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Introduction: Thyroid dysfunction has a great impact on lipids as well as a number of other cardiovascular risk factors. Though the effect of thyroid hormones on plasma cholesterol concentrations are well-recognized, however, there are conflicting reports about the effect of thyroid hormone on the metabolism of plasma triglycerides. We sought to determine the effect of hypertriglyceridemia on patient admitted with hyperthyroidism. **Methods:** We queried the National Inpatient Sample (NIS) databases from 2016 to 2017 for adults aged 18 and above with hypertriglyceridemia as a principle diagnosis with and without hyperthyroidism using ICD-10 codes. Multivariate logistic and linear regression analysis was used accordingly to adjust for confounders. **Results:** There were over 71 million discharges in the combined 2016 and 2017 NIS database. Out of 17,705 hyperthyroidism hospitalizations, 15% had hypertriglyceridemia. Hypertriglyceridemia with hyperthyroidism had a similar odds of inpatient mortality (AOR 0.37, CI 0.06–1.99, P=0.246), acute kidney injury (AOR 1.03, CI 0.706–1.510, P=0.868) and cardiogenic shock (AOR 0.96, CI 0.134–6.72, P=0.963). There was a statistically significant increase in odds of acute respiratory failure (AOR 0.46, CI- 0.21- 0.99, P=0.048) in those hospitalized with hyperthyroidism and hypertriglyceridemia compared to those with hyperthyroidism alone. **Conclusion:** Hypertriglyceridemia is associated with similar outcomes in patient admitted for hyperthyroidism in terms of mortality, acute kidney injury and acute cardiogenic shock with an increased odd of acute respiratory failure. More research is needed to explain the pathophysiologic mechanism underlying the effect of hypertriglyceridemia on hyperthyroidism.

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Improvement in Quality of Life and Psychological Symptoms After Treatment for Primary Aldosteronism

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Background: Primary aldosteronism (PA) is the most common treatable cause of secondary hypertension. In addition to increased cardiovascular risk, patients also suffer from impaired quality of life (QoL) and psychological symptoms. We assessed for changes in QoL and depressive symptoms in a cohort of Asian patients with PA, after surgical and medical therapy. **Methods:** We administered questionnaires to 34 patients with PA, mean age, 51.3 years, 29.4% females, in a prospective observational study from 2017 to 2020. QoL was assessed using RAND-36 and EQ-5D-3L, and depressive symptoms was assessed using Beck Depression Inventory (BDI-II) at baseline, 6 months, and 1 year post-treatment. **Results:** Significant improvement was observed 1 year after treatment in both physical and mental summative scores of RAND-36 from baseline, +3.65 (p = 0.023) and +3.41 (p = 0.033) respectively, as well as four subscale domains (physical functioning, bodily pain, role emotional and mental health). Significant improvement was also seen in EQ-5D dimension of anxiety/depression at 1 year post-treatment. Patients treated with surgery (N=21) had significant improvement in EQ-5D index score post-treatment, and better EQ-5D outcomes compared to medical group (N=13) at 1 year post-treatment. 37.9%, 41.6% and 60.7% of patients had symptoms in the cognitive, affective and somatic domains of the BDI-II respectively. There was significant improvement in the affective domain of BDI after 1 year of treatment. **Conclusion**: Appropriate treatment with surgical and medical therapy improves QoL and psychological symptoms in patients with PA, highlighting the importance of early diagnosis and treatment of this common condition.

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In a Mouse Model of Type 2 Diabetes and Peripheral Artery Disease, Modulation of MirR29a and ADAM12 Reduced Post -Ischemic Skeletal Muscle Injury, Improved Perfusion Recovery and Skeletal Muscle Function

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Diabetes Mellitus (DM) is a major risk factor for developing peripheral arterial disease (PAD) and individuals with DM have worse PAD outcomes but the molecular mechanisms involved are poorly understood. Previously, in a hind limb ischemia (HLI) model of PAD, we identified a disintegrin and metalloproteinase gene 12 (ADAM12) as a key genetic modifier of post-ischemic perfusion recovery. Moreover, we showed that expression of ADAM12 in mouse and human tissue is regulated by miR29a. In non-diabetic mice, miR29a expression is downregulated after HLI that allows increased expression of ADAM12. However, upon HLI in high fat diet feed (HFD) mice, a model of type 2 diabetes, miR29a expression remains elevated that prevents ADAM12 increase and results in poor reperfusion recovery, increased skeletal muscle injury and decreased muscle function. Hence, we hypothesized that inhibition of miR29a or augmenting ADAM12 would improve these functional outcomes.
Mice (male, 26–28 weeks old) were randomized into 3 treatment groups and their hind limbs were treated with saline (grp1), ADAM12 cDNA (grp 2) or mir29a-inhibitor (grp3), through targeted micro-bubble delivery. Mice were