hypertensive up to 190/115. Acute abdominal imaging revealed constipation but no obstruction. His symptoms resolved, returning a few hours later with another episode of acute nausea, vomiting, and severe lower abdominal pain, with blood pressure 210/126 and tachycardia. IV Metoprolol, Hydralazine and pain medications did not significantly improve his blood pressure, he was subsequently started on a Nitroglycerin drip. Abdominal workup was unremarkable, he was stabilized and discharged back to rehabilitation on increased oral medications. He continued to have blood pressure spikes up to 200/124 with nausea, vomiting, and severe abdominal pain until a Clonidine patch was started, after which his blood pressure was better controlled. He was discharged home with continued outpatient therapies. A few weeks later he returned to the ER with nausea and severe abdominal pain, blood pressure at home was 254/185. On exam he was diaphoretic, tachycardic, and tachypneic. A CTA scan was obtained without signs of dissection. A Nitro drip with IV push Hydralazine were not effective at controlling his blood pressure, and so Lisinopril, Amlodipine, and a Clonidine patch were added. Over the next few days he had progressively fewer hypertensive elevations and his symptoms were only present during hypertensive episodes. An extensive workup for secondary hypertension was started. 24-hour plasma and urine Metanephrines were within normal limits. Urine Normetanephrine was elevated to 1266 ug/24h (Ref 88-444). Urine Norepinephrine was elevated to 124 ug/24h (Ref 15-80), Urine Dopamine was elevated to 578 ug/24h (Ref 65-400), and total Catecholamines were elevated to 133 ug/24h (Ref 15-100).

Conclusion:

This case illustrates the variance in presentations for Pheochromocytoma and the importance of maintaining a high index of suspicion for secondary causes in patients with intractable hypertension. While commonly reported symptoms include nausea and hypertension, the presentation of acute abdominal pain as the primary complaint is also an important feature of this disease.

Neuroendocrinology and Pituitary

NEUROENDOCRINE & PITUITARY PATHOLOGIES

Development of a Local Reference Range for Hypertonic Saline-Stimulated Copeptin

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Abstract

Differentiating between primary polydipsia and central diabetes insipidus (DI) can be challenging. The water deprivation test has traditionally been used to diagnosis DI, however has poor diagnostic accuracy (1). Direct measurement of anti-diuretic hormone (ADH) is limited clinically. Copeptin is the C-terminal glycoprotein moiety of ADH prohormone, and correlates well with plasma ADH. Unlike ADH, copeptin is easy to measure (2).

Hypertonic saline stimulated copeptin measurements have recently been described for the diagnosis of central DI. A copeptin cut-off of >4.9 pmol/L has a diagnostic accuracy of 96.5% for distinguishing primary polydipsia from central DI (3). A copeptin assay has recently been established in our laboratory. Validation of hypertonic saline-stimulated copeptin concentrations in our local population is needed before this test can be used with confidence in patients presenting to our institution with polyuria-polydipsia syndrome. The aim of this study was to develop a local reference range for hypertonic saline-stimulated copeptin in healthy volunteers.

Twenty healthy volunteers (10 male and 10 female) were recruited. Subjects underwent a hypertonic saline test, as previously described (3). Hypertonic saline (3%) was administered as an initial 250 mL bolus followed by 0.15 mL/kg/minute until a target serum sodium of ≥150 mmol/L was reached. At this time, blood was drawn for copeptin.

Twelve healthy volunteers (7 females; 5 males) have undergone the study to date. Median age was 28 years (range 26-50); median body weight 75.7 kg (range 57.9 -94.5); median baseline plasma sodium 138 mmol/L (range 136 - 140) and median serum osmolality 289.5 (range 281-297). Median peak sodium was 152 mmol/L (range 150-154) with osmolality 314.5 mmol/kg (range 306-320). Median volume of hypertonic saline infused was 1583 mL (1230-2177) and median hypertonic saline stimulated copeptin was 29.2 pmol/L (9.6-167.4). Overall symptom burden was 5/10 (range 3/10-9/10). There were no serious adverse events.

Development of a local reference range for hypertonic saline stimulated copeptin measurements will assist in interpretation of the test in our local population of patients presenting with polyuria-polydipsia syndrome.

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Diabetes Mellitus and Glucose Metabolism

METABOLIC INTERACTIONS IN DIABETES

Progesterone Receptor Membrane Component 1 Suppresses Type II Diabetes (T2D) Progression via Induced Insulin Signaling in Muscle.

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Progesterone receptor membrane component 1 suppresses type II diabetes (T2D) progression *via* induced insulin signaling in muscle.