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Case Report

Recovery against the Odds: A Case Report of Post-Cardiac Arrest Brain Injury Management

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ABSTRACT:

Background: Post-cardiac arrest brain injury (PCABI) is a major contributor to mortality post-resuscitation. Management involves a multidisciplinary approach focusing on early recognition and aggressive intervention for neurological and cardiac complications. Tools like EEG, CT scan, and MRI aid in assessing neurological impairment. Despite improvements in acute-care treatments, standardized rehabilitation pathways for PCABI are lacking, highlighting the need for ongoing research and patient-centered care strategies.

Case Description: A 22-year-old male presented with ventricular fibrillation and subsequent a systole, leading to witnessed cardiac arrest. Post-resuscitation, he exhibited acute-anoxic encephalopathy (AAE) with a Glasgow Coma Scale (GCS) score of 3/15, requiring intensive care support, including mechanical ventilation and sedation. Initial imaging confirmed hypoxic-ischemic encephalopathy (HIE) and low ejection fraction (EF), which surprisingly improved after a short time interval. Following active neuroprotective therapy, the patient showed signs of cognitive and functional recovery, with improvements in GCS and the ability to perform basic daily activities, though challenges remain in fully assessing long-term outcomes. Neurological complications, such as brain edema and generalized epileptic activity, were managed with pharmacotherapy, while cardiac arrhythmias were treated using antiarrhythmic medications and direct current shocks.

Conclusions: This case underscores the potential for recovery in young patients following cardiac arrest, despite an initial poor prognosis. Early neuroprotective therapy and intensive care support were key in improving cognitive function and daily activities. While long-term outcomes remain uncertain, careful monitoring for neurological and cardiac complications is essential. Clinicians should be aware of the possibility of significant recovery, even in severe hypoxic-ischemic encephalopathy.

Keywords: Cardiac arrest, Coma, Hypoxia-Ischemia, Brain, Electroencephalogram (EEG)

INTRODUCTION:

In the US, over 400,000 people experience cardiac arrest every year. The overall survival rates are still quite low, at 10% for cases outside of hospitals and 22% for cases within hospitals [1]. Post-cardiac arrest brain injury (PCABI) is a critical concern, significantly impacting patient outcomes and quality of life. It arises from the complex response of the brain to reperfusion and its intolerance to ischemia, leading to secondary brain injury, which is a significant cause of mortality [1, 2]. Coma following resuscitation, known as acute-anoxic encephalopathy (AAE), is a fatal consequence. In the past 20 years, as cardiac resuscitation success rates have improved, the incidence of AAE has increased to 300,000 per year in the USA [1].

Even in cases where initial attempts at resuscitation are successful, up to 70% of hospital admissions result in death due to brain damage following cardiac arrest because the heart is more resilient to ischemia than the brain [1]. Restoring cardiac and cerebral function to pre-cardiac arrest levels is the ultimate aim of resuscitation. The likelihood of a favorable neurological outcome is increased by two to four times when high-quality cardiopulmonary resuscitation (CPR) and rapid defibrillation are initiated early to reduce primary brain injury [1]. To improve outcomes, several studies have been conducted in recent years to gain a better

understanding of the causes, consequences, and management of anoxic brain injury following cardiac arrest. Our aim is to update the reader about the management of anoxic brain injury in the acute setting in these patients.

CASE REPORT:

A 22-year-old Indian male with no known past medical history was taken to the emergency room after experiencing out-of-hospital cardiac arrest. He achieved Return of Spontaneous Circulation (ROSC) following three cycles of cardiopulmonary resuscitation (CPR) performed by the paramedic team. The patient was sleeping when his roommate noticed he was gasping and immediately began CPR, which continued for 10 minutes before the paramedics arrived. The initial cardiac rhythm observed was ventricular fibrillation, prompting the administration of a direct current (DC) shock. In the subsequent cycle, the ECG showed asystole, necessitating the continuation of CPR until ROSC was achieved during the third cycle.

Upon hospital arrival, immediate evaluation revealed a hemodynamically unstable patient with a Glasgow Coma Scale (GCS) score of 3/15, sluggish bilateral pupils measuring 3 mm, and poor respiratory effort characterized by a gasping pattern. This condition necessitated immediate intubation, mechanical ventilation, and sedation with fentanyl and midazolam to minimize the risk of secondary brain injury. A 12-lead ECG demonstrated sinus tachycardia. An initial echocardiogram revealed left ventricular enlargement and impaired ventricular contraction, with an ejection fraction of 30%, suggesting possible long-standing heart failure. An initial CT scan of the brain revealed diffuse brain edema as shown in (figure 1), necessitating the administration of a stat dose of methylprednisolone at 250 mg and the initiation of piracetam at 800 mg twice daily.

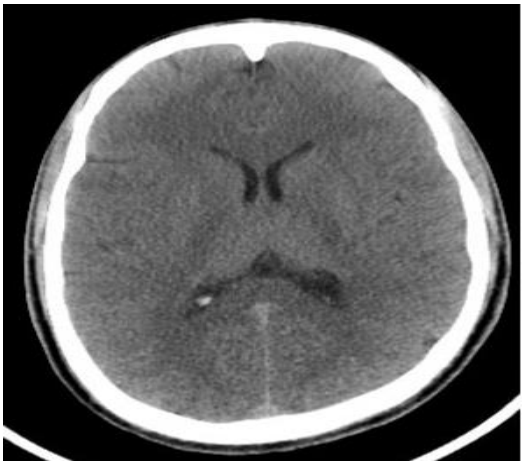

Figure 1. Mild Diffuse Brain Edema.

The patient was admitted immediately to the ICU, where the established post-cardiac arrest protocol was promptly implemented to ensure comprehensive care and monitoring. This included therapeutic hypothermia, optimal ventilation, hemodynamic support, and continuous neurological assessment to facilitate recovery and minimize potential complications.

On day 2 following admission, the patient experienced repeated episodes of abnormal jerky movements for which an EEG was done. EEG as shown in (figure 2) as epileptiform activity. This led to the initiation of treatment with 500 mg of levetiracetam twice daily, followed by the addition of 1 gram of sodium valproate. A follow-up CT scan of the brain performed on day 4 reported normal findings. Additionally, recurrent episodes of supraventricular tachycardia occurred on the fourth day, necessitating multiple direct current shocks and a dose of 150 mg of amiodarone, followed by a 900 mg infusion over 24 hours.

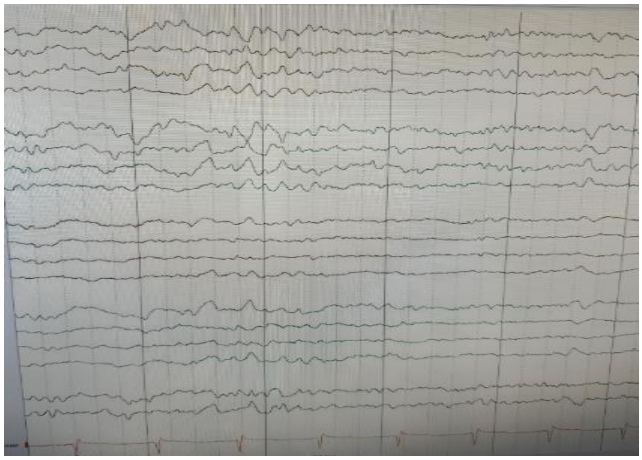

Figure 2 EEG shows epileptiform activities.

On day 5, a sedation vacation was performed, revealing a Glasgow Coma Scale (GCS) score of 6/10 + T (with "T" indicating intubation). The breakdown of the GCS was as follows: Eye response (E) 2, Verbal response (V) 2, and Motor response (M) 2. A diffusion MRI was performed on day 6 due to a lack of improvement in the Glasgow Coma Scale (GCS) score. The imaging revealed a finding suggestive of ischemic encephalopathy as shown in (figure 3).

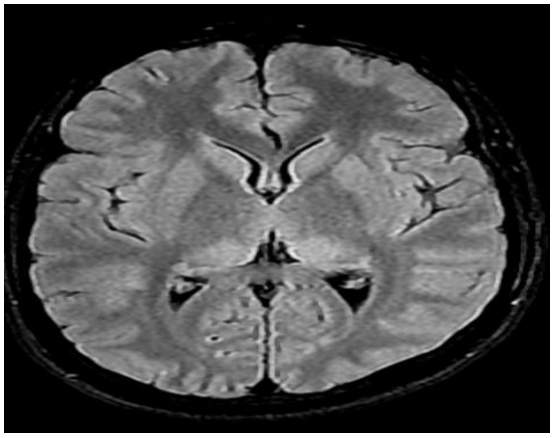

Figure 3 MRI brain with a finding suggestive of ischemic encephalopathy

By day 7, the GCS had improved to 10/10 + T, with an EVM breakdown of Eye response 3, Verbal response 3, and Motor response 4. The patient successfully passed the weaning procedure and was extubated. On day 8, the echocardiogram revealed no significant changes, while day 9 saw the GCS improve to 15/15. Holter monitoring indicated Unifocal Premature Atrial Complexes and spontaneously resolving supraventricular tachycardia (SVT) with a maximum heart rate of 210 beats per minute. The patient was then transferred from the ICU to the CCU. A follow-up echocardiogram conducted on day 13 revealed a 40% improvement in ejection fraction, with the coronary angiogram showing no significant findings. The cardiologist recommended ablation and defibrillator implantation to manage the SVT. On day 14, the patient exhibited intermittent mild episodes of abnormal jerky movements, prompting a repeat EEG. The EEG shown in (figure 4) reported as generalized slow waves.

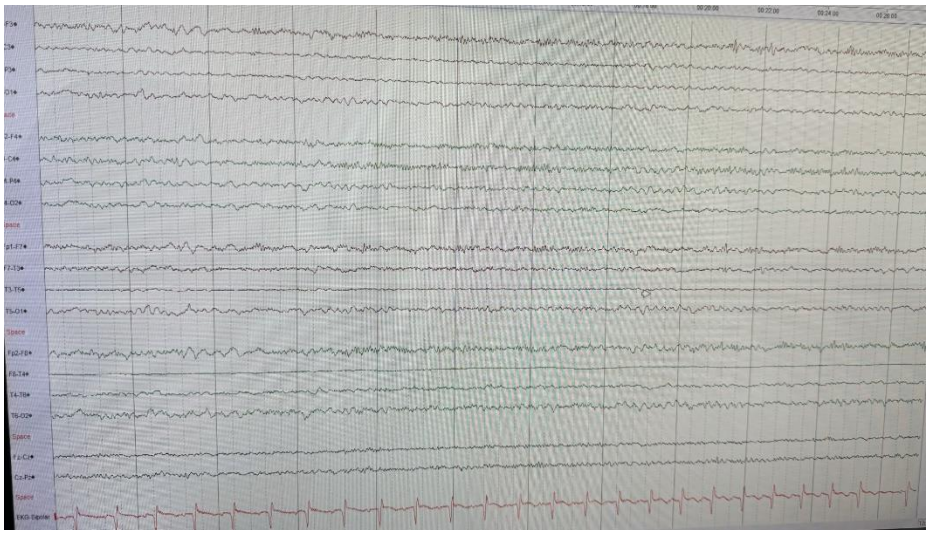

Figure 4 EEG reported as generalized slow waves.

The patient demonstrated significant functional recovery during his hospital stay. By day 7, he was able to follow simple commands and had regained motor control sufficient for sitting up in bed with assistance. By day 9, he was independent in activities such as eating and was able to sit without assistance. The patient's muscle strength improved over time, and he was able to walk short distances with minimal support by day 14. This functional recovery, in parallel with his GCS improvement, was an essential aspect of his rehabilitation process. While he had reached a GCS of 15/15 by day 9 and could make his own decisions, the patient was not bedridden, reflecting the importance of his functional progress in contributing to his overall recovery. The patient's EEG was not repeated after the finding of generalized slow waves on day 14. As such, there was no assessment of whether the brain wave activity had fully normalized post-recovery.

Regarding potential ischemic injuries, no significant ischemic damage was observed in other organs such as the kidneys or liver. There was no evidence of acute tubular necrosis (ATN) or ischemic hepatitis, both of which are vulnerable to anoxic injury and can complicate the recovery process. However, the patient's recovery was carefully monitored for any signs of multi-organ dysfunction.

On the 21st day after admission, the patient voluntarily signed a high-risk LAMA (Leave Against Medical Advice) form to be discharged and continue treatment in his home country. At that time, the patient was in stable condition and fully capable of making his own decisions.

DISCUSSION:

When a patient survives the acute phase of cardiac arrest, post-cardiac arrest brain injury (PCABI) is the primary cause of long-term disability and the primary cause of death [1]. Primary (ischemic) and secondary (reperfusion) injuries are included in PCABI pathophysiology, and they happen in turn during cardiac arrest, resuscitation, and the acute post-resuscitation phase [7]. To maintain tissue homeostasis, the brain receives 15–20% of total cardiac output, even though it only makes up 2% of body weight [8]. The survival of brain tissue is highly dependent on a steady flow of oxygen and glucose, which is the energy substrate. When cerebral blood flow (CBF) is interrupted, brain activity stops immediately. Human studies show that the electroencephalogram (EEG) becomes isoelectric after 10–30 s of asystole, and consciousness is lost between 4 and 10 s of absent CBF [9]. Similarly, the initial insult of hypoxic-ischemic injury in our patient resulted in a range of pathologies, including brain edema, seizures, and cardiac arrhythmias.

Several techniques, such as electroencephalography (EEG), computed tomography (CT scan), and magnetic resonance imaging (MRI) are used to assess and confirm the degree and kind of neurological damage in patients who had cardiac arrest. A head computed tomography (CT) scan should be performed as soon as possible following arrest to rule out any primary brain injury that could cause a coma or cardiac arrest [10, 11]. Brain edema, which manifests in our patient as sulcal effacement and diminished grey-white matter differentiation, is the primary CT finding of anoxic brain injury [12, 13]. The Glasgow Coma Scale (GCS) is frequently used to evaluate neurologic outcomes. A GCS score of five indicates mild to no disability and good cerebral performance. Lower scores correspond to more severe disabilities with a score of 1 indicating death or brain death [2]. Our findings indicate that a Glasgow

Coma Scale (GCS) score of 3 at the time of admission may function as a prognostic indicator in early neurological rehabilitation. This severely low GCS score suggests profound impairment, which can inform clinical decision-making and optimize rehabilitation strategies tailored to the patient's potential for recovery.

Approximately two-thirds of all deaths occurring after return of spontaneous circulation (ROSC) are attributed to post-cardiac arrest brain injury (PCABI), with cardiovascular instability and multiorgan failure contributing to the majority of fatalities within the first 48 to 72 hours post-ROSC [14]. A systematic review conducted in 2016, encompassing 23,388 patients from 26 studies, found that brain death was diagnosed in an average of 5% of patients who received conventional cardiopulmonary resuscitation (CPR) and in over 20% of those who underwent extracorporeal CPR, corresponding to 8% and 28% of all deaths, respectively [15].

Additionally, even after initial partial recovery from post-anoxic coma, delayed brain edema leading to brain death has been reported [24]. Massive cerebral edema is frequently observed in these patients, with post-resuscitation rates of brain death being higher among individuals with a neurological cause of arrest, those presenting with a non-shockable initial rhythm, and patients exhibiting lower serum sodium levels [16]. During hospitalization, there were no signs of brain death, and our patient demonstrated signs of cerebral edema, which progressively improved, along with notable recovery from anoxic brain injury. Methylprednisolone, a corticosteroid widely utilized to mitigate inflammation and edema in neurological conditions, was administered due to its mechanism of inhibiting pro-inflammatory cytokines and stabilizing cell membranes, thereby reducing intracranial pressure and limiting secondary neuronal damage [25]. Timely administration of methylprednisolone can be crucial in managing cerebral edema, potentially improving outcomes by preserving neurological function. In conjunction with this therapy, piracetam was initiated at a dosage of 800 mg twice daily. As a nootropic agent, piracetam has garnered attention for its neuroprotective properties, particularly in hypoxic-ischemic injury, where it may enhance neuroplasticity, improve cerebral metabolism, and increase blood flow to affected regions of the brain. Its role in reducing oxidative stress and improving mitochondrial function supports its potential in protecting brain tissue following acute neurological insults, promoting neuronal survival and cognitive recovery alongside the effects of methylprednisolone [26].

Hypoxic-ischemic encephalopathy can cause a wide range of disabilities, from full recovery to coma or even death. According to clinical trials, 64% of post-hypoxic coma patients passed away, 9% remained unconscious or in an unresponsive wakefulness syndrome (UWS), and 27% of patients recovered consciousness within 28 days [17, 18]. More patients are surviving acute care treatment and may be admitted to neurological early rehabilitation facilities as a result of improvements in pre-hospital and intensive care [19]. Similarly, the treatment approach in our patient includes prompt and effective resuscitation efforts by the paramedic team, aggressive management of cardiac arrhythmias with direct current shocks and antiarrhythmic medications, and successful weaning from mechanical ventilation. The multidisciplinary care team's coordination and quick adaptation to evolving clinical scenarios also contributed to the positive outcome.

Unlike for stroke, traumatic brain injury, or myocardial infarction, there are currently no widely accepted rehabilitation care pathways for patients with brain injury following cardiac arrest. Guidelines for treating seizures, maintaining normal physiology, and targeted temperature management (TTM) specifically address brain injury following cardiac arrest [20]. In our patient, the coordination of the multidisciplinary care team involving emergency medicine, neurology, cardiology, and critical care resulted in prompt interventions for cardiac and neurological problems. Upon admission to the intensive care unit (ICU) following cardiac arrest, the patient exhibited non-convulsive epileptic foci, characterized as interictal epileptiform activity, which were initially treated with sodium valproate and levetiracetam. Studies on animals have demonstrated the neuroprotective properties of LEV as well as its antiepileptic effects [21, 22]. LEV administration following hypoxia decreases neuronal apoptosis, as demonstrated by Kilicdag et al [23].

Limitations of the case include the lack of detailed information on the patient's pre-existing cardiac condition, which may have influenced the initial cardiac event. Additionally, the decision for the patient to sign a high-risk Leave Against Medical Advice (LAMA) form on day 21 raises questions about the long-term follow-up and continuity of care, especially considering the need for potential cardiac interventions such as ablation and defibrillator implantation. Implications of this case underscore the critical need for comprehensive post-cardiac arrest care, including neurological monitoring, seizure management, cardiac rhythm surveillance, and cardiac function assessment. Long-term follow-up and continuity of care are essential to address potential complications and optimize patient outcomes. Additionally, the decision-making process regarding discharge against medical advice highlights the importance of patient autonomy balanced with medical recommendations and informed consent processes. Future actions include continued research on PCAS

management strategies, enhancing interdisciplinary collaboration, and promoting patient-centered care.

CONCLUSION:

This case highlights the complexity of managing post-cardiac arrest brain injury (PCABI), particularly in a young patient, and underscores the importance of neuroprotective strategies. Despite the initial profound neurological impairment, the patient showed significant cognitive recovery, demonstrating the potential for functional improvement even after brain injury. The use of antiepileptic medications, corticosteroids, and piracetam played a crucial role in managing seizure activity, reducing brain edema, and supporting overall neurological recovery. This case emphasizes the importance of early neuroprotective interventions and a multidisciplinary approach in optimizing outcomes for post-cardiac arrest patients. It challenges the traditional prognosis for ischemic encephalopathy and demonstrates the potential for recovery.

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