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Diagnosis and treatment of hyponatraemia



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Hyponatraemia is the most common electrolyte abnormality encountered by physicians in the hospital setting. It is associated with increased mortality and length of hospital stay. However, the basis of the relationship of hyponatraemia with clinical outcome is not clear. Doubt remains as to whether the relationship is causal. It may reflect the association of two independent variables both of which are linked with disease severity.

Serum sodium concentration is regulated through integrated neuro-humeral mechanisms that overlap with those regulating circulating volume. A mechanistic approach to the classification of hyponatraemia can support a framework for investigation and differential diagnosis based on urine osmolality and urine sodium concentration. Such a framework is more reliable than those based on the clinical assessment of volume status.

In the emergency setting, the initial management of hyponatraemia is cause-independent. In other clinical contexts, a cause-specific approach is recommended. Over-rapid correction of serum sodium risks precipitating osmotic demyelination syndrome. Avoiding over-rapid correction is critical in any approach to patient care.

Sodium is the major circulating cation and thus a key determinant of overall plasma osmolality. Serum sodium concentration is maintained within a tight physiological range over time, despite

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wide variation in both sodium and water intake. Hyponatraemia (serum sodium concentration <135 mmol/L) is the most common electrolyte disturbance in clinical practice. All clinicians should be aware of the scope and scale of the problem.

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Epidemiology

Hyponatraemia is a clinical feature in 15–42% of non-selected emergency admissions to secondary care. Importantly, it is associated with increased length of hospital stay and morbidity in patients presenting with a range of conditions [1]. Moreover, it has prognostic significance. Hyponatraemia is a predictive indicator of outcome in malignancy and an independent predictor of all-cause in-patient mortality [2,3]. However, the relationship between serum sodium concentration and clinical outcome is not simple. While Chawla et al. (2011) demonstrated that a serum sodium concentration between 120 and 134 mmol/l was associated with an adverse patient outcome, further impact at lower levels of serum sodium was not clear [4]. A recent Danish study has confirmed the association of hyponatraemia with increased mortality but did not demonstrate a relationship between degree of hyponatraemia and mortality [5]. Taken together, these data raise questions over whether the relationship between serum concentration and clinical outcome is a causal one. Rather, it may simply reflect the association of two variables, both of which are linked (independently) with severity of illness and presentation [6].

Clinical impact of hyponatraemia: emerging themes

Bone is a major sodium reservoir and increased bone turnover (with consequent mobilisation of bone sodium) is a physiological response to persisting hyponatraemia. Population-based studies have confirmed a negative impact of hyponatraemia on bone density and association with increased fracture risk. Both osteoporosis and fragility fractures increase incrementally with categorical decrease in median serum sodium [7,8]. Hyponatraemia is also associated with gait instability [9]. The clustering of bone fragility with increased fall risk highlights the potential interaction of risk factors for common and

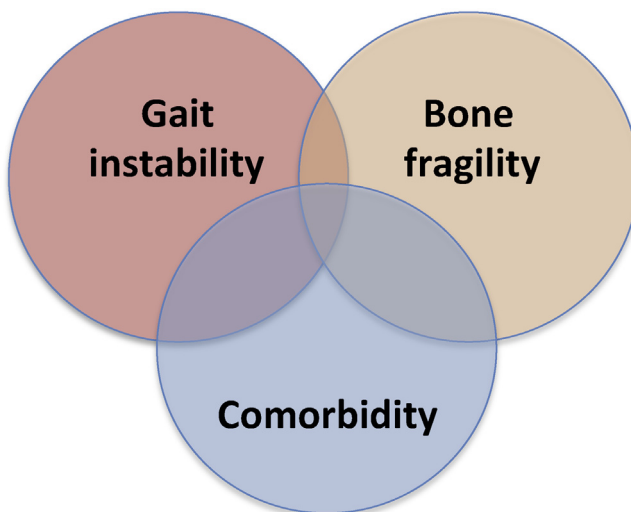


Fig. 1. The interaction of multiple, linked factors determine overall fracture risk in patients with hyponatraemia.

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