

## The Use of Vasopressin in the Management of Severe Traumatic Brain Injury: A Narrative Review

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**Abstract :** Introduction: Traumatic brain injury (TBI) is a leading cause of mortality among trauma patients. In the management of TBI, the main principle is avoiding cerebral ischemia, as this is a strong determiner of neurological outcomes. The use of vasoactive drugs, such as vasopressin, has an important role in maintaining cerebral perfusion pressure to prevent secondary brain injury. Current guidelines do not suggest a preferred vasoactive drug to administer in the management of TBI, and there is a paucity of information on the therapeutic potential of vasopressin following TBI. Vasopressin is also an endogenous anti-diuretic hormone (AVP), and pathways mediated by AVP play a large role in the underlying pathological processes of TBI. This creates an overlap of discussion regarding the therapeutic potential of vasopressin following TBI. Currently, its popularity lies in vasodilatory and cardiogenic shock in the intensive care setting, with increasing support for its use in haemorrhagic and septic shock. Methodology: This is a review article based on a literature review. An electronic search was conducted via PubMed, Cochrane, EMBASE, and Google Scholar. The aim was to identify clinical studies looking at the therapeutic administration of vasopressin in severe traumatic brain injury. The primary aim was to look at the neurological outcome of patients. The secondary aim was to look at surrogate markers of cerebral perfusion measurements, such as cerebral perfusion pressure, cerebral oxygenation, and cerebral blood flow. Results: Eight papers were included in the final number. Three were animal studies; five were human studies, comprised of three case reports, one retrospective review of data, and one randomised control trial. All animal studies demonstrated the benefits of vasopressors in TBI management. One animal study showed the superiority of vasopressin in reducing intracranial pressure and increasing cerebral oxygenation over a catecholaminergic vasopressor, phenylephrine. All three human case reports were supportive of vasopressin as a rescue therapy in catecholaminergic-resistant hypotension. The retrospective review found vasopressin did not increase cerebral oedema in TBI patients compared to catecholaminergic vasopressors; and demonstrated a significant reduction in the requirements of hyperosmolar therapy in patients that received vasopressin. The randomised control trial results showed no significant differences in primary and secondary outcomes between TBI patients receiving vasopressin versus those receiving catecholaminergic vasopressors. Apart from the randomised control trial, the studies included are of low-level evidence. Conclusion: Studies favour vasopressin within certain parameters of cerebral function compared to control groups. However, the neurological outcomes of patient groups are not known, and animal study results are difficult to extrapolate to humans. It cannot be said with certainty whether vasopressin's benefits stand above usage of other vasoactive drugs due to the weaknesses of the evidence. Further randomised control trials, which are larger, standardised, and rigorous, are required to improve knowledge in this field.

**Keywords :** catecholamines, cerebral perfusion pressure, traumatic brain injury, vasopressin, vasopressors

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