

CHAPTER I

INTRODUCTION

1.1. Latar Belakang

1.1 Background

The usage of corticosteroid as immunosuppressant and potent anti-inflammatory are widely use, some of the patient need to consume the medication for long term for more less 3 month. Especially patient with case of patient with chronic disease that will use corticosteroid as treatment, in general population 2% women younger than 50 years old are taking corticosteroid. From the whole population of people who received corticosteroid 19,6- 38% of the population get glucocorticoid induced osteoporosis.(Adami, Rahn and Saag., 2019). Some research from Europe and United states stated that the estimated prevalence of GC use is 0,7% to 17,1%. (Overman RA, Yeh JY, Deal CL., 2013) and (Laugesen K et al, 2017). It is also stated that the prescription rate of long-term GC has increased 34% over past 2- years (Fardet L, Petersen I, Nazareth I., 2011). Meanwhile in Korea, current prevalence of chronic oral *glucocorticoid* use for more than 30 days increase from 0,16% in 2002 to 0,54% in 2015, which is significant and that is why prevention measure is necessary for it (Oh TK, Song IA., 2020).

If the patient leave untreated, the adverse effect of *glucocorticoid-induced osteoporosis* will exist, and the bone resorption will hyperactive eventually will decrease the bone mineral and increase the calcium level in the blood. If the process continue for a long time, it will make the patient prone for fracture. Based on those consideration majority of the articles is proposing a bone mineral density monitoring for the patient that consume long duration of treatment of corticosteroid, for example like patient with *idiopathic thrombocytopenic purpura*. From the 10% of the patient

who received *glucocorticoid* treatment are diagnose with fracture and 30-40% have radiographic evidence of vertebral fracture (Buckley et al., 2017). GCs have been reported to responsible for decrease in bone density and fracture risk approximately chronic GC treatment are diagnose with symptomatic fracture (Curtis JR et al, 2006).

Based on the case of *glucocorticoid-induced osteoporosis* one of the solution is by doing monitoring of bone mineral density measurement to asses bone strength to know the risk of fracture. Based on the meta-analysis *bone mineral density* alone cannot be relied on to detect all of the treatment related effect on other important contributor to bone strength and fracture risk reduction. Other than BMD as prophylaxis of osteoporosis, risendronate is also important for postmenopausal women and salmon calcitonin (Small, 2005). Other paper also mention the guideline of glucocorticoid induced osteoporosis treatment when a patient have a plan to start glucocorticoid and chronically treated patient with GCs more than 3 months (Buckley et al., 2017). The guideline separate into 2 groups, which are those less than 40 years of old and those age more than 40. It also concerned GC status “initiating or continuing GCs treatment” and fracture risk (low or medium or high). The guideline identified adults more than 40 years old had a prevalent of *T score* less than -2,5 at the hip or spine or FRAX / *fracture risk assessment tool* risk of fracture more than 20% as a prediction for 10 years from now. The guideline also include children and adults less than 40 years of old , high risk of fracture in young groups was defined as history of prior fracture. The moderate risk of fracture is considered if the patient treated by GCs for more than 6 month with more than 7,5 mg/day for the concentration. Also BMD Z-score at hip or lumbar spine less than -3 or have rapid decline in bone mineral density of more than 10% over 12 month of GCs (Kanis JA et al., 2011).

Based on those considerations there have several mechanism in dealing with glucocorticoid-induced osteoporosis. Firstly monitoring system, by doing *Dual-Energy*

X-ray Absorptiometry DEXA scan for those people who planning to start the GCs, especially for the long term one in patient with chronic disease. Then asses the BMD Z score, by comparing the patient bone density to average bone density of people at the patient age and gender. From it, then we will calculate the T-score which (less than -2 will be one of indication of monitoring) to know whether the patient has low bone density or not, and of the patient have we will also calculate with FRAX to determine the prognosis of this patient after 10 years ahead. We will also considering patient (age, weight, BMD, family history, and other clinical risk factor, such as RA, Secondary osteoporosis and GCs exposure) in it.

1.2 Research Question

Is there any correlation of low dose and long term corticosteroid therapy with osteoporosis in patient with chronic disease?

1.3 Objectives of Study

1.3.1 General Objective

- Analyze the correlation of low dose and long-term corticosteroid therapy with osteoporosis in patient with chronic disease

1.3.2 Specific Objective

- To analyze the characteristic of the subject.
- To analyze patient distribution that consume long-term corticosteroid therapy.
- To analyze BMD of patient who consume low dose and long-term corticosteroid.
- To analyze the correlation of long-term corticosteroid therapy with osteoporosis in patient with chronic disease

1.4 Significance of Study

1.4.1 Theoretical Significance

- As an update of current treatment and strengthen the previous theory

1.4.2 Practice Significance

- For medical workers, this paper can be the source of SOP of treatment