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Depression and traumatic brain injury in Al Karaj

Ali Hassan A Ali[1,2*] Abdulaziz M M Alshamrani[3], Naif A H Albulayhid[3], Fahd M Alanazi[3], Mohammed Mazroua Almazroua[3], Turky N I Alhuwail[3], Waleed A H Aldholmi[3], Salman F A Alandas[3], Ali Mohammed Alotaifi[3]

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Authors' Affiliation:
[1]Anatomy Department, College of Medicine, Prince Sattam Bin Abdulaziz University, Alkharj 11942, KSA
[2]Anatomy Department, Faculty of Medicine, Al-Azhar University, Cairo, Egypt
[3]College of Medicine, Prince Sattam Bin Abdulaziz University, Al-Kharj, KSA

[*]**Corresponding author**
Anatomy Department, College of Medicine, Prince Sattam Bin Abdulaziz University, Alkharj 11942, KSA
Anatomy Department, Faculty of Medicine, Al-Azhar University, Cairo, Egypt
Email: alihassan3750@yahoo.com / a.ali@psau.edu.sa

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ABSTRACT

Depression is repeated sequel among patients with traumatic brain injury (TBI). The purpose of our research is to identify the prevalence of depression after TBI. It is a prospective, study carried out in the first two year after brain trauma occurred. In this study 213 patients with head trauma. All of them, MRI of the brain was done within 30 days after accident. Radiological, demographic, clinical, and data were gathered during hospitalization. Psychiatric and neurocognitive assessment were done two years after that time. In patients with lesions of the cerebral hemisphere and the corpus callosum, major depressive disorder is regularly recorded. Our research focuses attention on the importance of psychosocial factors as foretellers of those who will report severe depression after injury. Our data show a positive relationship between brain injury and depression.

Keywords: Brain injury, Depression, Psychiatric outcome, MRI, Al Karaj

1. INTRODUCTION

Hopelessness, loss, despair or feeling sad is symptoms of depression that conflict with daily activities. Depending on the severity of symptoms, depression can be categorized as mild, moderate or severe (National Collaborating Centre, 2010; Kessler and Brome, 2013). Although there is not a single trait in all depressed patients, common traits include sadness and persistent negativity. Other symptoms include apathy, lack of energy, cognitive distortions, inability to enjoy normal life events (Al Yousefi et al., 2021). The spread of depression has been increasing in Saudi Arabia between 2005 and 2015 (WHO, 2017). One of common psychiatric problems among patients with TBI (traumatic brain injury) is depression. It is higher than the reported rate in general population. The exact cause of depression after TBI is not known. This varies greatly from one to another. It may be of a physiological in nature, possibly due to the disruption of neural circuits. It may also be due to alterations of neurotransmitters after trauma in the cerebrum (Chen et al., 2007; Durish et al., 2018; Saberinia et al., 2020).

Depression can further occur as an emotional reaction to injury when a person fights to adapt to transient or permanent disorder or function alteration inside the family and community. In addition, there are unrelated factors because of individual causes, inherited genes, and other effects that were present before the trauma (Fann et al., 2009; Aletesh et al., 2021). After TBI, the psychosocial function is affected specially in the twelve months after injury among persons with major depression (Lin et al., 2010; Rapoport et al., 2006). Persons with cerebrovascular disorders and depression manifested by eminent administrative dysfunction, especially those persons with late-onset depressive disorders ischemic deep white matter and lesions in basal nuclei (Alexopoulos and Frontostriatal, 2002; Krishnan et al., 2002).

Brain injury due to trauma is considered a significant hazard for neurodegenerative diseases, including Alzheimer's and Parkinson's diseases as well as dementia (LoBue et al., 2018; Ma et al., 2019). About 65-89% of TBI patients continue to have neurological dysfunction, although many patients can resolve such problems within 12 months after trauma (Coronado et al., 2011). This research aimed to estimate and quantify the prevalence of clinical, neuroimaging, neuropsychological, and structural depression after brain trauma.

2. METHODS

This research is a prospective cross sectional. All the patients with head trauma were collected from January 2020 to January 2022. The study participants composed of 213 successive patients with head injury admitted to King Khalid and Prince Sattam Hospitals in Alkharj city. The Prince Sattam University Institutional Review Board approved the study (PSAU-2020 ANT 12/42PI). All cases were 13-15 score of Glasgow coma scale and their age from 18 to 72 years. Patients with injury spinal cord as well as those with severe lack of understanding were excluded from the study. Moreover, patients with history of psychic disorders before head trauma also excluded from this study. One hundred forty-nine (69.9%) of the 213 patients were injured in a motor car accident, thirty-eight cases (17.8%) by a falling from a height, twenty-six cases (12.2%) by struggle. 213 patients gave written consent to participate in this research.

Psychiatric status was assessed by SCAN (Schedules for Neuropsychiatric Clinical Assessment) scales by a psychiatrist. In addition, cognitive functions were assessed by MMSE (Mini Mental State Examination). Additionally, Neurological assessment was performed according to GOSE (Glasgow Expanded Outcome Score). At baseline, all patients were evaluated clinically using the (EDSS), and radiologically with MRI. Depression was evaluated using BDI and BAI (Beck's depression inventory -I and Beck's Anxiety Inventory respectively) (Beck et al., 1996). On the other hand, the depression severity was assessed via the Beck's depression inventory BDI-II (Beck et al., 1996), which consists of twenty-one elements scored from zero to three to give rise to an overall score ranging from zero to sixty-three. Higher scores refer to clinically significant depression or anxiety.

At the radiology department, magnetic resonance (MR) imaging was acquired as part of the standard clinical evaluation in the psychiatry as well as neurosurgery departments of the sharing hospitals. A neurologist trained in evaluating structural neuroimaging examinations, who was blind to the findings of a psychiatric examination, read all of the scans. SPSS 13.0 and also ANOVA (Analysis of variance) were used to analyze our results. Glasgow outcome scale extended in all cases. Analysis of the findings was done using a chi-square nonparametric and Glasgow coma score between both sexes, head trauma mechanism, pupillary anomalies.

3. RESULTS

213 cases with trauma to the head were included in our study. All the study participants have completed the inclusion criteria 115 (53.9%) males and 98 (46%) females. We found 64 (30%) suffering from depression after the accident (table 1). 15 patients were suffering from major depression occurs in approximately 7% of cases with head injury $p < 0.001$. Most of them were related to executive dysfunction and anxiety symptoms. 29 cases (13.6%) were suffered from mild depression. The remaining 20 cases had a moderate depression (9.3%). There were no significantly differences between both sexes (table 2, fig. 1). The mean Beck's depression inventory score was significantly higher at baseline set side by side to the two, four, eight and six weeks of follow up (table 3).

According to the results of magnetic resonance imaging, various injury to the axon with isolated cerebral hemisphere, lesions of the corpus callosum and the cerebral hemisphere as well. Major depressive disorder is commonly founded in cases with lesions of the cerebral hemisphere and the corpus callosum and in patients with lesions of the cerebral hemisphere and brainstem. The worst level of consciousness is noticed in cases with cerebral hemisphere lesions only and with epidural hematoma, lesions of the cerebral hemisphere and the corpus callosum with encephalomalacia (fig. 2-6) demonstrates different types of TBI as shown by axial MRI images.

Table 1 Comparison between frequency of different depression types after brain trauma. Statistically significant $p<0.05$

Depression	Frequency	Percentages
Mild depression	29	13.6 %
Moderate depression	20	9.3%
Major depression	15	7%
No depression	149	75.9 %
Total	213	100%

Table 2 Baseline and follow up periods after TBI; Expanded Disability Status Scale (EDSS). ∗ Significant difference (P = 0.02) between baseline and 8 weeks.

Beck's depression inventory Mean ± SD	EDSS	Beck's depression inventory
Base line	2.1 ± 0.6	22.34 ± 2.3
Two weeks	2.3 ± 0.7	20.8 ± 2.7
Four weeks	2.5 ± 0.8	20.9 ± 2.8
Six weeks	2.6 ± 0.6	20.1 ± 2
8 weeks	2.6 ± 0.7	20.7 ± 3

Table 3 Comparison between frequency of different depression types after brain trauma according to the site of the lesions. Cerebral Hemisphere (CH) Corpus Callosum (CC) Brain Stem (BS) Statistically significant $p<0.05$

Depression	CH	CC&CH	BS & CH	Combined lesions	Total no.
Mild depression	14 (6.5%)	4 (1.8%)	7 (3.2%)	4 (1.8%)	29 (13.6 %)
Moderate depression	8 (3.7%)	5 (2.3%)	4 (1.8%)	3 (1.4%)	20 (9.3%)
Major depression	9 (4.2%)	3 (1.4%)	2 (0.9%)	1 (0.4%)	15 (7%)
No Depression	46 (21.5%)	34 (15.9%)	36 (16.9%)	33 (15.4%)	149 (69.9%)

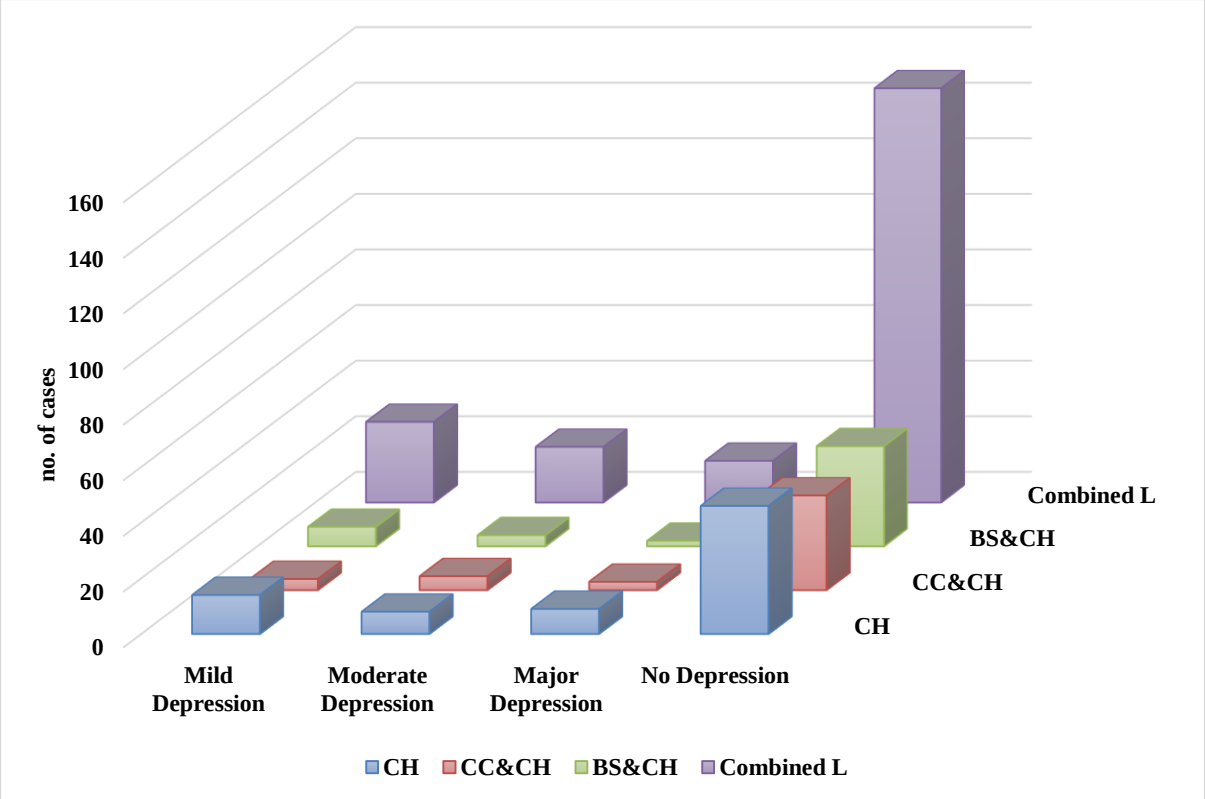

Figure 1 Different depression types after brain trauma according to the site of the lesions; Cerebral Hemisphere (CH) Corpus Callosum (CC) Brain Stem (BS).

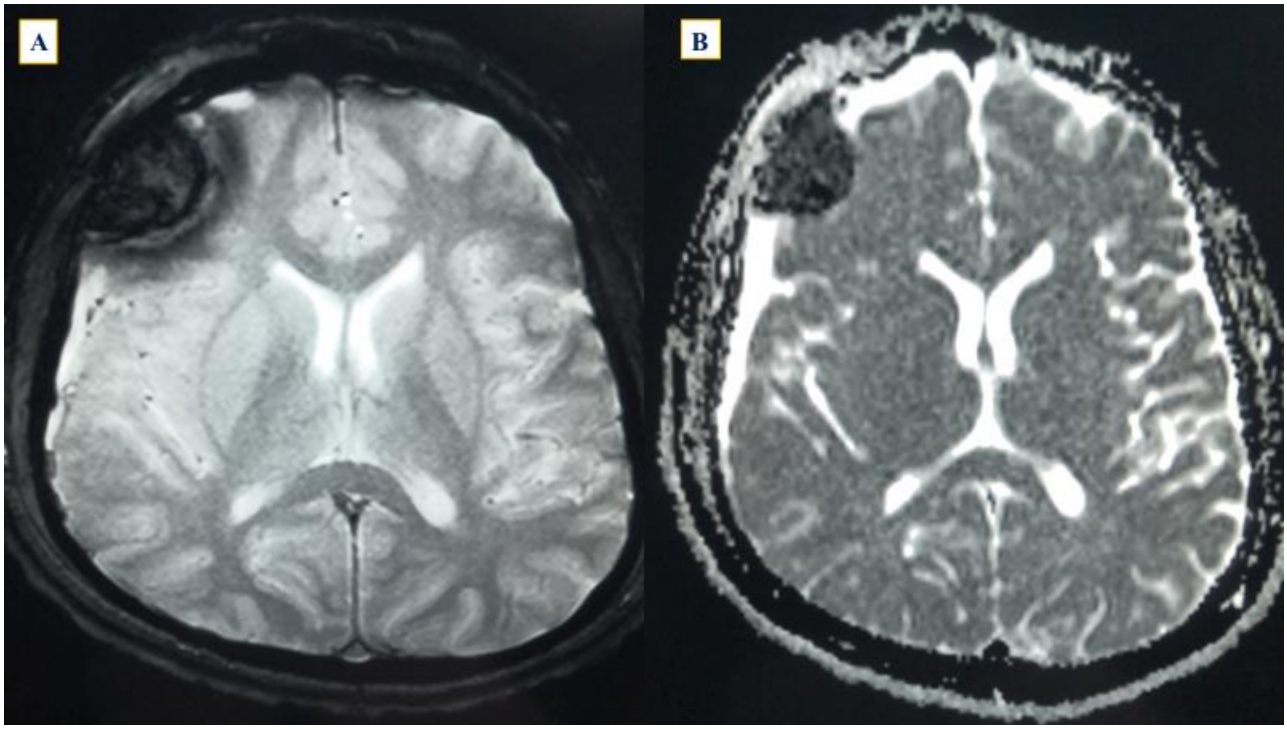

Figure 2 A and B Axial MRI of male 35 Years old with post traumatic right Frontal (EDH) epidural hematoma and ipsilateral Temporal (SDH) subdural hematoma.

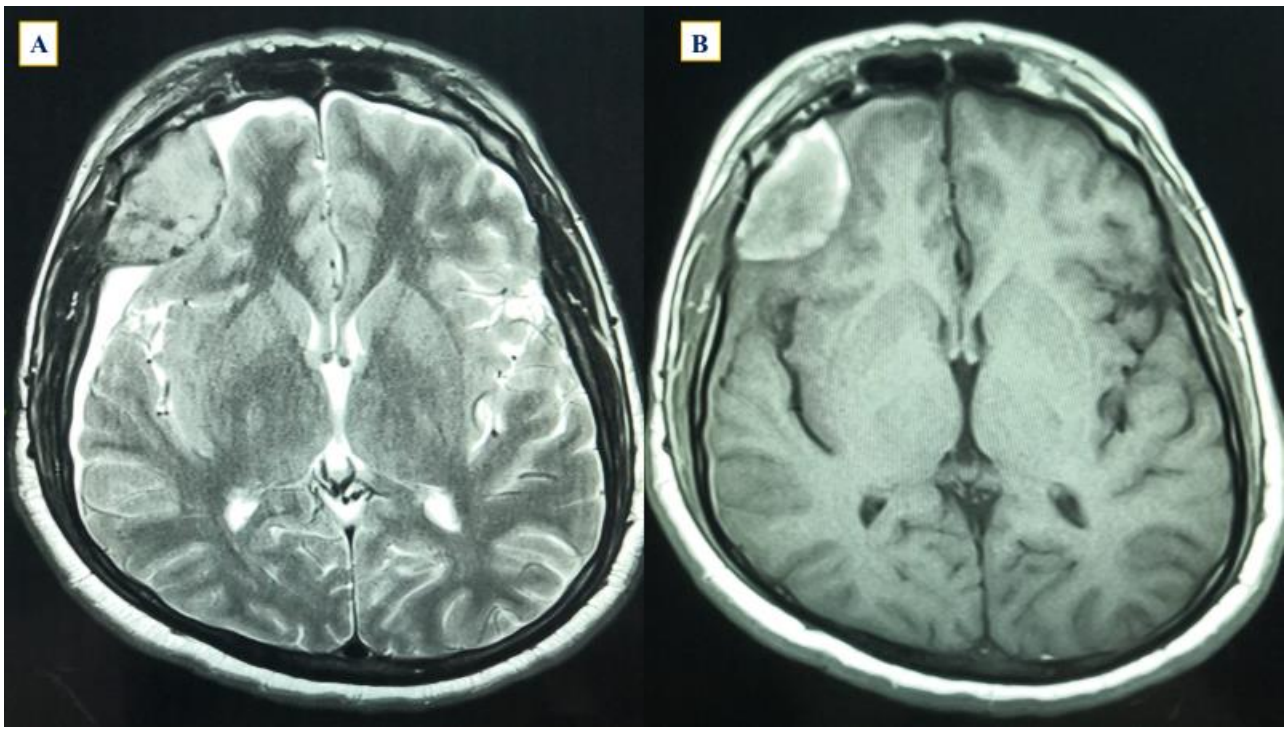

Figure 3 A and B Axial MRI of male 35 Years old with post traumatic right Frontal (EDH) epidural hematoma and ipsilateral Temporal (SDH) subdural hematoma.

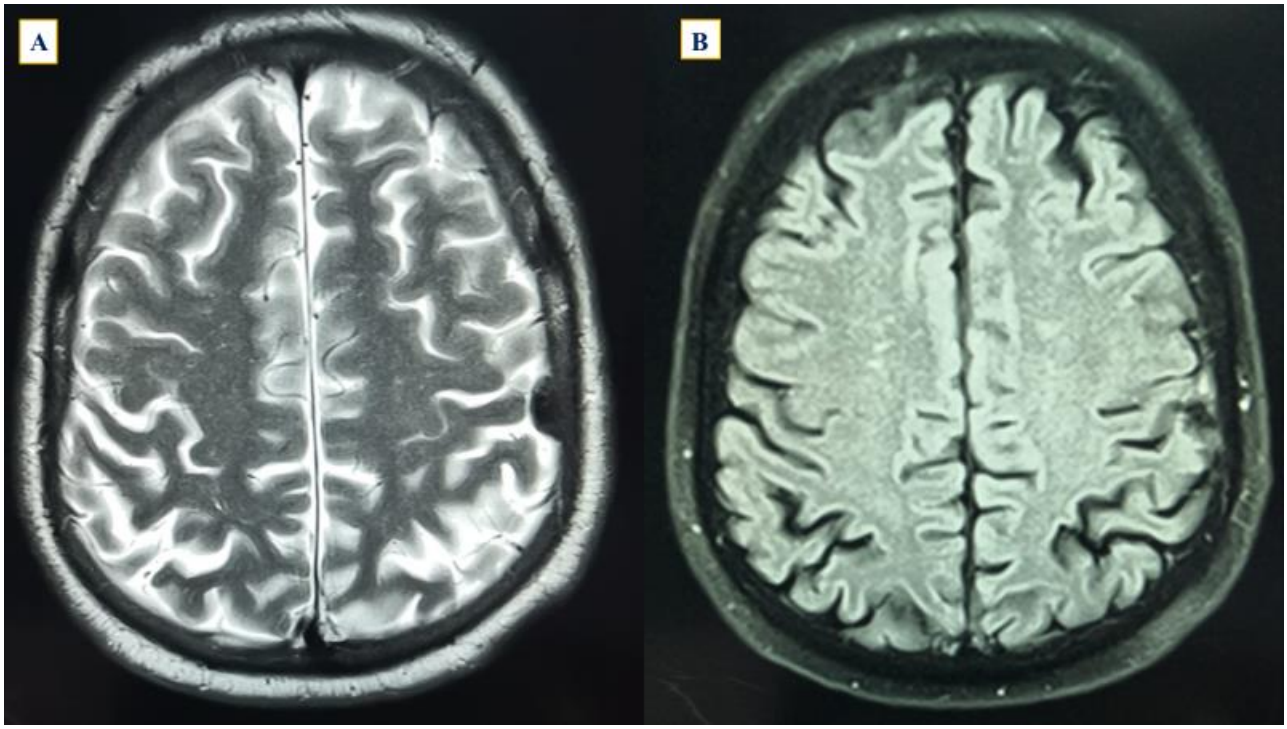

Figure 4 A and B Axial MRI of male 59-Year-old post trauma with Left parietal small EDH (epidural hematoma).

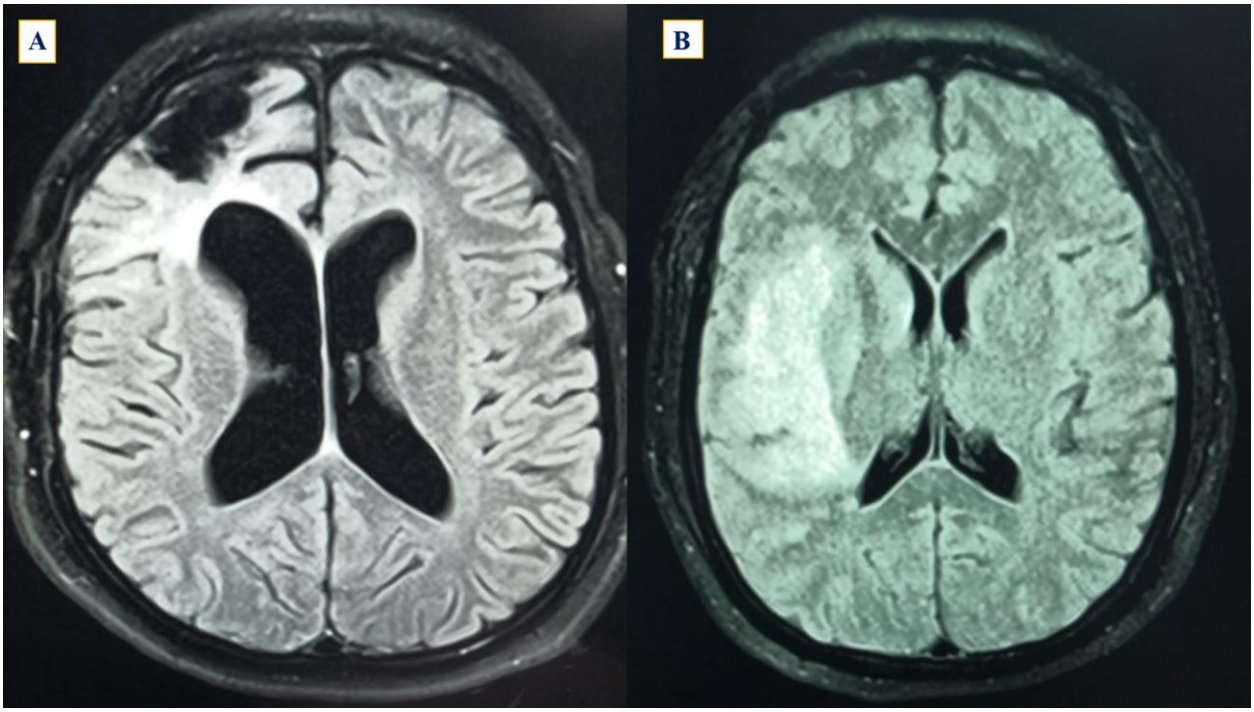

Figure 5 A and B Axial MRI of male 59-Year-old post trauma with Left parietal small EDH (epidural hematoma).

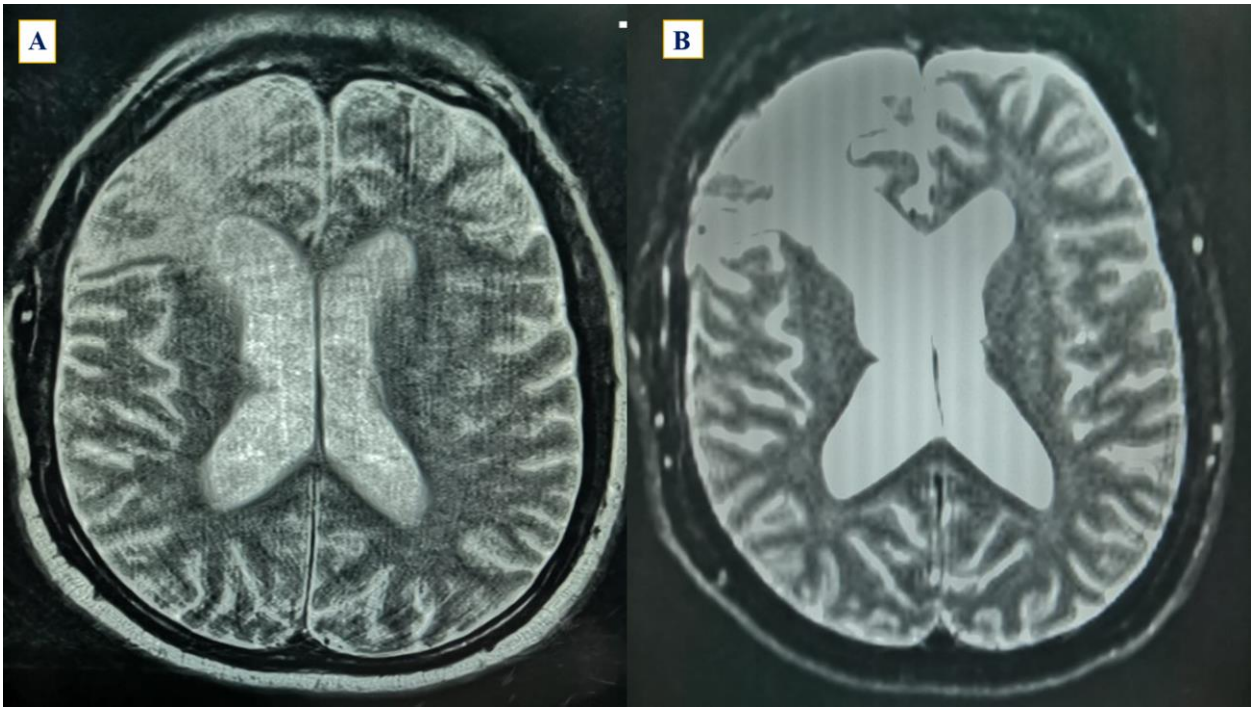

Figure 6 A and B Axial MRI of post old cranial trauma with sequel right frontal encephalomalacia.

4. DISCUSSION

After brain trauma, depression is one of the common outcomes. During the twelve months after injury, about half of those affected develop depression. Most (about two-thirds) are influenced during few years after brain injury (Fann et al., 2009). Generally, the average rate of depression is much less, affecting less than 1 in 10 people within twelve months period (Mubaraki et al., 2021). The mechanism of depression after head injury likely results from disruption of the biosynthetic neurons containing amine as they go through the basal nuclei or anterior subcortical white matter. Feelings of loss and frustration that arise soon after injury often follow symptoms of persistent dysphonia (Alqarny and Alsofyani, 2021). Common features of depression may be less noticeable in TBI patients due to the general flatness of personality. Irregular or poor recovery, or worsening of neurological shortfalls after initial recovery, may be signs of depression. Poor levels of pre-existing comorbid functions and a past history of mental illness are major hazards for depression (Hellewell et al., 2021).

In this study, those with an isolated cerebral hemisphere lesion, and lesions of the callosum and cerebrum, had the lowest level of consciousness at first. This was in part consistent with the findings of a previous study that reported a significant association between GCS and a lesion of the corpus callosum and that cases with a callosal lesion had more deterioration of consciousness than patients with other locations of the lesion (Cicuendez et al., 2017; Kotb et al., 2019).

Some investigators have reported selective involvement of lesions of the left frontal lobe and left basal nuclei in cases with acute depressive disorder after TBI (Fedoroff et al., 1992). Other researches of secondary depressive disorders have also found reduced metabolic rates in the lower frontal areas in Parkinson's disease patients and also in Huntington's disease. Other relevant researches of secondary depressive disorders have also demonstrated diminished metabolic rates in inferior frontal areas in patients with Huntington, (Fedoroff et al., 1992) and Parkinson diseases (Mayberg et al., 1990; Jorge et al., 2004).

5. CONCLUSION

Depression is a repeated sequel after brain trauma that has a detrimental effect on the healing process and psychosocial consequences of cases with TBI. The location of the lesion has an effect on the outcome of neurocognition. We recommend annual depression screening to detect the spread of depression among persons with severe brain trauma. The increased frequencies of depression after brain trauma are clearly identified. Rehabilitation and mental health professionals should improve the way depression is recognized, secondary prevention and treatment available.

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Authors' Contributions

All authors contributed to the research and/or preparation of the manuscript. Ali Hassan A. Ali, Abdulaziz M M Alshamrani and Naif A H Albulayhid participated in the study design and wrote the first draft of the manuscript. Fahd M Alanazi, Mohammed Mazroua Almazroua, and Turky N I Alhuwail collected and processed the samples. Waleed A H Aldholmi, Salman F A Alandas and Ali Mohammed Alotaifi participated in the study design and performed the statistical analyses. All of the authors read and approved the final manuscript.

Ethics Approval

All series of steps that were implemented in this study were in compliance with Ethics Committee of Prince Sattam bin Abdulaziz University Institutional Review Board (PSAU-2020 ANT 12/42PI).

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Conflict of interests

The authors declare that there are no conflicts of interests.

Data and materials availability

All data associated with this study are present in the paper.

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