Hong Kong College of Physicians
Case Report for Interim Assessment
Specialty Board of Advanced Internal Medicine (AIM)

For AIM Training, case reports should be submitted in the prescribed format together with the application form for Interim Assessment at least EIGHT Weeks before the date of Interim Assessment

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Date(s) and place (hospital) of patient encounter: Jul 2023, QMH
Date of report submission: 15 Sep 2025

Case report

Note: Failure to follow the prescribed format (including the number of words) results in a FAILURE mark (score between 0 and 4) for the Case Report.

Title: Dilution or Delusion? When hydration becomes harmful

Case history:

A 57-year-old previously independent Filipina domestic helper with a background of well-controlled hypertension on amlodipine was admitted to the intensive care unit (ICU), following acute neurological decline. She presented several days prior with symptoms of a presumed gastroenteritis, including nausea, vomiting, and diarrhoea. In response to these symptoms, she had markedly increased her oral water intake. On the 3rd of April, her co-worker witnessed an episode of vomiting followed shortly thereafter by generalized limb twitching and profound confusion. Upon presentation to the Accident and Emergency Department at 7am, she was drowsy, agitated, and had experienced a witnessed generalized tonic-clonic seizure.

Initial laboratory investigations revealed profound hyponatremia with a serum sodium of 114 mmol/L, accompanied by hypokalemia. She was given 100ml of 8.4% sodium bicarbonate in the AED, as well as saline infusions. She was later transferred to the ICU, where her biochemical profile was highly suggestive of syndrome of inappropriate antidiuresis (SIADH): plasma osmolality was markedly low at 244 mmol/kg, while urine osmolality was inappropriately concentrated at 378 mmol/kg with an elevated urine sodium. Computed tomography (CT) angiogram of the brain was urgently and revealed cerebral edema with effacement of the sulci and dilated bilateral superior ophthalmic veins, indicative of raised intracranial pressure.

Her hospital course was complicated by a markedly elevated creatine kinase (7100 U/L), consistent with rhabdomyolysis secondary to her seizure. She was managed with careful hypertonic saline infusion and fluid restriction.

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Her neurological status gradually improved as her sodium levels corrected, and subsequent imaging showed resolution of the cerebral edema. An electroencephalogram (EEG) captured electrographic seizures, prompting the initiation of levetiracetam. Blood cultures drawn on admission flagged positive for Methicillin-Sensitive Staphylococcus aureus (MSSA) and she was empirically started on intravenous co-amoxiclav (Augmentin) for a suspected sepsis. Repeat blood cultures on the 7th and 9th of April were sterile, confirming rapid clearance of the bacteremia. A transthoracic echocardiogram showed a normal ejection fraction with no evidence of valvular vegetations. A subsequent PET-CT scan, performed to look for a metastatic source of infection, was unremarkable aside from mild asymmetrical uptake in the left palatine tonsils.

Given the rapid sterilization, absence of endocarditis, and no evidence of other deep foci, her bacteremia was deemed uncomplicated. She completed a one-week course of intravenous beta-lactams and was stepped down to oral cephalexin. The final piece of the diagnostic puzzle was revealed when a stool PCR returned positive for Norovirus RNA, confirming the initial trigger for her excessive water intake. After a prolonged ICU and general medical ward stay, she was discharged on the 15th of April, without any neurological deficit.

Discussion and literature review

This case presents a fascinating and severe cascade of events, initiated by a common viral pathogen and complicated by a concurrent bacterial infection. The primary driver of the patient's critical illness was acute hyponatremic encephalopathy, supposedly secondary to water intoxication (primary polydipsia), but confounded by an initial biochemistry suggestive of SIADH.

The interpretation of this patient's initial biochemical profile is complicated the administration of intravenous sodium bicarbonate in the AED prior to the collection of urine studies. The reported values — a urine osmolality of 378 mmol/kg and a urine sodium of 111 mmol/L—which initially suggest SIADH, should therefore be viewed as potentially iatrogenically altered and not solely reflective of the patient's underlying pathophysiology. Sodium bicarbonate infusion directly contributes a significant solute load to the extracellular fluid. The kidneys will obligately excrete this excess solute, which requires accompanying water excretion. This process artificially elevates both urine osmolality and urine sodium concentration, effectively masking the classic biochemical signature of primary polydipsia (maximally dilute urine with a low sodium) [1, 2]. Therefore, what appears to be "inappropriate" concentration could, in fact, be an appropriate renal response to the administered bicarbonate, superimposed upon an

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underlying state of water intoxication.

This creates a diagnostic dilemma where the differential diagnosis realistically includes two possibilities. Firstly, a pure primary polydipsia; the patient's norovirus-induced vomiting provided a non-osmotic stimulus for ADH release, a physiologically appropriate response to volume depletion. Her subsequent excessive water intake against this backdrop of physiological ADH activity led to catastrophic water intoxication, i.e. an osmotic shift of water into brain cells, causing cerebral edema and the subsequent catastrophic neurological manifestations, including seizures and altered mental status [2, 3]. The CT finding of dilated superior ophthalmic veins is a recognised, sign of elevated intracranial pressure, further corroborating the severity of the cerebral edema, of which the bicarbonate infusion then confounded the urinary indices.

The alternative consideration would be a genuine SIADH complicated by water intake: The severe infection (either norovirus itself or the subsequently confirmed MSSA bacteremia) triggered a true SIADH. The patient's high water intake, while excessive, would have been particularly dangerous in the setting of this impaired free water excretion. However, the clinical narrative strongly favours the first scenario. The normalisation of sodium with fluid restriction and careful correction [4], alongside the complete resolution of cerebral edema on subsequent imaging, is highly characteristic of water intoxication. Furthermore, the inciting event—norovirus gastroenteritis prompting volitional overconsumption of water [5]—is a well-documented precipitant of hyponatremia.

The concomitant MSSA bacteremia, while a serious complicating factor, likely played a secondary role. The rapid sterilization of blood cultures and absence of a deep-seated focus suggest a transient bacteremia, perhaps from a gut translocation event facilitated by vomiting, rather than being the primary driver of a sustained SIADH. The systemic inflammatory response may have contributed to non-osmotic ADH release, but it is unlikely to have been the sole cause. The asymmetrical tonsillar uptake on PET-CT could suggest a cryptic pharyngeal focus, perhaps a breach in the mucosal barrier facilitated by vigorous vomiting, though this remains speculative.

This case is a stark reminder of the lethality of water intoxication and highlights norovirus as a common precipitant of this dangerous behaviour. Norovirus is a leading cause of acute gastroenteritis globally, responsible for an estimated 685 million cases and over 200,000 deaths annually, with a significant burden falling upon young children and the elderly [5, 6]. The clinical presentation, as seen in our patient, typically includes nausea, vomiting, and diarrhea, and is generally self-limiting. However, severe cases resulting in profound dehydration and electrolyte imbalances, occur

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most frequently in vulnerable populations, with available treatments limited to rehydration and supportive care, underscoring the substantial morbidity associated with this pathogen [6]. Public and patient education on the perils of over-hydration with plain water during illness remains a crucial public health message, advocating for balanced oral rehydration solutions instead [7].

Currently, management of norovirus is entirely supportive, as there are no FDA-approved specific antivirals or vaccines. However, the significant global human and economic burden—estimated at $60 billion annually in societal costs—has driven vaccine development, with several candidates currently in advanced clinical trials. The advent of an effective vaccine could potentially prevent not only the primary gastrointestinal illness but also its rare, severe secondary complications such as the one described here [6].

We are also reminded that this case demonstrates the necessity of a broad diagnostic approach in a critically ill patient, where common and rare aetiologies can coexist. The urinary sodium and osmolality are critical, low-cost tests that precisely differentiate between states of water excess and inappropriate antidiuresis, guiding correct management. We must also note that urinary electrolytes and osmolality must be interpreted in the full context of ongoing treatments, as common interventions can drastically alter their meaning. Ultimately, the clinical history often remains the most powerful diagnostic tool.

The systematic review corroborates that the median water intake associated with life-threatening hyponatraemia is 8.0 L per day [3], a volume our patient likely approached or exceeded, and which aligns with the 99th percentile of population intake [4]. Her presenting sodium of 114 mmol/L was near the median of 118 mmol/L reported in the literature, and her severe neurological manifestations (seizures, coma) were consistent with the 53% of cases that present with severe symptoms [3].

While psychogenic polydipsia —often in patients with chronic psychiatric disorders—was the most common cause, a significant proportion of cases (13%) were iatrogenic, resulting from misinterpreted medical advice [3]. Our patient's behaviour fits neatly into the "other reasons" category (11%), which includes using water as a "self-remedy" for gastrointestinal illnesses like norovirus. This highlights a crucial public health message: well-intentioned but non-specific advice to "drink plenty of fluids" during illness can be dangerously misconstrued, leading to catastrophic overconsumption of plain water instead of electrolyte-balanced solutions [1, 7].

The review underscores the significant morbidity and mortality of this condition, with a 13% mortality rate across all cases. Our patient's course

complicated by rhabdomyolysis was also a known entity, occurring in 7% of reviewed cases, often in association with prolonged seizure activity [3]. Furthermore, a subset of patients developed osmotic demyelination, a feared complication of rapid sodium correction, reinforcing the critical importance of the careful, controlled management she received in the ICU. The patient's survival and excellent neurological recovery are a testament to prompt critical care intervention, meticulous biochemical management, and appropriate antimicrobial therapy.

In conclusion, this case is a classical example of water intoxication as detailed in the largest systematic review on the topic. We are therefore reminded to always interpret investigations contextually; in this case, the administration of sodium bicarbonate created a diagnostic illusion of SIADH, illustrating how treatment can obscure the underlying diagnosis. But this scenario underscores the dangers of hyponatraemic encephalopathy secondary to excessive water intake and highlights norovirus as a common precipitant of this dangerous behaviour. Public and patient education on the perils of over-hydration with plain water during illness remains a crucial public health message, advocating for balanced oral rehydration solutions instead.

Tables and figures (where applicable) (no more than two figures)

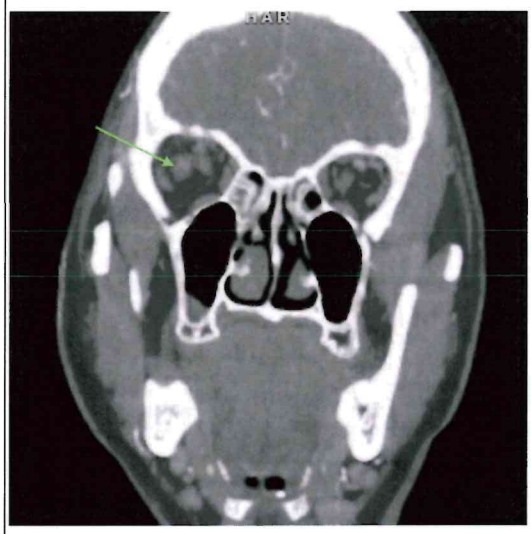

Figure 1. Sagittal view of CT showing dilated bilateral superior ophthalmic veins.

Clinical Diagnosis	/ hypoNa
	Nucleic acid testing
Specimen Type:	Stool
Norovirus RNA	Detected
Rotavirus RNA	Not detected
Astrovirus RNA	Not detected
Enteric adenovirus DNA	Not detected
Sapovirus RNA	Not detected

Figure 2. Stool PCR showing Norovirus RNA detection

Reference (not more than 10)

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5. Glass RI, Parashar UD, Estes MK. Norovirus gastroenteritis. N Engl J Med. 2009 Oct 29;361(18):1776-85. doi: 10.1056/NEJMra0804575. PMID: 19864676; PMCID: PMC3880795.
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7. McCotter L, Douglas P, Laur C, et al. Hydration education: developing, piloting and evaluating a hydration education package for general practitioners. BMJ Open 2016;6:e012004. 10.1136/bmjopen-2016-012004

No of words in Case History and Discussion (excluding references): 1597.
(should be between 1000-2000)

Declaration

I hereby declare that the case report submitted represents my own work and adheres to the prescribed format. I have been in clinical contact with the case selected. The case report has not been submitted to any assessment board or publication and it is NOT related to my second specialty(ies), if any. My consent is hereby given to the College to keep a copy of my case report, in written and/or electronic, at the College Secretariat and allow the public to have free access to the work for reference.

JKY Hooi

(signature of Trainee)

Endorsed by Supervisor *

(signature of Supervisor)

* Supervisors must go over the Case Report with the Trainees, advise Trainees whether further amendments are necessary, review the Originality/ Similarity Report prepared by Trainees, adherence to the required format, sign on the report and remind Trainees on issues related to copyright and plagiarism.

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