Dear Editor,

Glucocorticoids are a large class of drugs that have antiinflammatory and immunosuppressive properties and remain a necessary component of treatment for many disease [1]. In addition to therapeutic effects, corticosteroids have several side effects, including induction of hyperglycemia in nondiabetic patients and exacerbation of hyperglycemia in patients with diabetes [2]. Glucocorticoids also lead to increased insulin resistance in all patients [3]. The major mechanism that causes glucose intolerance after glucocorticoid administration is reduced insulin sensitivity [4]. Glucocorticoids impair glucose uptake through post receptor defects, such as diminished GLUT4 expression and migration and decreased glycogen synthase through reduction of glycogen synthase activity [5]. Moreover, glucocorticoids enhance hepatic gluconeogenesis by increasing phosphoenolpyruvate carboxykinase and glucose-6-phosphatase activity [6]. Furthermore, AMP-activated protein kinase (AMPK) mRNA expression is correlated negatively with glucocorticoid mRNA expression, which AMPK can reduce blood glucose level by reduction of glucose production in the liver [7]. In addition, glucocorticoids inhibit production and secretion of insulin by pancreatic β cells [8]. This glucocorticoid-induced hyperglycemia is associated with endothelial dysfunction and cardiovascular disease[9].

Currently, blood glucose-lowering agents, including sulfonylureas, metformin, thiazolidinediones (TZDs), and insulin, are the only option for treatment of diabetic patients [10]. These [11,12]. Previous studies suggested that some bioflavonoids such as quercetin could be beneficial in controlling blood glucose[13].

Quercetin is a common flavonol found in sources such as tea, onion, and apple. It has been shown to have antioxidant, antidyslipidemic, endothelial function-improving, and antihyperglycemic properties [14-17]. Antihyperglycemic mechanism of quercetin involves its effect on GLUT4 translocation from cytoplasm to cell membrane through increased phosphorylation of AS160 [18]. Moreover, this flavonoid protected pancreatic β cells from oxidative stress, resulting in increased insulin secretion [19]. Quercetin could also inhibit intestinal α-glucosidase activity and decrease postprandial hyperglycemia [20]. Furthermore, quercetin is an AMPK activator and affects energy metabolism and blood glucose levels [18].

Taken together, evidence shows that quercetin could potentially be an alternative hypoglycemic agent without side effects in diabetic patients. Given the fact that so far no study has been done on this issue, this can be the subject of future studies.

Conflict of interest:
The authors have nothing to declare.

References:
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