECT group had 15 of 24 (62.5%) who met response requirements; the ECT group had 10 of 24 (41.7%), and the MST group had 13 of 29 (44.8%) who fulfilled remission criteria. Remission rates and response rates did not significantly differ between MST and ECT. The HDRS-24 score significantly decreased in both the MST and ECT groups from the beginning of therapy to the end of treatment (Figure 1). Over a follow-up period of six months, both MST and ECT demonstrated a persistent benefit, with no significant difference between them.

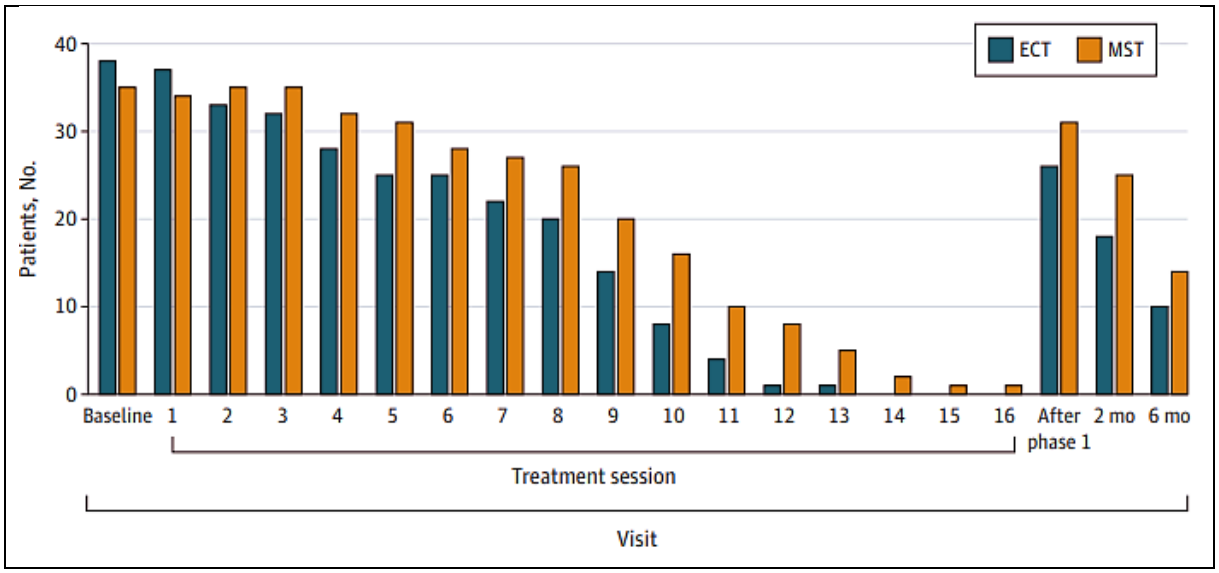


Figure 1: Hamilton Depression Rating Scale (HDRS-24) 24-item scores for the probability of remission between the groups receiving magnetic seizure therapy (MST) and electroconvulsive therapy (ECT).

Discussion

In comparison to MST, ECT exhibited a significantly extended period of time required for orientation following treatment, along with a more severe degree of subjective side effects, specifically in the physical and cognitive domains. After MST, the average time for orientation was only a few minutes, whereas the average time for ECT was about twenty minutes.[1] According to a study by Youssef NA, magnetic seizure therapy had fewer adverse effects on cognition and a Time to Reorientation of 1.8 minutes, which was much faster than ECT (RUL: 18.9 minutes; BT: 50.2 minutes).[3]

Conclusion

The current study concluded that MST's effectiveness was similar to that of ultrabrief pulse RUL ECT, the safest type of ECT currently in use. These outcomes demonstrated the benefits of MST over advanced ECT and encourage its further development.

Magnetic seizure therapy's (MST) quicker time to orientation and increased autographic memory recall, suggested that it may have significant antidepressant effects while retaining a high level of cognitive safety.

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2. Comparison of efficacy of electronic cognitive behavioral therapy with pharmacotherapy and combination therapy for generalized anxiety disorder

Introduction

Anxiety disorders are the most frequent mental health disorders, with a lifetime prevalence of 30%, with generalized anxiety disorder (GAD) being the most common anxiety illness. Less than half of those with mental health illnesses can receive treatment, despite the significant frequency of these conditions. In order to address the significant need for treatment, the mental health care system has to provide adaptable, reasonable, and easily accessible alternatives.[1] Pharmacotherapy and/or psychotherapy are now the gold standard treatments for generalized anxiety disorder. The first-line pharmacotherapies that were recommended are selective norepinephrine reuptake inhibitors (SNRIs) and selective serotonin reuptake inhibitors (SSRIs). The first-line treatment in psychotherapy was individual cognitive behavioral therapy (CBT), which should be administered for 12 to 20 sessions. Individual CBT has long-term benefits for patients, including increased quality of life and reduced psychological discomfort at times of crisis. Pharmacotherapy was not a treatment that many patients choose because of its widespread stigma, possible side effects, polypharmacy, and personal preferences. Individual cognitive behavioral therapy (CBT) was frequently inaccessible due to exorbitant fees, protracted waiting lists, privacy issues, and treatment periods that were limited to regular business hours. One possible way to overcome CBT's drawbacks was to offer it asynchronously online via electronic cognitive behavioral therapy (e-CBT). Clinical efficacy, treatment adherence, high treatment satisfaction, and results that were equivalent to in-person CBT have all been demonstrated with e-CBT.[2] The purpose of this study was to determine whether e-CBT for GAD was as effective as medication alone or in combination with it.

Methods

A total of 115 patients selected their preferred course of therapy in this quasi-experimental approach. Individuals diagnosed with generalized anxiety disorder (GAD) were divided into three groups: e-CBT (N = 41), medication (N = 41), or combination (N = 33). A digital platform was utilized to provide the 12-week e-CBT curriculum. The drugs were administered in accordance with clinical

recommendations. Each arm's efficacy was assessed using questionnaires that assessed depression, anxiety, and stress intensity, as well as quality of life.

Results

The baseline scores and the number of completed weeks did not differ significantly between the arms. Following therapy, anxiety levels improved in all arms. Depression scores improved in the combination and medication arms. Quality of life improved in the e-CBT and combination arms, whereas stress ratings were better in the combination arm. (Figure 1) Post-treatment scores for stress, anxiety, or depression did not differ across the groups. The quality of life improvement was significantly higher in the combination arm. Completers in the medication group exhibited notably lower baseline stress and depression levels than dropouts.

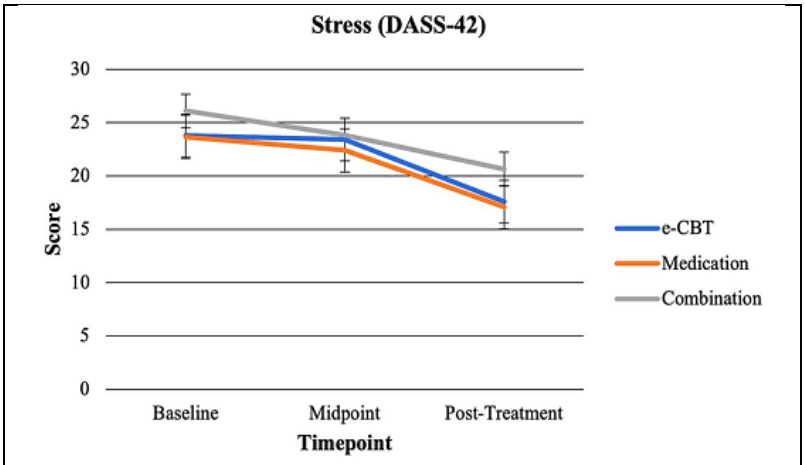

Figure 1: The averages of the symptom severity questionnaire scores for each arm and time interval.

Discussion

This study demonstrated that e-CBT was a viable treatment option for GAD, with patients in the e-CBT arm demonstrating significant improvements in GAD-7 scores. Patients with GAD showed improvements in their symptoms when treated with e-CBT, medication, or a combination of both.[1] Zhang W et al. revealed that internet-based cognitive behaviour therapy (ICBT) and cognitive behavioral therapy (CBT) had equal final mean differences (MDs), indicating that their respective effects on the decrease of anxiety scores were comparable. This confirmed that the web-based cognitive-behavioral therapy was effective. However, the outcomes showed that the treatment effects of ICBT and CBT were equivalent.[3]

Conclusion

The current study concluded that using e-CBT to treat GAD, either alone or in conjunction with medication therapies, was a feasible choice. Care professionals must prioritize considering the patient's favoured course of therapy, taking into consideration their lifestyle, personality, and values.

Generalized anxiety disorder (GAD) treatment with electronic cognitive behavioral therapy (e-CBT) or medication appeared to provide considerable improvements in symptoms.

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3. Assessment of childhood cumulative trauma, social support, and stress as determinants of bipolar disorder illness outcomes and quality of life

Introduction

Bipolar disorder (BD) is a chronic psychiatric disorder characterized by recurrent mood episodes separated by periods of remission. People with BD often experience symptoms for half of their time during the course of the disease. A number of adverse consequences, such as decreased functioning and quality of life, were linked to BD.[1] Environmental stressors have an impact on brain development, and inadequate and damaging stimuli have a different impact. It suggested that childhood trauma was linked to improper neurodevelopment as well as disorders and abnormalities of the brain.[2] Because social support was linked to better results for mental health; it was frequently hailed as a protective

factor. However, people with BD report lower levels of social support than people in general, and people who combine BD with trauma report even lower levels of social support perceptions. Another significant component that needs to be taken into account was stress. Social support and stress have been found to be important predictors of depressive recurrence, and some research has drawn attention to the connections between trauma and stress. However, no study has evaluated the three factors' (cumulative trauma, social support, and stress) combined potential to forecast future bipolar illness characteristics or quality of life. It was especially crucial to include a quality of life measure for chronic illnesses like BD since it sheds light on experiences of wellbeing that go beyond the existence or absence of disease symptoms.[1]

The purpose of this study was to investigate if childhood cumulative trauma, perceived social support, and perceived stress influence the outcomes and quality of life of people with BD.

Methods

A total of 114 people with BD-I (41.2%) or BD-II (58.8%) were included in this study. All self-report measures were completed by participants using Qualtrics at baseline and the 6-month follow-up, with the exception of the Childhood Trauma Questionnaire (CTQ), which was only completed at baseline. At both timepoints, participants also finished clinical interviews. The Montgomery-Asberg Depression Rating Scale (MADRS) and the Young Mania Rating Scale (YMRS) were used to measure manic and depressive symptoms, respectively. The Structured Clinical Interview for DSM-5 Research Version (SCID-5-RV) was adjusted at the 6-month follow-up interview to determine the type and severity of mood episodes encountered in the previous 6 months, with only the criteria for a major depressive, hypomanic, and manic episode being employed.

Results

A significant regression equation was computed for self-reported depression (DASS) at baseline: $F(3,110) = 222.55$, $p<0.001$. The model explained 49.6% of the variance in self-reported depression severity. Perceived social support (MSPSS), perceived stress score (PSS), and childhood cumulative trauma (CTQ) all made significant contributions to the model. All three predictors significantly contributed to the model, which at 6-month follow-up still accounted for 26.7% of the variation in self-reported depression severity ($F(3,110) = 13.339$, $p<0.001$) (Figure 1). The factors that

Vol 2
ISSUE 12
Petril Times
NEWSLETTER
INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

significantly and substantially explained the variation in quality of life (62%; 23.6%) and anxiety intensity (34.6%; 24.5%) at baseline and 6-month follow-up were childhood cumulative trauma, social support, and perceived stress. Social support and perceived stress both contributed significantly to depression and quality of life in bipolar disorder, and both were very distinctive in their predictions of all outcomes.

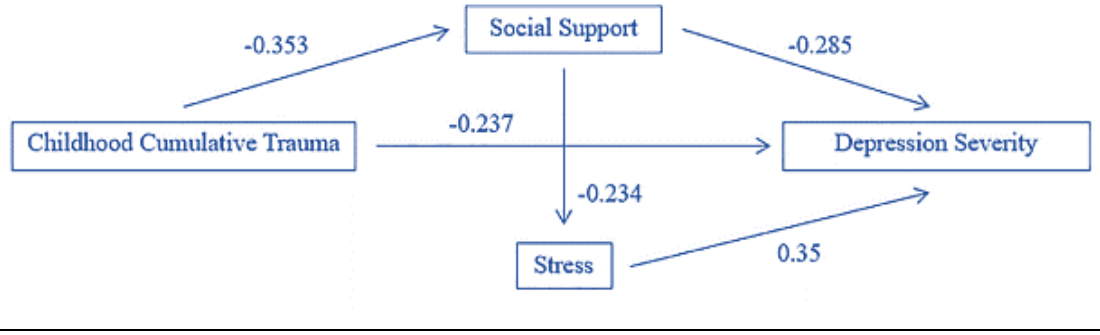

Figure 1: The actual path diagram for self-reported depression severity.

Discussion

Among all the outcome factors in this study, social support and stress were the most significant predictors of quality of life at the 6-month follow-up, and they accounted for the greatest amount of variance.[1] Faurholt-Jepsen M et al. showed that stress levels were a predictor of quality of life throughout a 6-month follow-up, as it found that greater levels of stress were linked to lower quality of life in a BD population. In order to increase wellbeing, physicians can target controllable factors like perceived stress and social support, which made this discovery especially significant.[3]

Conclusion

The current study concluded that quality of life, anxiety, and depression severity in BD patients at baseline and six months later were strongly predicted by the combination of childhood cumulative trauma, perceived social support, and perceived stress. These results hold promise for improving the lives of BD patients since social support and stress were changeable characteristics that can be targeted by therapists.

Stress and social support were crucial factors in the quality of life of those living with bipolar disorder.

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4. Case Report on Psychosis ahead of Parkinsonism in Wilson Disease

Introduction

Wilson's disease (WD) is a hereditary autosomal disorder characterized by disruptions in copper metabolism. It results in a pathological build-up of copper in many organs and tissues, primarily the liver, brain, cornea, and kidneys, which can produce symptoms. The condition differs in terms of the age at which symptoms first appear and the number of damaged organs. The second and third decades of life were when clinical symptoms of WD typically appear.[1] Copper build-up in the brain can cause neurological and psychological problems. While personality changes were frequently the initial sign in individuals with Wilson disease, severe psychosis was present in 1.4% to 11.3% of cases. Patients with neurological complaints always have Kayser-Flesicher rings. Serum copper and ceruloplasmin levels offer a strong positive predictive value for identifying Wilson illness in the relevant clinical context, but they were not often regularly tested.[2]

This case study described a 48-year-old man who had Parkinsonism and previously undetected Wilson disease. It also highlighted the findings on brain magnetic resonance imaging (MRI), which can support a thorough clinical history and examination to confirm the diagnosis.

Vol 2
ISSUE 12
Petril Times
NEWSLETTER
INFORMATION BULLETIN ISSUED AS A SERVICE TO THE MEDICAL PROFESSION

Case presentation

A 48-year-old man who had been experiencing increased mobility falls, dysarthria, and sialorrhea over the previous three months self-presented to the emergency room. His previous medical history was relevant for psychotic depression, which was identified 15 years earlier after a suicide attempt. At the age of thirty, the patient began to exhibit symptoms of psychosis while concurrently abusing cannabis, amphetamines, and other illegal substances. After being admitted to the hospital several times for episodes of substance misuse, psychotic episodes, and personality abnormalities, he was eventually diagnosed with schizophrenia. Two years before the current presentation, he had received a depot injection of 100 mg of paliperidone. After receiving paliperidone for eighteen months, extrapyramidal symptoms were observed. These were believed to be caused by drug-induced parkinsonism; thus, the dosage was lowered to 75 mg before the medicine was stopped. At the time of admission, the patient was not taken any other drugs. Examination showed axial rigidity, bradykinesia, hypomimia, risus sardonicus, dysarthria (almost anarthria), and sialorrhea, a hunched posture with a shuffling stride in both upper limbs. During the presentation or admission, there were no positive symptoms of schizophrenia, such as delusions, hallucinations, or thought disorders. After the results of the initial examinations were received, an additional ophthalmological examination was ordered, and it was discovered that the patient had Kayser-Fleischer rings (Figure 1).

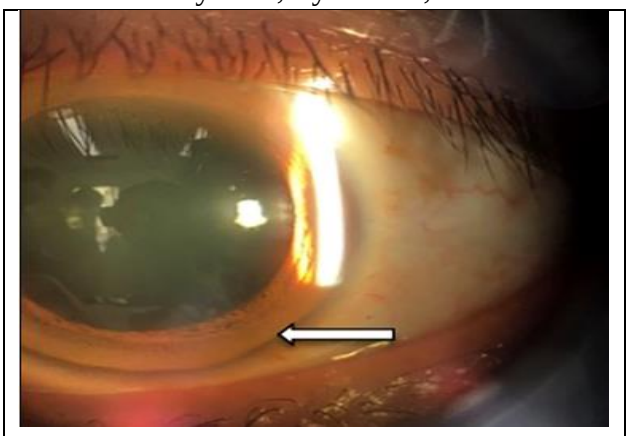

Figure 1: Kayser-Fleischer ring (white arrow) seen under a slit light examination.

The results of the liver function tests showed just a slight elevation in gamma-glutamyl transferase (GGT) level (57 U/L, reference range: 5–50). Studies on iron and liver synthetic function were normal. Ceruloplasmin was measured at 0.02 µmol/L (reference range: 0.17–0.31), serum copper at 4.3 µmol/L (reference range: 12.0–22.0), and 24-hour urine copper at 2.0 µmol (reference range: <1.6). Liver ultrasonography revealed cirrhosis but no sign of portal hypertension. A brain MRI revealed elevated FLAIR/T2 signals in the basal ganglia, midbrain, and pons (Figure 2).

As investigations were ongoing, the patient's paliperidone was stopped, and benztropine therapy was started. Chelation therapy with zinc and penacillamine was started as soon as Wilson disease was

confirmed. To treat the severe parkinsonism, levodopa medication was added. His stiffness and gait showed some improvement during the 6-month follow-up. However, after a year, things become worsen; the patient was bedridden, dysarthric, trembling in all four limbs, and suffering from acute dysphagia that was causing malnourishment and necessitating the installation of a percutaneous gastrostomy tube.

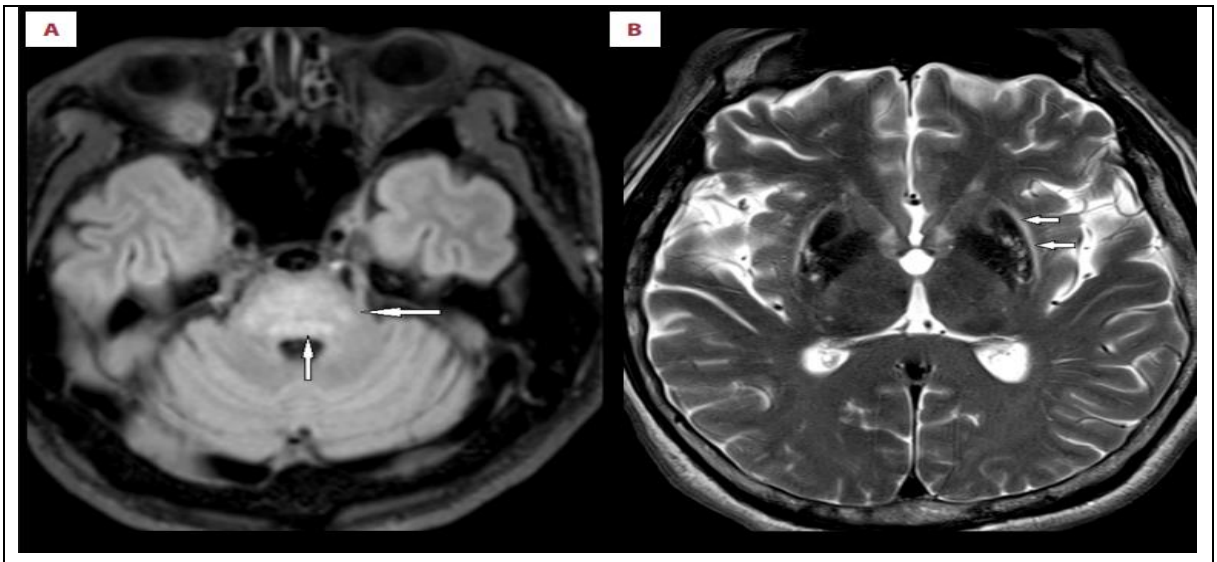

Figure 2: Brain magnetic resonance imaging. Axial FLAIR sequence (A) shows diffusely enhanced signal (white arrows) across the pons, while axial T2 sequence (B) shows mixed signal in the lentiform nuclei and increased signal (white arrows) in the external capsule.

Discussion

An MRI of the brain in this instance revealed an elevated FLAIR/T2 signal in the basal ganglia, midbrain, and pons.[2] According to Szafrański T et al., the brain's magnetic resonance imaging (MRI) showed symmetric alterations in the putamen and globi pallidi, which were hypointense on T1 pictures and hyperintense on T2-weighted images, which were suggestive of WD.[1] According to Yu XE et al., Wilson disease's MRI spectrum includes abnormalities in the putamen, pons, midbrain, and thalamus.[3]

Conclusion

Serum copper testing was inexpensive and should be included in the first evaluation of new neuropsychiatric disorders. Wilson disease was an uncommon but potentially curable and reversible

cause of psychosis if detected early. Extrapyramidal signs on examination were correlated with MRI brain alterations in advanced disease, which helped the clinical assessment distinguish the illness from drug-induced Parkinsonism.

The first-line examinations for all young adult psychiatric patients presenting with extrapyramidal and/or hepatic symptoms should include measuring serum copper and ceruloplasmin levels because these were readily accessible.

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