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Effect of Bilirubin in Diabetic Kidney Disease

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Introduction

Historically, bilirubin was thought to be a non-functional byproduct of heme catabolism, a sign of liver disease, or, worst of all, potentially neurotoxic molecules. Because mammals expend energy and resources converting biliverdin (a non-toxic compound that is relatively easy to secrete) into bilirubin (which must be further metabolised for excretion via a biliary system), it is reasonable to assume that bilirubin is more than just a byproduct of heme catabolism [1].

Description

Clinicians began to notice mild hyperbilirubinemia presented by Gilbert Subjects (but also levels in the upper quartiles of the currently accepted physiological serum bilirubin range) protected against the rise of civilization diseases (cardiovascular diseases, diabetes, obesity, metabolic syndrome) based primarily on oxidative stress in the last few decades. Meanwhile, basic scientists began to investigate the mechanisms involved in this molecule's protective role, and they discovered that bilirubin is an important modulator of various biological functions in the human body, and it is capable of acting as a hormone directly targeting its receptor to exert its effect [2]. Because a small increase in serum bilirubin levels appears to significantly reduce the impact of oxidative stress-related diseases, scientists are considering increasing serum bilirubin levels as a preventive method against civilization diseases. Diabetic Kidney Disease (DKD) affects approximately 20% to 40% of patients with type 1 or type 2 diabetes mellitus. Early detection of DKD is critical to preventing the disease from progressing to end-stage renal failure [3]. The current focus of research is on developing methods to predict DKD progression and improving treatment.

Endogenous bilirubin appeared to be important as a potential marker for progression as well as a therapeutic target for DKD prevention. According to various recent studies, which are well explained in this review, total serum bilirubin level could be considered a marker of DKD progression, useful in detecting low- and high-risk patient groups. Patients with low–normal total bilirubin concentrations may be aggressively managed to postpone the progression to kidney failure. Bilirubin, as a marker, has the advantage of being easily, cheaply, and routinely measured in most medical centres [4]. More research is needed to determine whether total bilirubin concentration is a potential therapeutic target for CKD prevention. There are numerous measures that can mildly raise serum bilirubin levels, such as lifestyle changes, the use of natural compounds as nutraceuticals or chemical drugs, and the inclusion of bilirubin in nanoparticles [5].

Conclusion

Recent basic and clinical studies have demonstrated its effects in the onset and progression of renal diseases, demonstrating that only minor or very mild increases in serum bilirubin concentrations provide real clinical benefits. Endogenous bilirubin appeared to be important as both a potential marker for progression and a therapeutic target for DKD prevention. Patients with low–normal total bilirubin levels may be managed more aggressively in order to postpone the progression to kidney failure. More research is needed to determine whether total bilirubin concentration is a potential therapeutic target for CKD prevention. Scientists are considering modulating plasma bilirubin concentrations to prevent a variety of oxidative stress and inflammation-mediated diseases, including DKD.

References

1. Torres, Antoni, Willy E Peetermans, Giovanni Viegi, and Francesco Blasi. "Risk factors for community-acquired pneumonia in adults in Europe: a literature review." *Thorax* 68 (2013): 1057-1065.
2. Small, William Jr, Jonathan B Strauss, Anuja Jhingran, and Catheryn M Yashar, et al. "ACR Appropriateness Criteria® definitive therapy for early-stage cervical cancer." *Am J Clin Oncol* 35 (2012): 399-405.
3. Kaye, Walter H, Angela Wagner, Julie L. Fudge and Martin Paulus. "Neurocircuitry of Eating Disorders." *J Eat Disord* 6 (2011): 59–80.
4. Agarwal, Pooja, Mahesh Prakash, Manphool Singhal, and Sanjay Kumar Bhadada, et al. "To assess vascular calcification in the patients of hypoparathyroidism using multidetector computed tomography scan." *Indian J Endocrinol Metab* 19 (2015): 785–790.
5. Fitzpatrick, Thomas B, and MA Pathak. "Historical aspects of methoxsalen and other furocoumarins." *J Invest Dermatol* 32 (1959): 229–231.

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