Treatment of subarachnoid haemorrhage complicated by hyponatraemia

Jordyn A Butler

Abstract:

Background statement: Developing hyponatraemia after a subarachnoid haemorrhage is common, however it is known to worsen patient outcomes. This paper aims to review the practice of managing hyponatraemia in acute subarachnoid haemorrhage patients with administration of 3% hypertonic saline solution.

Aim: To enquire into the practice and policy of one of Melbourne's large Metropolitan hospital's current management of hyponatraemia in subarachnoid haemorrhage patients, and determine if the policy is both current and evidenced based.

Methods: A search of the terms "subarachnoid haemorrhage", "hyponatraemia" and "hypertonic saline" was used in databases including Pubmed, Medline and CINAHL. Literature was included if it discussed the use of hypertonic saline for hyponatraemia, the effect of hyponatraemia on subarachnoid haemorrhage patients and the potential causes of acute hyponatraemia. The articles and literature reviews were assessed for inclusion by the author.

Results: Patients with a subarachnoid haemorrhage and hyponatraemia should not be fluid restricted, as this is contraindicated. Patients should be administered 3% hypertonic saline to avoid hypovolaemia and slowly increase serum sodium to prevent onset or exacerbation of cerebral oedema.

Limitation: Lack of evidence based data and studies in regard to the dosing of hypertonic saline resulted in the lack of consensus with prescribing rates and volumes to be infused for severe hyponatraemia.

Key words: *Subarachnoid haemorrhage, hyponatraemia, hypertonic saline*

Introduction:

Aneurysmal subarachnoid haemorrhage can be complicated by acute hyponatraemia in neurosurgical patients. De Oliveira Manoel et al., (2016, p. 1) define aneurysmal subarachnoid haemorrhage (SAH) as *'a complex neurovascular syndrome with profound systemic effects and is associated with high disability and mortality'*. An aneurysmal SAH is the result of cerebral aneurysm rupture or trauma, thus resulting in bleeding in the subarachnoid space. Rupture of cerebral aneurysms commonly occurs at bifurcations and branches within the Circle of Willis (Hickey 2014).

Questions or comments about this article should be directed to Jordyn Butler, Registered Nurse, Austin Health Melbourne.
Jordynbutler@hotmail.com

DOI: 10.21307/ajon-2017-002

Patients with a SAH commonly develop hyponatraemia within two weeks of cerebral rupture (Vrsajkov, Javanovic, Stanisavljevic, Uvelin, & Vrsajkov, 2012). Hyponatraemia is the most common electrolyte abnormality to develop in patients with a SAH. It is defined by Hickey (2014, p. 203) as *'serum sodium less than 135mEq/L'.* High-grade SAH patients with anterior circulation aneurysms have a 50% incident rate of developing acute hyponatraemia (De Oliveira Manoel et al., 2016), yet the pathophysiology linking SAH and hyponatraemia is not fully understood (De Oliveira Manoel et al., 2016; Manzanares, Aramendi, Langlois & Biestro, 2014; Mapa et al., 2016; See, Wu, Lai, Gross, & Du, 2016).

This enquiry into practice focuses on the treatment and management of hyponatraemia in SAH patients using hypertonic saline, with a structured discussion and analysis of current evidence based practice. The following will be explored; importance of a

high standard of care, a review of local policy relevant to SAH and hyponatraemia management and the impact on future nursing practices including a need for increased education. For this paper, the hospital has been de-identified, the reviewed policy is from a large metropolitan hospital with a 32 - bed ward with twelve dedicated neurosurgical beds including a 4 - bed neurosurgery high dependency unit (HDU).

Pathophysiology of SAH and hyponatraemia:

According to See et al. (2016) 30% of SAH patients develop hyponatraemia 1-week post rupture. Hannon et al., (2014) report 56% of patients admitted with SAH will develop hyponatraemia during their hospital admission. Due to the significant percentage of patients that develop hyponatraemia post rupture, these patients are at high risk for deterioration. It is crucial to observe for complications and symptoms associated with both SAH and hyponatraemia, with care provided being in accordance to current evidence based practice and local hospital policies. Hyponatraemia can occur in patients with a SAH due to syndrome of inappropriate anti-diuretic hormone (SIADH), cerebral salt wasting (CSW), glucocorticoid insufficiency and excessive use of diuretics (Hannon et al., 2014; Saramma, Menon, Srivastava & Sarma, 2013; See et al., 2016; Walcott, Kahle & Simard, 2012). According to Vrsajkov et al., (2012) & Mapa et al., (2016) the location of the aneurysm could potentially influence the patient's risk of developing hyponatraemia as aneurysms that rupture within the anterior circulation can affect the hypothalamic-pituitary region of the brain, consequently resulting in SIADH or CSW. SAH can lead to either an increase in secretion of antidiuretic hormone (ADH) causing SIADH or CSW due to the enhanced release of atrial natriuretic peptide, brain natriuretic peptide and noradrenaline. SIADH and CSW are fundamentally different conditions that can be difficult for clinicians to differentiate in regards to treatment (De Oliveria Manoel et al., 2016). It is crucial to initiate treatment targeted to the correct aetiology to ensure serum sodium levels are corrected appropriately; with a treatment plan reflective of the clinical situation and consideration of potential adverse effects (Ball & Iqbal, 2015; Hannon et al., 2014). Hyponatraemia can be life threatening if incorrectly treated and managed.

Intracellular and extracellular osmolarity must be equal. If there is a low serum sodium level, cells will begin to swell as fluid moves from the extracellular compartment to the interstitial fluid, resulting in intracellular oedema due to changes in osmolarity. When hyponatraemia develops rapidly the brain can be slow to adapt to the hypotonic environment (Mapa et al., 2016; Spasovski et al., 2014; Verbalis et al., 2013). When low serum sodium levels are over corrected too rapidly it can cause blood brain barrier breakdown and injury to myelin in the central nervous system, precipitating osmotic demyelination syndrome (Ball & Iqbal, 2015; Sterns, Hix & Silver, 2010; Verbalis et al,. 2013). Acute symptomatic hyponatraemia secondary to a SAH can have severe complications, with symptoms including cerebral oedema, seizures and cerebrovascular spasm (Saramma et al., 2013). Patient outcomes can vary significantly from a full recovery to severe disability or death post SAH, depending on severity of the bleed and associated complications (De Oliveira Manoel et al., 2016).

Nurses must accurately assess patients for changes in Glasgow Coma Scale (GCS) score and neurological condition. Whilst concurrently observing for signs and symptoms related to acute hyponatraemia including headache, nausea and vomiting, the nurse must be aware that the patient's condition can rapidly deteriorate leading to confusion, seizures, respiratory arrest and severe cerebral oedema resulting in death (Rafat et al., 2014; Verbalis et al., 2013).

TABLE 1: Serum sodium levels

This table lists serum sodium ranges of hyponatraemia and associated symptoms (Stern, 2015).

Hyponatraemia	Serum sodium range	Symptoms
Mild hyponatraemia	130-135 mmol/L	Nausea, vomiting, short-term memory loss & dizziness.
Moderate hyponatraemia	121-129 mmol/L	Confusion, muscle weakness, generalised malaise & headaches.
Severe hyponatraemia	<120 mmol/L	Lethargy, agitation, increased ICP, respiratory depression, cerebral oedema & disorientation.

Treatment of hyponatraemia:

Treatment and management of acute hyponatraemia in SAH patients should comply with hospital local policies, in conjunction with global evidence-based practice. The local policy at this hospital for 'IV infusion of hypertonic saline for hyponatraemia management'

has recently been reviewed and updated based on current evidence-based journal articles. Although medical professionals are prescribing the treatment, it is nurses administering the medication therefore it is paramount nurses administering hypertonic saline for severe hyponatraemia have sufficient knowledge and understanding of the high-risk infusion. According to See et al., (2016) the proportion of patients that developed hyponatraemia post clipping or coiling of an aneurysm, was almost equal. Hannon et al., (2014) also reiterated there was no difference in the incidence of hyponatraemia based on patients that had an aneurysm clipping or endovascular coiling. It is understood hyponatraemia may develop in response to hypothalamic injury as a result of SAH, consequently leading to aforementioned complications (Dority & Oldham, 2016; Vrsajkov et al., 2012). Due to increased renal reabsorption of free water in SIADH, fluid restriction is considered the gold standard of treatment. However, treatment of SIADH with fluid restriction in the setting of SAH is contraindicated and potentially detrimental to patient outcomes due to the increased risk of hypovolaemia-associated cerebral infarct and worsening vasospasm (De Oliveira Manoel et al., 2016; Hickey, 2014; Manzanares et al., 2014; Saramma et al., 2013; Walcott et al., 2012). Management of hyponatraemia in patients with a SAH includes preventing hypovolaemia and administration of isotonic fluid to prevent onset or exacerbation of cerebral oedema (De Oliveira Manoel et al., 2016; Raya & Diringer, 2014).

Hypertonic Saline:

The local hospital policy recommends for acute symptomatic hyponatraemia, an IV 3% hypertonic saline bolus of 100-250ml over 10 -20 minutes to correct low serum sodium levels, aiming for a sodium increase of 5mmol/L. The bolus can be repeated twice at 10-minute intervals if serum sodium remains unchanged (Adrogue & Madias, 2012; Verbalis et al., 2013; Grant et al., 2015; Spasovski et al., 2014). Starke & Dumont (2014) discuss the effects of hypertonic saline, due to its ability to move fluid from the interstitial and intracellular spaces via osmotic gradient into the intravascular system, thus reducing associated symptoms. The administration of hypertonic saline in SAH patients has been shown to increase arterial blood pressure, cerebral perfusion pressure and flow velocity whilst simultaneously reducing intracranial pressure and cerebral oedema (Starke & Dumont, 2014; Thongrong et al., 2014; Walcott et al., 2012). It is crucial to re-check serum sodium levels and urine osmolarity simultaneously post IV bolus and then repeat 2-4 hours post administration. To ensure accurate interpretation of values urine osmolarity and bloods should be taken at the same time (Spasovski et al., 2014). According to Adrogue & Madias (2012) 2-4 hourly neurological observations including GCS and vital signs should be performed, as well as serum sodium and urine electrolytes post-hypertonic saline administration, to ensure rapid overcorrection has not occurred. As per the reviewed hospital policy, patients administered with hypertonic saline require continuous cardiac monitoring and pulse oximetry in the HDU. In addition, an indwelling urinary catheter is inserted for accurate fluid balance due to the potential for large diuresis and this ensures the ability to obtain frequent urine osmolarity samples. Due to the high risks associated with hypertonic saline administration for SAH patients with severe symptomatic hyponatraemia, patients should not be left unattended whilst receiving the IV infusion. It can be observed that hospital policies are medical based, and can often lack guidance towards nursing practice and responsibilities. This signifies the need for policy change in conjunction with further nursing education for managing acute symptomatic hyponatraemia patients.

Hypertonic saline indications, infusion rates and target sodium concentrations have been described by Spasovski et al., (2014) as unclear, which can be challenging for nurses when prescribed and administered to patients. Currently there are no consensus guidelines for optimal concentration, infusion rates and dose. This is debated both in Australia and internationally regarding the administration and dosage of hypertonic saline for acute hyponatraemia, and remains an ongoing area of research due to inconsistencies in clinical recommendations. However evidence -based clinical practice guidelines are utilised to provide recommendations for clinically appropriate treatment and pathology testing (Nagler et al., 2014; Starke & Dumont, 2014; Thongrong et al., 2014). A review of current evidence-based practice at this hospital indicated that this local policy is in compliance with research findings, where a senior neurosurgical registrar decides rate and dosage for SAH patients.

Despite hypertonic saline improving symptoms associated with hyponatraemia and raising sodium levels, it can result in side effects including hypernatraemia, hypokalaemia and acute renal failure (Manzanares et al., 2014). It is important nurses administering hypertonic saline are aware of these side effects especially to observe for hypernatraemia, as rapid changes in serum sodium can have detrimental and permanent

neurological effects on the patient. Hence, serum sodium should not rise more than 10mmol/L within 24 hours and 18mmol/L within 48 hours to prevent osmotic demyelination syndrome (Ball & Iqbal, 2015; Grant et al., 2015; Rafat et al., 2014; Sood, Sterns, Hix, Silver, & Chen, 2013). Osmotic demyelination syndrome typically occurs 2-7 days post treatment and is clinically characterised by irreversible neurological damage (Sood et al., 2013). Manzanares et al., (2014, p. 236) reports, *'…in SAH, triple H therapy (hypertension, hypovolaemia and haemodilution) as an anti-vasospasm strategy promotes natriuresis and the risk of hyponatraemia'.* Thus reiterating the contraindication of fluid restricting SAH patients, as hypovolaemia and a negative fluid balance will worsen patient outcomes. Therefore utilising 3% hypertonic saline is the preferred treatment (Dority & Oldham, 2016).

Impact on future nursing practice:

It is imperative for nurses and clinicians to reflect upon current practices, challenge nursing interventions and management. Reflecting on current hyponatraemia management ensures the care provided to patients is based on current published research, whilst continuing to have a diagnostic approach in regards to accurate interpretation of serum sodium values. It was observed by McKeever et al., (2016, p. 85) that, '*delivering evidence-based nursing care contributes to improved patient outcomes, a superior quality of care, and potential cost efficiencies'.* Nurses can feel empowered and engaged when given the opportunity to contribute to improvements in nursing care and future practices within their speciality area, therefore improving the quality of care provided to patients. Although the local neurosurgery policy reflects current evidence based practices, there is a need for further nursing education. This is due to the infusion being high risk and may not be commonly administered in HDU settings, rather in the Intensive Care Unit. Increasing education will ensure nurses caring for SAH patients whom are at risk of developing hyponatraemia have a comprehensive knowledge of the condition, appropriate treatment as well as identifying signs and symptoms of deterioration. In conjunction, awareness of the risk factors of osmotic demyelination syndrome when administering hypertonic saline, such as malnutrition, liver disease and hypokalaemia (Rafat et al., 2014). Hyponatraemia can often lead to increased length of hospital stay, increased costs and associated complications, consequently highlighting the need for close monitoring of sodium levels and implementing appropriate treatment to reduce morbidity and mortality (Ball & Iqbal, 2015; Hannon et al., 2014; See et al., 2016). Increased knowledge of risk factors associated with SAH and hyponatraemia include advanced age, smoking, re-bleeding and cerebral vasospasm. This knowledge may prove to be vital in monitoring for sodium changes post – rupture in patients with pre-existing risk factors (Saramma et al., 2013).

Conclusion:

In conclusion, enquiry into practice is vital in continuing to develop and improve upon professional nursing practice whilst maintaining a high standard of evidence-based care. The incidence of SAH patients developing hyponatraemia is 50%, illustrating the importance of close monitoring of serum sodium concentrations and associated symptoms. Neurosurgical patients that become hyponatraemic during inpatient admission can result in increased length of hospital stay, increased morbidity and mortality, signifying the importance of monitoring serum sodium levels promptly, as well as for prevention of clinical consequences that can occur with untreated acute symptomatic hyponatraemia, such as cerebral oedema and increased intracranial pressure.

It can be concluded the reviewed local hospital policy for the monitoring, management and treatment of acute symptomatic hyponatraemia with administration of 3% hypertonic saline is in accord with current evidence based practice and global standard of care. However currently there remains a lack of consensus regarding the dosage and infusion rate of hypertonic saline, thus referring to clinical practice guidelines for recommendations in regards to treatment and management with SAH patients. Therefore to improve upon future local practice, increased nursing education is needed to be able to identify and determine signs and symptoms associated with hyponatraemia, monitoring for changes in serum sodium levels and in a timely manner escalating this to medical teams for appropriate intervention that is reflective of the clinical situation. Reflection into clinical practices and local policies proves invaluable to patient outcomes.

Acknowledgements:

This paper was completed as course work for the Graduate Certificate of Neuroscience, University of Tasmania

References:

Adrogue, HJ & Madias, NE. (2012). The challenge of hyponatraemia. *Journal of the American Society of Nephrology,* 23(7), 1140-1148.

Ball, SG & Iqbal, Z. (2016). Diagnosis and treatment of hyponatraemia. *Best Practice & Research Clinical Endocrinology & Metabolism,* 30(2), 161-173.

De Oliveira Manoel, AL, Goffi, A, Marotta, TR, Schweizer, TA, Abrahamson, S & Macdonald, RL (2016). The critical care management of poor-grade subarachnoid haemorrhage. *Critical care,* 20 (21), 1-19.

Dority, JS & Oldham, JS. (2016). Subarachnoid Hemorrhage: An Update. *Anesthesiology Clinics,* 34, 577 – 600.

Grant, P, Ayuk, J, Bouloux, PM, Cohen, M, Cranston, I, Murray, RD, Rees, A, Thatcher, N & Grossman, A (2015). The diagnosis and management of inpatient hyponatraemia and SIADH. *European Journal of Clinical Investigation,* 45(8), 888-894.

Hannon, MJ, Behan, LA, O'Brien, MMC, Tormey, W, Ball, SG, Javadpour, M, Sherlock, M & Thompson, CJ 2014. Hyponatraemia Following Mild/Moderate Subarachnoid Haemorrhage is due to SIAD and Glucocorticoid Deficiency and not Cerebral Salt Wasting. *Journal of Clinical Endocrinology and Metabolism,* 99(1), 291-298.

Hickey, JV (2014). *The Clinical Practice of Neurological and Neurosurgical Nursing* 7th edn. Philadelphia: Lippincott Williams & Wilkins.

Manzanares, W, Aramendi, I, Langlois, PL & Biestro, A (2014). Hyponatraemia in the neurocritical care patient: An approach based on current evidence. *Medicina Intensiva,* 39(4), 234-243.

Mapa, B, Taylor, BES, Appelboom, G, Bruce, EM, Claassen, J & Connolly, ES (2016). Impact of Hyponatraemia on Morbidity, Mortality, and Complications after Aneurysmal Subarachnoid Hemorrhage: A Systematic Review. *World Neurosurgery*, 85, 305- 314.

McKeever, S, Twomey, B, Hawley, M, Lima, S, Kinney, S & Newall, F (2016). Engaging a Nursing Workforce in Evidence – Based Practice: Introduction of a Nursing Clinical Effectiveness Committee. *Worldviews on Evidence – Based Nursing*, 13(1), 85-88.

Nagler, EV, Vanmassenhove, J, van der Neer, SN, Nistor, I, Van Biesen, W, Webster, AC & Vanholder, R (2014). Diagnosis and treatment of hyponatraemia: a systematic review of clinical practice guidelines and consensus statements. *BMC Medicine,* 12(231).

Rafat, C, Schortgen, F, Gaudry, S, Bertrand, F, Miguel-Montanes, R, Labbe, V, Ricard, JD, Hajage, D & Dreyfuss, D (2014). Use of Desmopressin Acetate in Severe Hyponatraemia in the Intensive Care Unit. *Clinical Journal of the American Society of Nephrology,* 9, 229-237.

Raya, AK & Diringer, MN (2014). Treatment of Subarachnoid Hemorrhage. *Critical Care Clinics,* 30(4), 719 – 733.

Saramma, P, Menon, RG, Srivastava, A & Sarma, PS (2013). Hyponatraemia after aneurysmal subarachnoid haemorrhage: Implications and outcomes. *Journal of Neurosciences in Rural Practice,* 4(1), 24-28.

See, AP, Wu, KC, Lai, PMR, Gross, BA & Du, R (2016). Risk factors for hyponatraemia in aneurysmal subarachnoid haemorrhage. *Journal of Clinical Neuroscience,* 32, 115- 118.

Sood, L, Sterns, RH, Hix, JK, Silver, SM & Chen, L (2013). Hypertonic saline and desmopressin: A simple strategy for safe correction of severe hyponatraemia. *American Journal Kidney Diseases*, 61(4), 571-578.

Spasovski, G, Vanholder, R, Allolio, B, Annane, D, Ball, S, Bichet, D, Decaux, G, Fenske, W, Hoorn, EJ, Ichai, C, Joannidis, M, Soupart, A, Zietse, R, Haller, M, van deer Veer, S, Biesen, WV & Nagler, E (2014). Clinical practice guideline on diagnosis and treatment of hyponatraemia. *Nephrology Dialysis Transplantation*, 29, 1-39.

Starke, RM & Dumont, AS (2014). The role of hypertonic saline in Neurosurgery. *World Neurosurgery,* 82(6), 1040-1042.

Sterns, RH. (2015). Overview of the treatment of hyponatraemia in adults. *Up To Date,* last updated April 6th, 2015. Retrieved from <http://www.uptodate.com/contents/overview-of- thetreatment-of-hyponatremia-in-adults?source=search_result&search=overview+of+the+treatment+of+hyponatremia+in&se lectedTitle=1%7E150>

Sterns, RH, Hix, JK & Silver, S. (2010). Treatment of Hyponatraemia. *Current opinion in Nephrology and Hypertension*, 19(5), 493-498.

Thongrong, C, Kong, N, Govindarajan, B, Allen, D, Mendel, E & Bergese, SD (2014). Current purpose and Practice of Hypertonic saline in Neurosurgery: A review of the literature. *World Neurosurgery,* 82(6), 1307-1318.

Verbalis, JG, Goldsmith, SR, Greenberg, A, Korzelius, C, Schrier, RW, Sterns, RH & Thompson, CJ (2013). Diagnosis, Evaluation and Treatment of Hyponatremia: Expert Panel Recommendations. *The American Journal of Medicine,* 126 (10A), 12-30.

Vrsajkov, V, Javanovic, G, Stanisavljevic, S, Uvelin, A & Vrsajkov, JP (2012). Clinical and Predictive Significance of Hyponatraemia after Aneurysmal Subarachnoid Hemorrhage. *Balkan Medical Journal,* 29, 243-246.

Walcott, BP, Kahle, KT and Simard, JM (2012). Novel treatment Targets for Cerebral Edema. *Neurotherapeutics,* 9, 65-72.