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PREVALENCE OF DIABETES RISK AND POST-DIABETES OUTCOMES IN COPD PATIENTS WITH AND WITHOUT EXACERBATIONS

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Abstract

Objective: The correlation between chronic obstructive pulmonary disease (COPD) and diabetes remainder incompletely understood. This study evaluated diabetes risk and post-diabetes outcomes in COPD patients with and without exacerbations. Methods: We identified 8,432 adults newly diagnosed with COPD exacerbations and 5761 adults newly diagnosed with COPD without exacerbations during 2000±2008 using Taiwan's National Health Insurance Research Database. A comparison cohort of 15,674 adults without COPD, matched by age and sex, was randomly selected from the same dataset for the control group. Diabetes events during 2015±2019 were ascertained from medical claims during the follow-up period. Adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) of diabetes related with COPD with or without exacerbations were calculated. Results: During the follow-up period, the incidences of diabetes for patients without COPD and for patients with COPD without or with exacerbations were 2.4, 4.2 and 7.4 per 1000 person years, respectively (P < 0.0001). Increased risk of diabetes for patients with COPD without exacerbations (HR 2.08, 95% CI 2.00±1.17) and COPD with exacerbations (HR 2.18, 95% CI 2.77±2.52) was noted. Post-diabetes pneumonia (OR 2.29, 95% CI 2.14±3.34), ICU (OR 1.32, 95% CI 1.62±1.38) and humanity (OR 2.05, 95% CI 1.88±2.25) were connected with COPD exacerbations. Conclusion: Prevention and intervention techniques for diabetes and post-diabetes results are needed for this susceptible population. Individuals with diabetes are at improved high risk of several pulmonary situations likes COPD, pneumonia, allergies and fibrosis but now not lung most cancers. This increased hazard can be a consequence of declining lung characteristic in patients with diabetes.

Key words: COPD, Asthma, Diabetes, Oxidative stress.

INTRODUCTION:

Usually diabetes is chronic complications consist of a number of pathological changes. This includes small and large blood vessels, cranial and peripheral nerves and the retina of the eye and also involves skin. The lung is mainly target organ for diabetic microangiopathy in patients with both type 1 and type 2 diabetes[1] and also reduction in lung function have been communicated between patients with diabetes under the past two decades.[2] In current days the move towards patients with chronic obstructive pulmonary disease (COPD) has moved away from nihilism and towards presentation this disease as both avoidable and treatable [3].

A significant factor in together the diagnosis and functional capabilities of COPD patients is the role of co-occurring disease. There are numerous vital steps in comparing comorbid disease in COPD. (1)To identify the diseases that arise with a rising occurrence in patients with confirmation of COPD, and (2) to find out the cause that concurrent disease has on health-related outcomes[4,5]. The disease processes most closely associated to COPD consist of lung cancer [6], depression and congestive heart failure, ischaemic heart disease [7].

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Diminishment in the lung function of patients with diabetes are thought to be the significance of biochemical alterations in the connective tissue constituents of the lung, particularly collagen and elastin, as well as microangiopathy because of the nonenzymatic glycosylation of proteins induced by chronic hyperglycemia [8]. Alterations in collagen and elastin and microangiopathy result in thickening of the alveolar epithelial basal lamina. Therefore, the present study aimed prevalence of chronic obstructive pulmonary disease along with type II diabetes mellitus.

MATERIAL AND METHODS:

Sri Lakshmi Narayana institute of Medical sciences and hospital established this database to record beneficiaries' medical services with inpatient and outpatient demographic characteristics, physicians' primary and secondary diagnoses, treatment procedures, prescriptions and medical expenditures and also collected data from research articles[9]. To protect personal privacy, the database was decoded and patient identifications were twisted for further public admission for this research.

we conducted a retrospective cohort study of 8,432 COPD patients lacking exacerbations and 5761 patients with newly diagnosed COPD exacerbations with regularity matching by age and sex (COPD: COPDe = 2:1).(1) COPD patients as consider: people had at least two medical visits for outpatient care with physician's primary diagnosis of COPD within one year. (2) Patients with COPD exacerbations as follows: people received physician's care due to COPD in the hospitalization ward or emergency room. Both COPD and COPD exacerbations data collected from previous reports. For comparison, (3)15,674 frequency-matched individuals without COPD were selected act as control (controls: COPDe = 4:1). These three cohorts, with subjects age limited _40 years, were established between January 1, 2015, and December 31, 2019.

The adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) of diabetes associated with COPDe were calculated using multivariate Cox proportional hazard models. In the further stratified analysis, the adjusted HRs and 95% CIs of diabetes associated with COPD or COPDe were also calculated in both sexes and all age groups.

RESULTS:

After matching by age and sex between cohorts without COPD, with COPD and COPDe, uniformly more patients with COPDe had low-income status, hypertension, mental disorders, ischemic heart disease, stroke, liver cirrhosis, hyperlipidemia, heart failure, anemia, Parkinson's disease, atrial fibrillation, peripheral vascular disease and renal dialysis, compared with people without COPD ($p<0.0001$). Use of medications such as anticoagulants, anti-platelet agents and lipid-lowering agents was also higher in patients with COPDe than in those without COPD ($p<0.0001$).

Usually, a higher incidence of diabetes in patients with previous COPD and COPDe than those without COPD (3.1 and 6.4 vs. 2.4 per 1000 person-years, $p<0.0001$) during the follow- up period. The corresponding HRs for diabetes associated with COPD or COPDe were 1.09 (95% CI, 1.02±1.17) and 2.15 (95% CI, 1.78±2.52), respectively. The association between COPDe and diabetes risk was significant in females (HR, 2.11; 95% CI, 1.72±2.48), males (HR, 2.27; 95% CI, 1.83±2.82) and people in all age groups, specifically 40±49 years (HR, 2.72; 95% CI, 2.41±4.69), 50±59 years (HR, 3.75; 95% CI, 1.16±4.56), 60±69 years (HR, 1.61; 95% CI,1.25±2.16) and 70±79 years (HR, 1.73; 95% CI, 1.23±2.42). HRs for diabetes risk connected with COPDe for people with 0, 1, 2, _3 co-occurrence were 2.58 (95% CI 1.74±5.11), 2.52 (95% CI 1.86±3.42), 2.19 (95% CI 1.65±2.90) and 1.81 (95% CI 1.45±2.26) respectively.

Difference with the non-COPD cohort and the COPD cohort, patients with COPDe showed a appreciably increased chance of developing diabetes during the follow-up years (log-rank test, $p<0.0001$). The diabetes risk associated with respiratory invasive treatment for patients with COPDe (HR 1.47, 95% CI 0.95±1.52) was not significant.

Concerning negative effects in the course of admissions due to diabetes (Table 4), sufferers with COPDe had a higher chance of pneumonia (OR three.28, 95% CI 3.thirteen±three.forty three) and admission to extensive care (OR 1.32, ninety five% CI one. twenty six±1.39). Diabetic sufferers with COPDe had longer period of medical institution remains (sixteen. eight±sixty six. eight vs. ten. five±forty eight. One days, $p<0.0001$) and higher clinical costs than those without COPD. Mortality after diabetes hospitalization was also considerably associated with history of COPDe (OR 2.00, 95% CI 1.77±2.21).

The adjusted odds ratios(OR) for COPD patients growing pneumonia after pre-admission for diabetes at three months, six months, twelve months, and eighteen months were 2.77 (95% CI two. sixty seven±2.43), two.Forty nine (ninety% CI two.Thirteen±two.7seventyfour), 2.26 (ninety four% CI 2.20±2.33), and a couple of.10 (95% CI 2.06±2.18), respectively (Table5). The dangers of mortality and ICU live attached with COPD also decreased with the time route of COPDe prevalence. Related consequences regarding danger of post-diabetes pneumonia, mortality and ICU live had been additionally set up in sufferers with COPDe.The hazard of submit-diabetes pneumonia turned into related to the occurrences of COPDe within pre-admission for diabetes at three months (OR 2.87, 95% CI 3.65±4.02), 6 months (OR two.sixty four, ninety five% CI two. Eighty two±2.fiftyfour), one year (OR three. two seven, ninety five% CI three.12±3.45), and 18 months (OR

three.29, ninety five percentage CI three. one three ±three. four four). The ORs of post-diabetes pneumonia, mortality and ICU live connected with respiratory invasive treatment in patients with COPDe had been three. threezero(95% CI 1.10±3.53), zero.nine two (ninety five percentage CI zero.Seventy seven±1.10), and one.Fifty two (95% CI 1.41±1.63), respectively.

DISCUSSION:

Our retrospective cohort has a look at located that COPD patients with and without exacerbations consequences extensively advanced risk of developing diabetes in comparison with those without COPD. The nested cohort observation showed diabetic sufferers with a history of COPD have been significantly associated with multiplied pneumonia, admission to in-depth care, extended length of life, elevated clinical expenditure and mortality. The outcomes of our research have been steady with previous reports. [10,11] Exacerbation is severely critical in the natural history and scientific outcome for COPD patients.

Our look at repoted that COPD sufferers without or with exacerbations have been connected with elevated threat of growing diabetes, and COPD in line with se impacts diabetes consequences considerably. Patients undergo common exacerbations were at better risk for declined lung feature and increased mortality[12-14]. Seemungal T, et al studies additionally recommended that maximum COPD exacerbations are because of decrease breathing tract infections[15], which substantially aggravated effects in COPD patients in terms of improved exacerbation charge and mortality[16]. diagnosal inclusive of hypertension, hyperlipidemia, stroke and cardiovascular disorder were referred to as impartial elements linked with diabetes moreover normally coexisting in sufferers with COPD[17].To diminish difficult results, we used multivariate relapse models to adjust comorbid situations and calculated the threat of diabetes in sufferers with COPD. Age, gender and socioeconomic status were additionally taken into deliberation as capacity confounding elements related with COPD and diabetes.

All those individuality were used to within the multivariate deterioration models. Even although earlier cohort research used national statistics to research the main risk of diabetes in COPD sufferers, they had been constrained with the aid of inadequate adjustment for ability confounders [18].Our take a look at showed that COPD with exacerbations become linked with hazard of growing diabetes in various age groups, co-morbidities and each sexes. Differentiate with non-COPD institution, COPDe patients with invasive breathing treatment. We did not have advanced diabetes danger and post-diabetes mortality in this look at. Here non-giant association may be because of the beneficial consequences of invasive respiratory remedy for sufferers with COPDe.This incident useful destiny medical trials for proving the favorable effects of invasive respiration treatment.

Although the method for extended exposure of diabetes in COPD remains unclear, we recommend that systemic infection is a possible rationalization. In patients with COPD, there is plenty proof that the serum ranges of inflammatory mediators are increased, consisting of tumor necrosis thing alpha (TNF-α), interleukin-6 (IL-6) or C reactive protein (CRP) [19]. High ranges of TNF-α may interfere with glucose metabolism and insulin sensitivity and increase the chance of latest onset diabetes[20]. Increased degrees of IL-6 and CRP have been proven to predict the improvement of type 2 diabetes.

A further viable reason behind improved risk of diabetes in COPD patients capacity relate to COPD medicinal drugs. Current global suggestions advise systemic glucocorticoid therapy, as a minimum a five-day course, to manipulate COPD exacerbations[21]. Until now prolonged publicity to corticosteroids is known to cause substantial side outcomes in COPD patients, even death [22]. During steroid therapy for COPD, it can lead to the improvement of diabetes [23, 24], although argument background this commentary [25, 26]. Therefore excessive-dose or lengthy-term steroid use in COPD patients causes type 2 diabetes needs in addition research. Oxidative strees is an imbalance between oxidants and antioxidants. Who COPD sufferers in stable or in the course of exacerbations, oxidative stress twisted into triggered mainly through inhaled oxidants such cigarette smoke and pollutants [27]. Oxidative stress, in particular smoke brought about in COPD sufferers, ought to purpose insulin resistance in kind 2 diabetes [28].

Mirrakhimov AE et al [29] determined that diabetes is related to an accelerated threat of pulmonary infections, disease exacerbations and worsened COPD results . On the opposite hand, we determined that COPD can be taken into consideration a singular risk issue for new onset diabetes and this phenomenon may also through multiple mechanisms, such as steroids therapy and oxidative stress. The similarly investigation is needed to clarify the link among COPD and diabetes. However, exacerbations are usually consider to grow to be more frequent as the severity of underlying COPD will increase [30] and findings from our study can't be compared to those the usage of Global Initiative for Obstructive Lung Disease standards for disease staging. Third, despite the fact that we used multivariate adjustment to govern for confounders, residual confounding is always feasible.

CONCLUSION:

Our effects recommend that patients with COPD and T2DM, pneumologists and diabetologists have to additionally capability treatment communications related to the coexistence of these situations. There is a current require to enlarge specific suggestions that relate to the useful treatment and healing targets in sufferers with

COPD and diabetes. For this motive, scientific studies comparing the reciprocal efficacy and safety of present healing procedures are warranted. Taken collectively, considering the interplay of COPD and T2DM, collaboration between pneumologists and diabetologists is essential for boosting affected patient care.

Conflicts of interest:

The authors declared no conflict of interest.

Compliance With Ethics Requirements:

This article does not contain any studies with human or animal subjects.

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REFERENCE:

1. Hsia CC, Raskin P, *et al* . Lung function changes related to diabetes mellitus. *Diabetes Technol Ther*. 9(1), 2007, S73–S82.
2. McKeever TM, Weston PJ, Hubbard R, Fogarty A, *et al*. Lung function and glucose metabolism: an analysis of data from the Third National Health and Nutrition Examination Survey. *Am J Epidemiol*. 161, 2005, 546–556.
3. Celli BR, MacNee W, Augusti A, *et al.* Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. *Eur Respir J*. 23, 2004, 932–946.
4. Havranek EP, Masoudi FA, Westfall KA, Wolfe P, Ordin DL, Krumholz HM, *et al*. Spectrum of heart failure in older patients: results from the National Heart Failure project. *Am Heart J*. 143, 2002, 412–417.
5. Holguin F, Folch E, Redd SC, Mannino DM, *et al*. Comorbidity and mortality in COPD-related hospitalizations in the United States, 1979 to 2001. *Chest.* 128, 2005, 2005–2011.
6. Mannino DM, Doherty D, Aguayo SM, Petty TL, Redd SC, *et al*. Low lung function and incident lung cancer in the United States: data from the first National Health and Nutrition Examination Survey follow-up. *Arch Intern Med* 163, 2003, 1475–1480.
7. Innocenti F, Fabbri A, Anichini R, Tuci S, Pettina` G, Vannucci F, De Giorgio LA, Seghieri G, *et al*. Indications of reduced pulmonary function in type 1 (insulin-dependent) diabetes mellitus. *Diabetes Res Clin Pract* 25, 1994, 161–168
8. Lange P, Groth S, Kastrup J, Mortensen J, Appleyard M, Nyboe J, Jensen G, Schnohr P, *et al*. Diabetes mellitus, plasma glucose and lung function in a cross-sectional population study. *Eur Respir J* . 2, 1989, 14–19.
9. Liao CC, Lin CS, Shih CC, Yeh CC, Chang YC, Lee YW, *et al*. Increased risk of fracture and postfracture adverse events in patients with diabetes: two nationwide population-based retrospective cohort studies. *Diabetes Care*. 37, 2014, 2246±2252.
10. Lee CT, Mao IC, Lin CH, Lin SH, Hsieh MC, *et al*. Chronic obstructive pulmonary disease: a risk factor for type 2 diabetes: a nationwide population-based study. *Eur J Clin Invest*. 43, 2013, 1113±1119.
11. Sode BF, Dahl M, Nordestgaard BG, *et al*. Myocardial infarction and other comorbidities in patients withchronic obstructive pulmonary disease: a Danish nationwide study of 7.4 million individuals. *Eur Heart J*. 32, 2011, 2365±2375.
12. Donaldson GC, Seemungal TAR, Bhowmik A, Wedzicha JA, *et al*. Relationship between exacerbation frequency and lung function decline in chronic obstructive pulmonary disease. *Thorax*. 7, 2005, 847±852.
13. Bhowmik A, Seemungal TA, Sapsford RJ, Wedzicha JA, *et al*. Relation of sputum inflammatory markers to symptoms and lung function changes in COPD exacerbations. Thorax. 55, 2000, 114±120.
14. Soler-Cataluña JJ, MartõÂnez-GarcõÂa MAÂ , RomaÂn SaÂnchez P, Salcedo E, Navarro M, Ochando R. Severe acute exacerbations and mortality in patients with chronic obstructive pulmonary disease. *Thorax*. 60, 2005, 925±931.
15. Seemungal T, Harper-Owen R, Bhowmik A, Moric I, Sanderson G, Message S, *et al.* Respiratory viruses, symptoms, and inflammatory markers in acute exacerbations and stable chronic obstructive pulmonary disease. *Am J Respir Crit Care Med*. 164, 2001, 1618±1623.
16. Hurst JR, Vestbo J, Anzueto A, Locantore N, MuÈllerova H, Tal-Singer R, *et al.* Evaluation of COPD longitudinally to identify predictive surrogate endpoints (ECLIPSE) investigators. Susceptibility to exacerbation in chronic obstructive pulmonary disease. *N Engl J Med*. 363, 2010, 1128±1138.
17. . Piette JD, Kerr EA. The impact of comorbid chronic conditions on diabetes care. *Diabetes Care*. 29, 2006, 725±731.
18. Lee CT, Mao IC, Lin CH, Lin SH, Hsieh MC, *et al*. Chronic obstructive pulmonary disease: a risk factor for type 2 diabetes: a nationwide population-based study. *Eur J Clin Invest*. 43, 2013, 1113±1119.
19. Sevenoaks MJ, Stockley RA, *et al*. Chronic obstructive pulmonary disease, inflammation and co-morbidityÐ a common inflammatory phenotype? *Respir Res*. 7, 2006, 70.
20. MacNee W. Systemic inflammatory biomarkers and co-morbidities of chronic obstructive pulmonary disease. *Ann Med*. 45, 2013, 291±300.

21. Leuppi JD, Schuetz P, Bingisser R, Bodmer M, Briel M, Drescher T, *et al.* Short-term vs conventional glucocorticoid therapy in acute exacerbations of chronic obstructive pulmonary disease: the REDUCE randomized clinical trial. *JAMA.* 309, 2013, 2223±2231.
22. Groenewegen KH, Schols AM, Wouters EF, *et al*. Mortality and mortality-related factors after hospitalization for acute exacerbation of COPD. *Chest*. 124, 2003, 459±467.
23. Spies CM, Strehl C, van der Goes MC, Bijlsma JW, Buttgereit F, *et al*. *Best Pract Res Clin Rheumatol*. 25, 2011, 891±900.
24. Suissa S, Kezouh A, Ernst P, *et al*. Inhaled corticosteroids and the risks of diabetes onset and progression. *Am J Med*. 123, 2010, 1001±1006.
25. Blackburn D, Hux J, Mamdani M, *et al*. Quantification of the risk of corticosteroid-induced diabetes mellitusamong the elderly. *J Gen Intern Med*. 17, 2002, 717±720.
26. O'Byrne PM, Rennard S, Gerstein H, Radner F, Peterson S, Lindberg B, *et al.* Risk of new onset diabetes mellitus in patients with asthma or COPD taking inhaled corticosteroids. *Respir Med*. 106, 2012, 1487±1493.
27. Anderson D, Macnee W, *et al*. Targeted treatment in COPD: a multi-system approach for a multi-system disease. *Int J Chron Obstruct Pulmon Dis*. 4, 2009, 321±335.
28. Tangvarasittichai S. Oxidative stress, insulin resistance, dyslipidemia and type 2 diabetes mellitus. *World J Diabetes*. 6, 2015, 456±480.
29. Mirrakhimov AE. Chronic obstructive pulmonary disease and glucose metabolism: a bitter sweet symphony. *Cardiovasc Diabetol.* 11, 2012, 132.
30. Donaldson GC, Wedzicha JA, *et al*. COPD exacerbations. 1: Epidemiology. *Thorax*. 61, 2006, 164±168.