Case #48

NAME Educational Activities Committee

Case provided by:

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Natural Ice
Natural
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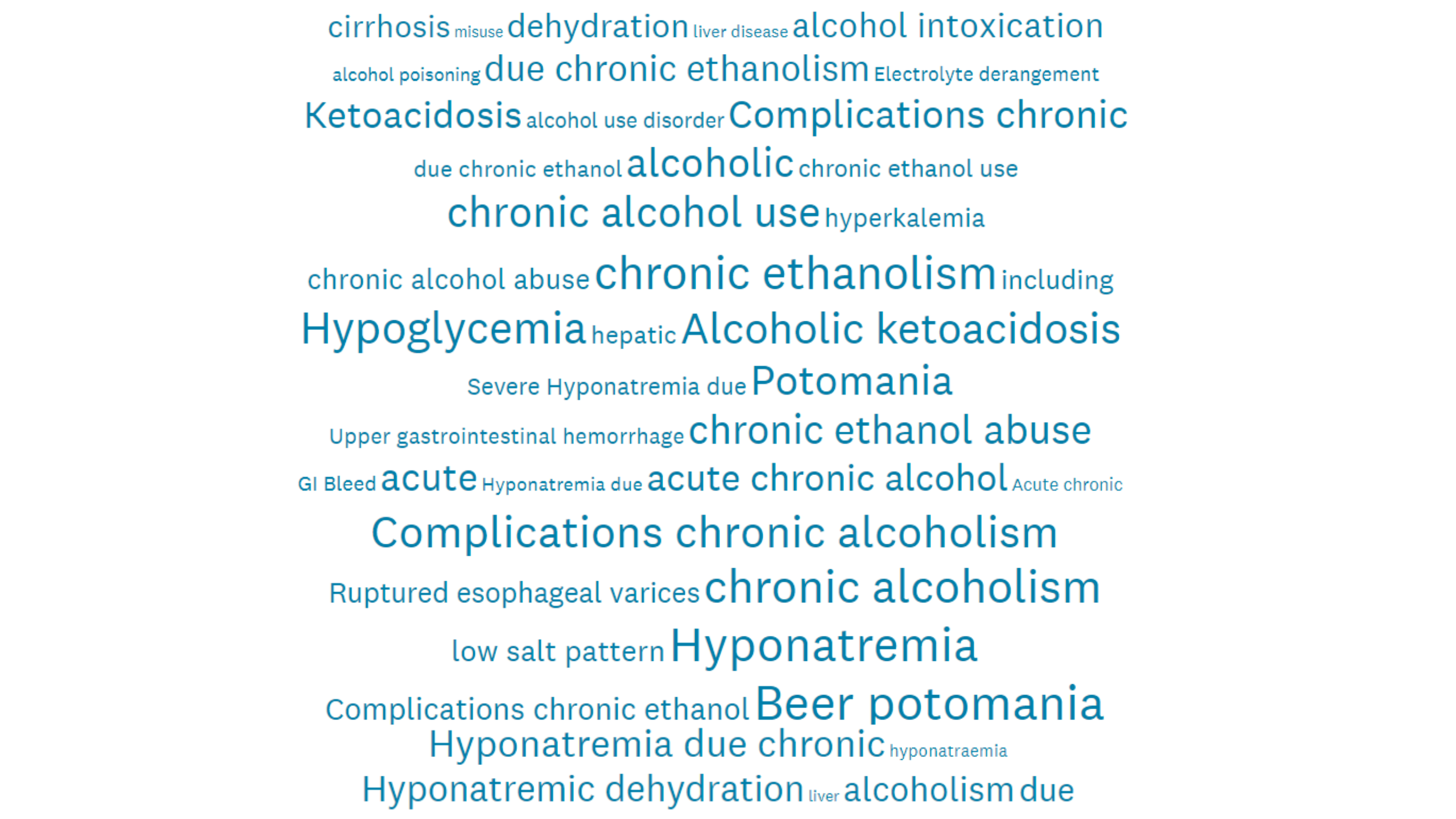
CHEMISTRY PANELS

Test	Result	Flag	Units	Reference Values
Specimen: SST				
Creatinine with eGFR, S				
Creatinine, S	0.59	L	mg/dL	0.74 - 1.35
eGFR Non-Black/African American	>90		mL/min/BSA	>=60
Estimated GFR calculated using the 2009 CKD_EPI creatinine equation.				
eGFR-Black/African American	>90		mL/min/BSA	>=60
Estimated GFR calculated using the 2009 CKD_EPI creatinine equation.				
CHEMISTRY				
Sodium, S	106	CL	mmol/L	135 - 145
Glucose, Random, S	<2	XL	mg/dL	70 - 140
Bld Urea Nitrog(BUN), S	9		mg/dL	8 - 24
Chloride, S	80	L	mmol/L	98 - 107
Potassium, S	13.7	CH	mmol/L	3.6 - 5.2

1. The decedent is a 70-year-old male with a history of chronic alcoholism who was found dead in bed during a welfare check. Autopsy showed steatosis and toxicology showed an alcohol level of 187 mg/dL. Based on the scene and vitreous electrolytes, what is most likely cause of death? (FREE TEXT ANSWER)


cirrhosis misuse dehydration liver disease alcohol intoxication
alcohol poisoning due chronic ethanolism Electrolyte derangement
Ketoacidosis alcohol use disorder Complications chronic
due chronic ethanol alcoholic chronic ethanol use
chronic alcohol use hyperkalemia
chronic alcohol abuse chronic ethanolism including
Hypoglycemia hepatic Alcoholic ketoacidosis
Severe Hyponatremia due Potomania
Upper gastrointestinal hemorrhage chronic ethanol abuse
GI Bleed acute Hyponatremia due acute chronic alcohol Acute chronic
Complications chronic alcoholism
Ruptured esophageal varices chronic alcoholism
low salt pattern Hyponatremia
Complications chronic ethanol Beer potomania
Hyponatremia due chronic hyponatraemia
Hyponatremic dehydration liver alcoholism due

Answer...

Beer Potomania

This case is a classic example of “Beer Potomania Syndrome,” a syndrome of severe hyponatremia seen occasionally in chronic alcoholics with excessive intake of alcohol (particularly “light” beer), together with poor dietary solute intake. The low solute content of beer and suppressive effects of alcohol on proteolysis result in reduced solute delivery to the kidneys. Free-water clearance is dependent on osmole excretion, therefore decreasing dietary osmole intake leads to a decrease in the excretory ability of the kidneys, causing dilutional hyponatremia secondary to excess fluid within the body. The hyponatremia is usually slowly progressive and can reach impressive levels as seen on our case.

These patients can present with fatigue, dizziness and muscular weakness. If rapid correction of the hyponatremia is attempted, they can develop pontine or extrapontine myelinolysis, also known as osmotic demyelination syndrome (ODS), resulting in dysphagia, dysarthria and ataxia. Microscopically these lesion shows symmetrical myelin destruction affecting all the fiber tracts, with a loss of oligodendrocytes.

The isolate nature of vitreous humor (VH) makes it one of the most stable and less affected fluids by post-mortem changes. This case brings up important considerations when evaluating postmortem VH:

VH Sodium, Chloride and Creatinine

These values remain relatively stable in their concentrations in post-mortem samples, and thus may be useful in determining the cause of death, as is seen in this case.

VH potassium

VH potassium levels have been well documented to gradually rise in the postmortem period, with multiple studies attempting to relate this somewhat linear increase to postmortem interval.

Antemortem potassium concentrations are maintained by the action of the ATPase pump. Rapidly after death, potassium leaks from the cells around the body, increasing serum concentrations. Due to breakdown of the potassium ATPase pump and loss of selective membrane permeability, potassium in the serum then diffuses from the retina and lens into the VH. Therefore, the “increased” potassium on our case is a postmortem artifact and is not reflective of antemortem values.

VH glucose

An increase in glycolysis is seen in VH during the early postmortem period, leading normal glucose levels to drop to near zero within a short period after death. However, when blood glucose levels are abnormality high, not all the vitreous glucose will be metabolized. Therefore, a postmortem VH glucose in the normal or elevated antemortem range would indicate a state of hyperglycemia.

In those settings, markers such as beta-hydroxybutyrate (BHB) can be used to assess for ketoacidosis. Increased VH glucose, to a lesser extent, can also be seen with prolonged agony, trauma, resuscitation attempts or surgery.

The “hypoglycemia” seen in our case is therefore a postmortem artifact.

REFERENCES

1. Abbott R, Silber E, Felber J, Ekpo E. Osmotic demyelination syndrome BMJ 2005; 331 :829 doi:10.1136/bmj.331.7520.829
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3. Lodhi MU, Saleem TS, Kuzel AR, et al. "Beer Potomania" - A Syndrome of Severe Hyponatremia with Unique Pathophysiology: Case Studies and Literature Review. *Cureus*. 2017;9(12):e2000. Published 2017 Dec 29. doi:10.7759/cureus.2000