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radioimmunoassay or immuno-turbidimetric method. When urinary albumin excretion exceeds >300 mg/day it is considered as overt proteinuria [3].

DN is characterized by hyperglycemia which is a primary cause of glomerular injury in patients with diabetic nephropathy as it responsible for the development and progression of diabetic nephropathy through metabolic derangements, including increased oxidative stress and accumulation of advanced glycation end products, as well as such hemodynamic factors as systemic hypertension and increased intraglomerular pressure [4].

Glycosaminoglycans (GAGs) are long unbranched mucopolysaccharides consisting of a repeating disaccharide unit. It has been proposed that hemodynamic alterations and structural changes in glomerular basement membrane glycosaminoglycans may play a role in the pathogenesis of DN [5]. Moreover, GAGs strongly in uence thickness, integrity and permselectivity of the endothelial glycocalyx which its composition is strongly alterated in diabetes patients, who typically show early sign of renal damage [6]. Glucosamine is a precursor in the synthesis of GAGs. Glucosamine also is a component of the hexosamine pathway, which has been demonstrated to be a mechanism by which glucose leads to diabetic complications [7]. In the hexosamine pathway, glucose is metabolized to hexosamine via the transfer of an amide group from glutamine to fructose 6-phosphate to form glucosamine 6-phosphate. is subsequently is metabolized to hexosamines such as UDP-N-acetylglucosamine (UDP-GlcNAc) and UDP-N-acetylgalactosamine, which are the building blocks for glycosaminoglycan synthesis. e rate-limiting step in the hexosamine pathway is the initial transfer of the amide group from glutamine, which is catalyzed by the enzyme glutamine: fructose-6-phosphate amidotransferase (GFAT; EC 2.6.1.16). GFAT activity is upregulated by high glucose and insulin levels [8]. N-Acetyl-β-D-glucosaminidase (NAGase) is an enzyme in lysosome which is contained abundantly in the renal tubular epithelia and is related to the degradation of mucopolysaccharides and glycoproteins.

Citation:

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In this study, we report the beneficial e ect of HMF in controlling the progression of kidney damage by modulating the diabetes induced e ects on hexosamine pathway, renal oxidative stress and glycoprotein deposition in STZ induced diabetic rats.

E HMF

Enlargement of Kidney is an early feature in experimental and diabetes due to an increase in the capillary length and diameter and was correlated with the degree of glycaemic control [32]. We observed partial yet significant reduction in the kidney weight with HMF administration.

Hyper functional -8(y)-208(i)3(s)-2T ( )TjEMC80ial yet lenlenlenlenlenlenal dC /idneniD 510 >>BDC -1.575 -1.2 Td[(e)-6(a)9(r)4(l)7(y)-42(s)5(
of HMF [34]. In addition to stimulatenay e ect of AGE [35] or BME [36]

content.

been noted in diabetic conditions. In addition, the measurement of TBA-reactive substances is frequently used to determine the oxidative stress level in diabetic patients [51].

As shown by present study, the levels of TBA-reactive substance in kidney of diabetic rats were significantly increased, whereas the administration of HMF significantly decreased these TBA-reactive substance levels compared to diabetic control rats. erefore, the administration HMF was suggested to alleviate oxidative stress of diabetic pathological conditions through the inhibition of lipid peroxidation.

Protective e ect of GSE against renal damage in experimental diabetic rats due to antioxidant properties of its constituents that include Flavonoids, Phenols, Tannis (Phenolic compounds) and Triterpenoids [52].

Also, the presence of glycosides, saponins, triterpines, steroids, vitamin C and A, phytochemicals such as momorchins, momordinol, momordicins, charantin, cucurbitacins, diosgenin, goyaglycosides, goyasaponins in BME that are characterized by their antioxidant activity [53].

On other hand, Ginsenoside the major constituent in AGE content that could induce the antioxidant enzymes which are important for maintaining cell viability by lowering the level of oxygen radical generated from intracellular metabolism.

AGE, also has the ability to intercalate into the cell membrane, changing its uidity and inhibit lipid peroxidation by chelating genera54]T,BDC -1.575 (l)-5(2260(i))7(li)3(sm10(lece)477yo)1226012(r)-7(co)1le(P)5-4pd1l0(radic)-HMFl0(radro)1le-3(y)-40l 453].

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