Renal Protective Effect of Scutellarin Based Antioxidation on Diabetic Patients

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Abstract: Diabetic nephropathy is the most common chronic microvascular complication of diabetes mellitus, which is one of the main causes of end-stage renal failure. Breviscapine can prevent diabetic nephropathy by inhibiting oxidative stress.

1. Introduction

Diabetic nephropathy is the microvascular complication of diabetes mellitus and one of the main causes of end-stage renal failure. The increase of renal oxidative stress and the decrease of antioxidant capacity play an important role in the pathogenesis of diabetic nephropathy[1]. Barbital can prevent diabetic nephropathy, improve the body's antioxidant function and inhibit PKC by inhibiting oxidative stress.

2. Oxidative Stress in Diabetic Nephropathy

Various factors of diabetes mellitus, the body generates too much reactive oxygen species, destroys the normal balance of normal oxidation-reduction reaction in renal tissue, and becomes the cause of oxidative stress in renal tissue. ROS can activate the major enzyme protein kinase C and microthermal activated protein kinase, activate NF - JB and activator protein 1, and start the transcription of various cytokines and growth factors[2]. It can stimulate the proliferation or hypertrophy of renal tissue cells, increase collagen synthesis, reduce cell lysis, induce renal hypertrophy, extracellular matrix deposition, glomerular basement membrane hypertrophy, and promote diabetic nephropathy. Because of the increase of renal blood flow, hydrostatic pressure of glomerular capillaries and the increase of glomerular filtration rate (GFR), the dynamic of renal blood circulation was changed in the early stage in diabetic patients. The existing data show that high mitochondrial perfusion, high internal pressure and high perfusion are one of the initiation factors of diabetic mitochondrial sclerosis[3]. The main reason for the increase of glomerular filtration rate of DN is the results of the study on the intrahepatic dynamics of DN animal model with the help of the technique of renal celiac puncture. In the intravascular renal resistance, especially the tolerance of arterioles and arterioles, the centrifugal confirmation is reduced, and the single medium isoionic flow will increase. Compared with the distal arteriole, the resistance of the distal arteriole is more obvious, which increases the glomerular filtration pressure and makes the glomerulus tense. The formation of abnormal renal blood circulation in DN is the result of a combination of multiple factors. It is generally believed that hyperperfusion and hyperfiltration are related to hyperglycemia in the first place. In animal experiments and clinical studies, strict management of blood glucose value was confirmed as diabetic animals and diabetic patients. GFR decreased, renal hypertrophy decreased, and microalbumin excretion decreased by 21%. Prostaglandin (PG), nitric oxide (no), to reduce endothelin peptide (ET), atrial natriuretic peptide (ANF), growth hormone (GH), glucagon, insulin, transforming growth factor β 1 (TGF s 1) and many other factors involved in DN excessive flow, resulting in high filtration. At present, the blood

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of glomerulus is changing dynamically, the glomerular filtration rate and high intracranial pressure, the expansion of glomerular basement membrane and the hypertrophy of glomerular basement membrane, eventually leading to glomerulosclerosis, which may cause the damage of human blood circulation mechanics changes in endothelial cells and epithelial cells, it may increase the protein filtration according to the damage of normal filtration barrier[2]; the glomerular capillary pressure may The activation of direct PKC.

3. Breviscapine

Eigeron breviscaps is a perennial herb of Compositae in Yunnan and Guangxi. Its main active ingredient is flavonoids containing flavonoids and flavonoids. According to the compendium of Materia Medica, "the lamp is thin and bitter, slightly bitter and sweet. It has the functions of dispersing cold, rheumatism, promoting blood circulation and removing blood stasis, unblocking collaterals, eliminating inflammation and pain, etc. it is often used to treat rheumatism. The formation of cerebral thrombosis, cerebral ischemia and sequelae of cerebrovascular disease. According to the report of life science in 1999, the vascular endothelial function of diabetic rats has been protected, the data is - 8943; more and more reports about lighting therapy are reported in DN. For example, Wu Yonggui et al. 73, STZ induced DN mice model on the impact of flavonoids were discussed, the kidney index of diabetic rats decreased, 24-hour urinary albumin excretion rate decreased, renal TGF-b 1 and CTGF expression were inhibited, the activity of differential superoxide enzyme (s d) increased. Catalase (CAT) Lin Hui et al[3]. For the protection of kidney in diabetic rats, cyanin and specific PKC Inhibitor (ly 33531) are similar to ly 33531, which can inhibit PKC activity in renal tissue and reduce PKC activity in cells. The plasma is transferred to the cell membrane. Clinical research shows that anthocyanin can reduce blood lipid and blood viscosity, improve microcirculation and renal function, and reduce urinary protein excretion in DN patients. Well, these studies show that cyanidin has a protective effect on DN. The mechanism may be related to the inhibition of PKC activity and oxidative stress, the inhibition of TGF BL over expression and the regulation of lipid [4].

4. Effect of Breviscapine on Diabetic Nephropathy

4.1. Breviscapine is the Whole Grass of Chrysanthemum Family

The active components are flavonoids, such as diacid, picric acid, tricarboxylic acid, etc. In addition, breviscapine is also effective in the prevention and treatment of DN[5]. Prevention of diabetic nephropathy antioxidant action a breviscapine inhibits oxidative stress: the body's oxidative response reacts with many organic compounds and biofilms to produce lipid peroxides, and the amount of highly reactive oxygen free radicals produces it. In particular, it destroys the barriers to myocytes and mitochondria, calcium + transport, intracellular calcium + overload and production. The chemical name of rutin is 4,5,6-trihydroxyflavonoid 7-glucoside. Several phenolic hydroxyl groups combine to form a ring structure. The reduction potential ratio of flavonoid radicals is low. The ability to remove reactive oxygen species. In the experimental study, breviscapine effectively inhibited the production of O 2 by GSH and Na 2SeO 3, and increased the dose rate and dose dependence. Breviscapine can remove harmful free radicals and prevent cell peroxidation[6]. In the initial stage of the action of antioxidant free radicals, the activity of drugs can eliminate oxygen free radicals through peroxides and glutathione peroxidases. It has an antioxidant system that activates the body and shows the effect of antioxidant free radicals.

Table 1 Changes of renal index of 12w diabetic rate

Group	n	Renal index
Con	10	6.36±0.46
DM	10	9.05±1.08
Cap	9	7.78±1.21

Rh	8	8.48±1.25
Br	10	7.85±0.98
TL	8	7.97±0.99

4.2. Breviscapine Inhibits Lipid Peroxidation

The increase of lipid peroxidation is not only through direct oxidation of electron unit recombinants, but also through oxidation of lipid, protein, nucleic acid, oxygen, cell function and integrity, and destruction of neutral granulocyte and lymphocyte macrophage. In addition, stellate cells can induce macrophages and stellate cells' inflammatory self-regulation and promote fibrosis. Heterobiotics and free radicals are the membrane lipid peroxidation of cells, which can cause the formation of biopolymers and Schiff bases, such as lipid peroxides, proteins and dissociated amino acids, leading to the bridge of biomolecules, protein denaturation and damage of cell membrane function. The membrane acid response (tbar) induced by superoxide and biopolymer free radicals has capacity dependent blocking effect[7]. The drug can prevent the formation of free radicals or directly capture free radicals and reduce lipid peroxidation. This effect is related to the free phenolic hydroxyl in the molecule. Flavonoids contain phenolic hydroxyl groups. Lipid peroxidation occurs on the cell membrane, providing electrons to free radicals and effectively preventing free radical chain reaction. Bupitazapine effectively inhibited mitochondrial lipid peroxidation induced by chitin oxidase and fesso 4 - H 2O 2, and was able to scavenge O 2 produced by xan XO. Vitamin is formed by the reaction of oxygen free radicals and mitochondrial membrane. Lipid peroxidation hinders lipid peroxidation in two stages. The capture of oxygen free radicals and chelation of Fe2 + are the mechanisms by which fibrin blocks mitochondrial lipid peroxidation. Modern molecular biology research shows that various cytokines are involved in the pathogenesis of DN, TGF - B1 is the most significant. TGF - B 1 is a unique fibrocytokine. Most tissue cells synthesize and secrete TGF - B 1. Among them, the expression of kidney is the most abundant. Kidney is mainly distributed in renal tube and kidney. In addition, the kidney is also the main target organ[8]. A number of studies have shown that TGF - B 1 levels significantly increased in diabetic state. For example, 81 is STZ induced diabetes in the blood glucose increased after 24 hours, TG and B 1 mRNA expression began to increase in the renal cortex, reached Plato after 96 hours, and then maintained at a high level. The expression of TGF - B 1 in the cortex of rats was about 2 times higher than that in the normal group. Human studies have found the same result - TGF - B 1 in DN is mainly to inhibit cell proliferation, promote cell hypertrophy, promote the accumulation of ECM. TGF - B 1 prevents cells from invading g phase and entering S phase, increases the amount of DNA and protein in cells, and increases cell volume. TGF - B 1 mainly promotes the synthesis of various collagen and other ECM components[9]. FN, LN and various proteoglycans, etc. (2) hinder the decomposition of ECM, not only the synthesis of enzymes that decompose the components of ECM, such as collagenase and plasmids activating factor. However, the discovery of protease inhibitors, which are the same as tissue inhibitors, can promote the expression of ECM receptor, increase the adhesion of extracellular matrix, and promote the calm of ECM. The use and use of TGF - 01 neutralizing antibody were effectively inhibited. At present, TGF - 01 has been considered as a new therapeutic target.

5. Conclusion

Oxidative stress and low antioxidant capacity play an important role in the pathogenesis of diabetic nephropathy[10]. Diabetic patients have obvious oxidative stress and renal tissue is damaged by ROS and PKC. SOD, GSH Px, cat and other antioxidant enzymes are glycosylated or oxidized, the content of which is sharply reduced, so that the body's antioxidant capacity is reduced, and promote the onset of diabetic nephropathy. Barbital can prevent diabetic nephropathy, improve the body's antioxidant function and inhibit PKC by inhibiting oxidative stress. The improvement of the theoretical basis of the detailed research on the defense mechanism of diabetic nephropathy and

the development of traditional Chinese medicine and the promotion of clinical application and consciousness are really promoted in the application program.

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