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Protective effect of omeprazole and lansoprazole on β-receptor stimulated myocardial infarction in Wistar rats

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Abstract

We investigated the effect of omeprazole (OPZ) and lansoprazole (LPZ) on the pathophysiology of myocardial necrosis in rats by inspecting a series of indicators like hemodynamic parameters, biochemical estimations and histopathological changes in the myocardial tissue. Rats received either OPZ, LPZ (50 mg/kg/day, p.o.) individually for 7 days with concurrent administration of isoproterenol (ISO) (150 mg/kg, s.c.) on 6th and 7th day of study period to induce myocardial infarction. On the 8th day after measuring hemodynamic parameters, rats were killed and parameters were evaluated. ECG waves were found to be normal in the treatment group. ISO control rats revealed escalation in the oxidative stress as evidenced by depletion in the content of SOD, GSH, catalase and increase in the level of MDA and NO as compared with the normal rats. Treatment with OPZ and LPZ significantly reduced the ROS, indicated by an increase in the endogenous antioxidants and a decrease in NO and MDA levels. ISO control rats showed a significant elevation in the levels of pro-inflammatory cytokine TNF-α as compared to the normal and treatment group of rats. Administration of OPZ and LPZ does not exhibit any significant toxicity. Our findings reveal that multiple doses of OPZ and LPZ may have distinctly minimized the ISO-induced myocardial necrosis by declining the hmodynamic parameters, oxidative stress and pro-inflammatory cytokine TNF-α in myocardial infarcted rats.

Keywords Myocardial infarction · Omeprazole · Lansoprazole · ROS · Cytokines

Introduction

Myocardial infarction (MI) is a state of cardiac necrosis, results due to imbalance between blood supply and demand, evidences have assigns MI to specific pathways including cytosolic calcium overload, increase in superoxide anions and free radicals (ROS), cardiomyocyte apoptosis and disruption in the mitochondria [1]. MI is associated with an irreversible necrosis of the myocardial tissue. MI is one of the main causes of morbidity and mortality all over the world in both the sex [2]. As per WHO report in the future, heart disease and stroke will become main reason of death and disability worldwide up to 2020 [3]. Incidence of death with myocardial infarction is due to severe increase in ROS and inflammation in the myocardium. The treatments for MI include the reinstatement of blood supply to the ischemic tissue and maximal recovery of the functional myocardium [4]. It is very challenging to mimic the human MI in animal model with isoproterenol (ISO), which is commonly used chemical to induce MI in rats [2]. ISO is a synthetic β-adrenergic receptor agonist, which induces MI through excess generation of highly cytotoxic free radicals [3]. ISO leads to development of ROS via its auto-oxidation and reported to cause cardiomyocyte necrosis and fibrosis by accelerating the intracellular calcium overload through opening of mitochondrial permeability transition pore channels [4]. Plenty of reports suggest the positive effects of different molecules on the MI. Proton pump inhibitors (PPIs) are backbone treatment for gastroesophageal reflux disease (GERD) [5]. The reported activities of omeprazole (OPZ), lansoprazole (LPZ) and other PPIs are better proven to be an anti-ulcerogenic, anti-inflammatory, anti-apoptotic and antioxidant in GERD [6, 7]. OPZ also possesses anticancer

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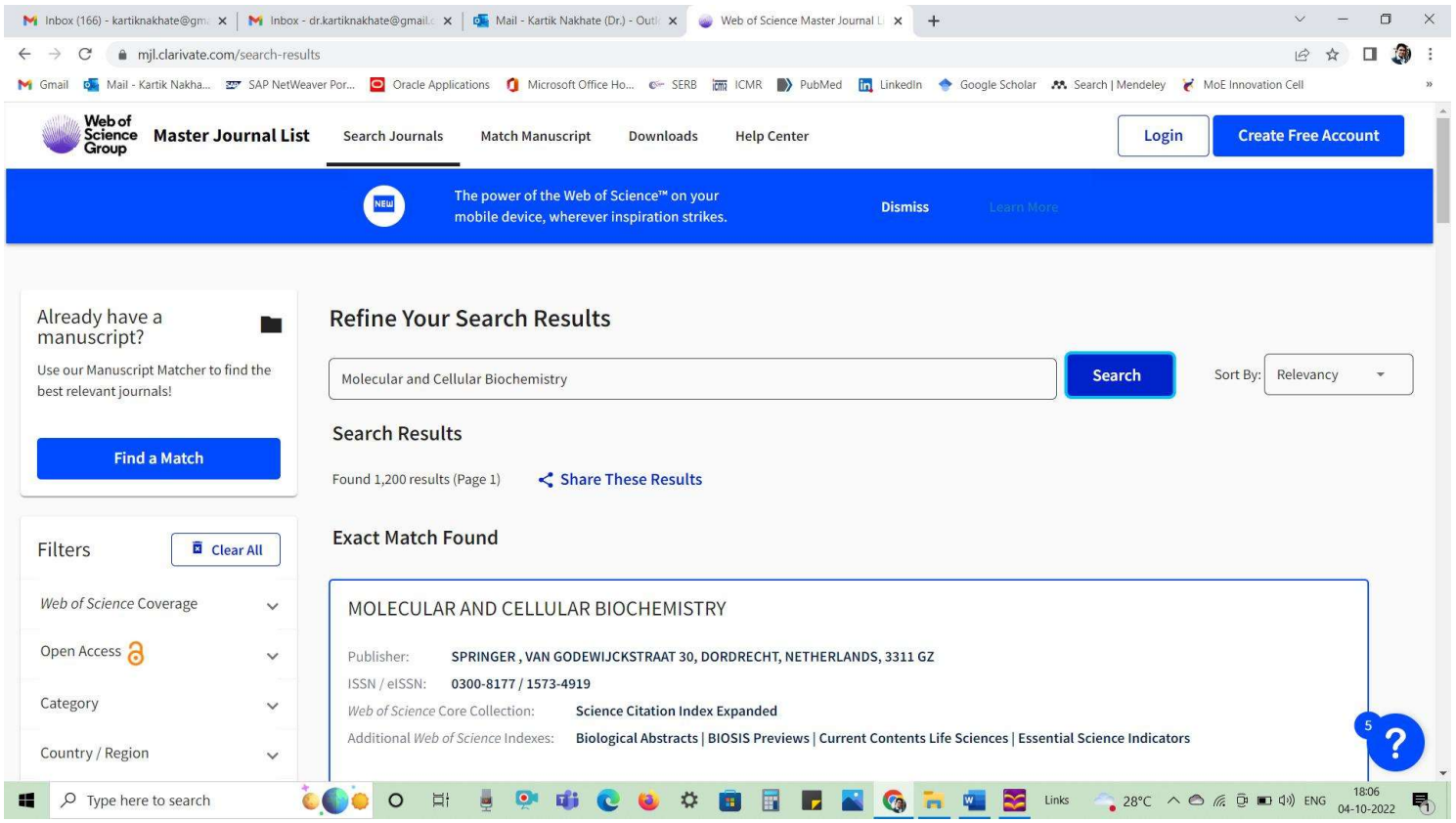
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