patients without night shifts was 2.4% within 24 hours (min 0-max 6.6).
Correlation analysis between carbohydrate metabolism and glucose variability revealed a significant strong relationship between the level of postprandial glycemia and modd (r=0.87, P=0.001) and MAGE (r=0.82, P=0.01), and also established a mean significant correlation between the level of postprandial glucose and Conga (r=0.52, P=0.01) and SD (r=0.61, P=0.05).
Fasting glycemia and congas were moderately correlated (r=0.4, P=0.01).
Weak reliable correlation was found only between HbA1c level and Conga variability index (r=0.27, P=0.04).
Conclusions: the results of the study indicate the lack of adequate glycemic control in persons working night shifts, high variability of glycemia, which is an independent risk factor for cardiovascular disease in patients with DM2. The associative relationship of fasting glycemia and postprandial with the indicator of variability Conga shows that glucose fluctuations during the day in patients are constant.

Neuroendocrinology and Pituitary

CASE REPORTS IN NEUROENDOCRINOLOGY BEYOND THE PITUITARY

Tachycardia and Myocardial Injury Induced Natriuresis - a Rare Clinical Encounter

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MON-248

Introduction:
Hyponatremia in the context of cerebral salt wasting secondary to intracranial events has well been described due to increased release of natriuretic peptides. We describe a case of natriuresis leading to acute symptomatic hyponatremia associated with tachyarrhythmia and myocardial infarction.
Case:
A 72-year-old lady was admitted with atrial fibrillation with rapid ventricular rate between 130-150 beats per minute, troponin positive chest pain, with high sensitivity troponin T of 1307 ng/L (<14 ng/L) and managed as type II myocardial infarction. 12 hours after admission she became acutely confused, agitated, associated with visual hallucinations and myoclonic jerks. On Examination she was dehydrated and there were no focal neurological features. Investigations showed acute hyponatremia with serum sodium (Na^+) of 117 mmol/L (135 - 145 mmol/L) which 12 hours earlier, on admission, was 137 mmol/L, serum osmolarity 249 mosm/kg (275 - 295 mosm/kg), urine osmolarity 486 mosm/kg, urinary sodium of 160 mmol/L, pro-NTBNP 6575pg/ml (<450 pg/ml), 9am serum cortisol 575 nmol/L (140 - 690 nmol/L), TSH 4.72 mu/L (0.2 - 5.0 mu/L), FT4: 18.6pmol/L (10.0 - 24.0 pmol/L) and normal chest x-ray. She was treated with hypertonic saline (2.7%) 200ml followed by 0.9% saline infusion that corrected her serum sodium levels to 136 mmol/L over next 48-72 hours along with clinical symptoms gradually went back to normal. Repeat pro-NTBNP after recovery was 3153 pg/ml. Echocardiography showed normal left ventricular systolic function with normal atrial size and moderate mitral regurgitation with pulmonary artery pressure of 30mmHg. Based on acute onset hyponatremia and raised urinary sodium, we proposed diagnosis of hyponatremia secondary to salt wasting and we believe that in absence of acute intracranial pathology and raised pro-NTBNP, renal salt wasting was induced by acute rise in natriuretic peptides of cardiac origin either secondary to myocardial infarction or tachyarrhythmia. Paroxysmal SVT is a known cause of transient polyuria after termination but is not known to cause hyponatremia. Although well described in context of intracranial events, this is the first case of myocardial injury or tachyarrhythmia induced natriuresis leading to acute hyponatremia.
Conclusion:
Salt wasting due to natriuretic peptides is not exclusive to intracranial events. Any cardiac event leading to sudden increase in natriuretic peptides can lead to natriuresis which if prolonged enough can lead to acute symptomatic hyponatremia.
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Thyroid

THYROID DISORDERS CASE REPORTS II

Myxedema Madness: A Rare Case of Severe Hypothyroidism Presenting as Psychosis

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SAT-480

Myxedema Madness: A Rare Case of Severe Hypothyroidism Presenting As Psychosis
Introduction
Myxedema coma is a rare, life-threatening medical emergency resulting from uncontrolled hypothyroidism. Myxedema coma refers to the neurological sequelae of severe hypothyroidism, which classically manifests as depressed mental status. Rarely, myxedema coma can present with a hyperactive mental state and psychosis. We present an unusual case of a drug overdose secondary to myxedema coma-induced psychosis.
Clinical Case
A 48 year old woman with a history of seizure disorder and hypothyroidism presented to the hospital after lamotrigine overdose. The patient's spouse witnessed her ingest forty-five tablets of lamotrigine after an argument. The patient had no previous psychiatric diagnoses or suicide attempts. On examination, the patient was hemodynamically stable but was agitated, disoriented, and uncooperative. She had a normal neurologic exam and no peripheral edema. Her lamotrigine level was 25.4 ug/ml (2.5-15.0 ug/ml). The patient's mental status did not improve with lamotrigine