

UNIVERSITI TEKNOLOGI MARA

**PREVALENCE AND FACTORS ASSOCIATED WITH
DYSGLYCEMIA IN CHRONIC OBSTRUCTIVE
PULMONARY DISEASE (COPD) PATIENTS WITH
AND WITHOUT EXACERBATIONS**

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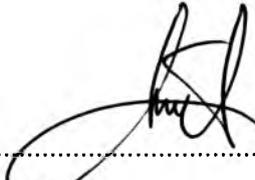
May 2017

AUTHOR'S DECLARATION

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I hereby, acknowledge that I have been supplied with the Academic Rules and Regulations for Post Graduates, Universiti Teknologi MARA, regulating the conduct of my study and research.

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ABSTRACT

Background and Aims:

Dysglycemia, consisting of diabetes mellitus (DM), impaired glucose tolerance (IGT) and impaired fasting glucose (IFG), is a common comorbid of COPD. High prevalence of dysglycemia in COPD is well-recognized, however data comparing COPD exacerbation with incidence of dysglycemia is scarce. Thus, this study aimed to determine whether COPD exacerbation is a risk factor for dysglycemia.

Materials and Methods:

This is a cross-sectional study involving patients with established COPD aged 30 years and above (n=186) attending respiratory specialist clinics in UiTM campuses. Those on long-term corticosteroid therapy, active malignancy or in peripartum periods were excluded. Patients were divided into those with or without hospital admission for exacerbation in the past year. All participants had glycated hemoglobin level and inflammatory marker Interleukin-6 (IL-6) concentration assessment, and patients with no prior diagnosis of dysglycemia underwent oral glucose tolerance test (OGTT).

Results:

76 patients (40.9%) had history of admission for COPD exacerbation, 27 (35.5%) of them were known diabetics. Among the remaining 110 patients with stable COPD, 29 (26.4%) were known diabetics. Mean age was 67.85 ± 9.08 years, mean body mass index (BMI) 25.17 ± 6.08 kg/m², and majority were male (95.2%). Baseline HbA1c in the exacerbation group was higher at 6.51 ± 1.48 % compared to 6.22 ± 1.01 % in the stable group (p=0.135). Among patients subjected to OGTT (n=130), mean fasting blood glucose was 5.43 ± 1.05 mmol/l with no difference between groups. 2-hour post prandial (PP) level was higher in the exacerbation group at 7.91 ± 3.79 mmol/l compared to 7.35 ± 2.82 mmol/l in the stable group (p=0.333). The incidence of new dysglycemia were 40.8% (n=20) and 34.6% (n=28) respectively (p=0.574). Cumulative days of admission ≥ 6 days/year (OR 4.76, CI 1.47-15.45) and weight

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CHAPTER ONE

INTRODUCTION

1.1 RESEARCH BACKGROUND

Chronic obstructive pulmonary disease (COPD) is a group of chronic inflammatory pulmonary disorders characterized by progressive partially reversible airflow obstruction and airways inflammation (Decramer & Janssens, 2013). Its main cause is tobacco smoking, although other factors have been identified as well including exposure to air pollution and ageing. Its comorbidities, or concomitant chronic diseases, include diabetes, cardiovascular disease, osteoporosis and lung cancer.

Acute exacerbation of COPD is defined as sudden worsening of a patient's condition from a stable state into a state that necessitates a change in medication or hospitalization (I. D. Pavord, P. W. Jones, P.-R. Burgel, & K. F. Rabe, 2016). It has cumulative detrimental effect on lung function, which affects patient's exercise capacity and overall health status and carries significant rate of morbidity and mortality.

According to the World Health Organization (WHO), COPD is set to be the third leading cause of death worldwide by 2030 (WHO, 2013). Its burden to the healthcare has also been on the rise, owing to the cost of treatment and prolonged hospitalizations.

Dysglycemia, a broad term referring to abnormalities in blood glucose level, usually refers to diabetes mellitus and pre-diabetes conditions - impaired fasting glucose and impaired glucose tolerance. It is known to be one of the major comorbidities of COPD, with several previous studies shown an established association between the two (Lee, Mao, Lin, Lin, & Hsieh, 2013; Yamane et al., 2013).

While acute exacerbations have been accepted as part of the natural course of COPD, it is still arguable if the nature of exacerbation, namely the severity, frequency and its treatment, played a role in development of dysglycemia, and patients' overall glycaemic control.