Editorial

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Journal of Metabolism

2017
Vol.1 No.1:2

Could Pulmonary Hypertension Be the Cause of Renalase Insufficiency?

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Received Date: October 25, 2017; **Accepted Date:** October 27, 2017; **Published Date:** October 30, 2017

Citation: Aydin S, Ugur K, Aydin S (2017) Could Pulmonary Hypertension Be the Cause of Renalase Insufficiency? J Metab. Vol. 1 No 1: 2.

Editorial

Pulmonary hypertension is a serious, progressive and life-threatening disease associated with increased blood pressure in cardiac patients, defined as >25 mmHg and Effort >30 mmHg, while the pulmonary artery mean pressure is normally 14 mmHg [1]. This hypertension associated with interstitial lung disease recognized and is frequently associated with substantial morbidity and mortality. Congestive heart failure, other heart diseases, birth defects of the heart, chronic lung disease, obstructive sleep apnea, and certain autoimmune disease (for example, rheumatoid arthritis) cause pulmonary hypertension. The right heart failure and sudden death were directing a cause of death of pulmonary hypertension [2]. This hypertension is also more common among women, non-Hispanic blacks, and its risk increases in older patients aged 75 or older.

It has been recently showed that the prototype of selective pulmonary vasodilator - Nitric Oxide (NO) is currently the most effective option in the management of this hypertension. There is a direct connection between the amount of circulating dopamine and epinephrine as well as norepinephrine. The nerves around the kidney veins cause extreme adrenaline secretion from the kidneys, causing hypertension. Prevention of excessive adrenalin secretion using the renal denervation method partially or completely prevents hypertension. The mechanism underlying pulmonary hypertension is not known but it may be linked with renalase.

Renalase, first discovered in 2005, is a flavin/adenine/dinucleotide-dependent amine oxidase [3] that is primarily synthesized from the kidneys and other biological tissues such as heart, intestine, liver, skeletal muscle, endothelium and brain and is secreted into the blood and urine [4,5]. Circulated normal concentration of renalase is approximately 3–5 μg/mL [6]. Its levels are regulated by at least three factors: renal function, renal perfusion and catecholamine (dopamine, epinephrine and norepinephrine) levels. It has been reported that renalase reduces blood pressure and heart rate and protects cells and organs against ischaemic and toxic injury, states, including cardiac injury and acute pancreatitis by modulating calcium transport.

Over all given information above this article hypothesized that dopamine as well as epinephrine- and norepinephrine-metabolizing renalase [3,7,8] may be linked to pulmonary hypertension as that described below. Since the main function of renalase in biological systems is metabolization of epinephrine and norepinephrine to 3-methoxy-4-hydroxymandelic acid (VMA) and dopamine to homovanilic acid. Failure of these cathacolamines to metabolize to their final metabolites may cause headache, vomiting, nausea, dizziness and especially hypertension. Therefore, renalase enzyme quantities below physiological concentrations may cause pulmonary hypertension, and there may be an association between renalase and pulmonary hypertension. Even though now there is no exactly cure for pulmonary hypertension [2]. However, renalase treatment in case of pulmonary hypertension might be also another option in place of pulmonary vasodilators (such as NO) in the future. Diagnosis of pulmonary hypertension is also currently difficult until the last stage of the disease, and measurement of renal enzymes in these patients may help diagnose pulmonary hypertension. Briefly, pulmonary hypertension is a rare, lethal disease that occurs in isolation or in association with myriad diseases [2], and so that measurement of renalase might help to monitor treatment response and progression of disease in the future.

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