Original Research Paper

Volume - 15 | Issue - 12 | December - 2025 | PRINT ISSN No. 2249 - 555X | DOI : 10.36106/ijar

Neurosurgery

POST-TRAUMATIC HYDROCEPHALUS: AN OVERVIEW OF INCIDENCE, PATHOPHYSIOLOGY, DIAGNOSIS, TREATMENT AND NEWER APPROACHES

Hem Chander Pandey* MS (Surgery), MCh, Assistant Professor & Neurosurgeon, Working in Armed Forces, India *Corresponding Author

Jyoti Sharma MD, Pathologist, Working in Armed Forces, India

ABSTRACT Dandy's 1914 paper was the first to identify the syndrome known as post-traumatic hydrocephalus (PTH). Although the exact pathophysiology of PTH is unknown, it is thought to be caused by barriers to the secretion, absorption, and circulation of cerebrospinal fluid (CSF). There is an absence of definitive diagnostic criteria. The criteria and investigations currently in use lack adequate sensitivity and specificity, making it difficult to acquire clear epidemiological data. In this work, we examined the epidemiology, etiology, pathophysiology, diagnosis of PTH and concentrated on its treatment and imaging assessment. A review of literature was also done keeping in mind to historically find out how the ideas regarding the condition and its treatment progressed. This review may offer a standard for diagnosing PTH and serve as a foundation for future PTH prevention and treatment. It also paves way for further research in this area.

KEYWORDS : Post-traumatic Hydrocephalus, Traumatic Brain Injury, Cerebrospinal Fluid

INTRODUCTION

Hydrocephalus is a condition resulting from an excessive accumulation of CSF within the ventricular system. Hydrocephalus can be described as obstructive, communicative, hypersecretory, or of normal pressure[1].The exact cause of PTH remains unclear even though it was first identified in 1914[2]. There is an absence of definitive diagnostic criteria. The criteria and investigations currently in use lack adequate sensitivity and specificity, making it difficult to acquire clear epidemiological data. Moreover, choices for treatment are fairly restricted, and results continue to be subpar. This assessment thoroughly explores the pathophysiological processes, clinical symptoms, epidemiological knowledge, risk determinants, and modern treatment approaches.

Epidemiology

There isn't a unified diagnostic criterion for PTH to be adequately reported; its prevalence ranges from 0.7% to 45%[3].

Risk Factors

Etiology of PTH is not fully understood, but a review of previous studies have identified many risk factors which include higher age, lower Glasgow Coma Scale (GCS) score at admission, intraventricular/subarachnoid hemorrhage (IVH/ SAH), decompressive craniectomy (DC), and meningitis with positive CSF culture[4].

Table 1 : Risk Factors for Post Traumatic Hydrocephalus

Risk Factors for Post traumatic Hydrocephalus
1, Higher Age
2. Lower GCS
3. Severity of Initial Traumatic Brain Injury (TBI)
4. Subarachnoid Haemorrhage
5. Midline Shift
7. Intraventricular Haemorrhage
8. Decompressive Craniectomy
9. Subdural hygroma

Pathophysiology

Primary injury, referred to as direct physical harm to the brain from TBI, can be localized, such as contusions, or extensive, like Diffuse Axonal Injury (DAI). Following the primary injury, secondary injuries can emerge, potentially triggered by excitotoxicity, mitochondrial dysfunction, oxidative stress, or various other elements. PTH can result from several different factors. Clot development following SAH or IVH can block the ventricular system, leading to diminished CSF drainage and consequently causing acute hydrocephalus. Scar tissue in the arachnoid granulations could possibly affect CSF drainage and cause PTH.

Review of Literature

Marmarou was the first to analyze the natural history of intracranial pressure and outflow resistance in patients with post-traumatic ventriculomegaly[5]. Nearly 20 years following Marmarou, De Bonis et al[6] released the initial study on CSF analysis in individuals with post-traumatic hydrocephalus and sought to determine whether a combination of infusion test metrics (initial pressure, outflow resistance, and intracranial elastance index) could assist in identifying shunt responders. In 2017, several Indian authors[7] assessed opening pressure, pressure-volume index, and contrasting patients with normal pressure hydrocephalus against those with post-traumatic hydrocephalus.

Clinical Features

Acute PTH typically presents with clinical signs such as headache, nausea and vomiting, cognitive decline, papilledema, and/or visual deficits. The most frequent headaches occur in the frontal region, as the flow of cerebrospinal fluid decreases when lying flat, leading to increased pain while lying down or upon waking in the morning, which can be alleviated by raising the head. Additionally, nausea and vomiting frequently occur alongside headache, irrespective of where the headache is located. Papilloedema is a key indicator of intracranial hypertension, with visual issues such as diplopia, reduced vision, and vision loss resulting from abducens nerve palsy.

Investigations

Initially, common signs and requirements for PTH include progressive expansion of the ventricular system (with a diameter exceeding 2 mm or an Evans' Index greater than 0.3), enlargement of the frontal angle of the lateral ventricle, enlargement of the temporal horn, rounding of the third ventricle, and in some patients, asymmetric ventricular enlargement[8]. Secondly, certain patients with PTH might exhibit low density surrounding the enlarged ventricular system on head Computed Tomography (CT), or heightened signal of CSF exudation in Magnetic Resonance Imaging (MRI) T2-weighted images, aiding in the diagnosis of PTH; meanwhile, narrowing of cerebral sulci can be useful for differentiating Normal Pressure Hydrocephalus (NPH)[9]. Additionally, other symptoms that may be present include enlarged temporal horns, rounded posterior horns, reduced mamillopontine distance, and flattened cerebral sulcal patterns[10].

Treatment

Pharmacologic

Mannitol and diuretics (Furosemide) that block the production of CSF are the primary medications employed in practice to lower intracranial pressure. Owing to the risk of rebound intracranial hypertension, Mannitol should not be used for more than 72 hours; Dexamethasone at 12–16 mg/day may be administered for 4–6 weeks; and Acetazolamide (100 mg/kg) alongside Frusemide (1 mg/kg) can be continued for as long as one month[11]. The joint use of acetazolamide and furosemide was markedly more effective than acetazolamide by itself in reaching normal ICP. Atorvastatin treatment helps manage hydrocephalus and aids in the recovery of the nervous system[12]. Isosorbide serves as a useful supplementary approach to temporarily manage intracranial pressure prior to shunt placement, but it cannot replace surgical intervention[13].

Surgical

PTH is surgically managed with either an endoscopic third ventriculostomy (ETV) or a ventriculoperitoneal shunt (VPS).

Newer Approaches

Blood that leaks into the ventricular system or subarachnoid space in the early stages of hydrocephalus creates blood clots, which impair normal CSF flow and lead to hydrocephalus. Pang et al.[14] demonstrated the effectiveness of fibrinolytic in a dog model of IVH for the first time in 1986. Research in pig and cat models demonstrated a reduction in ventricular dilation after hemorrhage[15]. On the other hand, a clinical trial indicated that patients undergoing intra-ventricular tissue plasminogen activator (tPA) infusion experienced significant inflammatory reactions. They made an intriguing observation that the intensity of this reaction was linked to the degree of fibrinolysis[16]. It's important to highlight the importance of an older thrombolytic medication, Urokinase.tissue plasminogen activator (uPA)[17], which has demonstrated comparable effects in dissolving clots but with notable enhancements in patient outcomes relative to tPA.

Numerous studies have demonstrated thrombin's role in the initial phase of brain injury after hemorrhage and the development of hydrocephalus after IVH. In a rat model, notable ventricular injury and extensive damage to ependymal cilia were observed after the infusion of thrombin into the ventricles. The treatment of this injury with a protease activated receptor 1 (PAR.1), SCH79797, indicates that PAR.1 might be involved in the development of hydrocephalus[18]. Additionally, research using rat models indicated that thrombin led to an increase in TGF.beta1 expression and significant fibrosis of the subarachnoid membrane, which was fully reversed with the treatment of a TGF beta1 inhibitor[19]. This discovery indicates that thrombin-stimulated TGF-beta1 might contribute to hydrocephalus development after SAH.

Numerous studies have demonstrated the importance of iron and hemoglobin in the development of PTH. A study found that administering hemoglobin intraventricularly led to ventricular enlargement and increased levels of the iron-handling protein lipocalin 2 (LCN2)[20]. The administration of hemoglobin into the ventricles demonstrated considerable ventricular enlargement at 24 hours in rat models, suggesting that hemoglobin contributes to hydrocephalus after intraventricular hemorrhage (IVH)[21].

Therapeutic approaches can also be like antioxidative stress therapy[22] and stem cell transplantation[23] which offer great promise for further study and improved therapeutic results.

Table 2 : Treatment of Post traumatic Hydrocephalus

Treatment of Post traumatic Hydrocephalus		
Pharmacologic	Surgical	Newer Approaches
Mannitol	Ventriculoperitoneal Shunt	Intraventricular Tissue Plasminogen Activator
Frusemide	Endoscopic Third Ventriculostomy	Urokinase Tissue Plasminogen Activator
Acetazolamide		Thrombin
Isosorbide		Iron
Dexamethasone		Haemoglobin
Atorvastatin		Antioxidative Stress Therapy
		Stem Cell Transplantation

CONCLUSION

PTH is a serious complication that has a high rate of mortality and morbidity following TBI. There is no single diagnostic standard for PTH to be properly documented. Recent preclinical studies have significantly advanced our understanding of the pathogenesis and treatment options for PTH. Pharmacologic treatment options include mannitol, Frusemide, Acetazolamide, Atorvastatin and Isosorbide while surgical options include ETV or VP Shunt. Fibrinolytics, Iron, Haemoglobin, Free radicals, antioxidative stress therapy, stem cell transplantation are newer treatment modalities.

Conflict of Interest

The authors have no conflicts of interest to declare. All co-authors have seen and agree with the contents of the manuscript.

REFERENCES

1. Koleva M, De Jesus O. Hydrocephalus. StatPearls. StatPearls Publishing Copyright © 2022, StatPearls Publishing LLC; 2022.
2. Guyot LL, Michael DB. Posttraumatic hydrocephalus. Neurol Res. 2000;22(1):25.28.
3. Svedung Wettervik T, Lewén A, Enblad P. Posttraumatic hydrocephalus—incidence, risk factors, treatment, and clinical outcome. Br J Neurosurg. 2022;36(3):400.406.
4. Tian HL, Xu T, Hu J, Cui YH, Chen H, Zhou LF. Risk factors related to hydrocephalus after traumatic subarachnoid hemorrhage. Surg Neurol 2008;69:241–6; discussion 246.
5. Marmarou A, Foda MA, Bandoh K, et al. Posttraumatic ventriculo megaly: hydrocephalus or atrophy? A new approach for diagnosis using CSF dynamics. J Neurosurg. 1996 Dec;85(6):1026–1035.
6. De Bonis P, Mangiola A, Pompucci A, et al. CSF dynamics analysis in patients with post-traumatic ventriculomegaly. Clin Neurol Neurosurg. 2013 Jan;115(1):49–53.
7. Ramesh VG, Narasimhan V, Balasubramanian C. Cerebrospinal fluid dynamics study in communicating hydrocephalus. Asian J Neurosurg. 2017 Apr-Jun;12(2):153–158.
8. Damasceno, B.P., 2015. Neuroimaging in normal pressure hydrocephalus. Dement Neuropsychol. 9 (4), 350–355.
9. Kim, H., Jeong, E.J., Park, D.H., Czosnyka, Z., Yoon, B.C., Kim, K., Czosnyka, M., Kim, D. J., 2016. Finite element analysis of periventricular lucency in hydrocephalus: extravasation or transependymal CSF absorption? J. Neurosurg. 124 (2), 334–341.
10. Bradley Jr., W.G., 2016. Magnetic resonance imaging of normal pressure hydrocephalus. Semin Ultrasound CT MR 37 (2), 120–128.
11. Rajshekhar, V., 2009. Management of hydrocephalus in patients with tuberculous meningitis. Neurol. India 57 (4), 368–374
12. Zhu, J., Ma, Y., 2022. Atorvastatin treatment in a patient with post-traumatic hydrocephalus: a case report. Brain Inj. 36 (9), 1204–1206.
13. Lorber, J., 1972. The use of isosorbide in the treatment of hydrocephalus. Dev. Med. Child Neurol. (Supplement 27), 87–93.
14. Pang D, Sclabassi RJ, Horton JA. Lysis of intraventricular blood clot with urokinase in a canine model: part 3. Effects of intraventricular urokinase on clot lysis and posthemorrhagic hydrocephalus. Neurosurgery. 1986;19(4):553.572.
15. Brinker T, Seifert V, Dietz H. Subacute hydrocephalus after experimental subarachnoid hemorrhage. Neurosurgery. 1992;31(2): 306.312; Discussion 311.312.
16. Kramer AH, Jenne CN, Zygun DA, et al. Intraventricular fibrinolysis with tissue plasminogen activator is associated with transient cerebrospinal fluid inflammation: a randomized controlled trial. J Cereb Blood Flow Metab. 2015;35(8):1241.1248
17. King NKK, Lai JL, Tan LB, et al. A randomized, placebo.controlled pilot study of patients with spontaneous intraventricular haemor rhage treated with intraventricular thrombolysis. J Clin Neurosci. 2012;19(7):961.964.
18. Gao F, Liu F, Chen Z, Hua Y, Keep RF, Xi G. Hydrocephalus after intraventricular hemorrhage: the role of thrombin. J Cereb Blood Flow Metab. 2014;34(3):489.494.
19. Li T, Zhang P, Yuan B, Zhao D, Chen Y, Zhang X. Thrombin.induced TGF.β1 pathway: A cause of communicating hydrocephalus post subarachnoid hemorrhage. Int J Mol Med. 2013;31(3):660.666.
20. Shishido H, Toyota Y, Hua Y, Keep RF, Xi G. Role of lipocalin 2 in intraventricular hemoglobin.induced brain injury. Stroke Vascular Neurol. 2016.
21. Strahle JM, Garton T, Bazzi AA, et al. Role of hemoglobin and iron in hydrocephalus after neonatal intraventricular hemorrhage. Neurosurgery. 2014;75(6):696.706; Discussion 706.
22. Turgut M, Erdogan S, Ergin K, Serter M. Melatonin ameliorates blood.brain barrier permeability, glutathione, and nitric oxide levels in the choroid plexus of the infantile rats with kaolin.induced hydrocephalus. Brain Res. 2007;1175:117.125.
23. Ahn SY, Chang YS, Sung SI, Park WS. Mesenchymal stem cells for severe intraventricular hemorrhage in preterm infants: phase I dose.escalation clinical trial. Stem Cells Transl Med. 2018;7(12): 847.856.