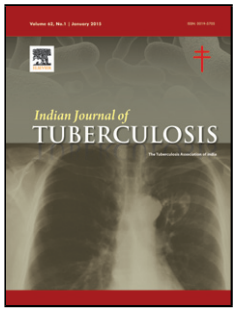

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A Case Risk-Study of Lactic Acidosis Risk in Metformin Use in Type 2 Diabetes Mellitus Tuberculosis co-Infection Patients

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ABSTRACT

Metformin (MET) has possibilities to be utilized as an adjunct of TB therapy in controlling the growth of Mycobacterium tuberculosis (M.tubeculosis). MET enhances the production of mitochondrial reactive oxygen species and facilitates phagosome-lysosome fusion, those mechanism are important in *M.tuberculosis* elimination. Moreover, MET associated lactic acidosis (MALA) needs to be considered and the incidence of MALA in patients with type 2 DM-TB co-infection remains unknown. This result contributes much to our understanding about the clinical effect of MET use in type 2 DM-TB co-infection.

In a purpose to know the MET effect as an adjuvant therapy in TB therapy and Insulin simultaneous therapy, an observational clinical study was done in type 2 DM newly TB co-infection outpatients at Surabaya Paru Hospital. Patients were divided into two groups. First group was MET group, whom was given MET accompanying insulin and TB treatment regimens, the golden standar therapy of DM-TB co-infection. MET therapy was given for at least 2 months. Second group was non MET group, were given insulin and TB treatment regimens. The lactate levels in both group were measured after 2 months.

From 42 participants, there was no case of lactic acidosis during this study period. Data was normal distribution, thus we continued analysis the difference using paired T-test with 95% confidence. There was no difference in lactate levels (p = 0,396) after MET therapy compared to non MET group.

In this study, involving of patients with TB pulmonary diseases, there is no evidence that MET therapy is induced lactic acidosis event nor increased of lactate blood level. Thus we concluded that MET use in type 2 DM-TB co-infection not induced lactic acidosis.

Keyword : type 2 diabetes mellitus-tuberculosis co-infection, metformin, lactic acidosis

1. INTRODUCTION

Tuberculosis (TB) remains a major source of morbidity and mortality throughout the world; one-third of the world's population is estimated to be infected with *M.tuberculosis* whereby approximately nine million people develop the disease each year, and almost two million die annually as a result[1]. DM-TB co-infection is associated with poor glycemic control in DM patients, thus elevated proinflammatory state[2–4]. People with DM had approximately three times the risk of developing TB disease as people without[2,5–10].

Metformin hydrochloride (MET), biguanide, use in type 2 diabetes mellitus for more than 60 years. MET works by inhibiting the production of hepatic glucose, reducing intestinal glucose absorption, and improving glucose uptake and utilization[11–14]. Recently, by a comprehensive in silico study, MET known has possibilities of utilizing as a combination