NEWPORT INTERNATIONAL JOURNAL OF SCIENTIFIC AND EXPERIMENTAL SCIENCES (NIJSES)

Volume 6 Issue 2 Page 31-36, 2025

©NIJSES PUBLICATIONS
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ONLINE ISSN:2992-5819
PRINT ISSN:2992-6149

https://doi.org/10.59298/NIJSES/2025/62.313600

Hepatic effects of Traumatic Brain Injury using Wistar Rats

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ABSTRACT

Over the years, studies have shown that head injuries can lead to various traumatic effects on the brain. However, these injuries not only impact the brain but also affect the liver. This study examined the histopathological effects of traumatic brain injury (TBI) on the liver. Fifty Wistar rats, weighing between 150g and 250g, were divided into five groups (A–E). Group A served as the negative control, with no trauma induced. Group B received a 50g weight impact on the head, Group C was subjected to a 100g weight, Group D to a 150g weight, and Group E to a 200g weight. The injured animals were sacrificed at intervals of 5 hours, 24 hours, 7 days, and 24 days post-trauma. Their tissues were processed using paraffin embedding and Hematoxylin-Eosin staining. Histological analysis of the control group revealed normal tissue morphology. In contrast, the traumatized groups exhibited pathological changes such as edema, necrosis, apoptosis, gliosis, inflammation, cellular infiltration, and hemorrhage. These findings suggest that TBI indirectly exerts toxic effects on the liver, with the extent of damage correlating to the severity of the trauma.

Keywords: hepatic, traumatic brain injury, peripheral abnormalities, and inflammation

INTRODUCTION

Traumatic brain injury (TBI) is a widespread medical issue, affecting approximately 1.5 million people in the United States each year [1] and [2]. These injuries commonly result from sports activities, traffic accidents, and military operations. While clinical studies increasingly report significant peripheral abnormalities in TBI patients, the underlying mechanisms remain poorly understood. The liver serves as a key organ for detoxification and plays a crucial role in nutrient metabolism, as well as the synthesis of proteins and lipids essential for both the brain and body. There is a bidirectional communication between the brain and peripheral organs, facilitated by the autonomic nervous system, hormones, and metabolites [3] and [4]. Recent research indicates that brain pathogenesis can lead to liver steatosis and metabolic dysfunction, highlighting the significant impact of peripheral disturbances in TBI patients.

MATERIALS AND METHODS

Table 1: Experimental Design

Groups	Induction weight (g)	Rats per group	Sacrificed rats (5hrs)	Sacrificed rats (24hrs)	Sacrificed rats (7days)	Sacrificed rats (21days)	Extra Rats
Group A	0g	10	2	2	2	2	2
Group B	50g	10	2	2	2	2	2
Group C	100g	10	2	2	2	2	2
Group D	150g	10	2	2	2	2	2
Group E	200g	10	2	2	2	2	2

The protocol for the conduct of the study was reviewed and approved by the Faculty of basic medical sciences Research Ethical Committee of Enugu State University, College of Medicine ESUCOM/FBMS/ETR/2025/010.

Experimental Design

Animals were grouped into five groups A to E. Two animals are kept in each group. Group A animals were not induced any injury. Group B animals were induced with 50g weight. Group C animals were induced with 100g weight. Group D animals were induced with 150g weight. Animals in all groups were sacrificed at intervals of 5hrs, 24hrs, 7 days, and 21 days.

TBI Induction

Marmarou's weight drop apparatus was used to carry out the induction of traumatic brain injury. It comprises of a long cylindrical plastic guide of about 2.2cm (diameter), 1.5m which was attached to a clamp stand. A clear transparent U-shaped plastic stage covered with foil was placed underneath the guide tube. A fishing line was attached to metal masses of different weight, the line was passed through the plastic guide tube and attached to the clamp stand. The masses was left hanging freely at about 2.5cm about the tin foil. The rats were placed in isoflourane chamber and lightly anesthetized until they become unconscious. Each rats was placed face down on the tin foil, with their head placed directly in the path of the falling weight. A rat was returned back to the isoflourane chamber if it begins to move before being placed on the tin foil. The Allen key was pulled allowing the weight to fall vertically through the plastic guide tube and hit the rat on the head. Immediately the rats were removed, topical lidocaine (analgesic) was applied on its head to relieve pain. The procedure was carried out for each group with their corresponding weights.

Anaesthetizing the animal

Syringe and ketamine were used. The animals were injected 1ml of ketamine on the thigh to make them unconscious. Pinch response method was used to determine the depth of the anaesthesia. The animals were unresponsive before dissection was done.

Sacrifice/Organ Harvest

A rat were selected from each group at 5hours, 24hours, 7 days amd 21 days post trauma. Before sacrificing, the final weight of the animal were taken. The animals were placed vertically in anatomically supine position on the dissecting table. The hands and foots were pinned for easy dissection. A vertical incision on the thoracic and abdominal region was made with a scalpel. Normal saline was passed through the apex of the heart for easy circulation for ten minutes. 10% formaldehyde was fixed for twenty minutes to prevent autolysis and putrefaction. The cranium of the rats were dissected, the brain harvested and stored in the organ tube containing formalin.

Histology Analysis

The harvested brains were processed using routine paraffin technique [5]. Staining was done using Hematoxylin and Eosin counterstaining method. Magnification for snapping: X300.
Camera used for snapping**:** AMSCOPE 3.7 (5.0Megapixel)
Scale bar**:** 0.4\8µm.

RESULTS

Group A

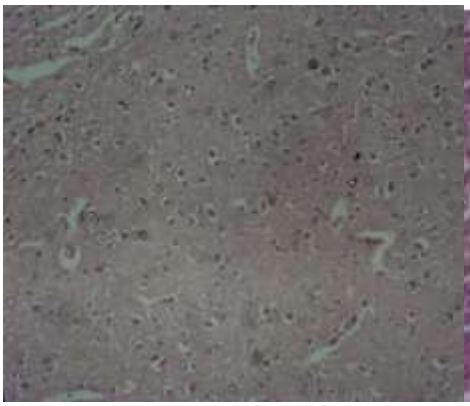

Plate A1: Temporal Lobe of the Brain showing normal architecture of the temporal lobe. H & E.

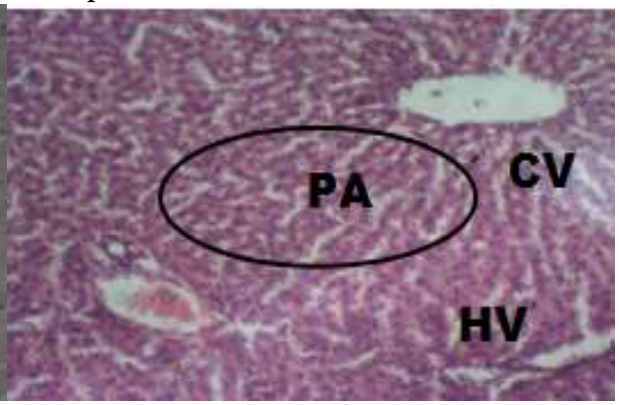

Plate A2: Liver showing the normal hepatic histo-architecture of a laboratory rodent revealing hepatic lobules made up of normal hepatocytes arranged in radial in reconnecting hepatic cords separated by hepatic sinusoids around the central veins (V). Also seen are the portal areas (PA-Circled) consisting of the hepatic artery, hepatic veins and bile duct. H & E.

Group B

Plate B1, temporal lobe of the Brain

Showing mild edema (black arrow) of the temporal lobe. H & E.

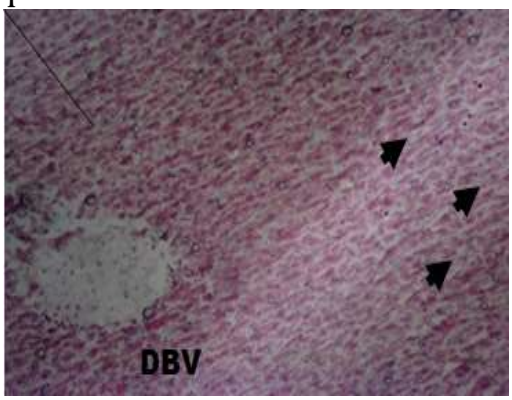

Plate B2: Liver showing normal hepatic histo-architecture of a laboratory rodent revealing hepatic lobules made up of normal hepatocytes arranged in radial in reconnecting hepatic cords separated by hepatic sinusoids around the central veins (V). Also seen are the portal areas (PA-Circled) consisting of the hepatic artery, hepatic veins and bile duct. H & E.

Group C

Plate C1, cortical area of Temporal lobe of the Brain showing gliosis and mild edema (black arrow) of the temporal lobe H & E.

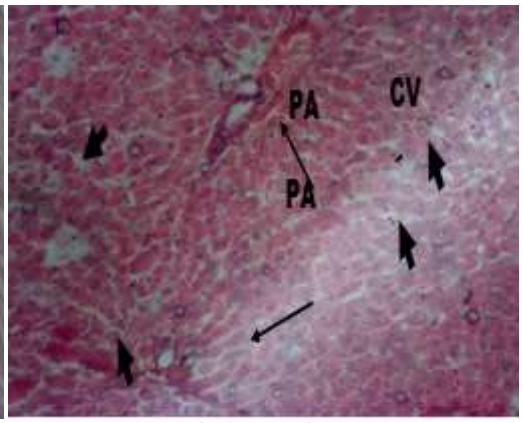

Plate C2, Liver showing a hepatic histo-architecture with regions of hepatocellular necrosis (thick arrow), congested veins (CV).Portal areas (PA). H & E.

Group D

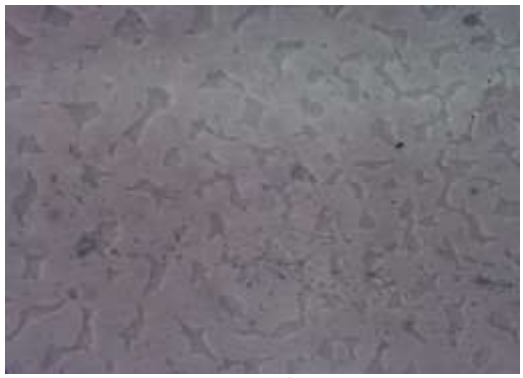

Plate D1, cortical area of the temporal lobe of the Brain Showing gross architectural apoptosis of the temporal Lobe. H & E

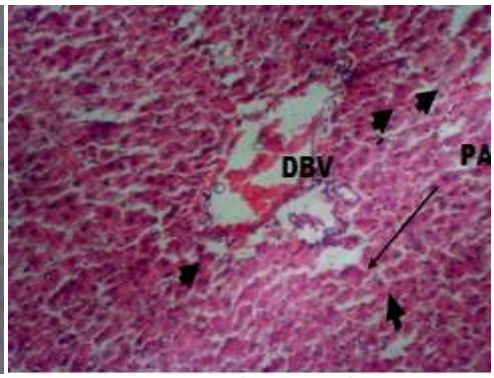

Plate D2, LIVER showing a hepatic histo-architecture with widespread hepatocellular inflammation, leucocytic infiltration (Arrow) distorted veins (DV) and mild hepatocellular necrosis. Portal areas (PA). H & E

Group E

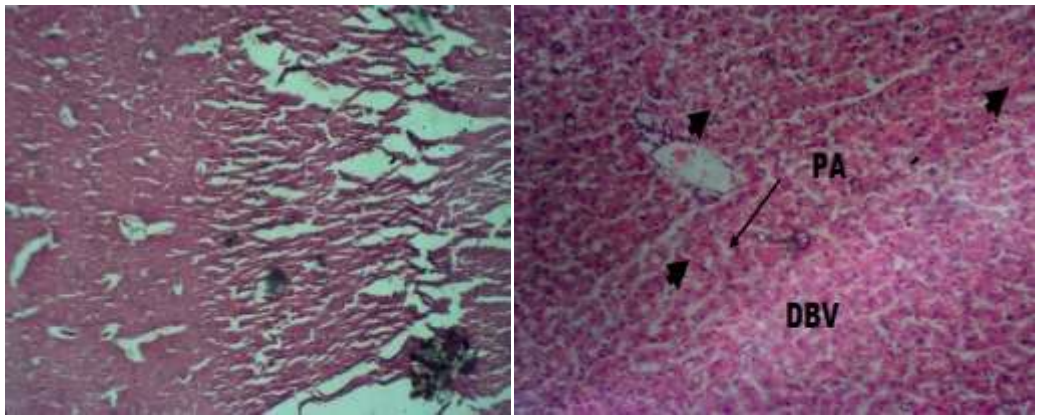

Plate E1: slide of the cortical area of the temporal lobe of the Showing edema, cerebral hemorrhage and Apoptotic architecture. H & E

Plate E2, LIVER showing a hepatic histo-architecture with widespread hepatocellular inflammation, leucocyte infiltration (thick arrow) and distorted blood vessels (BV). Portal areas (PA), H & E.

DISCUSSION

Histological analysis of the negative control group revealed normal brain and liver architecture, whereas the traumatized groups exhibited signs of toxicity in both tissues. These findings align with previous studies, as TBI has been directly linked to a systemic acute-phase response, characterized by the accumulation of inflammatory cells from circulation into the liver [5]. Following acute brain injury, the liver's chemokine expression triggers neutrophil recruitment, leading to hepatic damage and contributing to multi-organ dysfunction [5]. The severity of these toxic effects depends on the nature and timing of the trauma, a conclusion consistent with the findings of [6].

CONCLUSION

This study demonstrated that traumatic brain injury (TBI) not only affects the brain but also has indirect pathological consequences on the liver. Histopathological analyses of liver tissues from Wistar rats subjected to varying degrees of head trauma (50 g, 100 g, 150 g, and 200 g) revealed significant alterations, including edema, necrosis, apoptosis, gliosis, inflammation, cellular infiltration, and hemorrhage. The severity of these liver pathologies was found to correlate directly with the intensity of the induced trauma. Thus, this investigation underscores the systemic impact of TBI, highlighting the liver as an important secondary target organ affected by traumatic brain injuries. These findings have critical implications for comprehensive clinical management and therapeutic strategies for TBI patients, necessitating attention beyond primary neurological assessments.

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CITE AS: Ebuoh Maryann Chiamaka; Uzoefuna Chima Casmir; Ugwu Valentine Ifebuchechukwu; Ozor Ignatius Ikemefuna; Ani Peace Nkechi; Okoli Adaora Ukamaka; Ogbuka Janet N; Ugbor Chidera Victoria, Okpala Precious Onyedika and Nwankwo Stella Nneka (2025). Hepatic effects of Traumatic Brain Injury using Wistar Rats. NEWPORT INTERNATIONAL JOURNAL OF SCIENTIFIC AND EXPERIMENTAL SCIENCES, 6(2):31-36. https://doi.org/10.59298/NIJSES/2025/62.313600