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Just How Common is Pseudohyponatraemia? Retrospective Survey of 1,041 Patients with Serum Sodium 125 mmol/L or Less

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ABSTRACT

Introduction: Hyponatraemia is usually associated with a low serum osmolality <280 mOsm/kg. Iso-osmolar (serum osmolality 280-300mOsm/kg) or hyperosmolar (serum osmolality >300 mOsm/kg) hyponatraemia should alert the clinician to one of two possibilities: pseudohyponatraemia, a laboratory artefact that occurs when patients with severe hyperlipidaemia or severe hyperproteinaemia have their sodium checked using indirect ion selective electrodes; or dilutional hyponatraemia which reflects the presence of solutes that increase osmolality such as glucose and alcohol, causing water to move from the intracellular to extracellular space.

Objectives: To assess the prevalence and aetiology of pseudohyponatraemia in an unselected series of patients in southwest Scotland; and to determine the other causes of iso and hyperosmolar hyponatraemia in this patient population.

Design: Retrospective cohort study.

Setting: Southwest Scotland.

Participants: 84,108 patients who had serum urea and electrolytes measured during an 18-month period from August 2023 to January 2025.

Main Outcome Measures: Serum sodium, serum osmolality, serum lipids and serum immunoglobulins.

Results: 1,041(1.2%) patients had serum sodium 125 mmol/L or less. Iso-osmolar and hyperosmolar hyponatraemia accounted for 33(4%) of 824 patients who had simultaneous measurements of serum osmolality. We identified one probable case of pseudohyponatraemia due to hyperlipidaemia in this group. Alcohol excess and hyperglycaemia were the likely causes of dilutional hyponatraemia in eight and four patients respectively. We further identified one possible case of pseudohyponatraemia among the 217 patients who did not have their serum osmolality measured.

Conclusions: Despite its rarity, the recognition of pseudohyponatraemia remains important because it is a laboratory artefact for which no specific treatment is required.

Keywords: Pseudohyponatraemia; Dilutional hyponatraemia; Myeloma; Hypertriglyceridaemia; Serum osmolality

INTRODUCTION

Hyponatraemia is the commonest electrolyte abnormality encountered in clinical practice.[1] It is usually associated with a serum osmolality that is less than 280 mOsm/kg, when it is described as hypotonic or hypo-osmolar hyponatraemia. Less frequently, hyponatraemia can be iso-osmolar (serum osmolality 280-300 mOsm/kg) or hyperosmolar (serum osm >300 mOsm/kg). Iso-osmolar and hyperosmolar hyponatraemia should alert the clinician to one of two possibilities: of pseudohyponatraemia, a laboratory artefact that occurs when patients with severe hyperlipidaemia or severe hyperproteinaemia have their sodium checked using indirect ion selective electrodes;[2,3] and of dilutional hyponatraemia, which reflects the presence of solutes that increase osmolality such as glucose and alcohol, causing water to move from the intracellular to extracellular space.[3-5]

The prevalence of severe hyponatraemia (serum sodium <=125 mmol/L) is known to lie between 1% and 3% of all samples submitted for measurement of urea and electrolytes.[6,7] There are, surprisingly, no studies of the prevalence of pseudohyponatraemia. This is despite a number of case reports of pseudohyponatraemia in patients who are either known to have, or are found to have, hyperlipidaemia or myeloma[2,8-16] (Table 1).

Author	Age/ Gender	Serum Sodium mmol/L	Pseudohyponatraemia confirmed by serum Osm and/or direct ISE	Cause of Pseudohyponatraemia	Serum Lipids mmol/L Serum Immunoglobulins g/L
Song 2019	41M	118	Yes	Hyperlipidaemia	Chol 37.5, Trig 10.8
Yu 2005	56M	123	Yes	Myeloma	IgG 55.3, IgA 5.8, IgM 3.9
El Hage 2018	69M	119	Yes	Hyperlipidaemia	Chol 34.7, Trig 3.2
Hashem 2024	59M	119	Yes	Hyperlipidaemia	Chol 21.3, Trig 77.6
Garcia 2025	39M	114	Yes	Hyperlipidaemia	Chol NG, Trig >56.5
Wani 2025	69M	<100	Yes	Myeloma	IgG 71.0, IgA 0.19, IgM 0.24
Dawson 2021	26M	112	Yes	Hyperlipidaemia	Chol NG, Trig 65.5
Ballard 2023	55M	128	Yes	Hyperlipidaemia	Chol NG, Trig 13.5
Ballard 2023	68F	130	Yes	Myeloma	Total Protein 120g/L*

Legend: Osm = Osmolality. ISE = ion selective electrodes. Chol = cholesterol. Trig = triglyceride. NG = not given.

Normal ranges: Cholesterol 3.9 - 5.5mmol/l, Triglyceride 0 - 1.7mmol/L, IgG 6 - 16g/L, IgA 0.7 - 4.5g/L, IgM 0.4 - 2.5g/L, Total Protein 60-80g/L. Conversions: Cholesterol mmol/L to mg/dL x 38.67, Triglyceride mmol/L to mg/ dL x 88.57. *Serum electrophoresis confirmed diagnosis of myeloma.

Against this background, the aim of our study was threefold: to assess the prevalence of pseudohyponatraemia in an unselected series of patients who either attended their general practitioners or were admitted to hospital in southwest Scotland; to document the causes of said pseudohyponatraemia; and to determine the other causes of iso- and hyperosmolar hyponatraemia in this patient population.

METHODS

Serum sodium was analysed on the Cobas 8000 ISE module (Roche Diagnostics) using indirect ion-selective methodology. The analytical precision (CV%) for the sodium assay was 1% at a level of 114mmol/L, in agreement with Roche's technical specifications. EQA performance (UK NEQAS Clinical Chemistry) was also satisfactory.

Serum osmolality was analysed on the Advanced® Micro-Osmometer Model OSMO1 using freezing point depression methodology. The analytical precision (%CV) for the assay was <1% at all levels of IQC (240 – 400mOsm/kg), in agreement with Advanced Instrument's technical specifications. EQA performance (UK NEQAS Clinical Chemistry) was also satisfactory.

This was a retrospective cohort study. We measured serum sodium as part of a urea and electrolytes profile 338,870 times in 84,108 patients during an 18-month period between August 2023 and January 2025. We chose this date range to create a cohort of over 1,000 patients with a serum sodium of 125 mmol/L or less. One thousand and forty-one patients were included. Serum osmolality was measured within 24 hours of serum sodium in 824 (79%) of these patients and was 280 mOsm/kg or more (range 280-342 mOsm/kg) in 33, indicating iso- or hyperosmolar hyponatraemia.

We considered alcohol a likely contributor to normal or raised serum osmolality if patients were known to Drug and Alcohol Services, had a history of heavy alcohol use noted in their case record and/or a documented alcohol intake >50 units/week. We did not measure blood ethanol in these patients as they did not appear intoxicated when they presented with severe hyponatraemia.

We included blood glucose measurements if they were taken within a week of the documented hyponatraemia. Serum urea values in Table 2 and Table 3 were measured on the same sample as the serum sodium. We referenced serum lipids and immunoglobulins if these were taken within a month of hyponatraemia as we felt these analytes were less likely to undergo acute change. We noted if patients were receiving intravenous dextrose in the 24 hours preceding hyponatraemia by reading patient fluid charts. When patients had more than one serum sodium of 125 mmol/L or less, we chose the lowest value for inclusion in Table 2 and Table 3.

RESULTS

Our analytical cohort of 1,041 patients comprised 597 (57%) women, age range 0-97 years (only 11 of whom were under 20 years) and 443 (43%) men, age range 0-96 years (only 15 under 20 years). These patients were

representative of a largely white Scottish population as over 96% of the population in Dumfries and Galloway identify as a white ethnic group.

Table 2 shows serum osmolality, plasma glucose, serum urea, serum lipids, and serum immunoglobulins, when requested, for the 22 iso-osmolar patients whose serum sodium was 125mmol/L or less with serum osmolality 280 – 300 mOsm/kg. Hyperosmolar (serum osm >300 mOsm/kg) (n=11) patients are listed separately in Table 3.

Isoosmolar hyponatraemia

There were no cases of pseudohyponatraemia among the 12 patients whose serum lipids were measured or among the 10 patients with serum immunoglobulins. We identified five cases of dilutional hyponatraemia likely due to alcohol excess and one to hyperglycaemia (31.3 mmol/L). Uraemia (serum urea 38.8 mmol/L) may have accounted for one other case of iso-osmolar hyponatraemia, which was unexplained in the remaining 15 patients. None were receiving intravenous dextrose when found to be hyponatraemic.

Hypersomolar hyponatraemia

We measured serum lipids in 8 of the 11 patients, one of whom had hyperlipidaemia that was severe enough to cause pseudohyponatraemia (see case history). Serum immunoglobulins measured in five patients were normal. Alcohol excess was the likely cause of dilutional hyponatraemia in three patients, while hyperglycaemia was a probable cause in a further three patients (plasma glucose 52.3, 37.2, 34.4 mmol/L). It is unlikely that any hyperglycaemia in the remaining patients could account for serum sodium 125 mmol/L or less after applying Hillier's correction.[18] Uraemia may have been the cause of two other patients with hyperosmolar hyponatraemia (serum urea 34.9 mmol/L and 74.9 mmol/L).

We measured blood lactate in eight patients, only two of whom had lactate greater than 4 mmol/L: one was thought to have had alcoholic lactic acidosis while the other had diabetic ketoacidosis. It is unlikely therefore that lactate per se contributed to the hyperosmolality in these patients. The cause of the hyperosmolarity could not be determined in the remaining two patients. None of these patients were receiving intravenous dextrose when found to be hyponatraemic.

Case history

A 67-year-old male, who had no symptoms, was found to be hyponatraemic with serum sodium 123 mmol/L on routine blood testing. This was on a background of type 2 diabetes, ischaemic heart disease and atrial fibrillation. He was significantly overweight at 92.4 kg and his diabetes was poorly controlled with random glucose 30.9 mmol/L and HbA1c 150 mmol/mmol (normal 27-42 mmol/mol), although he was neither acidotic nor ketotic.

His serum was noted to be lipaemic, following which serum cholesterol was measured at 19.7 mmol/L (normal range 2.5-5.0 mmol/L) with serum triglyceride 63.1 mmol/L (normal range 0.8-3.0 mmol/L). Serum osmolality was raised in the hyperosmolar range at 317 mOsm/kg, suggesting a mixed picture of pseudo and dilutional hyponatraemia, as it is unlikely that glucose alone could account for his hyponatraemia even after applying Hillier's correction (Hillier 1999).

We treated his diabetes by switching from oral hypoglycaemics to insulin and his hyperlipidaemia with a combination of fenofibrate and ezetimibe. He had previously been unable to tolerate statins. He received one litre of 0.9% saline over 12 hours only. Serum sodium returned gradually to the normal range three weeks later. He subsequently received the GLP1 agonist Mounjaro, which facilitated a drop in weight to 75.8 kg. When last reviewed at the clinic, 21 months after his presentation, he was well with serum sodium 137 mmol/L, serum cholesterol 3.9 mmol/L, serum triglyceride 6.9 mmol/L and HbA1c 44 mmol/mol.

Table 2: Iso-osmolar Hyponatraemia

Age/Gender	Serum Sodium mmol/L	Serum Osm mOsm/kg	Osmolal Gap mOsm/kg	Blood Glucose mmol/L	Serum Urea mmol/L	Alcohol Excess	Serum Cholesterol mmol/L	Serum Triglyceride mmol/L	Serum IgG g/L	Serum IgA g/L	Serum IgM g/L
83M	125	280	8.9	5.7	15.4	No	4.3	0.6	13	5	1
66M	121	280	28.9	5.7	3.4	No					
85F	125	280	15.9	7.1	7	No	2.3	1			
51M	124	281	16.3	11.6	5.1	Yes					
83F	124	284	4.4	23.1	8.5	No					
73F	122	287	14.1	16.7	12.2	No	7.5	3.1	10.4	1.7	0.5
81M	124	288	8	8.3	23.7	No	3.4	2.7	14.5	4.7	1.4
78M	120	289	6	7.5	35.5	No	3.2	1.7	21.1	1.7	0.5
39M	123	289	16.3	15.5	11.2	Yes					
82F	121	289	10.4	19.2	17.4	No	2.3	1.1	13.4	8	1.1
93F	125	290	11.3	6.2	22.5	No	1.9	0.7	20.1	<0.1	0.7
60M	121	290	32.9	10.6	4.5	Yes					
60M	124	291	12.5	17.2	13.3	Yes	3.7	2.4			
72M	125	292	-10.4	24.9	27.5	No					
76M	125	292	12.5	5.5	24	No					
81F	125	293	3.1	12.7	27.2	No					
85M	122	294	5.7	5.5	38.8	No					
58F	124	295	13.5	6.2	27.3	No	5	1.6	8.7	2.1	1.4
82M	124	295	22.5	9.9	14.3	No					
66F	118	298	6.3	31.3	24.4	No	3	2.3	11.6	5.3	1
61M	121	300	50.5	5.4	2.1	Yes	4.2	1.7	9.4	3	2.3
74F	123	300	12.4	13.8	27.8	No	2.5	0.8	12.7	3	0.9

Table 2: Table showing serum sodium, serum osmolality, glucose, urea, alcohol excess, lipids and immunoglobulins in 22 patients with isoosmolar hyponatraemia.

Table 3: Hyperosomolar Hyponatraemia

Age/Gender	Serum Sodium mmol/L	Serum Osm mOsm/kg	Osmolal Gap mOsm/kg	Blood Glucose mmol/L	Serum Urea mmol/L	Alcohol Excess	Serum Cholesterol mmol/L	Serum Triglyceride mmol/L	Serum IgG g/L	Serum IgA g/L	Serum IgM g/L
73M	121	303	42.9	11.9	6.2	No			8.5	2.5	0.4
59F	119	308	43.2	34.4	10.2	No	2.5	1.9			
67M	122	308	56.6	4.7	2.7	Yes					
65M	122	310	55.8	5.3	4.9	No	5.3	1.3	9.2	1	1.1
83M	124	311	10.3	37.2	15.5	No	4	2.3	7.8	1.3	1.1
71M	125	312	17.6	9.5	34.9	No			3.6	0.7	0.3
67M	123	317	29.1	30.9	11	No	19.7	63.1	9.6	4.3	0.3
67M	120	319	71	5.9	2.1	Yes	3.7	0.5			
63M	123	326	52.4	13	14.6	Yes	3.5	1.8			
41M	116	339	38.4	52.3	16.3	No	4.8	2.4			
65M	120	342	19.2	7.9	74.9	No	2.4	0.6			

Table 3: Table showing serum sodium, serum osmolality, glucose, urea, alcohol excess, lipids and immunoglobulins in 11 patients with hyperosmolar hyponatraemia.

Hyponatraemic patients with no measurement of serum osmolality

We examined the case records of 217 patients with serum sodium 125 mmol/L or less who did not have a concurrent measurement of serum osmolality, to determine whether any had hyperlipidaemia or myeloma before, during or after their presentation with severe hyponatraemia. There were no cases of hypertriglyceridaemia greater than 17 mmol/L. There was one case of possible pseudohyponatraemia: an 87-year-old male with IgG myeloma, serum IgG of 78.8 g/L, serum IgA <0.1 g/L, serum IgM 1.9 g/L and an associated total protein of 147 g/L, who was found to have serum sodium 123 mmol/L.

DISCUSSION

The main findings of our study are that iso- and hyperosmolar hyponatraemias are uncommon, accounting for only 33 (4%) of a consecutive series of 824 patients with serum sodium 125 mmol/L or less whose serum osmolality was measured simultaneously, or at the very least within 24 hours. We identified one probable case of pseudohyponatraemia due to hyperlipidaemia in this group and further identified one possible case of pseudohyponatraemia due to myeloma among the 217 patients who did not have their serum osmolality measured. Alcohol excess and hyperglycaemia were the likely causes of dilutional hyponatraemia in eight and four patients respectively. Some were iso-osmolar and some hyperosmolar. No clear cause was identified in 15/22 patients with iso-osmolar and 2/11 patients with hyperosmolar hyponatraemia.

Pseudohyponatremia is a rare laboratory artefact characterised by hyponatraemia in the setting of a normal or raised serum osmolality (>280mOsm/kg).[3] This phenomenon is created by the use of indirect ion selective electrodes (ISEs) which report the sodium concentration measured within the water fraction of a diluted serum sample, relative to the total serum volume. Most NHS laboratories use indirect ISEs, in contrast to point of care blood gas analysers which generally employ direct ISEs to measure sodium within the water traction of an undiluted whole blood sample.[19] When serum proteins and lipids are within the normal physiological range, direct and indirect ISE methods are calibrated to produce equivalent sodium concentrations.

The key to understanding pseudohyponatraemia is as follows. An Indirect ISE (including the Roche Cobas 8000 ISE module) assumes that plasma water is ~93% of plasma volume, corresponding to normal protein and lipid concentrations. This assumption is fixed within the calibration model. The analyser doesn't measure the patient's water fraction or adjust for it. Because the method works on a diluted sample, the final sodium value is calculated per litre of total plasma, not per litre of plasma water. It follows that the differences in sodium results between indirect and direct ISEs only become apparent when there is an abnormally high or low total protein and/or lipid concentration in a patient sample that decreases or increases plasma water, respectively.

Pseudohyponatraemia is more likely to occur when serum hypertriglyceridaemia exceeds 17 mmol/L or hyperproteinaemia is greater than 100 g/L (21). Pseudohyponatraemia is physiologically unimportant and does not require treatment because the main complication of hyponatraemia, namely hyponatraemic encephalopathy, only occurs when the concentration of sodium in serum water becomes dangerously low. Failure to recognise this may lead to serious, and occasionally fatal, consequences.[11,22]

Glucose and alcohol can both cause another form of non-hypotonic hyponatraemia, namely dilutional hyponatraemia.[4] High blood glucose increases the osmolality of the blood, often to greater than 300 mOsm/kg, causing water to move from the intracellular to extracellular space. This dilutes the sodium concentration in the bloodstream causing hyponatraemia. As a rough guide, serum sodium will fall by 2.4 mmol/L for every 5.6 mmol/L rise in blood glucose.[18] This form of hyponatraemia resolves by controlling the blood glucose.

Alcohol[5] and lactate[23] are solutes that, like glucose, can increase serum osmolality and cause a dilutional hyponatraemia. Uraemia may also be associated with non-hypotonic hyponatraemia, with high levels of urea contributing to osmolality and loss of the ability to regulate water balance leading to water retention.[24] Urea is, however, an ineffective osmole because it freely crosses cell membranes. This means that, unlike hyperglycaemia, it does not cause water to move from the intracellular to extracellular fluid space.

Our study has strengths and weaknesses. The main strength is that we were able to conduct a survey of all patients whose serum sodium was measured during an 18-month period in a well-defined population in southwest Scotland and then access the medical records of all those found to have iso- and hyperosmolar hyponatraemia. Limitations are those necessarily expected of a retrospective survey. Not everyone with serum sodium 125 mmol/L or less had serum osmolality, blood glucose and blood ethanol measured simultaneously, nor did everyone with iso- and hyperosmolar hyponatraemia have recent measurements of lipids or immunoglobulins. We acknowledge that this could lead to an underestimation of pseudohyponatraemia in this patient population, though we feel this is unlikely given that serum sodium invariably returned to normal in these patients during follow up.

A further limitation is that we chose a sodium threshold of 125 mmol/L rather than the lower limit of the normal range for serum sodium (135 mmol/L). We may therefore have missed cases of pseudohyponatraemia in patients who were only mildly hyponatraemic. We feel we are unlikely to have missed many, given that most cases of pseudohyponatraemia described in the literature have serum sodium 125 mmol/L or less (Table 1). A future study could consider the prevalence of pseudohyponatraemia in patients with milder hyponatraemia in the range 126-135mmol/L.

CONCLUSIONS

Pseudohyponatraemia is uncommon. We found only one probable case due to hyperlipidaemia and one possible case due to myeloma in a consecutive series of 1,041 patients whose serum sodium was 125 mmol/L or less. Despite its rarity, the recognition of pseudohyponatraemia remains important because it is a laboratory artefact for which no specific treatment is required. Pseudohyponatraemia should be suspected when a patient with low serum sodium has normal or elevated serum osmolality. Point of care testing using a direct ion selective electrode should then be requested to provide immediate and direct measurement of serum sodium.

STATEMENTS AND DECLARATIONS

Contributions: CI had the idea for the project, KH and DF provided the biochemical data, WH extracted the clinical data from the electronic case record. CI and WH wrote the first draft, KH and FG contributed to subsequent drafts and all five authors collaborated in responding to our referees' comments.

Ethical considerations: We did not seek ethical approval as we did not include patient identifiable data, in keeping with our health board's policy.

Consent to participate: Not applicable

Consent for publication: We obtained written, informed consent from our case study (box 1) and have provided this to the editorial team as per the submission guidelines.

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REFERENCES

1. Spasovski G, Vanholder R, Allolio B et al. on behalf of the Hyponatraemia Guideline Development Group, Clinical practice guideline on diagnosis and treatment of hyponatraemia, *Nephrology Dialysis Transplantation* 2014; 29: i1–i39.
2. Ballard M, Chen L. Pseudohyponatremia: A Concise Guide to Diagnosis and Management in Clinical Practice. The Journal for Nurse Practitioners. 2023;19:104800.
3. Theis SR, Bhutta BS, Khandhar PB. Pseudohyponatremia. In: StatPearls[Internet]. Treasure Island (FL): StatPearls Publishing. 2025.
4. Dineen R, Thompson CJ, Sherlock M. Hyponatraemia: presentation and management. Clin Med (Lond). 2017;17(3):263-269.
5. Mathias J, Lovekar S, Yap J. Unmeasured Osmoles: The Hidden Solutes Obscuring a Hyponatremia Evaluation. Cureus. 2024;16(8): e66833.
6. Asadollahi K, Beeching N, Gill G. Hyponatraemia as a risk factor for hospital mortality. *QJM* 2006; 99(12): 877-80.
7. Hoorn EJ, Lindemans J, Zietse R. Development of severe hyponatraemia in hospitalized patients: treatment-related risk factors and inadequate management. Nephrol Dial Transplant. 2006;21:70-6.
8. Yu Z, Parker KM, Blick KE. Markedly decreased serum sodium concentration in a patient with multiple myeloma. Laboratory Medicine. 2005;36:224-6.
9. El Hage L, Reineks E, Nasr C. Pseudohyponatraemia in the setting of hypercholesterolaemia. AACE Clin Case Rep 2018; 5(2): e172-e174.
10. Song L, Hanna RM, Nguyen MK, Kurtz I, Wilson J. A Novel Case of Pseudohyponatremia Caused by Hypercholesterolemia. Kidney Int Rep. 2018;4(3):491-493.
11. Dawson A, Kanukuntla A, Kata P, et al. Pseudohyponatremia Leading to a Fatal Outcome in a Patient With Familial Hypertriglyceridemia. *Cureus* 2021; 13: e17066.
12. Calder M, Duch JM. X Marks the Salt: A Case of Pseudohyponatremia Due to Lipoprotein X: TH-PO329. Journal of the American Society of Nephrology. 2024;35(10S).
13. Hashem N, Abid A, Sola SC, Lewis A. Triglycerides and Sodium: Unmasking Pseudohyponatremia in a Clinical Case. *Cureus.* 2024;16:e74220.

14. Ahmad A, Joseph M. A Case of Pseudohyponatremia in the Setting of Pegaspargase-Induced Hypertriglyceridemia in an Adult T-Cell Lymphoblastic Leukemia Patient. Case Reports in Hematology. 2024.
15. Wani A, Gupta V, Shah KA, Ali I. Intricacies of presentation: pseudohyponatraemia, hyperviscosity and the diagnostic odyssey leading to IgG multiple myeloma. Kidney International Reports 2025; 10: S730.
16. Garcia LP, Frost L, Tucker BM. Hyponatremia with an Osmolar Gap, Pseudohyponatremia or Hyper-Osmolar Hyponatremia?. Am J Med Open. 2024;13:100081.
17. Anonymous. Osmolality Gap: calculation and Interpretation. The University of Iowa. Accessed at www.healthcare.uiowa.edu/path_handbook/Appendix/Chem/OSMO_GAP.html on 14 Sep 2025
18. Hillier TA, Abbott RD, Barrett EJ. Hyponatraemia: evaluating the correction factor for hyperglycaemia. Am J Med. 1999;106:399-403.
19. Holbek CC. Understanding the different values in electrolyte measurements. *Acute Care Testing*. 2025.
20. Weisberg LS. Pseudohyponatraemia: A Reappraisal. Am J Med. 1989; 86: 315-8.
21. Higgins C. Pseudohyponatraemia. Acute Care Testing. 2007.
22. Frier BM, Steer CR, Baird JD, Bloomfield S. Misleading plasma electrolytes in diabetic children with severe hyperlipidaemia. Arch Dis Child. 1980;55(10):771-5.
23. Sood MM, Richardson R. Lactate and the osmolar gap. *CMAJ* 2007;177(5):489.
24. Stern SC. Hyponatremia with Normal or Hyper-osmolality & Pseudohyponatremia. In: Stern SC, Cifu AS, Altkorn D. eds. *Symptom to Diagnosis: An Evidence-Based Guide*, 4e. McGraw-Hill Education; 2020. Accessed August 21, 2025.