from 2011-2013 we selected a cohort of men diagnosed with hypogonadism who was evaluated by each group. Kaplan-Meier curves with Wilcoxon tests were used to compare time to event for each disease. RESULTS: 1625 cases were identified as well as 4794 Greedy matched controls. T- test and chi-square analysis on all matching criteria showed no difference between the two groups at baseline. Kaplan-Meier analysis associated testosterone therapy with decreased time in months to event in osteoporosis (T1:7 months, NT4:months, p0.01), osteoarthritis (T9:months, NT7:months, p0.04), and MI (T13:months, NT10:months, p0.001). CONCLUSIONS: Testosterone therapy was shown to delay time to event for osteoporosis, osteoarthritis, and MI while not increasing the occurrence of stroke or MI. This study has shown that with long follow up injectable testosterone therapy delays the negative effects of hypogonadism in older men.

PD84

UTILIZATION OF NEGATIVE CONTROLS TO EXAMINE ASSOCIATION BETWEEN RARE GENETIC DISORDERS AND TYPE 2 DIABETES IN FOUR LARGE OBSERVATIONAL DATABASES

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OBJECTIVE: The association between genetic disorders, hereditary disorders, hyperfructose intolerance (HFI) or alpha-1 antitrypsin deficiency (AAT), and type 2 diabetes (T2D) has not yet been investigated. Therefore, the objective of this undertaking was to examine the association between both disorders in both types of diabetes using four large observational databases and adjust for ascertainment bias. METHODS: Patients with a HFI diagnosis (ICD-9: 271.2) or AAT diagnosis (ICD-9: 274.3) and T2D were identified in the Truven MarketScan Claims Database (2007-2012), Optum Claims Database (2002-2012), Humedica Electronic Health Records (EHR) Database (2007-2012), and GE Centricity EHR Database (1995-2012). The association between both genetic disorders and T2D was compared to the association between both disorders and type 2 diabetes without a genetic disorder (control) using the beta coefficients and 95% CI for each database. RESULTS: The unadjusted association between both genetic disorders and T2D was positive and heterogenous (p0.001) in all four databases. The unadjusted pooled odds ratio (OR) calculated using a random-effects model meta-analysis was 3.48 (95% CI: 21.5-5.46) for HFI and 2.71 for AAT (95% CI: 1.75-4.20). After pooling all patients and adjusting for the negative controls using a random-effects model meta-analysis, it was found that the genetic disorders have a 7.3 times increased risk for T2D (ORR=1.73, 95% CI: 1.08-2.75) compared to patients with negative control diseases; the association was stronger when utilizing a fixed-effects model meta-analysis (OR=2.19, 95% CI: 2.07, 2.31). The adjusted association between AAT and T2D was statistically significant in the fixed-effects (OR=1.33, 95% CI: 1.27-1.40) model meta-analysis but not the random-effects model meta-analysis (OR=1.35, 95% CI: 0.86, 2.12). CONCLUSIONS: HFI and T2D were positively associated after adjusting for the negative controls. The fixed-effects analysis identified a 21% increase in T2D due to HFI and a 35% increase due to AAT and T2D. Future research is needed to elucidate the mechanisms underlying the effects of these genetic disorders with T2D.

PD85

PREDICTING CHRONIC COMORBID CONDITIONS OF TYPE 2 DIABETES IN NEWLY-DIAGNOSED DIABETIC PATIENTS

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OBJECTIVES: Type 2 Diabetes(T2D) and its comorbid conditions are imposing large burdens on health-care systems worldwide. Once a patient is diagnosed with T2D, their risk for immediate chronic comorb conditions is less quantified. We aim to build risk prediction models for onset of cardiovascular, cerebrovascular, renal, and eye severe conditions for newly-diagnosed patients with T2D. METHODS: Our cohort includes 4 million insurance beneficiaries of age≥18yrs between 2005 and 2013. Of these subjects, 34,411 patients were newly-diagnosed with T2D, had continuous enrollment for 6 months pre- and 5yrs post-onset, and did not have the comorbid condition diagnosis before T2D. We developed a prediction model for each condition based on L1-regularization method, which selects few(less than 350 predictors) relevant risk factors from approximately 27k general variables derived from claims data. We defined comorb conditions based on the validated diabetes complications severity index. RESULTS: For patients newly-diagnosed with T2D, we predict new onset of cardiovascular conditions with Area Under Curve(AUC)=0.61±1e-4, cerebrovascular conditions with AUC=0.71±1e-4, eye conditions with AUC=0.65±1e-4. Our method discovers risk factors, some of which have significant (p<0.001) differences in odds ratios between patients with/have subgroups with and without the conditions. A total of 10,610 cases of blindness, 27,390 lower extremity amputations (LEAs), and 19,010 end stage renal disease cases requiring dialysis (ESRDs) were investigated for the drifts are not considered. Our study compared the simulated clinical outcomes of type 2 diabetic mellitus (T2DM) treatments with and without drift. METHODS: Lifetime clinical outcomes of treatments were estimated using a Monte Carlo simulation model. Simulated cohort was newly diagnosed age 45 T2DM patients with an initial 8.5% HbA1c level. Demographic and clinical characteristics were sourced from NHANES data. Two scenarios were simulated. Both scenarios were compared with a non-drift scenario, and the benefit of metformin with and without drift over a stable HbA1c of 8.5%. The second compared metformin with drift to basal-bolus insulin. Drift data were extrapolated from clinical trials. RESULTS: Metformin treatment without drift gained 3.02 life years (LY), while with drift the LY gain was only 0.08. For 100,000 simulated patients metformin without drift prevents 1,960 myocardial infarctions (MIs), 320 strokes, 10,610 cases of blindness, 27,390 lower extremity amputations (LEAs), and 19,010 end stage renal disease cases requiring dialysis (ESRDs). When HbA1c drift was considered, the benefits of metformin treatment shrink to no additional strokes, prevention of 500 cases of blindness, 700 LEAs, and 410 ESRDs, with an increase of 100 mls. The direct comparison of LY of metformin with drift and insulin treatment showed that the insulin therapy was 1.51 LY over metformin. Compared with metformin treatment with drift, insulin treatment prevented more complication events including 2,110 MIs, 140 strokes, 8,340 cases of blindness, 20,460 LEAs, and 16,360 ESRDs. CONCLUSIONS: HbA1c drift in oral anti-diabetic drugs has substantial effects on clinical outcomes and should be incorporated into diabetes health model outcomes.