Diabetes Mellitus and Glucose Metabolism

DIABETES COMPLICATIONS II

An Overlooked Cause of Diabetic Pain

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MON-670

Persistent hyperglycemia has been associated with vascular damage in patients with uncontrolled diabetes. Special emphasis has been placed on the heart, kidneys, eyes, and brain since those major organs are vital. However, little has been studied in terms of the vascular supply to the muscle and how it could be affected by high blood glucose. Here we present a 26-year-old female with a history of uncontrolled Type 1 Diabetes Mellitus treated with insulin pump who presented with muscle aches on her right lower extremity. During the evaluation at the Emergency Department (ED), the patient was noted to have diabetes ketoacidosis, intravenous fluids and insulin drip were started. As part of the workup for the muscle aches multiple blood studies were ordered including Creatinine Phosphokinase (CPK) 26 IU/L (25 - 185 IU/L), Erythrocyte Sedimentation Rate (ESR) 102 (25 - 185 IU/L), Erythrocyte Sedimentation Rate (ESR) 102

Diabetes Mellitus and Glucose Metabolism

CLINICAL STUDIES IN OBESITY, DIABETES RISK, AND CARDIOVASCULAR OUTCOMES

Glucose Intolerance Modifies the Association Between Insulin-Like Growth Factor-1 and All-Cause Mortality

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Background: Despite an increase in literature on insulin-like growth factor-1 (IGF-1) and its impact on insulin sensitivity, there remains controversy over its association with all-cause mortality. Insulin interacts with IGF-1 and its binding proteins, forming a growth hormone/IGF-1/insulin axis that may be impaired in Type II diabetes and/or prediabetes. We hypothesized that the association between insulin and IGF-1 with all-cause mortality differs in those with glucose intolerance (GI) in a nationally representative U.S. population with long-term follow-up. Methods: A total of 5,283 non-pregnant adults >20 years from the National Health and Nutrition Examination Survey (NHANES)-III (1988-1994) were linked to the National Death Index through 2015. Glucose intolerance was classified as per fasting blood sugar (≥100 mg/dl), hemoglobin A1c (≥5.7%), medication use, or self-reported diagnosis. IGF-1 was categorized into 0 - 20), C-Reactive Protein (CRP) 3.4 mg/dL (≤0.80 mg/dL), Aldolase 7.5 U/L (≤8.1 U/L), White Blood Cell (WBC) 13.1 B/L (4.0 - 11.0 B/L). At this point, a muscle biopsy was considered given the lack of evidence to support a definitive diagnosis. Before proceeding with the biopsy, a Magnetic Resonance Imaging (MRI) of the low extremities was done, showing diffuse intramuscular edema, predominantly in the right vastus intermedius, with additional patchy intramuscular edema in the right vastus lateralis, vastus medialis, and biceps femoris, as well as the left gluteus maximus, vastus lateralis which were compatible with myositis. Also, discrete areas of myonecrosis in the right vastus intermedius (1.7 x 1.1 x 3.6 cm), left vastus lateralis (1.7 x 0.8 x 6 cm) and left gluteus maximus (2.8 x 3 cm x 6 cm). Given her previous history of uncontrolled diabetes, the clinical presentation with low CPK levels, lack of data to support another diagnosis, and MRI findings the possibility of diabetes myonecrosis was raised. The patient was managed with conservative therapy: intravenous fluids, pain control and aspirin with improvement in myalgias and muscle strength. Diabetic myonecrosis is a rare condition that appears to be related to vasculopathich changes on uncontrolled diabetes. The lack of specific diagnostic tools and the nonspecific symptoms could make this condition to be overlooked easily; leading to unnecessary studies like muscle biopsy with consequences from complications and increased health care expenditure. A high index of suspicion is essential for timely treatment, which is limited to rest, optimal glycemic control, pain control and patients who are candidates low-dose aspirin. This condition resolves spontaneously over a few weeks to months in most patients and acknowledging this condition could provide timely relief and reassurance.
sex-specific quartiles. To analyze the joint impact of insulin and IGF-1, we also categorized participants into four groups: Group I) IGF-1 <230 ng/mL & insulin ≥11 uIU/mL, II) IGF-1 <230 & insulin <11, III) IGF-1 ≥230 & insulin ≥11, and IV) IGF-1 ≥230 & insulin <11. Our primary outcome was all-cause mortality. We used survey design-adjusted Cox regression to estimate the risk of all-cause mortality, adjusting for confounders. Results: Among the 5,283 subjects, 2,214 (42%) had GI. Participants had a mean follow-up of 22.1 years, during which 1,835 (34%) of them died. Those with GI in the highest quartile of IGF-1 had an unadjusted 64% lower risk of all-cause mortality compared to the lowest quartile (GI = unadjusted OR [95% CI]: 0.37 [0.24,0.55]). This association, although protective, was significantly less protective than those with normal glucose tolerance (NGT) (unadjusted OR: 0.16 [0.12,0.23]). After adjusting for confounders, these associations became insignificant (GI = aOR: 1.02 [0.73,1.42], NGT = aOR: 1.04 [0.74,1.46]). When estimating risk of mortality among joint groups of insulin and IGF-1 levels, those in Group I with NGT had 20% increased adjusted odds of mortality (1.30 [1.01,1.71]), while in GI subjects, there was an insignificant increased odds of mortality (1.17 [0.84,1.62]). Neither subgroup in Group 2 had significant adjusted odds of mortality relative to Group 4. Group 3 subjects with GI had an adjusted, insignificant 24% increased odds of mortality (1.24 [0.91, 1.70]) compared to 70% increased odds in NGT subjects (1.69 [1.18, 2.42]). Conclusion: The differences in odds of all-cause mortality across IGF-1 quartiles in glucose tolerant vs. intolerant individuals suggests that IGF-1 may play less of a protective role in Type II diabetes and prediabetes. Among those with normal glucose, higher insulin levels, regardless of IGF-1 levels, was associated with all-cause mortality. This association did not hold in those with glucose intolerance.

Adrenal
ADRENAL CASE REPORTS II
A Case of Ectopic Cushing's Syndrome with Major Complications Leading to Treatment Dilemmas
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Background: Ectopic ACTH syndrome is an uncommon cause of Cushing’s syndrome and patients are at high risk for serious complications including infections, thromboses, cardiovascular and neuropsychiatric complications; therefore, swift diagnosis and treatment is needed. Clinical Case: A 63-year old woman presented with lower extremity edema and severe lower back pain. She also reported a 2 month history of polyuria, polydipsia, and headaches. Initial labs showed hypernatremia, hypokalemia, alkalosis and hyperglycemia and an 8AM cortisol of 61.9 mcg/dL (N 5-25 mcg/dL) with an ACTH of 367 pg/mL (N 3.5-45 pg/mL). Imaging showed a right middle lobe lung mass and 3 vertebral body compression fractures. A failed attempt at biopsy of the lung mass resulted in pneumothorax, air embolism, stroke, and cardiac arrest with shock liver. She was then transferred to our tertiary care center.

Tumor Biology
TUMOR BIOLOGY: GENERAL, TUMORIGENESIS, PROGRESSION, AND METASTASIS
A Tough NUT (Midline Carcinoma) to Crack
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SAT-125
Introduction
NUT midline carcinoma (NMC) is a rare, highly aggressive cancer with poor prognosis. To date, there is no established therapeutic strategy; further studies are necessary to compare treatment modalities as well as investigate novel immunotherapeutic agents.

Case presentation
A 39-year-old female presented with a rapidly enlarging right neck mass and a recent 40-lb weight loss and was found to have a 5 cm poorly defined infiltrative hypointensating mass on CT scan concerning initially for anaplastic thyroid malignancy. Endocrinology and ENT were consulted and the patient underwent thyroid biopsy, which was positive for NMC. PET scan demonstrated hypermetabolic