

ages 60-85y, with and without prior fracture. We used the National Osteoporosis Foundation (NOF) recommendations for treatment based on BMD (osteoporosis by BMD, or osteopenia by BMD with a 10-year risk of hip fracture $\geq 3\%$ or 10-year risk of major osteoporotic fracture $\geq 20\%$).

RESULTS: The mean BMD for this cohort was 0.670 g/cm² and the median T scores were -2.0 (male reference) and -1.7 (female reference). Using the male T-score, 29% of men were classified as having osteoporosis, while using the female T-score, only 21% were so classified. 36% of men age 70-79y and 19% of men age 80-85y with osteoporosis (using the male T-score) would be reclassified from osteoporosis to osteopenia when a female T-score is used. Hypothetical cases of men age 60-85y (height 170 cm, weight 70 kg, BMD 0.590 g/cm² equivalent to a male T -2.5 or female T -2.2) were used to calculate 10-year hip fracture risk using FRAX. For these hypothetical cases, the calculated 10-year risk of hip fracture exceeded the NOF treatment threshold of 3% (10-year hip fracture risk) for all cases, with or without prior fracture.

CONCLUSION: For elderly men with fracture with male-T osteoporosis and female-T osteopenia, the T-score reference population used does not alter treatment recommendations because the calculated hip fracture risk is already above the treatment threshold of 3%. This is also true for men age ≥ 70 without a prior fracture. Hence the debate pertaining to the appropriate T-score reference population for men has limited relevance for men age ≥ 70 years who are being screened for osteoporosis.

Diabetes Mellitus and Glucose Metabolism

DIABETES TECHNOLOGY

Glycemic Profile of Intravenous Glucocorticoid Induced Hyperglycemia Using Continuous Glucose Monitoring

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Background: Intravenous (IV) steroids are widely used in critically ill patients and with chemotherapy. It is well known that glucocorticoid-induced hyperglycemia (GCIH) occurs within 3 hours following oral administration of steroids with typical postprandial glycemic excursions lasting 24-36 hours. The recent increased availability of Continuous Glucose Monitoring (CGM) has allowed a detailed description of glycemic fluctuations in patients receiving steroids in different settings, however there is no reported observation of CGM findings following a single dose of IV Dexamethasone in a patient with diabetes. We present a case of glycemic pattern documented on CGM of a patient with type 2 diabetes, who had received 11 cycles of a single dose Dexamethasone-containing chemotherapy.

Clinical Case: The patient is 70 years old female with history of type 2 DM of 19 years duration and metastatic pancreatic adenocarcinoma, diagnosed in November 2018,

and treated with Fluorouracil and Dexamethasone 6mg IV on every other Wednesday since December 2018. Her diabetes was fairly controlled on Metformin, Repaglinide, Pioglitazone and Detemir insulin. Premeal Lispro was added while Metformin and Repaglinide were discontinued with the beginning of chemotherapy. She started using Freestyle Libre CGM in January 2019. During her visit in March 2019, the patient was taking Detemir Insulin 50 units in AM and 30 units at night, and Lispro 15 units before meals, in addition to correction insulin based on an Insulin Sensitivity Factor (ISF) of 1:25 for Blood Glucose (BG) above 200mg/dl. Unlike the reported postprandial hyperglycemic excursions associated with oral steroids, the patient's CGM data showed a reproducible triphasic glycemic pattern following IV Dexamethasone, consisting of a steady state of hyperglycemia reached within 3 hours and lasting around 18-30 hours, followed by a transient BG improvement for 18-20 hours, and ending with another hyperglycemic plateau of 10-16 hours on day 3 post chemotherapy, with no association to meal intake. Given this recurrent pattern, the patient was advised to increase her bedtime Detemir insulin from 30 to 45 units and her correction ISF from 1:25 to 1:18 on days 1 and 2 after chemotherapy, with subsequent attenuation and shortening of GCIH. **Conclusion:** Our case report is the first one to describe CGM documented glycemic profile following a single dose of IV Dexamethasone in a patient with type 2 diabetes treated with insulin. The CGM data reveals a consistent steady GCIH, lasting around 48 hours, and reflecting the prolonged action of Dexamethasone. The transient BG improvement seen on day 2 is likely due to the Detemir dose self-increase and the carbohydrates intake decrease in response to day 1 hyperglycemia. A 48 hours modified insulin regimen based on higher dose of long acting and correction insulin improved Dexamethasone induced hyperglycemia.

Tumor Biology

ENDOCRINE NEOPLASIA CASE REPORTS I

A Case of Coexisting Insulinoma and Islet Cell Hyperplasia

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Introduction

Insulinoma is the most common neuroendocrine tumour of the pancreas and cause of endogenous hyperinsulinemia hypoglycaemia. Islet cell hyperplasia is a rare cause of hypoglycaemia in adults.

Clinical Case

A 42-year-old lady presented with hyperphagia, giddiness, decreased concentration and weight gain of 10 kg over one year. Her symptoms occurred both during fasting and postprandial. She did not have any medical conditions and did not take alcohol.

A 72-hour fast confirmed the presence of endogenous hyperinsulinemia; serum glucose of 2.4 mmol/L paired with insulin 8.14 mU/L and C-peptide 0.71 nmol/L occurring after 16 hours of fasting. Screening for sulphonylureas and

meglitinides was negative. Serum beta-hydroxybutyrate was 0.1 mmol/L with a 1.6 mmol/L rise in serum glucose post 1 mg glucagon administration. Computed tomography (CT) of the abdomen showed a 13 X 11 X 15 mm exophytic lesion at the superior aspect of the pancreatic body and another exophytic projection measuring 9 X 8 X 6 mm arising from the tail. In view of possible multifocal insulinoma, a ⁶⁸Ga-DOTATATE scan was performed and it showed an intensely DOTATATE-avid exophytic nodule arising from the pancreatic body and a second indeterminate DOTATATE-avid nodule close to the pancreatic tail. In addition, there was diffuse DOTATATE uptake in the tail of pancreas.

She underwent enucleation of pancreatic body nodule and spleen-saving distal pancreatectomy as the pancreatic tail nodule was not seen intra-operatively. Histology showed an insulinoma; a well-differentiated neuroendocrine tumour (Grade 1, pT1 N0 Mx) that was positively stained for synaptophysin, CD56, insulin, SSTR2 and SSTR5. The pancreatic tail nodule and distal pancreatectomy specimen showed islet cell hyperplasia; the pancreatic parenchyma showed multiple foci of well-circumscribed nests of bland islet cells with similar morphology to those seen in the insulinoma. She did not have further hypoglycaemia episodes post-operatively.

Concomitant presence of fasting and postprandial hypoglycaemia may suggest underlying dual pathology. Clinical and biochemical differentiation between insulinoma and islet cell hyperplasia is difficult. Therefore, imaging for diagnosis and precise preoperative localisation is important for successful resection of suspected lesions. ⁶⁸Ga-DOTATATE scan can be as useful as ⁶⁸Ga-DOTANOC and ⁶⁸Ga-DOTATOC scan and is better than CT scan in localising not only insulinoma but also islet cell hyperplasia. In this case, islet cell hyperplasia-induced hyperinsulinemic hypoglycaemia may have persisted if distal pancreatectomy was not performed.

Conclusion

Adult-onset endogenous hyperinsulinemia hypoglycaemia can be caused by concurrent insulinoma and islet cell hyperplasia. ⁶⁸Ga-DOTATATE scan may be a useful, non-invasive investigation, especially in cases where CT imaging suggests multifocal disease.

Thyroid

HPT-AXIS AND THYROID HORMONE ACTION

Delayed TSH Elevation in Small for Gestational Age Infants: A Need for Second Screening?

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The incidence of CH with a delayed TSH elevation was higher in ELBW and VLBW infants compared with infants weighing ≥ 1500 grams. Second screening should be considered in preterm neonates, low birth weight (LBW) and very low-birth weight (VLBW) neonates, ill and pre-term newborns admitted to NICU, specimen collection within the first 24 hours of life, and multiple births (particularly same-sex twins). Purpose of this study was to determine incidence of delayed TSH elevation with or without congenital hypothyroidism in SGA infants and to

investigate necessity for second screening. Retrospective analysis was performed. 66 SGA newborns with 34-40 weeks' gestation born at Keimyung University Dongsan Medical Center from 2015 to 2018 were enrolled. Primary screening was performed 48 hours - 7 days after birth. Second screening including venous TSH and venous free T4 at postnatal 8-40 days. Exclusion criteria were infants with congenital hypothyroidism at primary screening (NBS), descendants of mothers with immune thyroid disease, congenital malformations, renal, hepatic, and metabolic diseases, history of steroid or dopamine usage. Initial NBS were collected onto pre-printed filter at the age of 2-7 days by heel prick. (normal TSH < 10 mIU/L). Second sample was obtained at the age of 8-49 days by venous sampling (normal TSH < 5 mIU/L). TSH and free T4 were measured on venous samples with Cobas 8000 e801 (electrochemiluminescence, Roche, Diagnostics, Basel, Switzerland) using standard methods.

Incidence of delayed TSH elevation was 27% (18/66). Of them number of transient hyperthyrotropinemia was 13. Mean TSH at initial elevation was 7.56 mIU/L and median age at initial TSH elevation was 18.6 days. Median age at resolution of TSH elevation was 41.5 days. Number of hypothyroidism undergoing l-thyroxine medication was 5. Mean TSH at initial elevation was 22.1 mIU/L. Median age at initial TSH elevation was 14 days. Mean peak TSH was 23.4 mIU/L.

The presence of delayed TSH elevation was not related to very low birth weight. SGA infants might be at a risk of delayed TSH elevation. Considering 2nd screening test within 1 month. Further study with more SGA infants are needed. Limitation of this study was relative small number of patients and iodine status was not considered

Bone and Mineral Metabolism

CLINICAL ASPECTS OF OSTEOPOROSIS AND VITAMIN D ACTION

Fracture Site in High-Energy Trauma Is Associated with Osteoporosis Risk.

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Background: In our recent studies, we noted that patients with history of high energy fractures commonly have underlying endocrine abnormalities and low bone mineral density (BMD). In this expanded patient population, we aimed to investigate whether the fracture site can better predict the risk of abnormal BMD.

Methods: We prospectively enrolled adult patients of both genders, with any history of high energy fracture. We measured serum PTH, vitamin-D and calcium and we performed BMD measurements with a DEXA scan. We split our subjects' BMD, based on the lowest T- or Z-score in "Normal" (≥ -0.9), "low bone mass" (LBM) (-1.0 to -2.4)