Metformin improves insulin resistance and corrects dyslipidemia in patients with diabetes mellitus

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Description

Chronic Kidney Disease (CKD) is a serious public health problem, with an estimated prevalence of 8-16% of the world's population. Treating this condition is expensive and many of these patients end up on renal replacement therapy (dialysis or transplantation). Furthermore, the accelerating increase in diabetes patients will have a significant impact on the development of Diabetic Kidney Disease (DKD). This is because it is one of the most common complications of both types of diabetes. It is also the leading cause of End-Stage Renal Disease (ESRD), accounting for nearly half of cases in developed countries. Therefore, intensive interventions in the metabolic control of the disease are needed to minimize the risk of ESRD and reduce the severe socioeconomic burden posed by this disease. DKD occurs in up to 40% of people with type 1 or type 2 diabetes. This not only poses a risk of progression to ESRD, but also leads to a significant increase in cardiovascular morbidity and mortality. The development of DKD is a complex process that can be altered by proper metabolic control and proper glycemic control is essential. The use of metformin has been integrated as a first-line treatment strategy.

Metformin inhibits hepatic glucose production, a primary mechanism of action that ameliorates hyperglycemia in type 2 diabetes. Biochemically, metformin not only suppresses gluconeogenesis and stimulates glycolysis, but also inhibits glycogenolysis, a pathway important for increasing hepatic glucose production. Apart from these beneficial effects on hyperglycemia, metformin improves insulin resistance and corrects dyslipidemia in patients with DM and various pleiotropic effects. Its anti-inflammatory properties are not fully understood. For decades, one of the major limitations imposed on metformin by regulatory authorities was renal function, with some creatinine cut-off point to contraindicate its use.

Metformin is primarily excreted without renal metabolism, glomerular filtration, and tubular secretion. Patients with renal failure are therefore susceptible to the accumulation and development of lactic acidosis, a fatal complication. Indeed, the trigger for lactic acidosis in patients receiving metformin is abrupt loss of tubular secretion. Such loss does not occur in stable CKD, but is a hallmark of rapid capacity depletion associated with acute kidney injury or inter-current illness. Therefore, patients with chronic kidney disease should be encouraged to be aware of potential side effects and to provide written information on metformin suspension if they have co-occurring medical conditions that may lead to rapid depletion of metformin doses. Until recently, an association between lactic acidosis and metformin accumulation was not established. This led to regulatory warnings, which unfortunately limited its use and encouraged an undue fear of the risk of such undesirable events.

For certain combinations containing metformin, recommendations for dose adjustments of other medications in patients with renal impairment should be considered. Dose adjustments may only be possible using individual metformin tablets and other drugs. Because no other active ingredients in the combination should be used, some fixed combination products may cause renal impairment. Not recommended for patients. The best way to avoid DKD is to prevent diabetes itself. However, once diagnosed, glycemic control can delay the onset of kidney disease.

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Conflict of interest

The author has nothing to disclose and also state no conflict of interest in the submission of this manuscript.

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