

# **The Relationship between Serum Sodium and Outcome of Elderly Admitted to The Acute Care**

**Thesis**

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Degree in **Geriatric Medicine and Gerontology**

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## **List of Abbreviations**

- ACTH: Adrenocortico-tropic hormone
- ADH: Anti-diuretic hormone
- ADL: Activities of daily living
- AVP: Arginine vasopressin
- BP: blood pressure
- CKD: chronic kidney disease
- CLD: chronic liver disease
- CNS: central nervous system
- CRH: corticotropin releasing hormone
- CT: computed topography
- DI: diabetes insipidus
- GDS: geriatric depression scale
- GFR: glomerular filtration rate
- GIT: gastro-intestinal tract
- IADL: instrumental activities of daily living

## **List of Abbreviations** (Cont.)

- ICU: intensive care unit
- KFT: kidney function tests
- LFT: liver function tests
- LOS: length of stay
- MMSE: mini-mental status examination
- ODS: osmotic demyelination syndrome
- RBF: renal blood flow
- SIADH: syndrome of inappropriate anti-diuretic hormone
- SPSS: statistical package for social science
- Uosm: urinary osmolarity

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## **Introduction**

Hyponatraemia is one of the most common biochemical abnormalities in clinical practice. Estimates of its prevalence in the literature understandably depend on the population studied (e.g. medical, surgical, intensive care etc), and the cut-off level for plasma or serum sodium used. A common lower limit for the reference range is 134 or 135 mmol/l (*Gill et al., 1998*).

Hyponatraemia is the most commonly observed electrolyte imbalance in hospitalized patients, occurring in up to 6%. In the vast majority of cases, it is due to hypotonic hyponatraemia. This arises when there is an excess of water in relation to sodium stores, and is traditionally divided according to the fluid status of the patient (*Adroque et al., 2000*).

Hyponatraemia has a wide variety of causes, but thiazide diuretics are one of the commonest causes in hospitalized patients as is the 'syndrome of inappropriate antidiuresis' (SIADH) related to a variety of underlying illnesses or prescribed drugs. Iatrogenic fluid overload (especially with hypotonic solutions) may be a significant problem (*Clayton et al., 2006*).



Mild hyponatraemia is generally asymptomatic, but where the decrease in serum sodium is marked (125 mmol/l) or acute (occurring over <48 h), serious neurological complications can ensue as a result of cerebral oedema. Early symptoms of headache, muscular weakness, nausea, lethargy, ataxia and confusion can progress to seizures. Ir-reversible neurological damage, coma and death, if unrecognized and untreated.

Hyponatremia is common in the hospitalized population but extremely heterogenous in terms of causes and the prognostic significance as well (*Decaux et al., 2008*).

Hyponatremia at admission has been associated with higher in-hospital mortality and longer LOS in other unselected patient populations, with risk generally increasing with lower serum sodium levels. Moreover, hyponatremia has been linked to higher mortality risk in numerous medical conditions, including heart failure, liver cirrhosis, cancer, congenital heart disease, community-acquired pneumonia, pulmonary arterial hypertension and pulmonary embolism, as well as in liver transplant candidates (*Zilberberg et al., 2008; Asadollahi et al., 2007; Whelan et al., 2009*).

Sodium levels are tightly controlled in a healthy individual by regulation of urine concentration and production and regulation of the thirst response. In patients with an intact

thirst response, hypernatremia (defined as a serum sodium level  $>145$  mEq/L) is a rare entity. When hypernatremia does occur, it is associated with a high mortality rate ( $>50\%$  in most studies) (*O'Connor et al., 2006*).

In general, hypernatremia can be caused by derangement of the thirst response or the behavioral response there to (psychiatric patients, and elderly patients who are institutionalized), by problems with the renal concentrating mechanism (diabetes insipidus [DI]) secondary to kidney pathology (nephrogenic DI) or difficulty with the neurohormonal control of this concentrating mechanism (central DI), or by losses of free water from other sources (*Adroque et al., 2000*).

The mortality rate from hypernatremia is high, especially among elderly patients. Mortality rates of 42-75% have been reported for acute changes and 10-60% for chronic hypernatremia. Because patients with hypernatremia often have other serious comorbidities, precisely evaluating the degree of mortality directly due to hypernatremia is difficult. Morbidity in survivors is high, with many patients experiencing permanent neurologic deficits. Most deaths are due to an underlying disease process, rather than the hypernatremia itself. Delaying in treatment (or inadequate

treatment) of hypernatremia increase mortality. In hospitalized patients, persistent hypernatremia and protracted hypotension have been associated with a very poor prognosis (*Borra et al., 1995*).

## **Aim of the Work**

- Assessment of pattern of sodium level in acute hospitalized elderly patients.
- Determination of relationship between sodium level and hospital outcome (length of stay and hospital mortality).

# Chapter 1

## Hyponatremia

Disorders of serum sodium concentration are the most common electrolyte abnormalities seen in the geriatric population (*Verbalis et al., 2007*).

Sodium is the principal solute in the extra -cellular compartment and hence the plasma osmolality largely depends on the serum sodium concentration (*Genanri et al., 2006*).

Plasma osmolality in turn is regulated tightly within a narrow range of 275 - 290 mosm/kg by various mechanisms. A decrease or increase in the serum sodium level will have an effect on the plasma osmolality and this can have deleterious effects on the whole body – in particular, the central nervous system (*Lin et al., 2005*).

Severe hypo- and hypernatraemia are associated with significantly high mortality and morbidity. Moreover, inappropriate treatment may result in treatment related complications such as osmotic demyelination syndrome (*Lin et al., 2005*).

Furthermore, the development of serum sodium abnormalities is associated with increased morbidity and mortality in affected patients (*Arinzon et al., 2005*).

### **Prevalence :**

Estimates of its prevalence in the literature understandably depend on the population studied (e.g. medical, surgical, intensive care etc)., and the cut-off level for plasma or serum sodium used, hyponatraemia is the most common electrolyte abnormality observed in hospitalised patients, occurring in 20–30% of acute admissions. Hyponatraemia tends to be more common in the elderly, in patients admitted with respiratory tract infections, in those with a history of alcohol excess and in patients treated with thiazide diuretics. A common lower limit for the reference range is 134 or 135 mmol/l (*Gill et al., 1998*).

Patients in outpatient settings exhibit hyponatremia in about 5% of those tested, with occurrence rates increasing to as high as 20% in hospitalized geriatric patients and 30% in patients seen in intensive care units (*Miller et al., 2006*).

The severity of the primary process contributing to the development of the abnormal serum sodium is responsible for the unsatisfactory outcome. The most common disorder of

serum sodium concentration in the geriatric population is hyponatremia (*Adroque et al., 2000*).

### **Sodium homeostasis**

Factors contributing to the development of hyponatremia in the elderly include age-associated decrease in glomerular filtration rate and free water clearance, as well as sodium losses from decreased activity of the renin-angiotensin-aldosterone system and increased activity of natriuretic hormones together with abnormalities of secretion of the pituitary hormone arginine vasopressin (AVP), antidiuretic hormone (ADH) (*Adroque et al., 2000; Miller et al., 2006*).

Although plasma osmolality is closely related to serum sodium concentration, hyponatraemia can be associated with low, normal, or high osmolality (*Genanri et al., 1984*).

Osmolality or tonicity refers to the contribution to osmolality of solutes such as sodium, glucose, and urea that cannot freely move across the cell membrane thereby reducing transcellular shifts in water (*Genanri et al., 1984*).

Plasma osmolality is preserved within the normal range by the effect of (ADH). Osmoreceptors near the hypothalamus sense plasma osmolality and modulate vasopressin release (*McKinley et al., 2004*).