International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

Management Prolong Dehydration Due to Water Depletion in Elderly Patient

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Abstract: ***We present a case of a 60yearold man who was admitted to the emergency room at Wangaya hospital with symptoms of weakness and dizziness over 3 weeks, indicative of malnutrition and dehydration. The patient was diagnosed with hypovolemic shock due to long term dehydration, acute kidney injury, and hypernatremia. This case study highlights the importance of early detection and treatment of dehydration to prevent severe complications such as hypernatremia, especially in elderly patients.***

Keywords: prolong dehydration, hypovolemic, dehydration

1. Introduction

A decrease in intravascular volume and a consequent decline in preload define hypovolemic shock. The body makes up for the volume loss by raising heart rate, stroke volume, oxygen extraction rate, and later by increasing 2,3-diphosphoglycerate concentration and shifting the oxygen dissociation curve to the right.

Sodium is a dominant cation in extracellular fluid and necessary for the maintenance of intravascular volume. By concentrating urine as a result of antidiuretic hormone (ADH) action and increasing fluid intake as a result of a potent thirst response, the human body maintains sodium and water homeostasis. Some vulnerable populations, vasopressin insufficiency, or renal tubular non-response hinder these processes to prevent the development of hypernatremia. Hypernatremia is defined as a serum sodium concentration of greater than 145 meq/L.

The general press claims that 75% of Americans suffer from chronic dehydration. Dehydration in older patients is prevalent, despite the fact that medical research does not support this. According to reports, it affects 17% to 28% of older persons in the US. Hospital admissions are frequently caused by dehydration. It complicates numerous medical problems and has the potential to increase morbidity and mortality on its own. Additionally, dehydration may be over diagnosed. This may result in an incorrect diagnosis of the patient's disease and excessive fluid administration. Both prevention and treatment of dehydration are simple. Dehydration's causes and diagnosis should be thoroughly understood to provide better patient care.

2. Case Report

60 years old man was delivered to the emergency room at Wangaya hospital with chief complain feel weak and dazed over 3 weeks. Patient was neglected with condition difficult to communicated, weak, malnutrition, had wound almost all over his body especially on his left body and the skin look like peel off and stiffness in his arms and legs. Patients were nursing at one foundation at bali before. Another complaint like nausea, vomiting, abdominal pain were denied. Past medical history was stroke, past medication was unknow.

On physical examination, patient's consciousness was compos mentis, blood pressure 90/69mmHg, heart rate 144x/menit, respiratory rate 22x/menit, axillary temperature 38, 7 with 95% oxygen saturation. There are boy eye, dry mouth, increase bowel movement, skin turgor return very slowly, damage skin more than 50% with skin loss.

From the laboratory examination, WBC 10.700/µL, Hb 18, 0 g/dL, Hematokrit 56, 4% SGOT 163, SGPT 186, Ur 179mg/dL, Cr 1, 7mg/dL, Natrium 145mmol/L, kalium 5, 0mmol/L, Klorida 121mmol/L, random blood sugar 100.

The ECG result obtained sinus tachycardia with a heart rate of 131 beats/minute.

From the examination result, patient was diagnosed with shock hypovolemic ec long dehydrationec skin loss and soft tissue infection, Acute kidney injury, Hypernatremia

Treatment given to patients as conservative therapy includes an infusion of Dextrose 5% first loading 1000cc then maintain 30 drops per minute, erythromycin 3x500mg (PO), esomeprazole 1x40mg (iv), ondancetron 3x4mg (iv), paracetamol 3x500mg (PO), curcuma 3x1 tablet

3. Discussion

This patient was diagnosed with shock hypovolemic ec long dehydration ec skin loss and soft tissue infection, Acute kidney injury, Hypernatremia.

Hypovolemic shock is a potentially life-threatening condition. Early detection and effective treatment are crucial. Circulatory failure brought on by an effective intravascular volume loss (of fluids or blood) is known as hypovolemic shock [8,9].

Non-hemorrhagic, on the other hand, results from bodily fluid loss and a decreased effective intravascular volume. Hemorrhagic shock is most frequently caused by trauma, by far. The gastrointestinal (GI), genitourinary, and bleeding

following surgical intervention are other causes of hemorrhagic shock[8].

Volume resuscitation must begin right away for individuals who are experiencing non-hemorrhagic hypovolemic shock in order to replenish effective circulatory blood volume. It might be challenging to identify the specific kind of fluid loss. In order to swiftly restore tissue perfusion, it is advisable to start with a warm, isotonic crystalloid solution of 30 ml/kg body weight. You may make this blouse more than time. Heart rate, blood pressure, urine output, mental state, and peripheral edema can all be used to assess effective resuscitation. As previously mentioned, there are several methods for determining fluid responsiveness, including pulse pressure variation, central venous pressure monitoring, and ultrasonography to evaluate IVC compressibility. Hypovolemic shock shouldn't be treated with vasopressors since they might impair tissue perfusion [8,9].

For severe volume depletion that is not the result of bleeding, crystalloid fluid resuscitation is favored over colloid solutions. Depending on the patient's lab results, the predicted volume of resuscitation, the acid-base state, and the preferences of the treating physician or institution, the type of crystalloid used to revive the patient can be customized. Large doses of isotonic saline might cause hyperchloremic metabolic acidosis during resuscitation because it is hyperchloremic in comparison to blood plasma. Other isotonic liquids with lower chloride contents are available, including lactated Ringer's solution and IV electrolyte replacement products. These solutions are frequently described as balanced crystalloids or buffered crystalloids. According to some research, using balanced crystalloids and restricting chloride may help patients who require large volume resuscitation avoid kidney damage. In comparison to colloid solutions, crystalloid ones are both more cost efficient and similarly effective. Albumin or hyperoncotic starch based colloid solutions are often utilized. Studies looking into albumin solutions for resuscitation have not demonstrated improved outcomes, while other studies have demonstrated that resuscitation with hyperoncotic starch results in higher mortality and renal failure [8,9].

As is known, excess solute and water shortage are the two main causes of hypernatremia. The most frequent cause of hypernatremia is total body water loss relative to solute loss. Hypernatremia is frequently accompanied by hypovolemia, which can happen when there is coupled water and solute loss, free water loss, or water loss that is larger than salt loss[6]. Through the skin, lungs, kidneys, and GI tract, body water is lost. Dehydration is brought on by a loss of bodily fluids without sodium. The epidermis, lungs, digestive system, and kidneys all lose water. When body water losses surpass water replacement, dehydration occurs. It might result from failing to replace necessary water losses[4, 7].

About 55 to 65 percent of the human body is water. That water is divided into two parts: an intracellular portion and an external portion. Extracellular water contains a fifth intravascular water. To sustain euvolemia, the body has a sophisticated system in place. Through the digestive system, water is absorbed. Osmoreceptors in the brain are the main regulators of water homeostasis. These osmoreceptors sense dehydration, which activates the hypothalamic thirst center and causes water consumption. The kidney's ability to conserve water is another effect of these osmoreceptors. Antidiuretic hormone (ADH), which promotes the kidneys to reabsorb more water, is released by the posterior pituitary when the hypothalamus senses reduced water concentration. Renin secretion from the kidney is sparked by low blood pressure, which frequently happens along with dehydration. Renin changes angiotensin I into angiotensin II, increasing the production of aldosterone from the adrenal glands. Aldosterone enhances the kidney's ability to absorb water and salt. The body controls body volume, salt and water concentration through these methods[3, 7].

Identification of the underlying disease and correction of the hypertonicity are necessary for proper care of hypernatremia. The aim of treatment is to balance the intravascular volume and serum sodium. When possible, fluids should be given orally or through a feeding tube. Fluid resuscitation with isotonic fluids is the first step in treating patients with severe dehydration or shock, followed by free water correction. One of the following formulas can be used to determine the free water deficit and treat hypernatremia[5, 6].

Water deficit (L) = [0.6 in men and 0.5 in women x body weight(kg)] x [(plasma sodium/140) -1]

It is crucial to keep in mind that cerebral edema can result from hypernatremia because water flows from the serum into the brain cells when hypernatremia is rapidly corrected. The aim is to achieve a maximum 12 - meq reduction in serum sodium in a 24 - hour period. Throughout the acute phase of correction, meticulous serial monitoring of serum sodium every 2 to 4 hours is crucial. Rapid osmolality changes can cause cerebral edema, which is why hypotonic fluid delivery should be stopped when seizures start happening while hypernatremia is being corrected. With a reduction in serum sodium not exceeding 0.5 meq per hour, the anticipated free water deficit should be rectified over 48 to 72 hours[5, 6.]

As for the case we can calculate that total body water had been lost was 1L that need to fill within 40 hours.

There are specific management base on type of hypernatremia

a) Hypernatremia with hypovolemic

- Prioritize handling the volume deficit rather than correcting the water deficit.
- Correct volume deficit by starting isotonic saline until improvement in orthostasis, tachycardia, and urine output occurs.
- Calculate and correct water deficit.
- Etiological treatment of fluid loss that occurs.
- After correcting the volume deficit, give saline 0.45%, 5% dextrose, or water orally to replace the deficit and ongoing losses.

Sepsis is a clinical state that falls along a continuum of pathophysiologic states, starting with a systemic inflammatory response syndrome (SIRS) and ending in multiorgan dysfunction syndrome (MODS) before death[10]. Below guidelines are derived from the Surviving Sepsis Campaign Guidelines.

Source Control

1. Broad-spectrum antibiotics within one hour of diagnosis for all patients. Initial empiric anti-infective therapy should have activity against all likely pathogens and adequate penetration of source tissue.
2. Removal of infected/necrotic tissue, if it is the source of septic shock, i.e. patients with cellulitis, abscess, infected devices, purulent wounds.

Management of Shock

1. Measures most effective if achieved within the first six hours of diagnosis
2. Restore central venous pressure (CVP) to 8 mmHg to 12 mmHg
3. Restore mean arterial pressure (MAP) greater than 65 mmHg
4. Restore superior vena cava saturation to 70% or mixed venous saturation to 65%
5. Fluid resuscitation with crystalloid (NS or albumin) and colloid (blood products) up to 80 ml/kg
6. Mechanical ventilation to reduce metabolic demand
7. First-line vasoactive agents (epinephrine in cold shock versus norepinephrine in warm shock) when fluid-refractory Note: dopamine as a first-line agent has fallen out of favor given its inhibitory effect on the HPA axis, namely prolactin and growth hormone, which can confer immunologic dysfunction

Enhancing Host Response

1. Corticosteroids indicated in vasoactive-refractory shock and or in patients with low (unstimulated) basal cortisol levels less than 150 ug/L)
2. Addition of vasopressin indicated in vasoactive-refractory shock

4. Conclusion

Hypernatremia is a serious consequence of dehydration, requiring immediate medical intervention to restore the bodies fluid balance. In this case study, the patients longterm dehydration led to a state of hypovolemic shock, acute kidney injury, and hypernatremia. This case underscores the importance of early detection and treatment of dehydration, especially in elderly individuals, to prevent severe complications such as hypernatremia. Furthermore, it highlights the need to address potential sources of fluid loss, such as skin tissue damage, in the management of dehydration.

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