

distal left femoral vein extending to the popliteal vein and was started on xarelto, but he developed pleuritic chest pain and dyspnea in 48 hours. CT scan confirmed a pulmonary embolism and patient was treated with heparin drip. Two days following heparin drip patient developed acute bilateral flank pain and hypotension; and CT abdomen showed 2 masses replacing the adrenal glands that were concerning for hematomas. Laboratory results: serum potassium 4.9 mmol/L, serum cortisol 3.3 mcg/dL (reference 7.2–19.4), ACTH level 319 pg/mL (reference 7–53), aldosterone <1.0 ng/dL (reference 0.0–3.0), and plasma renin activity 7.17 ng/ml/hr (reference, 0.15–3.95). Serum antiphospholipid antibody testing showed cardiolipin Ab Ig 140 GPL/mL (reference 0–14), cardiolipin Ab IgM 100 MPL/mL (reference 0–12) and cardiolipin Ab IgA >150 APL/mL (reference 0–11). Further testing showed beta-2 glycoprotein 1 Ab IgG 103 GPI units (reference 1–20), IgM 94 GPI units (reference 0–32), and IgA 150 GPI units (reference 0–25). His hypotension dramatically improved upon administration of IV hydrocortisone and the abdominal pain resolved in 3 days. Upon discharge he was placed on hydrocortisone and continued warfarin therapy. At a 6-week follow-up visit, patient was asymptomatic. Additional lab tests revealed normal plasma renin activity and aldosterone levels. Two years later an ACTH stimulation test confirmed persistent AI. Basal plasma ACTH level was 230 pg/mL (ref 5–50). Additionally plasma renin activity and serum aldosterone levels indicated no mineralocorticoid deficiency. An adrenal CT scan revealed significant long-term interval decrease in size of bilateral adrenals with hypo-attenuating focus in the right adrenal gland, favored to represent post hemorrhage changes without convincing evidence of underlying neoplasm especially given decrease in size compared to 4-years prior. Presently, patient is doing well on hydrocortisone and warfarin treatment. **Conclusion:** In all cases of adrenal hemorrhage and infarction with unknown etiology, screening with lupus anticoagulant and anticardiolipin antibodies is imperative. Recognition of this high mortality condition will allow for appropriate screening and confirmatory tests leading to prompt diagnosis and timely management.

## Diabetes Mellitus and Glucose Metabolism

### CLINICAL AND TRANSLATIONAL STUDIES IN DIABETES

#### *Diabetes Risk for Non-Obese Subjects in a Japanese Population*

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#### MON-662

[Background] Obesity is a major risk factor of developing diabetes and cardiovascular diseases, though not all obese people develop these conditions and diseases. Because Asian populations have a lower frequency of obesity in comparison

with populations in the United States and Europe, it is important to detect risk factors for developing diabetes in non-obese Japanese populations. [Objectives] To examine risk factors for diabetes, and to consider countermeasures against diabetes development in Japanese populations, especially non-obese individuals. [Methods] This study examined 1,794 individuals (514 males and 1,280 females) who participated in both Adult Health Study health examinations on A-bomb survivors and their controls in Hiroshima and Nagasaki between 1994–1996 (baseline) and 2008–2011. They were aged 48–79 years and had not been diagnosed with diabetes at baseline or cancer. Obesity was defined as a BMI of 23 kg/m<sup>2</sup> or greater based on the WHO recommendation for Asians. In accordance with AHA/NHLBI criteria for diagnosis of metabolic syndrome, we defined a diagnosis of metabolic abnormality as having at least two of the criteria other than abdominal obesity. The diagnostic criteria for diabetes were a fasting blood glucose  $\geq 126$  mg/dL, a non-fasting blood glucose  $\geq 200$  mg/dL, a self-report of a diabetes diagnosis, or the initiation of medical treatment for diabetes during the follow-up period. We compared presences of fatty liver and metabolic abnormality, BMI at baseline, and changes of body weight from baseline between the group that developed diabetes and the group did not over a 15-year follow-up. [Results] During the follow-up period until 2001, 66 (7.0%) individuals and 127 individuals (14.8%) from the non-obese and obese groups, respectively, developed diabetes. BMI at baseline and presences of fatty liver and metabolic abnormality were associated with developing diabetes in both non-obese and obese groups. Changes in body weight from baseline were not a significant risk factor of diabetes in this study. Furthermore, we analyzed the association between diabetes risk and appendicular lean mass/height<sup>2</sup> (ALM/H<sup>2</sup>) and handgrip strength based on the diagnostic criteria for sarcopenia among 676 subjects with information of these measurements at baseline. Occurrences of low ALM/H<sup>2</sup> were associated with developing diabetes, but an association between low handgrip strength and developing diabetes was not observed. [Conclusion] Regardless of whether obesity was observed or not, presences of metabolic abnormality and fatty liver were significant risk factors. Increased risk of developing diabetes was observed among non-obese individuals with suspected sarcopenia. This study suggests that maintenance of muscle mass may be an effective countermeasure to reduce the risk of developing diabetes.

## Adrenal

### ADRENAL CASE REPORTS II

#### *Primary Aldosteronism and Klinefelter's Syndrome: Two Cases*

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**SUN-161**

**Background:** Primary aldosteronism (PA) is more common than expected. Aberrant adrenal expression of LH receptor in patients with PA has been reported, however, its physiological role on the development of PA is still unknown. Herein, we report two unique cases of PA in patients with untreated Klinefelter's syndrome, characterized as increased serum LH, suggesting a possible contribution of the syndrome to PA development. **Clinical Cases:** Case 1 was a 39-year-old man with obesity and hypertension since his 20s. His plasma aldosterone concentration (PAC) and renin activity (PRA) were 220 pg/mL and 0.4 ng/mL/h, respectively. He was diagnosed as having bilateral PA by confirmatory tests and adrenal venous sampling (AVS). Klinefelter's syndrome was suspected as he showed gynecomastia and small testes, and it was confirmed on the basis of a low serum total testosterone level (57.3 ng/dL), high serum LH level (50.9 mIU/mL), and chromosome analysis. Case 2 was a 28-year-old man who had untreated Klinefelter's syndrome diagnosed in his childhood and a two-year history of hypertension and hypokalemia. PAC and PRA were 247 pg/mL and 0.3 ng/mL/h, respectively. He was diagnosed as having a 10 mm-sized aldosterone-producing adenoma (APA) by AVS. In the APA, immunohistochemical analysis showed co-expression of LH receptor and CYP11B2. **Conclusion:** Our cases of untreated Klinefelter's syndrome complicated with PA suggest that increased serum LH levels and adipose tissues, caused by primary hypogonadism, could contribute to PA development. The possible complication of PA in hypertensive patients with Klinefelter's syndrome should be carefully considered.

**Thyroid****THYROID DISORDERS CASE REPORTS II*****The Weary Beating Heart: Complications of Severe Hypothyroidism in a Mentally Ill Patient.***

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**SAT-489**

Myxedema coma is a severe form of hypothyroidism representing an endocrinologic emergency. It requires prompt identification and management, as mortality rates exceed 50%. Its rarity stems from early recognition and thyroid medication availability. Its presentation can be non-specific, making it a challenging diagnosis.

This is a 67-year-old male inmate who was brought to the ED due to hypoactivity. He had a long-standing history of bipolar disorder, and hypothyroidism receiving oral levothyroxine.

On evaluation, patient had slowed mentation, GCS 14/15, sluggish reactive pupils, macroglossia, diffuse non-pitting edema, and delayed relaxation of the deep tendon reflexes in the extremities. Vital signs were abnormal; T: 35.2 °C, RR: 10 rpm, SpO<sub>2</sub>: 84 %, BP: 137/89 mmHg and HR: 42 bpm without chronotropism. 12-lead ECG revealed a complete atrioventricular block (AV block), with non-conductive P waves and idioventricular rhythm. Patient became hemodynamically unstable, transcatheter pacemaker was placed. Dopamine infusion was initiated for adequate mean

arterial pressure. Subsequently, a femoral transvenous pacemaker was performed. However, neurological deterioration prompted mechanical ventilation.

Exploring reversible AV block etiologies, laboratory results were markedly elevated for TSH at 184.775 ng/mL and decreased T<sub>4</sub> at 1.5 ng/mL. Lithium levels were therapeutic. Myxedema coma was identified and timely treatment was provided with intravenous thyroid hormone replacement, intravenous hydrocortisone, and supportive care. Patient was transferred to an ICU where TSH was monitored. After 5 days of receiving IV thyroid hormone replacement therapy, TSH improved. However, patient remained dependent on transvenous pacemaker, for which permanent pacemaker had to be placed. With further therapy, patient's neurological status improved leading to extubation, and subsequent discharge.

Thyroid hormones play a vital role in the electrical current of the heart; hence, disturbances may potentiate cardiac arrhythmias. Sinus bradycardia and QT interval prolongation are commonly seen. As the severity of hypothyroidism progresses, high-grade AV block may be encountered, being third degree AV block the most challenging and severe.

Patients with high-degree AV block in the setting of reversible etiologies, commonly, do not need a permanent pacemaker. On the contrary, our patient developed complete dependence of the pacemaker for adequate cardiac synchrony, despite adequate replacement therapy.

With this case, we illustrate the importance of a thorough evaluation in patients with AV block of unknown origin, with special attention to reversible etiologies. Thyroid function abnormalities should be promptly identified and managed for better outcomes. Furthermore, it may decrease cardiac death risk and the need for invasive procedures, such as permanent pacemaker placement.

**Bone and Mineral Metabolism****BONE AND MINERAL CASE REPORTS II*****Hollow Bones: A Case Report of Immobilization Osteoporosis in a Young Female***

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**MON-359****Abstract****Introduction:**

Osteoporosis is defined as decreased bone strength due to reduced bone mineral density (BMD) and/or abnormal bone architecture leading to bone fragility and increased risk of pathologic fractures. Typically a disease of postmenopausal females, osteoporosis is uncommon in the young. We present a case of a 34-year-old woman, who was diagnosed with severe osteoporosis in the setting of prolonged immobilization.

**Clinical Case:**

A 34-year-old woman was admitted for treatment of bacteremia in the setting of IV drug use and right hip septic arthritis leading to femoral head osteonecrosis requiring a long course of antibiotic treatment and resulting in prolonged