DOI: https://doi.org/10.53350/pjmhs22165727

ORIGINAL ARTICLE

Frequency of Intracranial Hemorrhages in Medico-Legal Death Cases

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ABSTRACT

Objective: To analyze the occurrence of cerebral bleeding in medically assisted deaths.

Study Design: Cross-sectional

Place of Study: Department of Forensic Medicine & Toxicolgy, Peoples University of Medical & Health Sciences for Women (PUMHSW), SBA and Peoples Medical College Hospital (PMCH) Nawabshah, Sindh, Pakistan from 1st October 2021 to 31st March, 2022.

Materials and Methods: For this research, 94 people were enrolled. After obtaining written permission, all of the patient's demographic information was gathered. Analysis of all patients' tissue specimens. Toxicology and alcohol testing were performed on the blood sample. All cases were evaluated using X-rays and autopsy were carried out for all of them. Decomposed remains were not included in our research.

Results: Among 94 cases, there were 60 (63.8%) males and 30 (36.2%) females. Included patient had mean age 31.45±13.43 years and had mean BMI 22.19±20.48. Frequency of deaths with intracranial lesion was found in 14 (14.9%) cases. We found that frequency of traumatic cases were higher than that of non-traumatic (cerebro-vascular accidents). Frequency of intracranial lesions alone was found in 31 (32.9%) cases. Hemorrhages in the subarachnoid space were the most frequent intracranial pathology. The most prevalent cause of delayed death is pneumonia.

Conclusion: The number of deaths that were caused by cerebral trauma was found to be very low in this research; nevertheless, the bulk of deaths were caused by injuries to other anatomical regions.

Keywords: Trauma, Intracranial lesion, Mortality

INTRODUCTION

TBI (traumatic brain injury) or natural reasons may result in intracranial haemorrhage. Both are possible causes. Traumatic causes include road traffic accidents, falls from height, railway mishaps, assaults, and other accidents. Natural causes include strokes, tumours, and bleeding diseases, among others. Indians die from fatal cerebral haemorrhages per year. Intracranial haemorrhages may occur regardless of the severity of the head injury. Intracranial haemorrhage was found in two-thirds of the patients who presented in coma to Bullock and Teasdale.[1] Any of these may be present at the moment of injury, but they can also develop over time or grow in size. Extra Dural Haemorrhage (EDH) is most often related with skull fractures and occurs at the point of impact in most instances. [2] That which sits between the skull and the dura mater is an ovoid mass of blood clots.[3] Only 5 to 15 percent of fatal head injuries have it, and 85 percent of those injuries had fractures as a co-morbidity. It is very rare. In a study by Rowbotham, these instances accounted for between 3% and 5% of any large series of severe brain trauma[4,5]. As a result, the prevalence of these injuries amongst head traumas in post-mortem rooms is reduced. Trauma is the most common cause of extradural haemorrhage, with the notable exception of blood dyscrasias in infancy. [6]

Non-traumatic (natural) brain lesions may result from elevated intracranial pressure, cerebrovascular illness, metabolic problems, malignancies of the central nervous system (CNS), neurodegenerative diseases, infections of the CNS, and myelin diseases. [7] Intracranial haemorrhages (more than 50% of cases), seizures, neurodegenerative disorders, hypoxic-ischemic lesions with strokes, intracranial tumours, primarily CNS infections, multiple sclerosis and developmental disorders with predominance of the male gender in all age groups except for the neurodegenerative disorders (dementia disorders), which are primarily seen in older adults [8]

Intracerebral haemorrhages, according to Al-Qazzaz, are a common complication of severe brain injuries. Hemorrhages and skull fractures were two of the most common causes of mortality in his research of road traffic accidents. [17] Brain damage may be produced by a direct hit or just by acceleration alone. There are a number of events that may occur in the minutes and days that follow an injury to the brain, on top of the immediate harm. A number of mechanisms, including changes in cerebral blood flow and the pressure inside the skull, contribute significantly to the damage caused by the original injury. [9] An intracranial haemorrhage known as a traumatic intracerebral haemorrhage (TICH) happens when bleeding occurs inside the brain's tissue. Penetrating head trauma is the most common cause of (TICH), however it may also be caused by depressed skull fractures, acceleration/deceleration traum, or a combination of these. [10]. TICH's harmful consequences include a mix of primary damage (the destruction of local tissue caused by the hematoma itself and the associated rise in ICP) and secondary effects induced by the toxic effects of blood on nearby tissue, which are both caused by the procedure. To these processes, we should take into account the impact of internal barotrauma-induced diffuse cerebral fluid percussion damage as well. Increased intracranial pressure (ICP), mass effect, or herniation cannot necessarily explain the changes in awareness that occur following traumatic intracranial haemorrhage (TICH). As previously mentioned, brain alterations that persist for many days following acute (TICH) damage show that there is a prolonged window of opportunity for intervention to enhance neurological prognosis. [11,12]

To better understand the prevalence and forms of cerebral lesions in medical-legal death situations, we undertook the current research.

MATERIAL & METHODS

This interventional comparative study was carried out at Department of Forensic Medicine & Toxicolgy, Peoples University of Medical & Health Sciences for Women (PUMHSW), SBA and Peoples Medical College Hospital (PMCH) Nawabshah, Sindh, Pakistan from 1st October 2021 to 31st March, 2022 and comprised of 94 cases. Medical records and police reports both provided copious amounts of information on each victim, including their age, gender, cause of death, and a long list of other personal details. All patients had their blood drawn for toxicology and alcohol testing using a combination of NaF or KFl and potassium oxalate. Alcohol gas chromatography (ALC) was utilised for the detection of alcohol, while thin layer chromatography (TLC) was employed for toxicological analysis (TLC)

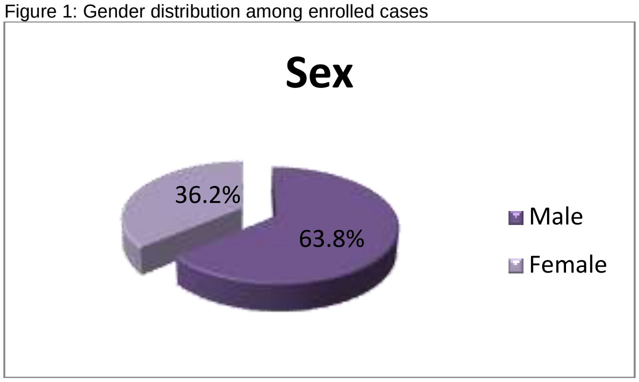
There were a number of different injuries and diseases that need digital imaging. Photographs were taken of injuries and diseases that were fascinating and unusual.

We performed an exterior inspection of the bodies, noting any external injuries, their locations, and the sorts of damage. A thorough classical autopsy, beginning with the head area, was done in certain instances after an X-ray scan. There were no hematomas or other abnormalities seen after removing the bony window from around the brain and meninges. It was necessary to remove the dura mater and extensively check the base of the skull to look for any signs of fractures or a pathological condition in the brain. Following a sagittal and coronal incision of one centimetre slices, the brain was divided into two sections, the cerebellum and cerebrum, to evaluate the ventricles and both white and grey matter; then a single swap movement was used to separate the cerebellum from the cerebrum. Slices of the cerebellum and the brainstem were also studied. Dissection of the chest and abdomen finished the autopsy, revealing any internal organ damage caused by the cerebral disease. SPSS-22.0 was used to analyse the data.

RESULTS

Among 94 cases, there were 60 (63.8%) males and 30 (36.2%) females.(figure-1)

Figure 1: Gender distribution among enrolled cases



Included patient had mean age 31.45±13.43 years and had mean BMI 22.19±20.48. We found traumatic cases were 73 (77.7%), frequency of non-traumatic was 17 (18.1%) and unknown cases were 4 (4.3%). (Table 1)

Table 1: Age and mortality mode among enrolled cases

Variables	Frequency	Percentage
Mean age (years)	31.45±13.43	
Mean BMI (kg/m^2)	22.19±20.48	
Death Mode		
Traumatic	73	77.7
Non-traumatic	17	18.1
Unknown	4	4.3

Frequency of intracranial lesions alone was found in 31 (32.9%) cases. Most common age of intracranial lesion was 20-40 years found in 26 (27.7%) cases and only 5 (5.3%) cases had age >60 years.(table 2)

Table-2: Frequency of intracranial lesions among enrolled cases

Variables	Frequency	Percentage
Intracranial lesion alone		
Yes	31	32.9
No	63	67.1
Age		
20-40 years	26	27.7
>60 years	5	5.3

RTA (road traffic accident) was the most common cause of death, followed by bullet injuries and traumatic wounds in traumatic deaths. And in non-traumatic cases, cerebrovascular diseases were the most common, followed by meningitis, sudden death and tumors. (Table3)

Table 3: Causes of death among traumatic and non-traumatic cases

Variables	Frequency	Percentage
Traumatic		
RTA	35	37.2
bullet injuries	17	18.1
traumatic wounds	10	10.6
Fall from height	7	7.4
Bomb explosion	6	6.4
Total	63	77.7
Non-traumatic		
cerebrovascular diseases	6	6.4
meningitis	4	4.3
sudden death	4	4.3
tumors	3	3.2
Total	17	18.1

Hemorrhages in the subarachnoid space were the most frequent intracranial pathology found in 45 cases. Frequency of complications was found in16 (17.02%). The most prevalent cause of delayed death is pneumonia 8 (50%), myocardial infarction found in 3 (18.6%) cases, gastric ulcer found in 3 (18.6%), gastroenteritis in 1 (6.3%), pancreatitis and Atrophied internal organs found in 1 (6.3%)cases of each.(table 4)

Table-4: Frequency of subarachnoid hemorrhages and complications

Variables	Frequency	Percentage
Subarachnoid hemorrhages		
Yes	45	47.9
No	49	52.1
Complications		
Pneumonia	8	50
myocardial infarction	3	18.6
gastric ulcer	3	18.6
gastroenteritis	1	6.3
pancreatitis and Atrophied internal organs	1	6.3
Total	16	100

As many victims may have been under the influence of benzodiazepines, barbiturates and antiepileptic medicines when they were driving, all toxicological laboratory tests were negative.

DISCUSSION

This research was comparable to a large number of other studies that were conducted in the past since intracranial injuries were discovered in 14.9 percent of the total fatalities. The findings of the research accord with those showing that cerebral lesions account for between 10 and 15 percent of all fatalities. [13,14]

Among 94 cases, there were 60 (63.8%) males and 30 (36.2%) females. Included patient had mean age 31.45±13.43 years and had mean BMI 22.19±20.48. We found traumatic cases were 73 (77.7%), frequency of non-traumatic was 17 (18.1%) and unknown cases were 4 (4.3%).These were comparable to the previous researches.[15,16]

Frequency of intracranial lesions alone was found in 31 (32.9%) cases. Most common age of intracranial lesion was 20-40 years found in 26 (27.7%) cases and only 5 (5.3%) cases had age >60 years. The mean ages of all of the patients were comparable to those found in two earlier studies [17,18] carried out by d'Avella D, Servadei F, et al., and by Al-Qazzaz, Muataz, and Mohammad et all. This was consistent with the findings of the prior research that Chute DJ and Smialek JE et all [19] had obtained.

In the course of our research, the vast majority of instances had severe injuries that required an autopsy, whereas the number of cases involving non-traumatic injuries was far lower. These findings, which showed that men were more likely to be impacted by both traumatic and non-traumatic modes of detah, were consistent with the findings of many other earlier retrospective studies on traumatic brain injuries. [20]

Natural fatalities were reported in a minority of instances, while fatal road traffic occurrences were observed more in frequency in traumatic mode, and these findings were quite comparable to those found in previous research. traumatic mode. [21] As a result of the traumatic method of injury, mixed intracranial and extracranial lesions were detected in the majority of patients. The intracranial group presented with a subarachnoid haemorrhage, which was then followed by an intracerebral haemorrhage. Although there were no fractures observed in half of the patients, skull fractures caused by gunshot injuries were the most prevalent kind that was discovered [11-15]. In this particular research, there were three times as many damage to internal organs as there were overall injuries.

A total of 17.02 percent of patients had complications, with pneumonia accounting for the majority of those who died after a prolonged illness. One explanation is that the patients were in the hospital for a longer period of time and had to rely on parental feeding since they were unconscious and contaminated, making them more susceptible to pneumonia. The most prevalent kind of damage to the head reflects the severity of the trauma (deaths from brain laceration), whereas edoema may be either naturally occurring or the result of trauma. [15] Half of the individuals in our research died instantaneously, which is consistent with earlier findings. [13] There were several situations when victims died on site and others where they were unable to go to a hospital for treatment because of the delay in getting them there. As a consequence of problems such as pneumonia and delayed bleeding after a rupture of a cerebral artery, which may occur following damage to its wall and the creation of post-traumatic aneurysm, the remainder of the patients who died in hospitals perished from complications such as pneumonia. [2] This is consistent with prior research, which found that alcohol and toxicity had only a little influence. [23]

CONCLUSION

In this research, we found that young guys were the most impacted, although both sexes were included in the list of people who died from stress. Accidental injuries were recorded, and subarachnoid haemorrhage was discovered to be the most prevalent pathology, while brain and brainstem lesions were found to be lacerated.

REFERENCE

1. Mason J.K (2000) Third edition, the Pathology of Trauma, London; Edward, Arnold, Closed Head Injury, 191-210.
2. Maloney and Whitmore (1969), Clinical and Pathological Observations in Fatal Head injuriesa 5 year study of 172 cases. British Journal of Surgery 56, 23-51. As cited by Mason J.K (2000) third edition, the pathology of trauma, London; Edward, Arnold, closed Head injury, 191-210.
3. Jamieson, K.G., Yelland, J D. Extra dural Haematoma, report of 167 cases, J. Neurosurgery, 1968. 29: 13-23.
4. Rowbotham, G.F Acute Injuries of Head. 4: 69:80. Edinburgh, Livingston, 1964, cited by Polson, CJ et al 1985, Essentials of Forensic medicine, Oxford: Pergamon press, 4:164.
5. Polson, CJ et al. (1985). The Essentials of Forensic medicine. Oxford: Pergamon press, 4:165.3.
6. Grant W. T (1964) Chronic Extradural hematoma, a case report;Bull. Los angeles. Neurology. As cited by Gradwohl in Trauma to central nervous system and its membranes. Gradwohls Legal Medicine. Bristol: John Wright & sons ltd. Second edition. Page no: 310-312. Forensic Medicine Edinburgh, Churchill Livingston: 1982.p. 250-359.
7. Yilmazlar S, Kocaeli H, Dogan S, et al. Traumatic epidural hematomas of non-arterial origin: analysis of 30 consecutive cases. Acta Neurochir (Wien) 2005;147 :1241–8 discussion 1248.
8. Graham DI, Nicole JAR, Bone I. Editors. Adams and Graham's introduction to neuropathology. 3rd ed. London: Hodder Arnold; 2006 chapter 2.
9. Milders, M.; Fuchs, S., & Crawford, J. R. "Neuropsychological impairments and changes in emotional and social behaviour following severe traumatic brain injury.". Journal of Clinical & Experimental Neuropsychology. 2003;25:157–72
10. Ownsworth, T.; Fleming, J. "The relative importance of metacognitive skills, emotional status, and executive function in psychosocial adjustment following acquired brain injury.". Journal of Head Trauma Rehabilitation . 2005;20:315–32.
11. Langlois JA, Rutland-Brown W, Wald MM. "The epidemiology and impact of traumatic brain injury: A brief overview". Journal of Head Trauma Rehabilitation. 2006; 21:375– 78.
12. Salomone JP, Frame SB."Prehospital care".In Moore EJ, Feliciano DV,Mattox KL. Trauma. New York: McGraw-Hill, Medical Pub.Division..Retrieved 2008-08- 15.2004:117–18.
13. Alexis RJ, Jagdish S, Sukumar S, Pandit VR, Palnivel C, Antony MJ. Clinical Profile and Autopsy Findings in Fatal Head Injuries. *J Emerg Trauma Shock*. 2018;11(3):205-210. doi:10.4103/JETS.JETS_127_17
14. Amitabh S, Meena RN, Vivek S, Nilesh K, Meena LP. Evaluation of the incidence, nature of injury and clinical outcomes of head injuries in North Eastern part of India. *Int J Med Sci Public Health.* 2013;2:287–9.
15. De Silva MJ, Roberts I, Perel P, Edwards P, Kenward MG, Fernandes J, et al. Patient outcome after traumatic brain injury in high-, middle- and low-income countries: Analysis of data on 8927 patients in 46 countries. *Int J Epidemiol.* 2009;38:452–8
16. Ownsworth, T.; Fleming, J. "The relative importance of metacognitive skills, emotional status, and executive function in psychosocial adjustment following acquired brain injury.". Journal of Head Trauma Rehabilitation . 2005;20:315–32.
17. Al-Qazzaz M, Jabor M. Mohammad. Medico-legal study of intracranial causes of death. Egyptian J Forensic Sci 2014; 4. 10.
18. Unterberg AW, Stover J, Kress B, et al. Edema and brain trauma. Neuroscience 2004; 129: 1021–9
19. d'Avella D, Servadei F, Scerrati M, et al. Traumatic acute subdural hematomas of the posterior fossa: clinicoradiologic analysis of 24 patients. Acta Neurochir (Wien) 2003; 145:1037–44.
20. Amit M, Walter VF. Pattern of fatal blunt head injury: A two year retrospective/prospective medico legal autopsy study. *J Indian Acad Forensic Med.* 2010;32:144–9
21. Dahlberg, C.A.; Cusick CP, Hawley LA, Newman JK, Morey CE, Harrison-Felix CL, & Whiteneck GG. "Treatment efficacy of social communication skills training after traumatic brain injury: A randomized treatment and deferred treatment controlled trial.". Archives of Physical Medicine & Rehabilitation. 2007;88:1561–73.
22. MUHAMMAD FAHEEM ASHRAF , MUHAMMAD IMRAN KHAN , MEHAK KHALID , SAMINA KANWAL , SHAMS MUNIR. Frequency of Intracranial Hemorrhages in Medicolegal Death Cases: A Cross Sectional Study. P J M H S Vol. 14, NO. 4, OCT – DEC 2020 949
23. Indian Road Accident Data. *Open Government Data (OGD) Platform India.* [Last accessed on 2017 Jan 24]. Available from: https://www.data.gov.in/keywords/indian-road-accident-data .