



Beer Potomania: Don't Just Stand There, Do Nothing



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INTRODUCTION

- **Beer potomania** is an unusual cause of hyponatremia in excessive beer drinkers who have low daily solute intake.
- **Patients** presenting with beer potomania are at increased risk of osmotic demyelination syndrome (ODS) due to rapid sodium correction.
- **The infrequency** with which beer potomania is encountered and the tendency for patients to present with severe symptomatic hyponatremia make its recognition and management challenging.

CASE PRESENTATION

History of Present Illness:

A 47-year-old man with **history of alcoholism** presented with wrist pain and **altered mental status after ground level fall**. Patient was a housing contractor who on day of hospital admission, had been **working outside in the heat**. He had skipped both breakfast and lunch but drank 1 gallon of water and 8 bottles of Gatorade throughout the day. After returning home from work, he **tripped and fell** in his garage. He denied head trauma or loss of consciousness but complained of severe wrist pain. His daily fluid intake included **1-2 gallons of water, 8 bottles of 32 oz Gatorade, 32 oz coffee, and 4-6 24 oz beers**. He often **skipped meals** and ate mostly toast, pretzels, and occasionally fast food. Recent history included **binge drinking** one day prior to admission.

Past Medical History

Hypertension

Past Surgical History

Hernia repair, L shoulder repair
R knee arthroplasty and ankle repair

Medications Prior to Admission:

Absorbic acid 500 mg daily

Social History:

Alcohol: Four to six 24 oz beers daily
Tobacco: 1 pack every 1-2 weeks
Drugs: Marijuana for 1 month 2 years ago
Occupation: Contractor

Physical Exam:

Vitals: afebrile, hypertensive to 150s/80s
General: well-appearing, awake, no acute distress, cooperative
Neuro: mild confusion, alert and oriented to person, place, and time, normal gait
Head, eyes, ears, nose, throat, heart, lung, abdomen, and extremity exam: benign

Imaging:

Chest, wrists, knees, feet Xray: No acute abnormality, fracture, or trauma



Labs:

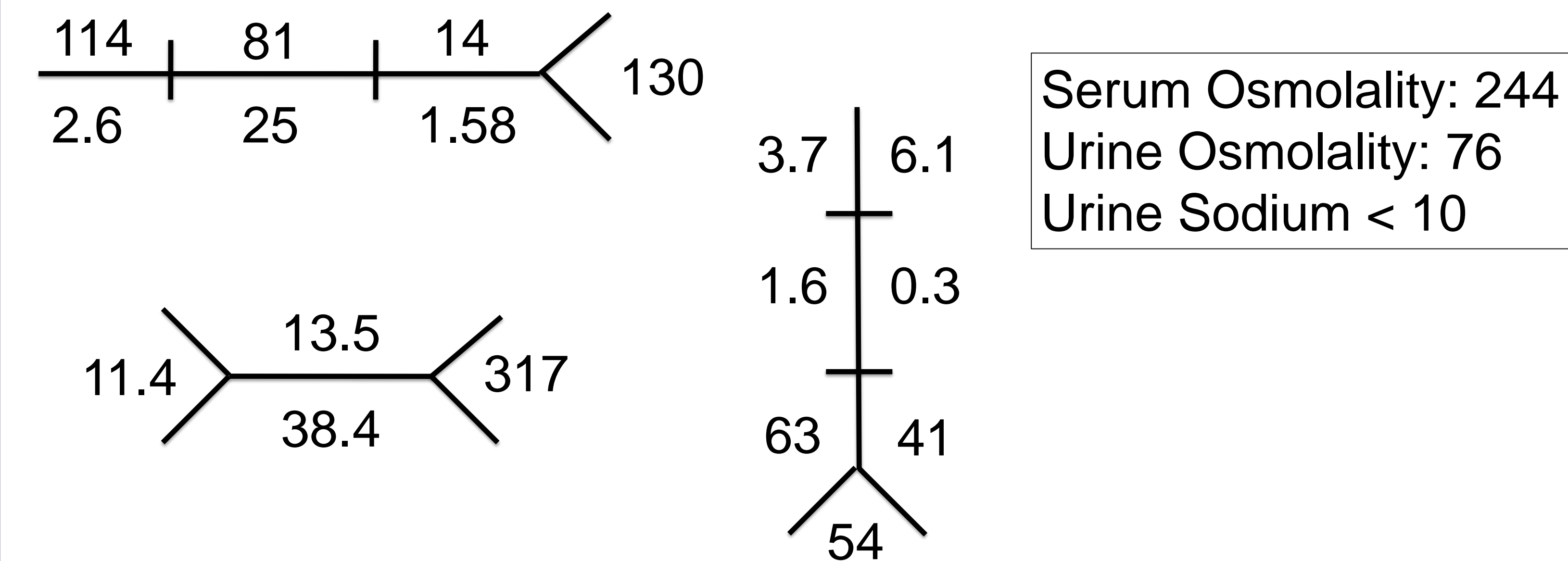


Figure 1. Progression of Serum Sodium

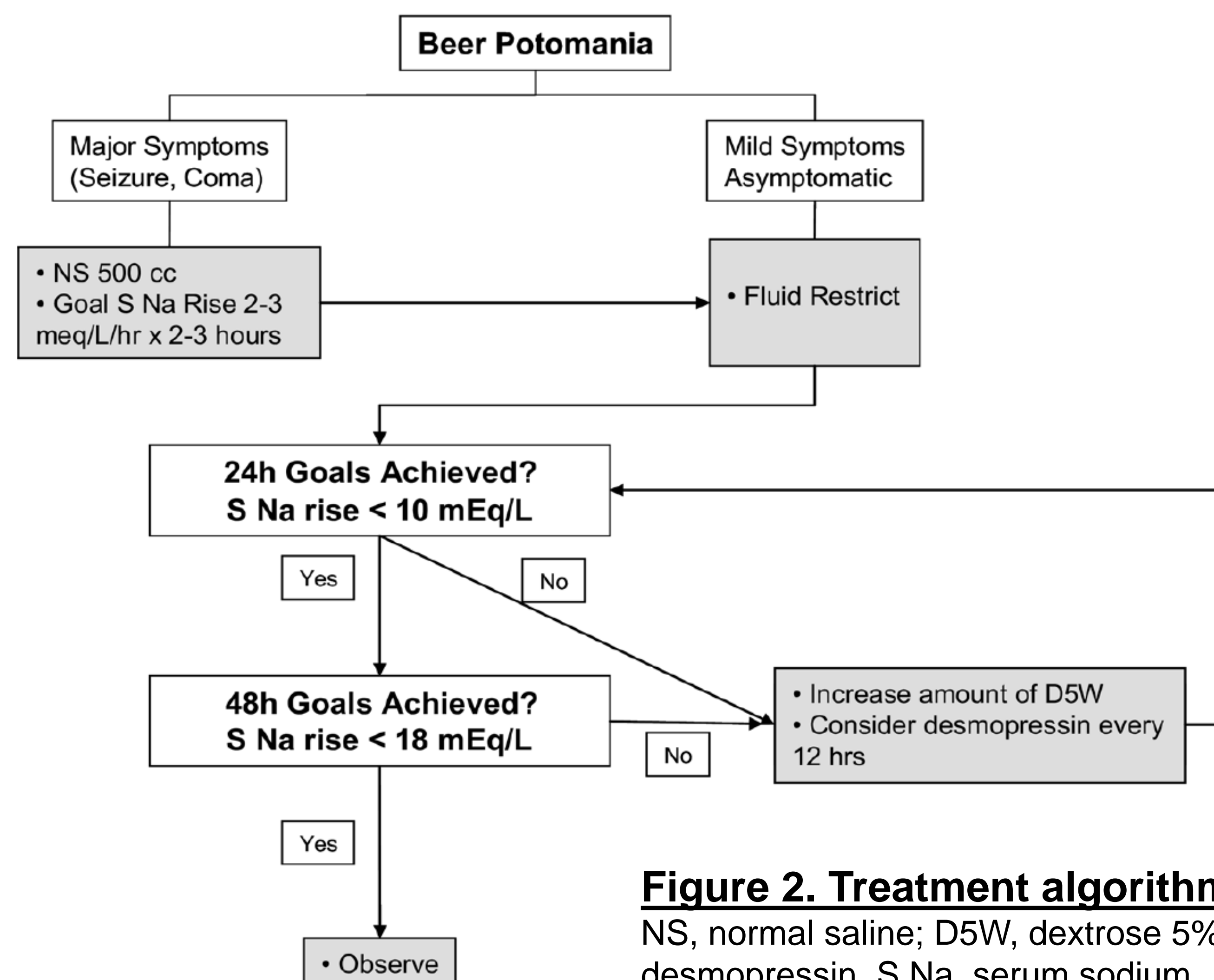
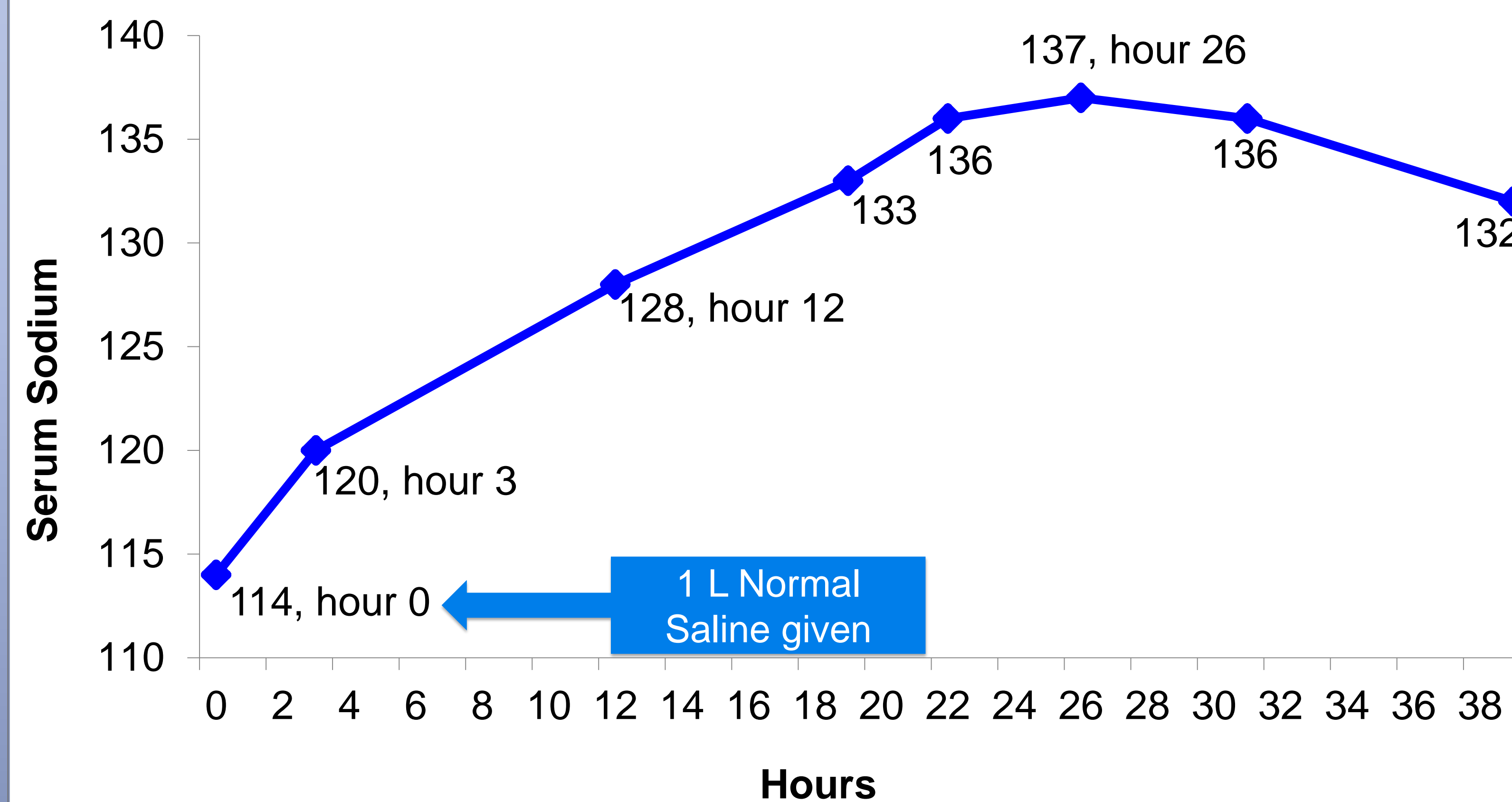


Figure 2. Treatment algorithm¹. Abbreviations: NS, normal saline; D5W, dextrose 5% in water; DDAVP, desmopressin, S Na, serum sodium

DISCUSSION

Pathophysiology

- Water excretion depends on solute excretion and urinary dilution capacity
- Beer has low sodium and protein + poor diet= low total body solute
- Obligatory solute loss is ~250 mOsm/day²
- Kidneys can dilute urine to 50 mOsm/L

250 mOsm / (50 mOsm/L) = 5 L of water used to excrete solute.

*Any fluid intake over 5 L will lead to hyponatremia

- ADH is suppressed³
 - Significant diuresis can occur after giving solute in low ADH state
- Normal saline can cause rapid correction of serum sodium → 18% ODS risk¹

Diagnosis

History and Physical:

- Excess beer drinking + recent binge drinking or illness
- Neurological symptoms- confusion, altered mental status, gait disturbance

Labs:

- Severe hyponatremia
- Hypokalemia
- Low serum osmolality
- Low urine sodium

Management

- Slow correction of sodium, no intravenous fluids unless symptomatic
- Obtain serum sodium levels every 2 hours
- Goal sodium correction:
 - First 24 hours: Increase < 10 mEq/L
 - First 48 hours: Increase < 18 mEq/L

CONCLUSION

1. Beer Potomania= euvolemic hyponatremia + low solute intake + excessive alcohol intake
2. The underlying pathophysiology of beer potomania puts patients at high risk of ODS
3. Early recognition this diagnosis is critical to instituting appropriate treatment and preventing adverse neurological sequelae of overzealous sodium correction.

REFERENCES

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