Section: Anaesthesiology

Original Research Article

TO EVALUATE VASOPRESSIN'S ROLE IN HYPERNATREMIA PATIENTS WITH CLINICAL BRAIN DEATH: A PROSPECTIVE STUDY

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ABSTRACT

Background: Many changes happen in the body when the brainstem doesn't work right. The point of this study was to find out how well intravenous vasopressin treats hypernatremia in people who have been identified with clinical brain death.

Material and Methods: From March 2023 to February 2024, this prospective experimental study was done at the Department of Anaesthesiology, Guntur Medical College, Guntur, Andhra Pradesh, India. It looked at how well vasopressin treated hypernatremia in people who were clinically brain-dead. 20 of these people had too much sodium in their blood.

Results: Vasopressin is a nanopeptide that is broken down in the liver and kidneys. Its half-life is between 10 and 15 minutes. It can be given through an intravenous or intramuscular shot. Desmopressin is a counterpart that works for a longer time and doesn't have as many vasopressor properties. It can be put under the skin, into a vein, or given through the skin. Patients who are brain-dead are affected by vasopressin in two main ways: 1. It helps people who are brain-dead deal with hypernatremia caused by hormonal failure after a posterior pituitary infarction. Organs that could be used for transplantation could be hurt by this situation, especially when the blood sodium level is above 55 mEq/L. The V2 receptor is what makes this action happen.

Conclusion: Using a needle or syringe to put something into the body. Vasopressin infusion is a very good way to treat hypernatremia, an electrolyte imbalance that often happens in brain-dead people and has a core cause.

Keywords: Vasopressin, hypernatremia, brain death, and endocrine dysfunction.

INTRODUCTION

Brainstem disorder changes many of the ways that the body normally works. It's possible that the changes in cell homeostasis will make donated organs less viable, which will make transplants less useful. Brain death can happen when the brain stem stops working properly because of an injury to the brain or a change in the brain's physiology. People in this group are able to give healthy organs to anyone who needs them.[1-3]

By understanding how brain death happens, we can deal with the bad effects in a way that makes the chances of success for the transplanted parts better. Endocrine and parasympathetic dysfunctions are two of the most important changes.[2-4] Center Diabetes insipidus is a common hormonal problem seen in people who are brain-dead. It happens when the posterior pituitary gland doesn't make enough vasopressin, the hormone that controls how concentrated urine is. Hypermagnesemia and high blood osmolality are signs of diabetes insipidus. If your blood sodium level is more than 145 mEq/L, you have hypernatremia. The liver, kidneys, heart, and heart valves are the organs that people give the most. The kidneys, liver, and heart can all be hurt by high blood sugar. When liver grafts from donors have

high amounts of sodium, they are more likely to be rejected during transplantation.[3-5]

When the sodium level goes above 155 mEq/L, bad things are more likely to happen, especially if the hypernatremia lasts for a longer time before the organ is harvested. Several studies have shown that high blood salt levels can affect the success of liver operations. Exogenous vasopressin given through an IV can improve the function of donated organs, especially the liver, when used to treat hypernatremia.[6-8] The goal of our work is to find the target. In this study, blood sodium levels were checked before and after vasopressin therapy was started to treat hypernatremia in patients who were clinically brain-dead and waiting for an apnea test to confirm brain death. Vasopressin raises blood pressure because low blood pressure is typical in people who have died or hurt their brain stem. The point of this study was to find out how well intravenous vasopressin treats hypernatremia in people who have been identified with clinical brain death.

MATERIAL AND METHODS

The Department of Anaesthesiology, Guntur Medical College, Guntur, Andhra Pradesh, India served as the location for this prospective experimental investigation, which was carried out between March 2023 to February 2024. The effectiveness of vasopressin in treating hypernatremia in patients who were clinically brain-dead was investigated in this study. There was an excessive amount of sodium in the blood of twenty of these individuals.

Inclusion Criteria

- Patient with traumatic injury;
- Serum Na+ level greater than 145 meq/L;
- Urine production greater than 4 ml/kg/hr.

Exclusion Criteria

- Individuals with established renal disease
- Drug allergy to the vasopressin class

Study Procedure used

Patients were checked out and given clinical certification of brain death twice, four to six hours apart, after any ward or ICU got information on a patient who was clinically brain dead. In order to rule out other possible causes of unconsciousness, like being drunk, the studies were looked into. The patient's caretakers gave their permission after the inclusion criteria were met and hypernatremia was diagnosed. Following this, vasopressin was started to be given through an IV drip at a rate of 0.01-0.04 U/min. Twenty units of vasopressin are mixed with 500ml of normal saline (NS) to get a dose of 0.04U/ml. As planned, the given traits were looked at at regular times for five and a half hours.

RESULTS

Fifty percent of the people who took part in the study were women and fifty percent were guys. Out of all the cases looked at in this study, only 15% were related to falls from heights. The other 85% were related to car crashes. The amount of sodium in the blood dropped steadily every hour. When they got to the end, there was a clear drop in blood salt levels. The amount of pee made steadily went down. After being steady for 5.5 hours, there was a clear rise in the average blood pressure. There was no statistically significant difference between the controls for blood sodium in men and women. In this trial, the type of damage didn't seem to change the way blood pressure changed or how salt was controlled.

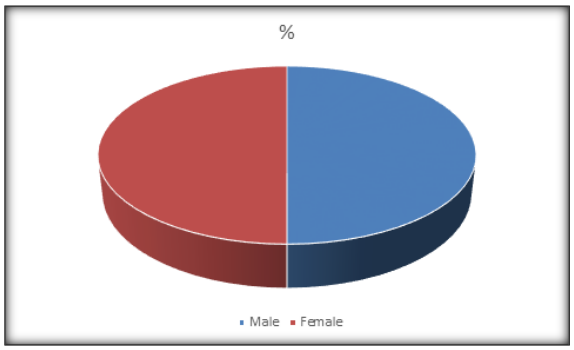

Figure 1: Gender wise distribution

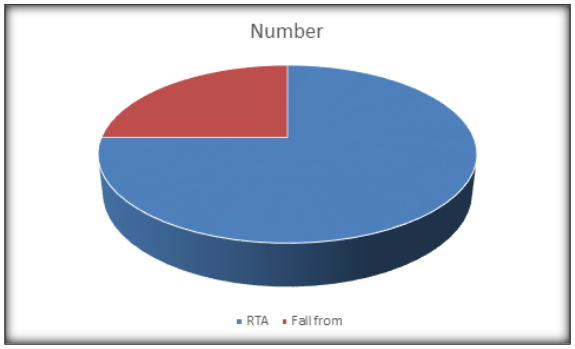

Figure 2: Mode of Injury

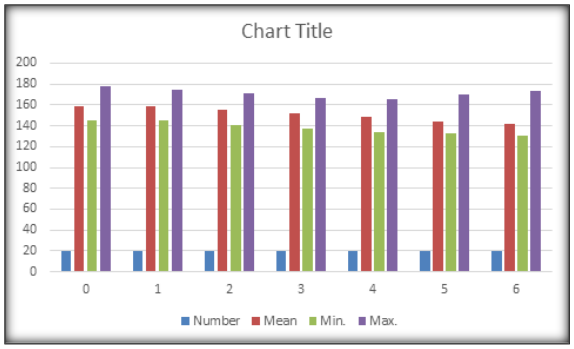

Figure 3: A comparison of the serum sodium levels every hour

Table 1: Gender wise distribution

Sr. No.	Gender	Number	%
1	Male	10	50.0
2	Female	10	50.0
Total	-	20	100

Table 2: Mode of Injury

Sr. No.	Mode of Injury	Number	%
1	RTA	15	75.0
2	Fall from	05	25.0
Total	-	20	100

Table 3: Age wise distribution

	Number	Min.	Max.	Mean
Age (yrs.)	20	16	61.0	30.0

Table 4: A comparison of the serum sodium levels every hour

Serum Na+ (in hrs.)	Number	Mean	Min.	Max.
0	20	159.2	145	178
1	20	159.1	145	174
2	20	155.6	141	171
3	20	152.2	137	167
4	20	148.8	134	166
5	20	144.3	133	170
6	20	141.4	131	173

DISCUSSION

Vasopressin is a nano peptide that is broken down in the liver and kidneys. Its half-life is between 10 and 15 minutes. It is given through an intramuscular or intravenous shot. Desmopressin is a counterpart that works for a longer time and doesn't have as many vasopressor properties. The medicine can be taken by mouth, injected, transdermal, or subcutaneously.[8-10] Vasopressin has two main effects on people who have brain death. To begin, it increases the absorption of free water through the V2 receptor, which helps control hypernatremia caused by hormone failure after a posterior pituitary infarction. If the amount of sodium in the blood is more than 55 mEq/L, hypernatremia is a serious condition that can threaten the health of organs that might be used for donation.[9-11] Brain-dead people can keep or improve their hemodynamic state by controlling vasoconstriction through the V1 receptor. Because of the common autonomic dysfunction that goes along with it, these people are more likely to have hemodynamic instability. Statistical analysis and direct observation both showed that the amounts of sodium in the blood dropped consistently in each hourly sample. At the end of the sixth hour, the salt levels of 31 people had dropped in a way that was statistically significant.[10-12]

The highest amount of sodium that was found was 142.48 mmol/L, and the normal amount of sodium at the start was 160.10 mmol/L. By the end of the sixth hour, the amount of pee that thirty-one people were producing had significantly decreased, and this trend continued every hour. On average, 240.50 L of urine were peed every hour, and 149.88 L were peed every hour on average.[13-15] It's possible that these 31 people have central diabetes insipidus because of a posterior pituitary injury. Nine of the people didn't have a major drop in urine production or serum salt levels, which suggests that their condition may be different and could be a sign of nephrogenic diabetes insipidus. The mean artery pressure, diastolic blood pressure, and systolic blood pressure all show that almost all patients' blood pressure gets a lot better.[16-19]

The way the drugs were given or how they responded to changes in blood pressure, pee output, or serum sodium level did not depend on the person's gender. So, giving a vasopressin infusion for four to six hours caused a significant and conclusive drop in the blood sodium level in patients with hypernatremia who were already brain dead. A big drop in the amount of urine produced.[18-20] It was clear that the hemodynamic state got better or stayed the same because the need for catecholamines slowly went down. The literature review shows that these findings are backed up by other research. What effect does vasopressin have on keeping blood sodium levels in check.[21-23]

Type 2 diabetes having central diabetes insipidus: If the pituitary gland and/or brain are damaged during surgery or an accident, central diabetes insipidus can happen. Brain infiltrations, infections, cerebral aneurysms, tumors in the hypothalamus or pituitary gland, and ischemia in the central nervous system are some of the other causes. Finally, some people may have genetic or idiopathic central DI. For short-term use, vasopressin is recommended, especially when blood pressure is low. Desmopressin should be used for a long time. Desmopressin is a chemical that is related to AVP.[22-24] It has strong effects on stopping urine production while having very little effect on blood vessel relaxation. Nephrogenic diabetes insipidus is a type of diabetes insipidus that has to do with the kidneys. It can be either learned or born with. Some drugs, like lithium, foscarnet, clozapine, and

demeclocycline, can cause nephrogenic diabetes insipidus.[23-25] It can also be caused by diseases like hypercalcemia, hypokalemia, and post-obstructive renal failure. The recommended treatment for nephrogenic DI is to make sure the person stays properly hydrated by drinking enough water. Interestingly, thiazide diuretics can lower the amount of pee that people with nephrogenic diabetes insipidus produce, which is the opposite of what lithium would normally do.[24-26]

CONCLUSION

Highly successful treatment for hypernatremia, an electrolyte imbalance that often happens in brain-dead people, is giving them a vasopressin infusion through an injection. In addition, it lowers the risk of hypotension, which is common in these people. In addition, intravenous fluids with a lot of free water and not much salt are a choice, as well as injectable desmopressin, which does not narrow blood vessels.

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REFERENCES

1. Todd PM, Jerome RN, Jarquin-Valdivia AA. Organ preservation in a brain dead patient: information support for neurocritical care protocol development. Journal of the Medical Library Association. 2007 Jul;95(3):238.
2. Arita K, Uozumi T, Oki S, Kurisu K, Ohtani M, Mikami T. The function of the hypothalamo-pituitary axis in brain dead patients. Acta neurochirurgica. 1993 Mar; 123:64-75.
3. Salim A, Martin M, Brown C, Rhee P, Demetriades D, Belzberg H. The effect of a protocol of aggressive donor management: implications for the national organ donor shortage. Journal of Trauma and Acute Care Surgery. 2006 Aug 1;61(2):429-35.
4. Richardson DW, Robinson AG. Drugs five years later: desmopressin. Annals of internal medicine. 1985 Aug 1;103(2):228-39.
5. Debelak L, Pollak R, Reckard C. Arginine vasopressin versus desmopressin for the treatment of diabetes insipidus in the brain dead organ donor. InTransplantation proceedings 1990 (Vol. 22, No. 2, pp. 351-352).
6. Ralston C, Butt W. Continuous vasopressin replacement in diabetes insipidus. Archives of Disease in Childhood. 1990 Aug 1;65(8):896-7.
7. Keservani RK, Gautam SP. Formulación y evaluación de vesículas de liposomas de baclofeno usando lecitina. Ars Pharmaceutica (Internet). 2020 Sep;61(3):175-80.
8. Rostron AJ, Avlonitis VS, Kirby JA, Dark JH. Hemodynamic resuscitation of the brain-dead organ donor and the potential role of vasopressin. Transplantation Reviews. 2007 Jan 1;21(1):34-42.
9. Ahire ED, Sonawane VN, Surana KR, Talele SG, Talele GS, Kshirsagar SJ, Khairnar SJ, Thombre NA, Mahajan SK. Preventive Measures of Type 2 Diabetes via Nutrition. InThe Metabolic Syndrome 2023 (pp. 71-99). Apple Academic Press.
10. Tiozzo Fasiolo L, Manniello MD, Tratta E, Buttini F, Rossi A, Sonvico F, et al. Opportunity and challenges of nasal powders: Drug formulation and delivery. Eur J Pharm Sci. 2018;113(May 2017):2–17.
11. Khulbe P, Singh DM, Aman A, Ahire ED, Keservani RK. The emergence of nanocarriers in the management of diseases and disorders. Community Acquired Infection. 2023 Apr 19;10.
12. Thombre NA, Niphade PS, Ahire ED, Kshirsagar SJ. Formulation development and evaluation of microemulsion based lornoxicam gel. Biosciences Biotechnology Research Asia. 2022 Mar 31;19(1):69-80.
13. Kleindienst A, Hannon MJ, Buchfelder M, Verbalis JG. Hyponatremia in neurotrauma: the role of vasopressin. Journal of neurotrauma. 2016 Apr 1;33(7):615-24.
14. Sharma VK, Mishra DN, Sharma AK, Keservani RK, Dadarwal SC, Sharma VK. Development and optimization of vitamin E acetate loaded liposomes. Pharmacologyonline Newsletter. 2010; 2:677-91.
15. Ahirrao SP, Sonawane MP, Bhambere DS, Udavant PB, Ahire ED, Kanade R, Kuber D. Cocrystal formulation: a novel approach to enhance solubility and dissolution of etodolac. Biosci. Biotechnol. Res. Asia. 2022 Mar 1;19(1):111.
16. Zhang W, Li S, Visocchi M, Jiang J, Wang X. Natriuretic peptides, antidiuretic hormone and hyponatraemia after acute craniocerebral injury. Journal of International Medical Research. 2008 Jul;36(4):648-55.
17. Garrett RE, Wilhelm S, Maerz G, Bar-Or D. Vasopressin receptor antagonist use in a neurologic rehabilitation center. The Journal of Spinal Cord Medicine. 2010 Jan 1;33(1):65-7.
18. Human T, Onuoha A, Diringer M, Dhar R. Response to a bolus of conivaptan in patients with acute hyponatremia after brain injury. Journal of Critical Care. 2012 Dec 1;27(6):745-e1.
19. Blaine EM, Tallman JR RD, Frolicher D, Jordan MA, Bluth LI, Howie Mb. Vasopressin supplementation in a porcine model of brain-dead potential organ donors. Transplantation. 1984 Nov 1;38(5):459-64.
20. Lathiya MK, Pepperl E, Schaefer D, Al-Sharif H, Zurob A, Cullinan SM, Charokopos A. Vasopressin-induced hyponatremia in an adult normotensive trauma patient: A case report. World Journal of Critical Care Medicine. 2023 Jan 1;12(1):35.
21. Li YQ, Zhang YY, Luo HY, Yan Q, Zhang HY, Zhao X, Wang XH. Effect of total input fluid volume on the prognosis of patients with craniocerebral injury combined with hypernatremia. Int J Clin Exp Med. 2016 Jan 1;9(7):14749-53.
22. Jena SS, Venkatapura RJ, Christopher R, Shukla D, Chakrabarti D. Incidence and Outcomes of Hypernatremia in Adult Neurological Non–Brain-Dead Patients Admitted to Tertiary Care Neurologic Institute: A Retrospective Study. Journal of Neuroanaesthesiology and Critical Care. 2019 Mar;6(01):024-9.
23. Murphy T, Dhar R, Diringer M. Conivaptan bolus dosing for the correction of hyponatremia in the neurointensive care unit. Neurocritical care. 2009 Aug; 11:14-9.
24. Kinoshita Y, Yahata K, Yoshioka T, Onishi S, Sugimoto T. Long-term renal preservation after brain death maintained with vasopressin and epinephrine. Transplant international. 1990 Jan;3(1):15-8.
25. Kolmodin L, Sekhon MS, Henderson WR, Turgeon AF, Griesdale DE. Hypernatremia in patients with severe traumatic brain injury: a systematic review. Annals of intensive care. 2013 Dec; 3:1-7.
26. Langlois PL, Bourguignon MJ, Manzanares W. Hyponatremia in the neurologic patient in intensive care unit: Etiology and treatment. Médecine Intensive Réanimation. 2016 Nov; 25:203-13.