

CASE REPORT

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Isolated pure motor apraxia in the setting of depressed skull fracture: A case report and review of the literature

Cierra N Harper, Kavita Prakash, Damirez Fossett

ABSTRACT

Introduction: An isolated pure apraxia of speech following head trauma is a rare clinical presentation in the absence of any other neurologic deficit. It is more commonly seen in patients with vascular pathology. Confusion can exist in recognizing an apraxia of speech versus a true Broca's aphasia. We report such a case in a patient undergoing significant head trauma. The pathology and its management are discussed.

Case Report: A 41-year-old male presented to our Emergency Department with a 3 cm laceration to the left side of the head following an assault with an unknown object. The patient suffered a depressed skull fracture with underlying epidural hematoma, bilateral traumatic subarachnoid hemorrhages, and a resultant isolated pure motor apraxia of speech. He was treated effectively with an emergency frontoparietal craniotomy, elevation and repair of his skull fracture, and evacuation of his epidural hematoma. His pure motor apraxia was greatly improved post-operatively and the patient was referred to outpatient speech therapy with continued neurosurgical follow-up.

Conclusion: This case represents the unusual presentation of a pure motor apraxia of speech following traumatic assault to the left frontoparietal skull. An excellent therapeutic response was obtained following surgical intervention and speech therapy.

Keywords: Apraxia of speech, Broca's aphasia, Cranial trauma

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INTRODUCTION

Traumatic brain injury (TBI) occurs when a physical insult to the skull results in impairments of normal brain functioning. In the United States alone, TBI accounted for over 223,000 hospitalizations, 60,000 deaths, and 25% of all injury related deaths [1]. In 2019, men were nearly twice as likely to be hospitalized and nearly three times as likely to die from TBI compared to their female counterparts with violence as the leading cause of TBI-related death overall [1, 2].

The presence of skull fracture in TBI has been documented as an important indicator of underlying intracranial lesions such as hematoma or contusion [3]. Skull fractures are categorized as either linear or depressed, the latter of which is defined as those where the fractured segment is displaced below the interior table of the adjacent skull [4, 5]. Depressed skull fractures account for significant morbidity and mortality with 41.5% resulting in at least one intracranial lesion and the frontoparietal skull being the most likely to be affected [4–6].

Broca's area is one region that is particularly vulnerable during insult to the left frontoparietal skull. This region is primarily responsible for the motor function

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involved in speech production. Injury to this region can result in pathologies characterized by the inability to produce speech such as expressive aphasia or apraxia of speech with or without accompanying deficits in speech comprehension [7, 8]. Vascular compromise due to ischemic or hemorrhagic stroke is the most common mechanism of injury to Broca's area. Less frequently deficits to this region may arise in the setting of trauma, neoplasm, or infection but are rarely unaccompanied by additional neurologic and cognitive deficits [9].

Here we discuss the unusual presentation of a 41-year-old male with an isolated acquired apraxia of speech in the absence of aphasia and other neurologic deficits following an assault to the left frontoparietal skull.

CASE REPORT

History

The patient is a 41-year-old African American male who was brought in to the Emergency Department of our level 1 trauma center after being assaulted with an unknown object. Upon presentation he was observed to have a 3 cm frontoparietal laceration to the left side of his head and difficulty producing speech. On neurologic examination, the patient was able to understand and follow commands from the trauma team but was completely non-verbal. By report, prior to the assault, he had been able to speak with normal rate, rhythm, and tone and with a full vocabulary. Information obtained from his mother corroborated this history. In the emergency room, the patient's only methods of communication were through hand signals, written word, and text messaging.

The patient's past medical history was not significant for any previous neurologic deficits or surgical procedures. He had no known allergies. Further history was limited due to his inability to speak coupled with illiteracy. Laboratory testing was remarkable for a lactic acid of 2.9 and a metabolic lactic acidosis with a pH of 7.38. All other laboratory values were within normal limits. Initial vital signs were as follows: temperature = 98.2°F, blood pressure = 118/77, heart rate = 93, respiratory rate = 16, and oxygen saturation = 96%. Imaging was obtained including a non-contrast enhanced computer tomography (CT) of the head and cervical spine. These images revealed a large left frontoparietal depressed skull fracture with an underlying 5.71 mm epidural hematoma (Figure 1), along with a contusive hemorrhage and edema, and a right contrecoup temporal/parietal subarachnoid hemorrhage (Figures 2 and 3). No acute cervical spine pathology was identified.

Examination

On physical examination the patient had a 3 cm full-thickness laceration to the left frontoparietal region of the skull. He was awake, alert, and oriented with pupils that were equal and reactive to light and accommodation. His

extra-ocular movements were intact, and he had no other cranial nerve deficits. He had no gross motor, sensory, or cerebellar deficits. No other trauma was identified, and his Glasgow Coma Score (GCS) was documented as 11. Evaluation of his speech revealed a complete inability to produce words or sounds despite intact comprehension as evidenced by his ability to follow commands and respond appropriately to questioning. In addition to hand and head gestures, he was able to communicate through the use of text messaging and written word with appropriate syntax and vocabulary. The patient was illiterate, but given assistance with spelling had no difficulty in demonstrating his understanding of the consent process, providing a history, or asking questions of his own.

Operation

The patient received a loading dose of levetiracetam, cefazolin as a prophylactic antibiotic, and a 50 gram intravenous (IV) bolus of mannitol. He was taken directly from the CT scanner to the operating room. There, an emergency left frontoparietal craniotomy was performed with elevation and repair of the depressed skull fracture, evacuation of the epidural hematoma, and cauterization of the left middle meningeal artery (Figure 4). The dura was opened to see if there was any subdural bleeding. A diffuse subarachnoid hemorrhage (Figure 5) with mild edema was revealed. The dura was closed, the bone flap repaired and replaced, and a Jackson–Pratt (JP) subgaleal drain was placed before the scalp was closed.

Post-operative course

The immediate post-operative period was unremarkable. The patient was successfully extubated in the operating room and taken to the surgical intensive care unit (SICU). In post-operative day (POD) 1, his GCS remained at 11 as there was no improvement in his speech or sound production. He maintained the ability to communicate non-verbally using text messaging and written communication with appropriate syntax and full vocabulary without difficulty. His comprehension of written and verbal communication remained intact. The patient had no other neurologic or motor deficits on physical examination. POD 1 was notable only for an afebrile leukocytosis most likely due to an acute phase response (APR) and he was transferred to the neurosurgery floor. On POD 2, his GCS improved to 12 and the patient began to make incomprehensible utterances during speech attempts. His ability to communicate non-verbally and understand the treatment team remained intact as he continued to use his phone to type. No other neurologic or motor deficits were noted on physical examination. His white blood cell count began downtrending. By POD 4, his GCS had improved to 15 and he was able to produce a halting speech with full range of appropriate vocabulary and grammar. No other neurologic or motor deficits were noted on physical examination. On POD 5, the JP drain was removed, and the patient was discharged with

outpatient speech therapy and an appointment to follow up in the neurosurgery clinic.

Imaging obtained in the immediate post-operative period included a non-contrast enhanced CT. This revealed elevation and repair of the skull fracture with appropriately placed drain (Figures 6 and 7), resolving bilateral subarachnoid hemorrhages, and pneumocephalus with consistent with expected post-operative changes (Figure 8).

The patient was followed in clinic at 2, 6, and 8 weeks after surgery. During his last follow-up appointment his speech and cognition were at baseline, and he was discharged from our care. He returned to work with no deficits.

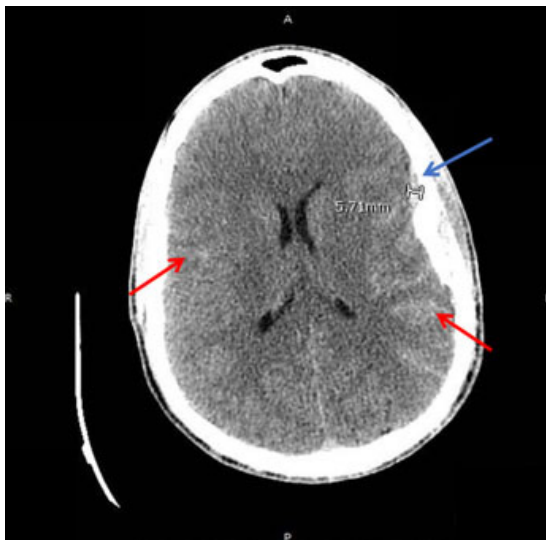


Figure 1: Pre-operative axial computed tomography (CT) of the skull with 5.71 mm epidural hematoma (blue) and diffuse bilateral subarachnoid hemorrhage (red).

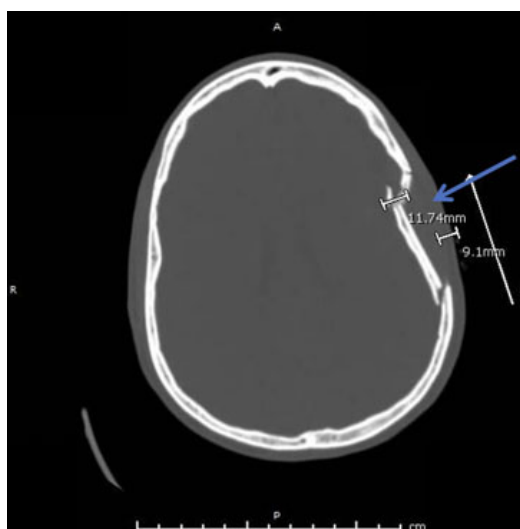


Figure 2: Pre-operative axial computed tomography (CT) of the skull with left calvarial frontoparietal depressed skull fracture measuring 20.84 mm in depth and 11.74 mm below the interior table (blue).



Figure 3: Pre-operative sagittal computed tomography (CT) of the skull with contusive hemorrhage and edema underlying the depressed fracture with right contrecoup temporal/parietal subarachnoid hemorrhage.

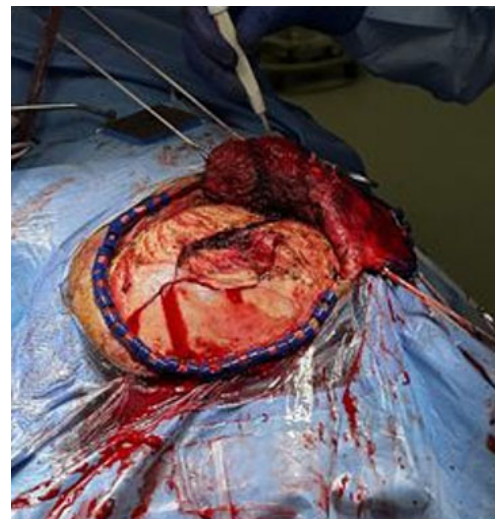


Figure 4: Intra-operative photo of depressed skull fracture after overlying skin has been reflected.

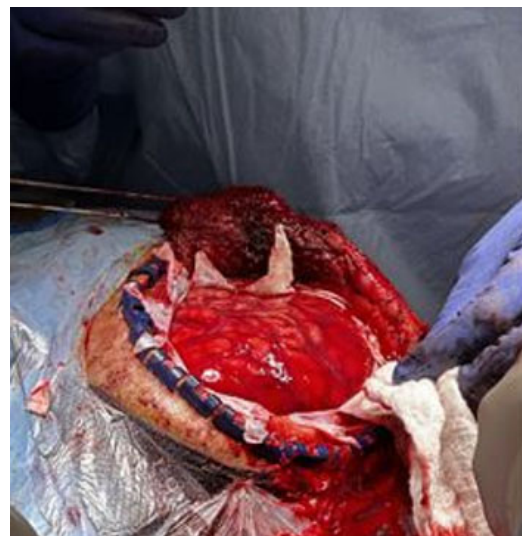


Figure 5: Intra-operative photo of diffuse subarachnoid hemorrhage underlying the sight of impact.

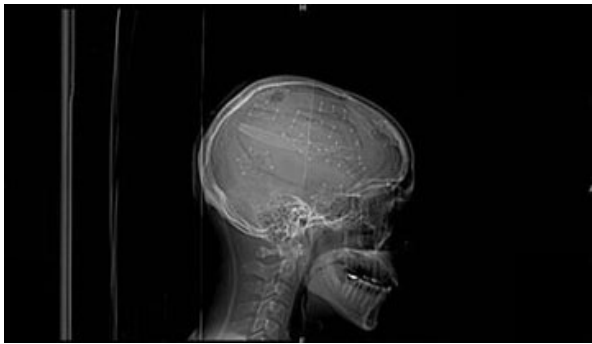


Figure 6: Post-operative sagittal computed tomography (CT) of the skull with reduced and reconstructed skull fracture and appropriately placed drain.

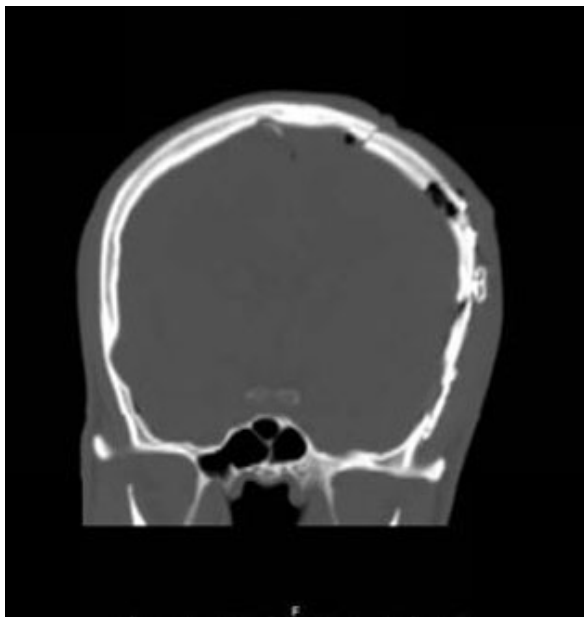


Figure 7: Post-operative coronal computed tomography (CT) of the skull with reduced skull fracture.



Figure 8: Post-operative coronal computed tomography (CT) of the skull with resolving bilateral subarachnoid hemorrhages (red) and pneumocephalus (blue).

DISCUSSION

Apraxia is a term used to describe a broad range of neurological deficits in which a previously learned task can no longer be performed and where the dysfunction cannot be explained by another sensory or motor deficit. Apraxia of speech (AOS) specifically refers to the loss of the ability to produce speech that occurs in the absence of aphasia or dysarthria and can either develop in childhood or be acquired [10, 11]. While AOS is most commonly the result of a cerebrovascular insult, it can also be developed in the setting of trauma, neoplasm, or neurodegeneration [10, 12].

The term “apraxia of speech” has occasionally been used synonymously with Broca’s aphasia. The misconception that the two disorders are one and the same may have arisen from the fact that AOS and Broca’s aphasia often occur together. However, the two disorders have been shown to be distinguishable, since AOS has been documented in non-aphasic patients who do not manifest linguistic deficits, such as agrammatism and naming deficits [12]. Apraxia of speech is differentiated from aphasia or dysarthria in that it is not caused by a muscle weakness and does not interfere with comprehension of auditory or written communication [13].

Speech apraxia refers to a purely motor deficit in speech production in the absence of muscle atrophy. Aphasia refers to overall difficulty in language comprehension, regardless of the motor ability to produce speech. When diagnosing speech apraxia, therapists look for differences in speech prosody, difficulty with complex pronunciations, and production of inconsistent sounds. When diagnosing aphasia, physicians assess language retrieval with tasks including labeling objects and following commands [12, 13].

Neuroanatomically, speech production aphasia is correlated to Broca’s area in the left inferior frontal gyrus. Few studies have been published on traumatic insult to Broca’s area, and no previous publication has described a pure motor speech deficit [14].

Here we presented the case of an isolated AOS in the absence of other neurologic or motor deficits secondary to a left frontoparietal depressed skull fracture. The treatment for this patient, as with many head trauma patients, was operative management followed by speech therapy [2, 15].

In patients with cerebrovascular injury to Broca’s area, recovery can be measured in months to years with younger patients and those that maintain higher levels of speech comprehension having the highest recovery rates [16]. Our patient was able to resume speech within a matter of days and was producing a halting speech with appropriate vocabulary and syntax by the time of discharge. Few cases have been published on traumatic insult isolated to Broca’s area with no prior studies showing a pure motor deficit [17, 18].

In our patient, who maintained full comprehension and could clearly convey his thoughts through written and

electronic communication, the deficit was purely motor. The distinction between an aphasia and an apraxia of speech is that in apraxia there is solely an impairment in the motor aspect of speech production, whereas in aphasia there is impairment in linguistic capabilities. The patient was completely alert, oriented, and both cognitively and neurologically intact beyond his new onset inability to produce speech. His resulting Glasgow Coma Score (GCS) of 11 did not provide an accurate representation of his level of consciousness. Some limitations to the GCS have been outlined previously in the literature and this case further highlights a need for an improved classification that incorporates the overall clinical picture and has specific modifications for unusual circumstances [19]. Challenges in communication represent an area in which a mismatch between clinical tools and their intended measures can lead to delays in care and obstacles to obtaining and documenting an accurate assessment of neurologic status [20]. Thus, it is imperative that thorough and detailed documentation outside of updated GCS be kept to assess for improvement or decline of these patients.

CONCLUSION

While often occurring concurrently, there is a distinction between an aphasia and an apraxia of speech. With apraxia there is solely an impairment in the motor aspect of speech production, whereas in aphasia there is impairment in linguistic capabilities. It is important to distinguish the two when determining management and prognosis. Here we present the unusual case of a pure motor apraxia of speech following an isolated traumatic insult to Broca's area.

REFERENCES

- Peterson AB, Thomas KE, Zhou H. Centers for Disease Control and Prevention. Surveillance Report of Traumatic Brain Injury-related Deaths by Age Group, Sex, and Mechanism of Injury – United States, 2018 and 2019. Center for Disease Control and Prevention, U.S. Department of Health and Human Services; 2022.
- Thurman DJ, Alverson C, Browne D, et al. Centers for Disease Control and Prevention. Report to Congress: Traumatic Brain Injury in the United States. Center for Disease Control and Prevention, U.S. Department of Health and Human Services; 1999.
- Hung CC, Chiu WT, Lee LS, Lin LS, Shih CJ. Risk factors predicting surgically significant intracranial hematomas in patients with head injuries. *J Formos Med Assoc* 1996;95(4):294–7.
- Bullock MR, Chesnut R, Ghajar J, et al. Surgical management of depressed cranial fractures. *Neurosurgery* 2006;58(3 Suppl):S56–60.
- Muhammad G, Aurangzeb A, Khan SA, et al. Dural tears in patients with depressed skull fractures. *J Ayub Med Coll Abbottabad* 2017;29(2):311–5.
- Yavuz MS, Asirdizer M, Cetin G, Günay Balci Y, Altinkok M. The correlation between skull fractures and intracranial lesions due to traffic accidents. *Am J Forensic Med Pathol* 2003;24(4):339–45.
- Keller SS, Crow T, Foundas A, Amunts K, Roberts N. Broca's area: Nomenclature, anatomy, typology and asymmetry. *Brain Lang* 2009;109(1):29–48.
- Le H, Lui MY. Aphasia. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.
- Stinnett TJ, Reddy V, Zabel MK. Neuroanatomy, Broca Area. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.
- Gowda SN, Kolton Schneider L. Apraxia. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2023.
- Ogar J, Slama H, Dronkers N, Amici S, Gorno-Tempini ML. Apraxia of speech: An overview. *Neurocase* 2005;11(6):427–32.
- Basso A, Scarpa MT. Traumatic aphasia in children and adults: A comparison of clinical features and evolution. *Cortex* 1990;26(4):501–14.
- Ogar J, Willock S, Baldo J, Wilkins D, Ludy C, Dronkers N. Clinical and anatomical correlates of apraxia of speech. *Brain Lang* 2006;97(3):343–50.
- Graff-Radford J, Jones DT, Strand EA, Rabinstein AA, Duffy JR, Josephs KA. The neuroanatomy of pure apraxia of speech in stroke. *Brain Lang* 2014;129:43–6.
- Patira R, Ciniglia L, Calvert T, Altschuler EL. Pure apraxia of speech due to infarct in premotor cortex. *Neurol Neurochir Pol* 2017;51(6):519–24.
- Kertesz A, McCabe P. Recovery patterns and prognosis in aphasia. *Brain* 1977;100 Pt 1:1–18.
- Galeoto G, Polidori AM, Spallone M, et al. Evaluation of physiotherapy and speech therapy treatment in patients with apraxia: A systematic review and meta-analysis. *Clin Ter* 2020;171(5):e454–65.
- Jang SH, Ha JW, Kim HY, Seo YS. Recovery of injured Broca's portion of arcuate fasciculus in the dominant hemisphere in a patient with traumatic brain injury. *Medicine (Baltimore)* 2017;96(51):e9183.
- Bodien YG, Barra A, Temkin NR, et al. Diagnosing level of consciousness: The limits of the Glasgow Coma Scale total score. *J Neurotrauma* 2021;38(23):3295–305.
- McHenry M, Wilson R. The challenge of unintelligible speech following traumatic brain injury. *Brain Inj* 1994;8(4):363–75.

Author Contributions

Cierra N Harper – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Kavita Prakash – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be

accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Damirez Fossett – Conception of the work, Design of the work, Acquisition of data, Analysis of data, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Guarantor of Submission

The corresponding author is the guarantor of submission.

Source of Support

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Consent Statement

Written informed consent was obtained from the patient for publication of this article.

Conflict of Interest

Authors declare no conflict of interest.

Data Availability

All relevant data are within the paper and its Supporting Information files.

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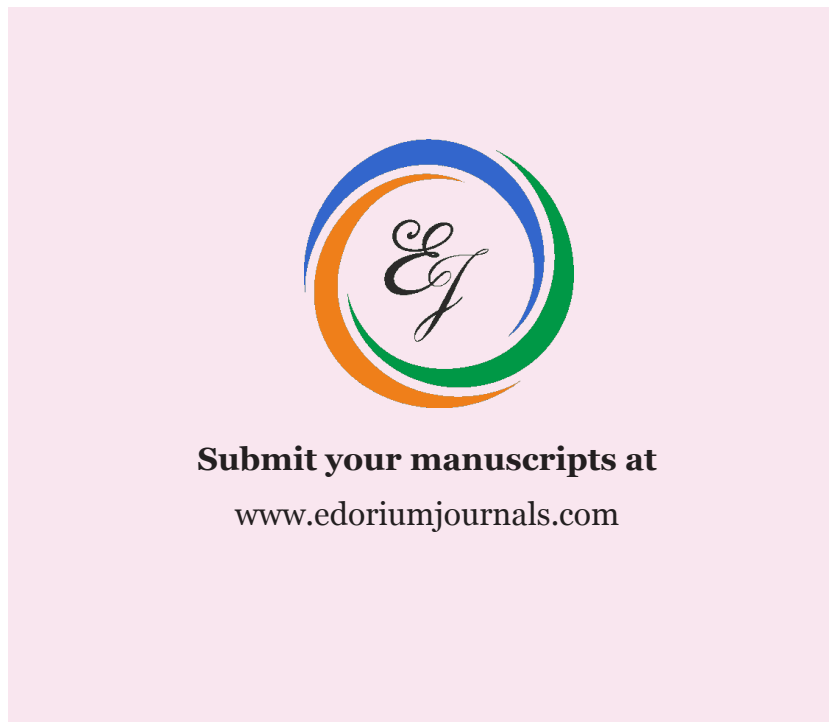
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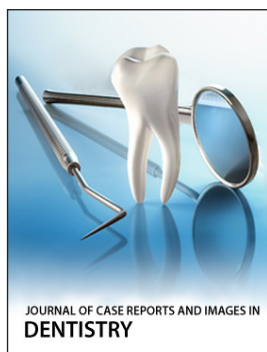
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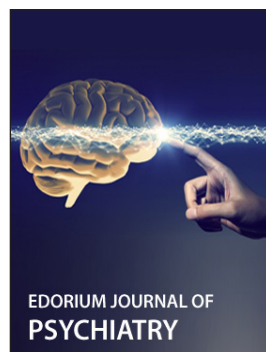
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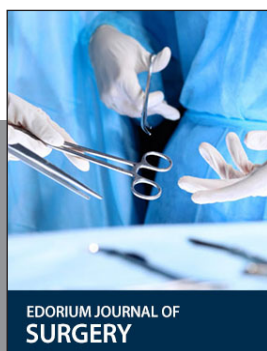
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