# Hyponatremia in the elderly: A primary care challenge

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## SUMMARY

**Hyponatraemia is common in the elderly. Given the broad differentials for hyponatraemia, a meticulous diagnostic approach and comprehensive laboratory investigations are needed. Fluid status assessment is vital to decide on the next management. Unidentified hyponatraemia may lead to complications such as seizure and coma. Mismanagement or overly aggressive correction of hyponatraemia can lead to morbidity, such as osmotic demyelination syndrome, which might cause mortality. This case report delineates a challenging instance of moderate hyponatraemia in an elderly patient, highlighting the complexities encountered in the primary care setting. The patient initially presented with lethargy and generalised weakness, which worsened during routine follow-ups. History, physical examination, and laboratory investigations lead to the diagnosis of SIADH with no clear cause identified. Treatment primarily consisted of fluid restriction, accompanied by regular electrolyte monitoring. This case underscores the importance of prompt diagnosis and tailored therapeutic interventions to optimise hyponatraemia management in the elderly patients at the primary care settings.**

## INTRODUCTION

Hyponatraemia has a prevalence of 47.9% among individuals aged 60 years and above.[1] In Malaysia, 6.9% of elderly in primary care have hyponatraemia.[2] The prevalence of hyponatremia among older inpatients in a general hospital in China was 24.7%.[3] Hyponatraemia is defined as a plasma sodium level of less than 135 mmol/L. Sodium is an electrolyte that regulates the balance of water and minerals, conducts nerve impulses, and contracts and relaxes muscles. Mild hyponatraemia may have signs and symptoms such as nausea and vomiting, headache, muscle weakness, spasm, and fatigue.[4] Severe hyponatremia may lead to life-threatening complications such as seizures and coma.[4] It is important to correct hyponatraemia, as it is associated with increased morbidity and mortality.[5] The most common causes of hyponatremia in adults are SIADH, thiazide and antidepressant therapy, and endocrinopathies.[6] The American Family Physician algorithm for hyponatremia assessment provides a structured approach in evaluating hyponatraemia.[7] Assessing fluid status will help to diagnose the underlying condition. In general, hyponatremia is treated with fluid restriction in isovolumic, isotonic saline given in hypovolemia, and diuresis given in hypervolemia. Hypertonic saline is used to treat severe symptomatic hyponatraemia.[8] Chronic hyponatremia is less likely to cause seizures and other severe complications. Correction of chronic hyponatremia should be performed gradually than acute to avoid osmotic demyelination.[4] Recognising and diagnosing hyponatremia is imperative in primary care settings. Hence, this case study is going to discuss the management of an elderly man presented to a primary care clinic with chronic hyponatremia.

## CASE PRESENTATION

A 64-year-old man is under six monthly follow-ups in a primary care clinic. He is a known case of hypertension, Parkinson's disease, benign prostatic hyperplasia, and a past episode of major depressive disorder with generalised anxiety disorder but defaulted psychiatry follow-up. During this follow-up, he presented with lethargy and generalised body weakness persisting for the past 5 months. His symptoms had worsened over the past 2 weeks, rendering him unable to climb stairs without assistance. As the presentation is non-specific, a systemic approach to history taking and physical examination is essential in ruling out broad differential diagnoses, including infection, dehydration, stroke, and anaemia. He denied experiencing symptoms of hyponatremia, such as headache, seizure, dyspnoea, nausea, vomiting, diarrhoea, muscle cramp, or paraesthesia. He takes about 2 litres of water per day and has no polyuria or polydipsia. There was no history of excessive fluid intake or fluid loss to rule out psychogenic polydipsia. No history of falls or trauma to the head. He has features of depression, such as low mood, anhedonia, and loss of concentration. His medications were Tab. Madopar (levodopa 200mg/benserazide 50mg) TDS, Tab. bisoprolol 5mg OD, Tab. atorvastatin 40mg ON, Tab. amlodipine 5mg OD, Tab. perindopril 4mg OD, and Tab. vitamin B1/B6/B12, 1 tablet OD. He did not smoke, take traditional medicines, or use alcohol or illicit drugs, and he was not taking diuretics.

During the initial assessment, his BP was 128/64, and other vital signs were stable. Physical examination showed no signs of dehydration and anaemia. There was no evidence of fluid overload, such as raised JVP, pulmonary oedema or bilateral lower limb oedema. He exhibited signs of Parkinsonism, including muscle rigidity, tremors, and reduced arm swing.

Investigations prior to this follow-up revealed moderate hyponatremia with a sodium level of 126 mmol/litre, prompting further evaluation. His sodium level has been ranging between 131 and 134mmol/L since 2007 throughout the follow-ups at primary care. Suggested relevant initial workups in primary care are thyroid function tests, fasting

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**Table I: Baseline investigations and results**

| Date | 2 Jan 2024 | 18 Jan 2024 | 22 Jan 2024 | 31 Jan 2024 | 19 Feb 2024 | 16 Apr 2024 | 27 May 2024 | 1 Nov 2024 | Ref range | Units |
|---|---|---|---|---|---|---|---|---|---|---|
| Na | 127 | 126 | 129 | 133 | 133 | 131 | 132 | 134 | 136-14 | mmol/l |
| K | 4.5 | 4.4 | 4.5 | 4.4 | 4.7 | 4.3 | 4.0 | 4.5 | 3.5-5.1 | mmol/l |
| Urea | 4.9 | 4.4 | 4.9 | 4.6 | 4.2 | 4.7 | 4.0 | 7.1 | 3.2-7.4 | mmol/l |
| Creat | 74.6 | 64.7 | 71.1 | 76.1 | 72.3 | 71.9 | 85.1 | 85.7 | 64-104 | umol/l |
| LDL | 1.98 | - | 1.92 | 2.40 | | | | 2.61 | <3.8 | mmol/l |
| Chol | 4.17 | - | 3.91 | 4.59 | | | | 4.42 | <5.2 | mmol/l |
| TP | 67 | - | 65 | 69 | | | | 65 | 64-83 | g/l |
| AST | 22 | - | 18 | 22 | | | | 24 | 5-34 | umol/l |
| ALT | 27 | - | 20 | 24 | | | | 23 | 0-55 | u/l |
| ALP | 84 | - | 88 | 85 | | | | 81 | 40-150 | u/l |
| FBS | 6.05 | - | - | - | | 5.79 | 5.92 | | 3.9-6.0 | mmol/l |
| T4 | - | 14.54 | | | | | | | 9-19.05 | pmol/l |
| TSH | - | 0.59 | | | | | | | 0.35-4.94 | uiu/ml |
| Cortisol | 331 | | | | | | | | >50 | nmol/l |
| Serum osmolality | 271 | | | | | | | | 275-295 | mosm/kg |
| Urine osmolality | 309 | | | | | | | | 50-1200 | mosm/kg |
| Urine sodium | 44 | | | | | | | | - | mmol/l |

blood sugar, fasting serum lipid, and liver function tests. At the same time, serum osmolality is sent to differentiate between isotonic, hypotonic, or hypertonic hyponatraemia. Given the patient's hypotonic hyponatremia (low plasma osmolality) and euvolemia, differentials such as SIADH, Addison's disease, hypothyroidism, psychogenic polydipsia, drug-induced causes, severe potassium depletion, and renal insufficiency need to be considered. Therefore, serum morning cortisol, urine osmolality, and urine sodium were sent. A chest X-ray was done to detect lung malignancy, and it was normal in this patient. A brain CT scan would be necessary if a neurosurgical condition such as subarachnoid haemorrhage or subdural haematoma were suspected.

Investigations showed low serum osmolality (271mOsm/kg), with raised urine osmolality (309 mOsm/kg) and random urine sodium of 44mmol/L. His serum T4 (14.54pmol/L), TSH (0.59uIU/ml), cortisol (331nmol/L), renal profile, liver function test, and urine albumin creatinine ratio were all normal (Table I). Pseudohyponatremia was ruled out with normal fasting blood sugar and total cholesterol value.

Syndrome of inappropriate antidiuretic hormone secretion (SIADH) was diagnosed by low plasma sodium (<130mmol/L), plasma osmolality (<275mOsmol/kg), urine Na (>20mmol/L), urine osmolality> plasma osmolality, no oedema or signs of hypovolemia, normal renal, thyroid, and adrenal function with low plasma urea level.

The patient was started on Tab Madopar in 2023, but hyponatremia has been recorded prior to that since 2007. The most common time levodopa causes hyponatremia is when it is newly started or after a dose increase.[9] The patient has tolerated Tab. Madopar well and is not keen to change the medication.

He was given a two-weekly follow-up under the care of family medicine specialists at a primary care clinic in view of moderate hyponatraemia. A fluid restriction regimen with 1 litre/ day was initiated alongside electrolyte monitoring for this patient.

Following fluid restriction of 1 litre/day and no restriction of salt intake, the patient's sodium gradually increased from 127mmol/l to 129mmol/l, then 133mmol/l within one month. The patient's sodium level was stable between 131 and 134mmol/l thereafter. With the increasing trend of sodium, his symptoms of lethargy and generalised body weakness improved. The patient is comfortable with current management.

## DISCUSSION

Hyponatraemia is defined as a plasma sodium level of less than 135mmol/L. Mild hyponatraemia is defined as serum sodium concentration (130-135mmol/L), moderate (125-129mmol/L), severe (<125mmol/L). In cases of severe hyponatraemia, a patient may present with seizure or coma. Referral to the hospital for immediate treatment is essential in severe hyponatraemia to prevent the risk of cerebral oedema and hyponatraemic encephalopathy. Plasma sodium concentration depends on the amount of both sodium and water in the plasma. Therefore, hyponatraemia does not necessarily imply sodium depletion. Plasma osmolality is maintained by strict regulation of the arginine vasopressin, also known as antidiuretics hormone (ADH) system, and thirst. If plasma osmolality increases, ADH is secreted and water is retained by the kidneys, thus reducing serum osmolality. If plasma osmolality decreases, ADH also decreases, resulting in diuresis of free water and a return to homeostasis. Failure of regulation of plasma osmolality leads to hyponatraemia. The ability to excrete a water load is delayed in the elderly. Therefore, the elderly are susceptible to alterations of water imbalance due to decreased renal mass, cortical blood flow, and glomerular filtration rate, thus affecting responsiveness to sodium balance.

Managing hyponatraemia in primary care involves initial investigations to facilitate ruling out common differential diagnoses such as hyperproteinaemia, hyperlipidaemia, hyperglycaemia, and hypothyroidism (Figure 1). Assessing fluid status is the key to diagnosis in addressing hyponatraemia. This patient has been diagnosed with

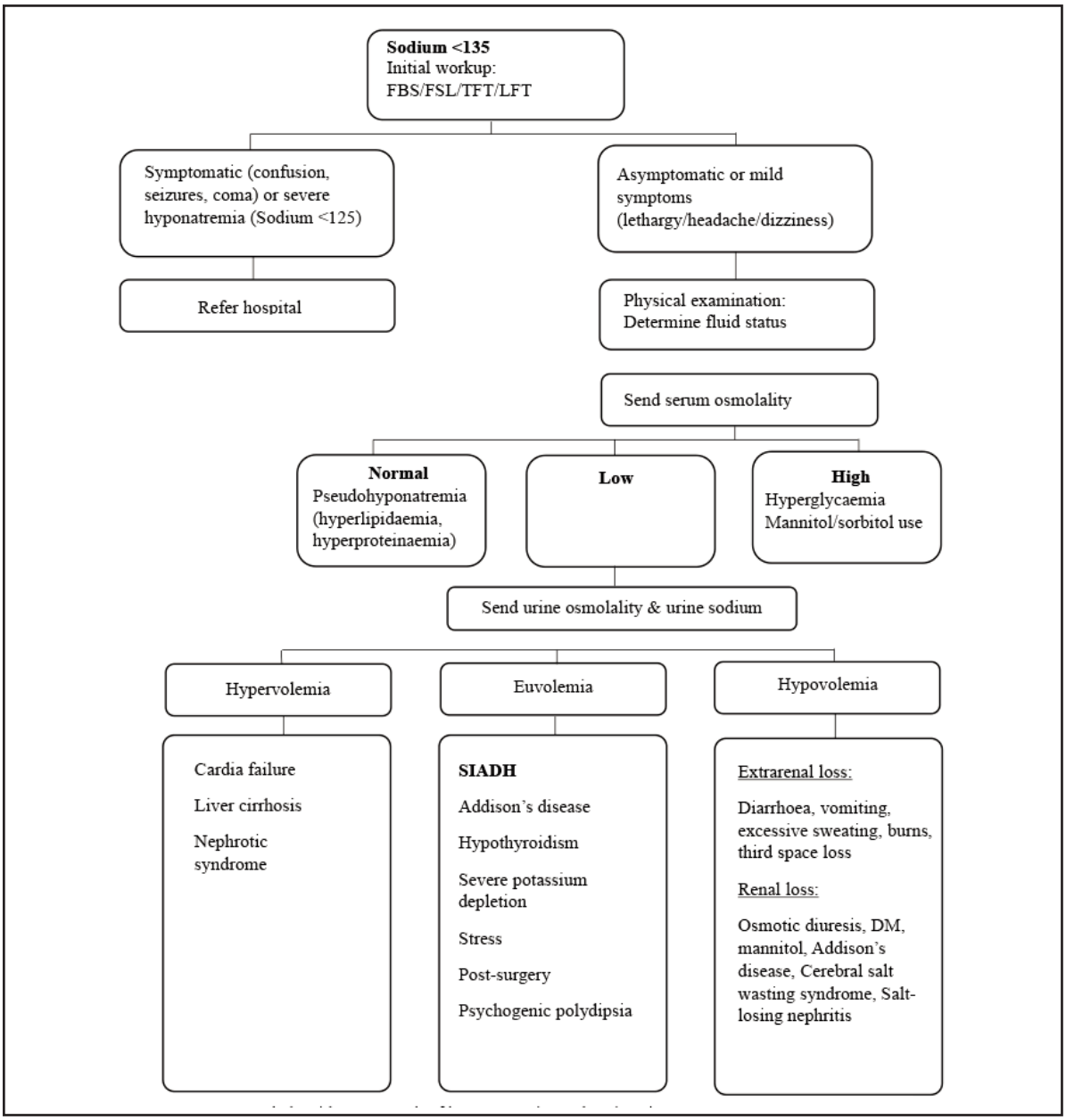


**Fig. 1:** Suggested algorithm approach of hyponatremia workup in primary care
Abbreviation: FBS = Fasting blood sugar; FSL = Fasting serum lipid; TFT = Thyroid function test; LFT = Liver function test; DM = Diabetes Mellitus; SIADH Syndrome of inappropriate antidiuretic hormone secretion

euvolemic (increased total body water with normal sodium level) hyponatraemia secondary to SIADH, as supported by concentrated urine sodium in the presence of hyponatremia and low plasma osmolality, in the absence of hypovolemia, oedema or diuretics.[4] The patient had no depressive symptoms during subsequent follow-up, thus, antidepressant was not started, considering the patient was already on polypharmacy. Despite the complexities associated with managing this patient's multiple comorbidities and non-compliance issues, a coordinated and patient-centred approach resulted in gradual improvements in his symptoms and overall well-being.

Hyponatraemia is more common in the elderly because they are more likely to have polypharmacy (more frequently thiazides and antidepressants). Levodopa is the effective antiparkinsonian agent. However, Parkinson's disease treatment with levodopa is known to cause SIADH. It is important to correct the underlying cause of hyponatraemia as it increases the risk of falls in the elderly. In general, serum sodium should not be increased by more than 10mmol/L in a 24-hour period in an asymptomatic patient and 12mmol/L in a symptomatic patient. In chronic hyponatremia (defined as a duration of more than 48 hours), overzealous correction should be avoided as it can lead to central pontine myelinosis. Fluid restriction is the mainstay of treatment and

preferred mode of treatment for SIADH. In this case, administration of normal saline is not appropriate, as the sodium may be rapidly excreted while the water is retained, therefore worsening hyponatraemia. We need to take into consideration that taking a high-sodium diet needs to be weighed against the drawback of raised high blood pressure, especially in this patient who has hypertension; hence, close monitoring of blood pressure is required for this patient.

The patient was not referred to the endocrine team, as he was managed by a family medicine specialist with close monitoring of sodium level. Emergency referral is recommended in a patient with a sodium level <125mmol/L or severe symptoms. Consultation with the endocrinologist in patients is required for a patient with a persistent sodium level of 125-129mmol/L.[10]

## CONCLUSION

In summary, this case exemplifies the diagnostic and therapeutic challenges posed by moderate hyponatraemia and polypharmacy in elderly patients within the primary care setting. Hyponatraemia, in this case, was attributed to the syndrome of inappropriate hormone secretion with no clear cause identified despite looking for common causes of SIADH (neurological, respiratory, neoplasia, and medication). Doctors in primary care settings may have difficulties treating patients who have hyponatraemia due to the complexity of the diagnostic workup. The diagnostic workup, including assessment of fluid status and blood investigations, is important to aid in confirming the underlying aetiology and guiding appropriate management strategies. While hyponatraemia is common in primary health care, SIADH may be underdiagnosed in primary care due to the complexity of the condition and limited understanding among primary care doctors. It contributes to increased morbidity in the elderly, including a higher risk of falls and altered mental status. Fluid restriction emerged as the cornerstone of treatment for SIADH, with gradual correction of sodium levels observed over subsequent follow-ups.

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## DECLARATION

The authors declare no conflict of interest.

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