Duke Charitable Foundation award #2015216 and the Yale Center for Clinical Investigation.

0584

IMPACT OF OBSTRUCTIVE SLEEP APNEA ON CHRONIC OBSTRUCTIVE PULMONARY DISEASE HOSPITALIZATIONS

Mohannad Al Khateeb[1], Mubarak Yusuf[1], Ali Horoub[1], Mohammad aldiabat[1], Yazan Al Jabiri[1]
Lincoln medical center [1]

Introduction: Obstructive sleep apnea (OSA) is a sleep disorder that has been linked to increase the risk for hypertension, ischemic heart failure, arrhythmia and heart failure. There are multiple similarities between OSA and Chronic Obstructive Pulmonary Disease (COPD); both are associated with hypoxia and hypercapnia, with different mechanisms of hypoxia; in COPD its chronic and slow progression, whereas it is suddenly intermittent hypoxia in OSA. Intermittent hypoxia was hypothesized to enhance the protective effect on subsequent hypoxia resulting in cardioprotective effect [1]. There is little data on rates of in-hospital mortality on patients with OSA and COPD using a nationwide study. In this study, we aim to analyze the impact on mortality and length of hospital stay of obstructive sleep apnea in patients with COPD.
Methods: Adults with principal diagnosis of COPD were selected from the 2019 US National Inpatient Sample, using ICD 10 code primary diagnosis on discharge. We queried the 2019 National Inpatient Sample for OSA, and other secondary diagnoses (hyperlipidemia, hypertension, heart failure, smoking, CKD, electrolytes disturbances). Confounders were adjusted for using multivariable linear regression analysis for other secondary diagnoses.
Results: In a total of 520,624 adult hospitalizations with COPD primary diagnosis on discharge were included from the 2019 national inpatient sample. 73,705 patients had concomitant secondary diagnosis with OSA. On weighted analysis, hospitalizations with primary diagnosis of COPD and secondary diagnosis of OSA had lower in-hospital mortality rates compared to hospitalizations with COPD alone (0.6% vs 1.08%, p= 0.000), .COPD hospitalizations with OSA had statistically significant lower odds for mortality compared to COPD patients without OSA (adjusted OR 0.73, 95% CI 0.57-0.93; p= 0.009).However, COPD hospitalizations with OSA showed increased in the mean length of stay by 0.21 days (95% CI 0.12-0.30, p=0.000) compared to patients without OSA.
Conclusion: Our analysis showed better mortality outcomes for COPD patients with OSA , supporting the protective effect hypothesis of intermittent hypoxia. COPD patients with concomitant secondary OSA diagnosis have increased in-hospital length of stay.
Support (If Any): 1- Murry CE, Jennings RB, Reimer KA (1986) Preconditioning with ischemia: a delay of lethal cell injury in ischemic myocardium. Circulation 74:1124–1136

0585

USE OF A HYBRID CLOSED LOOP INSULIN DELIVERY SYSTEM IMPROVES SLEEP AND GLYCEMIC CONTROL IN ADULTS WITH LONG-STANDING TYPE 1 DIABETES AND HYPOGLYCEMIA UNAWARENESS

Susan Malone[1], Austin Matus[2], Amy Peleckis[3], Anneliese Flatt[3], Laura Grunin[4], Gary Yu[1], Sooyong Jang[5], James Weimer[5], Insup Lee[5], Michael Rickels[3], Namni Goel[6]
Rory Meyers College of Nursing, New York University [1] Universtiy of Pennsylvania School of Nursing [2] Institute for Diabetes, Obesity, and Metabolism, Perelman School of Medicine, University of Pennsylvania [3] Rory Meyers College of Nursing [4] PRECISE Center, Department of Computer and Information Science, School of Engineering and Applied Sciences, University of Pennsylvania [5] Biological Rhythms Research Laboratory, Department of Psychiatry and Behavioral Sciences, Rush University Medical Center [6]

Introduction: Insulin delivery and continuous glucose monitoring systems (CGMs) have been reported to disrupt sleep in individuals with type 1 diabetes (T1D), potentially thwarting the adoption and continued use of diabetes therapeutic technologies. This study assessed changes in actigraphic sleep and glycemic outcomes in individuals at high risk for life threatening nocturnal hypoglycemia after initiating a hybrid closed loop (HCL) insulin delivery system with integrated CGM.
Methods: 10 adults (median age=51y) with long-standing T1D (median duration=34y) and hypoglycemia unawareness participated in an 18-month ongoing clinical trial assessing the effectiveness of a HCL system. Wrist actigraphs and CGMs measured sleep and glycemic control, respectively, at baseline (1 week) and at months 3, 6, 9, 12, 15, and 18 (3 weeks) following HCL initiation. Body mass index and hemoglobin A1c (HbA1c) were also collected at these timepoints. Hypoglycemia awareness was assessed using the Clarke hypoglycemia questionnaire, HYPO score, and glycemic lability index. Paired sample t-tests and Cohen's d effect sizes modeled changes in sleep, glycemic control, and hypoglycemic awareness and the magnitude of these changes from baseline to 18 months.
Results: Sleep improved from baseline to 18 months [shorter sleep latency ($p<0.01$), later sleep offset ($p<0.05$), and less wake after sleep onset (WASO) (<0.01)]. Medium effect sizes were found for later sleep onset (d=0.74) and later sleep midpoints (d=0.77). HCL also improved hypoglycemia awareness from baseline to 18 months [Clarke score ($p<0.01$), HYPO score ($p<0.01$), lability index ($p<0.05$)]. Medium to large effect sizes were found for reduced nocturnal hypoglycemia (percent time glucose was <54mg/dL,<60mg/dL,<70mg/dL; d=0.66 – 0.81), daytime and nocturnal hypoglycemia (percent time glucose was<54mg/dL,<60mg/dL,<70mg/dL; d=0.61 – 0.69), and glucose variability (coefficient of variability; d=0.62).
Conclusion: HCL insulin delivery with CGM improved sleep over time as indicated by shorter sleep onset latency, later sleep offset, and less WASO. HCL insulin delivery also improved hypoglycemia awareness and led to clinically significant reductions in hypoglycemia and glucose variability.
Support (If Any): NIH (R01DK117488 (NG), R01DK091331 (MRR), K99NR017416 (SKM), and UL1TR001878 (University of Pennsylvania Center for Human Phenomic Science)). National Aeronautics and Space Administration (NASA) (NNX14AN49G and 80NSSC20K0243 (NG)) and from the Pennsylvania Department of Health (SAP 4100079750 (IL)).