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MULTI-COMPARTMENTAL INTRACRANIAL HAEMORRHAGE WITH PNEUMOCEPHALUS FOLLOWING MINOR TRAUMA: A CASE REPORT AND ETHICAL REVIEW

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ABSTRACT Traumatic brain injury (TBI) in elderly individuals is associated with high morbidity and mortality, even trivial trauma. This can be attributed to cerebral atrophy, vascular fragility, and impaired autoregulation mechanisms seen commonly in the elderly. Management decisions are often complicated by poor prognostic indicators and ethical considerations regarding any form of intervention. We report a case of a 70-year-old female with multi-compartmental intracranial haemorrhage and pneumocephalus after a trivial fall. Despite the severity of injuries, which raises the absolute necessity for neurosurgical intervention for this particular patient, caregivers were reluctant to undergo any such intervention, which prompted medical management initially, but in the later stages, the caregivers withdrew their consent for life-saving measures as well. Through this case, we highlighted the severity of multi-compartmental intracranial haemorrhage after low-impact falls in elderly patients, the importance of individualised decision-making, ethical documentation, and family-centred communication in neurotrauma management, especially geriatric neurotrauma.

KEYWORDS :

INTRODUCTION:

Traumatic brain injury (TBI) remains one of the leading causes of morbidity and mortality worldwide. The elderly population is presently an increasingly vulnerable age group, more prone to severe neurotrauma, especially from low-impact incidents. Although ground-level falls are the most common cause of TBI in older adults in Western countries, studies from India have identified road traffic accidents (RTAs) as the predominant mechanism of traumatic brain injury among the elderly population [1]. Older adults are predisposed to TBI due to multiple factors, including chronic medical conditions, prior head injuries, medication side effects, visual impairment, cognitive decline, and balance or gait instability [2]. According to Menon and Girish, the increased severity of traumatic brain injury in this age group is attributed to strong dural adherence, cerebral atrophy, reduced brain compliance, reduced neuronal plasticity, and a higher incidence of cerebral microbleeds [1].

Initial evaluation of elderly patients presenting with traumatic brain injury typically follows standard protocols-Glasgow Coma Scale (GCS) assessment and non-contrast head CT. However, several studies have highlighted that current protocol models often fail to take into account the pre-existing comorbidities and baseline cognitive status, rendering them less accurately predictive for geriatric patients [1,3]. The proportion of elderly individuals requiring intensive care for traumatic brain injury has been steadily rising, reflecting both longer life expectancy and the increasing burden of chronic systemic diseases [3]. Given the variability of this group, predicting outcomes and tailoring management remain critical. Recently, the eTBI (elderly traumatic brain injury) scoring system has shown promise in improving prognostic accuracy for this population [4]. Nevertheless, elderly patients generally exhibit slower recovery and poorer cognitive outcomes compared to younger counterparts, even with optimal rehabilitation [2] and seemingly overall lesser severity of injuries compared to their non-elderly counterparts [5].

Thus, the decision to pursue aggressive neurosurgical intervention should be individualised, taking into account injury severity, pre-existing comorbidities, expected functional outcome, and patient or family preferences. In certain cases, as reported in Canadian studies, mortality among elderly TBI patients has been partly attributed to the withdrawal of life-sustaining treatment following multidisciplinary consensus and family consultation, which contributes significantly to overall mortality among elderly patients with TBI.

This report describes a similar scenario - a case of severe, multi-compartmental intracranial haemorrhage along with pneumocephalus in a 70-year-old woman following a trivial fall (low-impact trauma), where surgical intervention and later, medical life-sustaining measures were declined and the patient's condition was allowed to follow its natural course

CASE REPORT:

A 70-year-old female presented to the Emergency Department with a history of a skid and fall on a wet floor at around 8:30pm previous day, at her residency. The physical examination revealed active bleeding in the right ear and bloody vomitings, stable vitals. The patient was drowsy with a GCS score of 9 (E2V3M4) and sluggishly reacting pupils. Patient attenders were informed about complaints of headache and vomitings.

The patient was managed medically provisionally and admitted for further neurosurgical evaluation. CT scan of the brain showed extradural haemorrhage in the right occipital region, bilateral subdural haemorrhage in the frontal region, hemorrhagic contusions with peri-hemorrhagic oedema noted in the bilateral frontal and right temporal lobes, subarachnoid haemorrhage in the sulcal spaces of the bilateral frontal lobes, right temporal lobe, falx cerebri, and tentorium, pneumocephalus in the right temporal and occipital lobes, and an undisplaced fracture of the occipital bone which warranted consideration for neurosurgical intervention as per standard protocol. However, the patient's caregivers declined any such intervention after comprehensive and thorough counselling. The decision was documented; supportive and conservative management was continued.

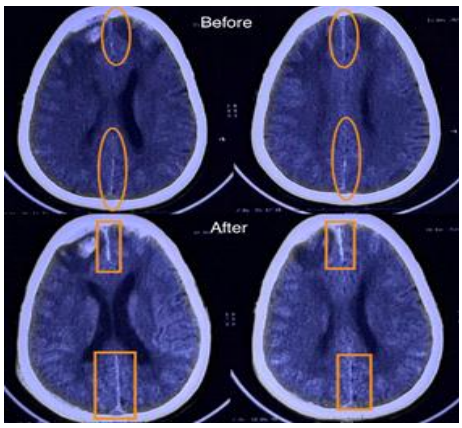


Figure 1: CT brain initial and follow-up respectively showing subarachnoid haemorrhage

The initial CT brain shows subarachnoid haemorrhage (orange circle), which seems to increased in follow CT brain (orange box).

Initial and follow-up CT brain images show bilateral frontal subdural haemorrhage have increased in size.

Figure 2: Initial (green circle) and follow-up (green box) CT brain images showing bilateral frontal subdural haemorrhage

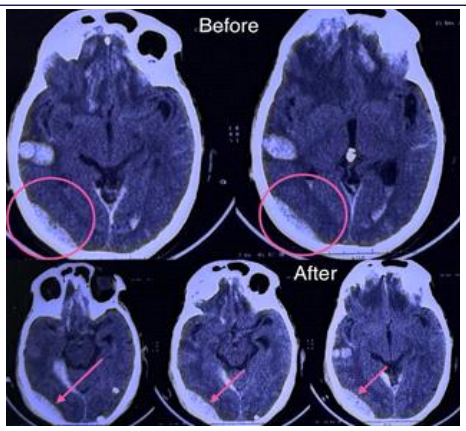
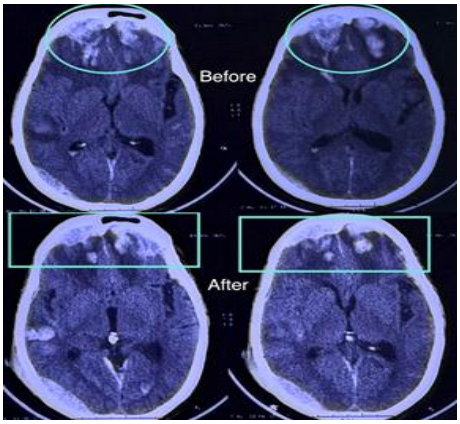
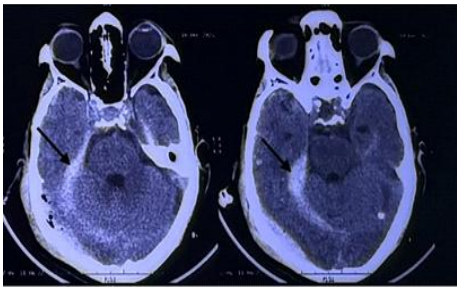


Figure 3: Initial (pink circle) and follow-up (pink arrow)CT brain showing right occipital extradural haemorrhage



Upon comparing the initial and follow-up CT images, we can see the increase in size of the extradural haemorrhage in the follow-up CT image.

Figure 4: CT brain initial (blue circle) and follow up(blue box) showing right temporal haemorrhagic contusion with peri haemorrhagic oedema



The initial CT shows right temporal haemorrhagic contusion with peri haemorrhagic oedema and air foci which are pneumocephalus which are resolved in the follow up CT

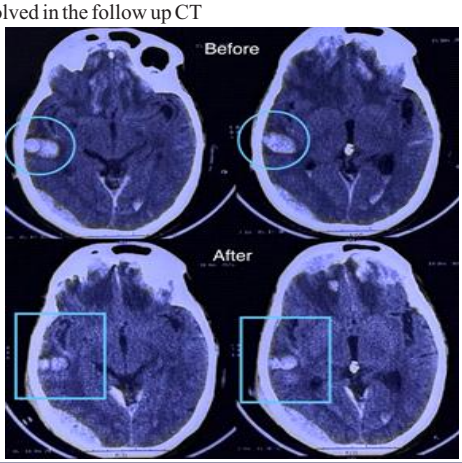


Figure 5: Follow-up CT brain which shows intraventricular haemorrhage (black arrow)

Follow-up CT brain which shows the intraventricular haemorrhage, which weren't present in the initial CT brain, which can be attributed to the expansion of intracranial haemorrhages into the ventricles.

Patient condition was closely monitored. After admission for further monitoring and conservation management, her vitals, evidence of her condition, showed further deterioration— elevated blood pressure, worsening GCS, tachycardia, and decreased blood oxygen saturations, occasional fever spikes. Consistently elevated blood pressure warranted a cardiologist consultation, who advised two-dimensional echocardiography revealing ejection fraction of 58–60%, elevated right ventricular systolic pressure (RVSP), mild pulmonary arterial hypertension (PAH), grade 1 left ventricular diastolic dysfunction, mildly dilated right atrium and ventricle, moderate tricuspid regurgitation (TR), concentric left ventricular hypertrophy (LVH), mild mitral regurgitation, moderate aortic regurgitation, and dilated coronary sinus. Follow-up CT scan showed resolution of pneumocephalus, an increase in the severity of intra-cranial bleeds, and their intraventricular extension.

During the course of conservative management, upon being informed and counselled about the patient's condition, the patient's caregivers refused any further intervention— procedural or otherwise. “Do Not Intubate” and “Do Not Resuscitate” consents were documented. The patient was taken off ventilatory support. Following these measures, the patient's condition took its natural course, and she succumbed to her injuries.

DISCUSSION:

Geriatric traumatic brain injury is a unique concern due to the high prevalence of comorbidities, frailty, and age-related physiological changes that influence injury severity, management, and outcomes [2,3]. Severity of injuries may be deceptively mild, contrary to what is usually and initially perceived as in geriatric TBI cases compared to younger counterparts with TBI [6]. Multi-compartmental intracranial bleeds with pneumocephalus, undisplaced occipital fracture, and de novo hypertension, as seen in our case, illustrate that geriatric TBI cases are more complex than they usually seem— not only prognostically but ethically as well.

Co-morbidities do play a significant role, especially in patients with traumatic brain injury, in determining the outcome of the patients, both long-term and short-term [7]. They may be unrecognised or undiagnosed at the time of presentation of traumatic brain injury, which would negatively contribute to the prognosis of the patient through impaired systemic and cerebral compensatory mechanisms. In this case, two aspects— persistent hypertension during hospitalization and two-dimensional echocardiography changes— support the likelihood of previously undiagnosed chronic hypertension. At the time of presentation, the patient was normotensive, but serial recordings have provided us with sustained elevated readings. Two-dimensional echocardiography findings were compatible with long-standing hypertensive cardiovascular remodelling [8]. Findings of normal ejection fraction, grade 1 left ventricular diastolic dysfunction, and dilated coronary sinus support the chronicity of the hypertension [8]. Thus, not only does 2D echocardiography provide indirect evidence of hypertension, but also of pre-existing hypertensive vasculopathy and cardiopathy, which may have further contributed to the severity and progression of the intracranial injuries. Chronic hypertension results in both structural and microvascular changes within the myocardium and cerebral circulation. These changes impair cerebral autoregulation and reduce vascular compliance, predisposing to multifocal intracranial bleeds and peri-hemorrhagic oedema following even minor trauma [9].

In the elderly, age-related physiological changes are observed, which include cerebral atrophy, vascular fragility, reduced neuronal compliance [10], reduced intracranial compliance and impaired autoregulation mechanisms. Cerebral atrophy increases the subdural space, which in turn increases the brain's mobility within the cranial vault, placing greater tension on cortical bridging veins, rendering them prone to rupture, even with low-impact trauma. It also diminishes neuronal reserve and reparative capacity, resulting in poorer neurological recovery. Reduced intracranial compliance and impaired cerebrovascular autoregulation in the elderly can render even small

haemorrhages clinically catastrophic. As noted by Czosnyka and Pickard (2004) [11], even minor rise in intracranial volume can provoke disproportionate rise in pressures in low-compliance systems (as seen in elderly), explaining the rapid deterioration observed in this patient. Vascular fragility paired with impaired autoregulation mechanisms affects the ability to maintain cerebral perfusion, especially after injury. Chronic hypertension, which eventually stiffens the arteries with age, may have contributed to vascular vulnerability, which in this patient could be observed as multifocal haemorrhages and progressive worsening of the patient's condition. Therefore, the constellation of lesions observed in this patient— epidural, subdural, and subarachnoid hemorrhages with pneumocephalus and an undisplaced occipital fracture— is suggestive of a combination of impact and acceleration-deceleration forces acting on an already structurally vulnerable brain. The epidural hematoma can be attributed to possible disruption of dural vessels at the site of occipital impact, whereas the bilateral frontal subdural and subarachnoid components indicate injury to bridging veins and cortical vessels due to shearing forces. The pneumocephalus noted over the right temporal and occipital lobes can be attributed to dural breach (laceration) at the fracture site caused by reduced cranial compliance, allowing ingress of air into the cranial cavity. The peri-hemorrhagic edema visualized on imaging reflects the cascade of secondary injury due to traumatic brain injury caused by disruption of the blood-brain barrier, ionic imbalance, and cytotoxic oedema. The neuronal and glial senescence seen in the elderly results in a reduced capacity for metabolic recovery. Experimental studies suggest that impaired calcium homeostasis and mitochondrial dysfunction contribute to secondary neuronal injury following TBI. Together, these lesions represent the combined effect of both primary mechanical insult and secondary pathophysiological responses that are amplified in elderly, fragile neural tissue.

Research into prognostic models for geriatric traumatic brain injury has evolved beyond conventional trauma scores. Traditional tools such as the IMPACT and CRASH models incorporate age, GCS motor score, pupillary reactivity, and CT findings and have demonstrated reasonable predictive value for mortality and functional outcome following TBI [12,13]. However, their accuracy may decline in elderly patients with significant comorbidity burden and frailty. Frailty-based approaches that incorporate physiological reserve and pre-injury functional status have demonstrated improved prognostic discrimination in geriatric trauma populations [14,15]. While newer approaches incorporating blood biomarkers show promise, further validation across diverse elderly populations is required before widespread clinical implementation [16].

Postoperative morbidity following neurosurgical intervention for traumatic brain injury is generally higher in elderly patients because of frailty, reduced physiological reserve, and multiple comorbidities. Neurological complications, infections, thromboembolic events, prolonged rehabilitation requirements, and functional dependence are commonly reported and may significantly affect long-term quality of life [17,18,19]. These considerations often influence shared decision-making when discussing aggressive surgical intervention in geriatric neurotrauma.

In this case, however, the decision to forego neurosurgical intervention initially, followed by withdrawal of life-supportive measures, reflects the complexity a geriatric neurotrauma case can pose in the present-day healthcare system— highlighting patient autonomy, surrogate decision-making, anticipated quality of life, socioeconomic limitations and family autonomy. After rigorous counselling regarding the prognosis, expected outcomes, and potential complications of surgical and intensive interventions, the patient's family declined neurosurgical management, pointing to the patient's pre-existing frailty and poor chances of meaningful recovery. This refusal was respected in accordance with ethical standards. Upon further decline in the patient's condition, discussion of the options for continued ventilatory and circulatory support was conducted by the team. The family, fully informed of the existing nature of the patient's condition, requested withdrawal of life-sustaining interventions. Consequently, “Do Not Intubate” and “Do Not Resuscitate” directives were documented. The family elected to forego continuation of supportive care and upon their request ongoing life-sustaining measures were withdrawn, while palliative and comfort measures were maintained.

CONCLUSION

This case throws light on the complexity of managing geriatric neurotrauma cases where comorbidities, physiological limitations, and ethical considerations play important roles in patient management. Age-related physiological changes likely magnify the effects of a minor impact injury, resulting in more severe injuries in a seemingly trivial incident of injury. It underscores the need for an individualised, multidisciplinary approach to geriatric neurotrauma that integrates clinical severity, comorbidity burden, prognostic uncertainty, and ethical considerations into management decisions.

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