Case Report *Parul University Journal of Health Sciences and Research*
ISSN: 2583-990X (E)
https://doi.org/10.62373/PUJHSR.2024.111

Unmasking Atrial Fibrillation: The Hidden Cardiac Risk in Traumatic Brain Injury

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ABSTRACT:

Background: Traumatic intracranial hemorrhage (ICH) often triggers a systemic inflammatory response and arrhythmias. Atrial fibrillation (AF), the most frequent arrhythmia, is linked with systemic inflammation, increasing cardiovascular mortality risk twofold.

Methods: This study explores the link between traumatic ICH and AF through a case study of a 70-year-old female with traumatic brain injury (TBI) leading to AF. Comprehensive clinical assessments, imaging, and laboratory tests were conducted to understand the relationship between TBI, ICH, and AF.

Results: The patient showed altered sensorium and irregular heart rhythm post-trauma. Imaging identified a subdural hematoma with mass effect and midline shift, along with subarachnoid hemorrhage. Initial ECG indicated AF with rapid ventricular response. Treatment with IV anti-arrhythmics and osmotic diuretics stabilized heart rhythm and reduced intracranial pressure (ICP). A subsequent ECG showed a return to normal sinus rhythm.

Conclusions: Findings suggest traumatic ICH significantly raises AF risk, likely due to systemic inflammation and elevated ICP. This underscores the necessity of vigilant cardiac monitoring in TBI patients. Further research should clarify the mechanisms connecting TBI, ICH, and AF to develop effective management strategies.

KEY WORDS: Intracranial hemorrhage(ICH), Atrial Fibrillation(AF), Traumatic Brain Injury(TBI), Intracranial Pressure(ICP),Subarachnoid hemorrhage(SAH).

BACKGROUND

Traumatic brain injury (TBI) is a critical health issue with varying etiologies across populations [1]. Traumatic intracranial hemorrhage (ICH) is defined as intracranial bleeding following moderate to severe TBI, a leading cause of death in young individuals and resulting in lifelong disabilities for survivors [2,3]. Additionally, TBI can initiate inflammatory cascades and vascular disruptions, including the release of pro inflammatory cytokines and chemokines, with long-term consequences [4,5].

These consequences may persist over time after the initial TBI [6-8]. Atrial fibrillation (AF) is the most prevalent sustained cardiac arrhythmia; it confers a twofold increased risk of cardiovascular mortality [9]. In recent decades, concerns have been raised regarding the role of inflammation in the pathogenesis of AF.

Several studies have demonstrated an increased risk of AF in patients with multiple systemic diseases, including in patients who have undergone cardiac surgery or have coronary artery disease, hypertension, obesity or sepsis [10]. New-onset AF is associated with detrimental consequences. Although the mechanism connecting these situations to AF is not completely understood, studies have proposed that systemic inflammation plays a major role in the pathophysiology of AF. Accordingly, we hypothesized that there is increased risk of AF in patients with traumatic ICH. However, limited evidence is available regarding the association between traumatic ICH and AF. In this case, how TBI affects the heart rate and rhythm, will be covered.

CASE REPORT

A 70-year-old woman, previously healthy, arrived at the ER of PSH following a bike fall on 11/07/2023 at around 2 PM. She sustained a left frontotemporal head injury. Post-injury, she experienced vomiting and altered sensorium. Clinical examination revealed:

- **Airway:** Patent, patient could speak.
- **Breathing:** Bilateral chest rise and air entry equal, SpO2 98% on room air, RR 24/min, regular.
- **Circulation:** PR 128/min, all peripheral pulses palpable but irregularly irregular, BP 170/90 mm Hg.
- **Disability:** Drowsy, GCS: E4V4M6, RBS 128 mg/dL, pupils reactive to light with bilateral plantar flexors.
- **Exposure:** No abnormalities detected (NAD).

Diagnosis: The 12-lead ECG indicated AF with RVR at 140/min (Figure 2). NCCT brain imaging showed a subdural hematoma on the left frontoparietotemporal lobe with a mass effect and a 3.7 mm midline shift to the right. Subarachnoid hemorrhage (SAH) was also noted (Figure 1). A 2D echo, blood tests including cardiac markers, chest X-ray (AP view), and E-FAST were normal.

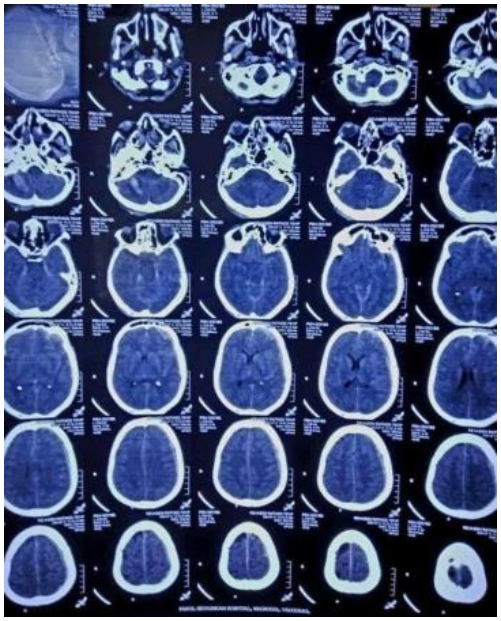

Fig. 1: NCCT brain imaging showing subdural hematoma involving the left frontoparietotemporal lobe convexity with a mass effect and midline shift.

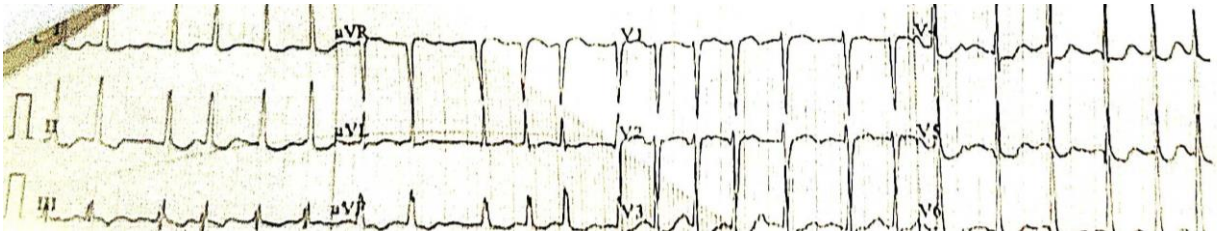

Fig. 2: Initial 12-lead ECG indicating atrial fibrillation with rapid ventricular response

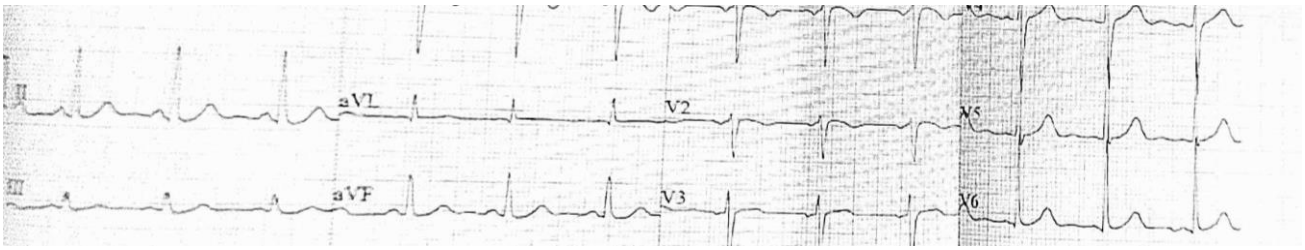

Fig. 3: Follow-up 12-lead ECG showing the return to normal sinus rhythm after treatment.

Management: The patient received IV anti-arrhythmic (Inj. Diltiazem 3 ml without dilution IV stat over 5 minutes), IV anti-emetics, and IV antacids. After 10 minutes, her pulse rate was controlled but remained irregularly irregular, and BP was 160/90 mm Hg. IV mannitol and IV anticonvulsant (Inj. Levetiracetam 1 gram in 100 cc NS IV stat over 20 minutes) were

administered. After 20 minutes, BP was 138/60 mm Hg, suggesting reduced ICP from mannitol's osmotic diuresis effect. A subsequent 12-lead ECG indicated a return to normal sinus rhythm. The patient was then moved to the ICU, where ECG monitoring showed consistent normal sinus rhythm.(figure:3)

DISCUSSION

Various mechanisms explain the TBI-AF link. TBI can damage the cerebral cortex, increasing hypothalamic pressure and causing sustained sympathetic stimulation and systemic catecholamine release. This catecholamine-mediated myocardial injury can lead to cardiac structural damage due to factors like tachyarrhythmia, coronary spasm, direct myocardial toxicity, and increased intracellular calcium accumulation, potentially causing heart failure and PV arrhythmogenesis, triggering AF.

Another hypothesis involves TBI-induced immune and inflammatory responses. Studies report increased levels of inflammatory factors like interleukin-1 and TNF-alpha in the CNS post-TBI. Other immune and inflammatory mediators include brain-derived antigens, danger-associated molecular patterns, immune signaling molecules, and autonomic nervous system signals.

Key points summarizing the TBI-AF link include:

1. **Sympathetic Stimulation and Catecholamine Release:** TBI elevates hypothalamic pressure, causing sustained sympathetic stimulation and systemic catecholamine release, leading to myocardial injury and arrhythmias.
2. **Inflammatory Responses:** TBI triggers proinflammatory cytokine and chemokine release, significantly contributing to AF pathophysiology.
3. **Intracranial Pressure (ICP):** Raised ICP from traumatic ICH can indirectly affect cardiac function, contributing to AF development.

The pathophysiological mechanism linking traumatic ICH and AF remains unclear but may involve increased sympathetic stimulation, inflammation, and raised ICP. Further research is needed to clarify these mechanisms and develop effective management strategies for TBI and AF patients.

CONCLUSION:

This study underscores the significant association between traumatic brain injury (TBI) and atrial fibrillation (AF). The findings indicate that traumatic intracranial hemorrhage (ICH) can trigger systemic inflammation and elevated intracranial pressure (ICP), contributing to AF development. The case study demonstrates that timely intervention and management of brain injury and cardiac arrhythmia can stabilize the patient's condition and restore normal sinus rhythm.

Clinical implications are substantial, necessitating vigilant cardiac monitoring in TBI patients. Early detection and management of AF in TBI patients can reduce cardiovascular morbidity and mortality risks, improving overall outcomes.

Future research should focus on elucidating the precise mechanisms linking TBI, systemic inflammation, and AF. Longitudinal studies are needed to explore TBI's long-term cardiovascular effects and preventive strategies' benefits. Additionally, investigating various anti-inflammatory and cardioprotective treatments in TBI patients could optimize care for this vulnerable population. Advancing our understanding of the heart-brain connection can lead to more effective interventions, mitigating the risks associated with traumatic brain injuries and improving patient prognosis.

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Article information

Manuscript Submitted: 20-06-2024 **Manuscript Revised:** 17-07-2024
Manuscript Accepted: 20-08-2024 **Manuscript published:** 20-12-2024

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