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Exploration of the Mechanism of Intimal Hyperplasia in Autogenous Arteriovenous Fistula

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Abstract: ***Objective:*** ***To explore the mechanism of intimal hyperplasia (IH) in autogenous arteriovenous fistula (AVF) from the perspective of integrated traditional Chinese and western medicine.*** ***Methods:*** ***This study reviewed the relevant literature, analyzed and summarized the mechanism of intimal hyperplasia of autogenous arteriovenous fistula from the perspective of integrated traditional Chinese and western medicine.*** ***Conclusion:*** ***Intimal hyperplasia of autogenous arteriovenous fistula involves many aspects. From any Angle, it provides a theoretical basis for clinical research and further guides clinical practice.***

Keywords: Autogenous arteriovenous fistula, Intimal hyperplasia, Proliferation of smooth muscle cells, Inflammatory cytokines**.**

1. Introduction

End-stage renal disease (ESRD) is a severe chronic kidney disease that usually requires renal dialysis or transplantation for treatment. The incidence of ESRD is increasing year by year, which has become one of the global public health problems. According to the World Health Organization, approximately 85 million people worldwide are affected by ESRD. The onset age of ESRD was mainly concentrated over 40 years old, and male patients were more likely to develop ESRD. The disease causes of ESRD are complex, including diabetes, hypertension, glomerulonephritis, polycystic kidney, etc. In addition, some drugs, toxins and genetic factors may also lead to ESRD. Over the past three decades, chronic kidney disease (CKD) has steadily climbed the ranks of the world's leading causes of death. In 2016, CKD ranked 13th in the list of causes of death. It is predicted to rise to the fifth cause of death by 2040. With 850 million people suffering from chronic kidney disease (CKD), CKD has been recognized as an important global public health event. When chronic kidney disease reaches end-stage renal disease, it must be replaced by renal replacement therapy (including hemodialysis, peritoneal dialysis, kidney transplantation, etc.) to replace normal renal function and maintain life. There are approximately 15 million patients with end-stage renal disease (ESRD) worldwide, and the rate of increase is 5% per year. The treatment of ESRD patients requires hemodialysis to remove uremic toxins and control blood volume [1]. Good vascular access is the "lifeline" for patients with end-stage renal disease to maintain hemodialysis [2].

In 1943, the application of hemodialysis (HD) to treat and prolong the survival time of patients with end-stagerenaldisease (ESRD) became a new and effective method [3]. Brescia et al. [4] first created autogenous arteriovenous fistula (AVF) surgery, which is the preferred vascular access technology for patients with long-term HD. At present, the failure rate of arteriovenousfistula (AF) is increasing gradually, and the patency rate is decreasing [5]. The key factor for the failure of AVF is intimalhyperplasia (IH). At present, studying the mechanism of intimal hyperplasia of autogenous arteriovenous fistula is of great significance for improving the prognosis of patients with chronic renal failure and improving their quality of life.

2. TCM Pathogenesis of Arteriovenous Fistula Stenosis

According to traditional Chinese medicine (TCM), Yang master initiates, generates, is agitated, and changes qi, and the Yang qi of the heart is the Yang of the heart, which drives the blood circulation back and forth. Therefore, the lack of outward expansion of AVF vessels is due to the insufficient encouragement and promotion of heart Yang. AVF anastomoses the arteries and veins, which dissipates the Yang qi of the arteries and flows into the Yin of the veins. In addition, hemodialysis patients have long-term spleen and kidney deficiency, qi and blood deficiency, so it is easy to "Yang slightly qi weak".

In clinical practice, patients with AVF often have complications such as stenosis and thrombosis after the establishment of AVF. Modern medicine mainly believes that it is related to insufficient expansion of the blood vessel outward and stenosis caused by intimal hyperplasia (or valve). In traditional Chinese medicine, it is regarded as "phlegm and blood stasis", so it forms "phlegm and blood stasis". Therefore, the pathogenesis of arteriovenous fistula stenosis in traditional Chinese medicine is "Yang weak qi, phlegm and blood stasis", and the treatment should be based on the principle of "warming Yang and tonifying qi, clearing phlegm and blood stasis".

3. Western Medicine Research on Arteriovenous Fistula Stenosis

Pathogenesis of arteriovenous fistula stenosis in recent years, the pathogenesis of AVF stenosis has not been unified. At present, it is believed that inflammation, toxins, cell proliferation, migration and phenotypic transformation, repeated dialysis puncture, AVF surgical injury, etc., lead to AVF stenosis and thrombosis [6-7]. In addition, hypertension, diabetes, hyperlipidemia and smoking are also important factors that cause AVF stenosis and shorten the service life of AVF [8].

3.1 Inflammation

Foreign studies have found that local or systemic inflammatory response is associated with intimal hyperplasia in AVF Birth and development are closely related. Local inflammation and hypoxia caused by surgical trauma of internal fistula and the whole body of uremic patients the role of inflammatory factors in AVF dysfunction is expressed by the state of inflammatory response. Back in 2000, Meeus. A large number of inflammatory cells infiltrated around AVF vessels in uremic patients, suggesting that inflammation may be involved in the development of renal failure. Vascular damage [9]. Patients who are treated with maintenance hemodialysis often have puncture at the site of internal fistula, which can produce inflammatory reactions. It is manifested as obvious infiltration of mononuclear macrophages at the puncture site, which activates inflammatory factors and promotes their secretion. Inflammation acts on the intima of the blood vessel, combined with the injury caused by multiple punctures, leading to intimal hyperplasia of the radial artery. The more severe the inflammation, the more obvious the intimal hyperplasia, and the stenosis or even blockage of the internal fistula, eventually leading to the failure of the internal fistula [10]. Local trauma of AVF surgery can lead to infiltration of macrophages and lymphocytes. This phenomenon is more significant in patients with chronic kidney disease. Macrophage infiltration may be mediated by its migration inhibitory factors, while lymphocyte infiltration may be mediated by extracellular signal-regulated kinase (ERK) and p38 mitogen activated protein kinase (p38 mitogen activated protein) kinase (MAPK) signaling pathway up-regulates vascular endothelial growth factor (VEGF), IL-8 and monocyte chemotactic protein-1 (monocyte chemotactic protein-1, IL-8). MCP-1), and then drive inflammatory cells to accumulate in the local vascular intima, and promote the proliferation of vascular media and intimal cells, resulting in vascular intimal thickening [11, 12]. The systemic inflammatory state of uremia patients can also promote AVF dysfunction. Studies have found that [13] inflammatory cytokines related to intimal hyperplasia, including IL-6, TGF-β1 and TNF-α, are significantly increased in uremia patients. And markers related to oxidative stress injury and lipid peroxidation, such as 8-hydroxy-2 ‘-deoxyguanosine and 4-hydroxy-2-nonenal, also increase [11], leading to the increase of mitogen level, which together with local inflammation and hypoxia, causes the pathological process of intimal hyperplasia in AVF.

3.2 Uremic Toxins

Renal insufficiency in hemodialysis patients can not clear toxins in time, which can cause a series of systemic reactions such as immune dysfunction. Studies have found that [14] uremic toxins can lead to vascular dysfunction and also have a certain impact on vascular pathways, and there is an undeniable link between the two. Uremic toxin can stimulate the migration and proliferation of VSMC, resulting in increased intimal thickness [15, 16]. ChenNX [17] et al. suggested that uremic serum could induce vascular calcification, which involved the process of metastasis and differentiation of VSMC into osteoblast-like cells. At the same time, uremic toxins (such as indoxyl sulfate and guanidine compounds) can inhibit endothelial cell proliferation and wound repair, leading to neointimal hyperplasia and severe narrowing of vascular lumen [18]. Therefore, reducing uremic toxin levels is essential to inhibit AVF stenosis and improve fistula longevity.

3.3 Cell Proliferation, Migration and Phenotypic Transformation

The proliferative intima involves the involvement of a variety of cells, such as endothelial cells, fibroblasts and macrophages [19]. In particular, the interaction between macrophages and inflammatory factors and VSMC can promote intimal hyperplasia and stenosis [20]. However, most of the current evidence supports that VSMC proliferation, migration and phenotypic transformation [21, 22] are involved in the intimal hyperplasia of AVF vessels [23, 24]. The phenotypic transformation of VSMCS, that is, the differentiated VSMCS with high expression of contractile related proteins such as SM22α, under the stimulation of various factors, the expression of contractile related SM22α, etc. is down-regulated, while the expression of proliferation and synthesis related OPN, etc. is increased, which makes VSMCS have stronger proliferation and migration ability. In turn, it can mediate vascular stenosis [25, 26]. Foreign scholars implanted polytetrafluoroethylene (PTFE) grafts into baboons with bilateral femoral arteriovenous fistulas, and found that intimal thickening of the arteriovenous fistulas was caused by VSMC proliferation and accumulation [27, 28]. Some studies have detected vascular tissue samples from AVF vein end of AVF patients with AVF dysfunction, and found that VSMC can migrate through the media to the intima to maintain continuous intimal hyperplasia [29]. The proliferation, migration and phenotypic transformation of VSMC are important for the study of the pathogenesis of AVF stenosis.

3.4 Repeated Dialysis Puncture

Hemodialysis patients need to puncture the arteriovenous fistula during each dialysis, and repeated puncture will cause mechanical damage to the inner wall of the blood vessel, which will lead to pathological thickening of the vascular intima [30]. In clinical practice, the rope ladder puncture and button-hole puncture are often used for dialysis puncture [31], among which the button-hole puncture has a higher incidence of intimal hyperplasia [32]. A number of studies have found that the rope ladder puncture method plays an advantage in reducing vascular proliferation and protecting vascular intima [33, 34]. Some scholars [35] found that compressing the distal internal fistula vessels of the puncture point during AVF puncture can reduce the incidence of intimal hyperplasia and improve the success rate of puncture. Repeated puncture of AVF, especially in the same location, can increase the incidence of intimal hyperplasia and stenosis of AVF.

3.5 Oxidative Stress

Internal fistula surgery can cause damage to local blood vessels. Changes in vascular hemodynamics can lead to oxidative stress response of the body, reduce superoxide dismutase (SOD) and heme oxygenase-1 (HO-1), and make superoxide anion, nitrotyrosine substance and peroxynitrite in the blood vessel Vascular endothelium and smooth muscle

cells accumulate in AVF [36]. SOD is an important antioxidant enzyme in the body, which is the primary substance to remove free radicals in the body. Nitrotyrosine and peroxynitroso can up-regulate the expression of metalloproteinases, further degrade the extracellular matrix, enhance the migration ability of smooth muscle cells, and widely participate in the phenotypic transformation, proliferation and migration of vascular smooth muscle cells in internal fistula [37]. Li Zezheng et al. [38] found that the expression level of metalloproteinase-9 was significantly increased in the hyperplastic intima of AVF patients, indicating that metalloproteinase-9 was involved in the occurrence and development of vascular intimal hyperplasia in AVF. It is suggested that metalloproteinase-9 plays a major role in the degradation of extracellular matrix and accelerates the process of intimal hyperplasia in maintenance hemodialysis patients.

3.6 Shear Force

Flow shear stress is the frictional force exerted by blood flow on the vessel wall [39], which can fluctuate within a physiological range. The different flow shear stress at the internal fistula has different effects on intimal hyperplasia. Flow shear stress on vascular endothelial cells can affect the activation, structure and function of endothelial cells and the development of intimal hyperplasia. In the external end-to-side anastomotic fistula model, the area of high blood flow shear stress is located at the anastomosis, the side opposite to the arterial blood flow and the lateral wall near the anastomotic vein, and the area of low blood flow shear stress is located at the medial wall near the anastomotic vein [40-42]. However, some experimental studies have shown that [43], Franzoni et al. found that the blood flow shear stress model of unidirectional pulsatile flow promoted the multifunctional transcription factor Kruppel-like factor (Kruppel-like factor) by establishing the extracorporeal circulation study of internal fistula factors-2, KLF-2) is significantly expressed and regulates endothelial cell function through multiple pathways. At this time, vascular endothelial cells are in a functional quiescent state and do not proliferate, which has vascular protection. However, the oscillating pulsatile flow can induce the expression of inflammatory, oxidative and apoptotic mediators [44], and the expression of MCP-1 and IL-8 increases in the blood flow shear stress model. As an inflammatory mediator, MCP-1 is also a strong monocyte chemotactic factor, and chemokines are important mediators of leukocyte activation and migration during inflammation [45], which can induce the migration and infiltration of monocytes, macrophages, memory T cells and NK cells to active inflammatory sites. It can activate vascular endothelial cells, promote the proliferation and migration of smooth muscle cells, and mediate the expression of various tissue factors [46]. IL-8 can attract monocytes and neutrophils to the inflammatory site and aggravate local inflammation. The flow shear stress itself has an impact on the inner wall of the blood vessel, which can cause damage to the inner wall of the blood vessel. It can also induce the secretion and expression of inflammatory factors, thereby affecting the intima of the blood vessel, leading to its proliferation and ultimately the failure of internal fistula stenosis.

3.7AVF Surgical Injury

AVF surgery is mainly the use of modern surgical methods to anastomose the patient's own peripheral artery and superficial vein, which may cause different degrees of prognosis in the process of surgical operation. ChenQ [47] et al. included 43 patients who underwent AVF reconstruction and 39 patients who underwent failed AVF. The venous vascular tissue of the same part of AVF was taken and evaluated by histopathological analysis. The results showed that there was a direct relationship between venous vascularization and neointimal hyperplasia in patients with failed AVF. It may be related to the surgical method of AVF and the surgical injury caused by AVF. Zhang Chunhua [48] et al. compared conventional AVF and No-touch technology to make AVF and found that the latter could reduce the intimal hyperplasia rate and stenosis [49]. The injury caused during AVF surgery plays a key role in the development of AVF in the later stage.

3.8 Hypertension

Renal damage is a common event in the course of hypertension [50]. RezapourM [51] et al. found that hypertension is one of the factors leading to AVF dysfunction. Due to high intravascular pressure in hypertensive patients, under continuous pathophysiological stimulation, vascular endothelial function is impaired [52, 53], resulting in increased AVF tension and proliferation of mediator smooth muscle cells, thereby promoting intimal hyperplasia and leading to vascular remodeling [54]. In the long-term use of AVF, the control of hypertension is of great importance.

3.9 Diabetes Mellitus

Hyperglycemia is an important factor in vascular injury [55]. FariesPL [56] et al. compared the cultured morphology of VSMCS from diabetic and non-diabetic patients and showed that VSMCS from diabetic patients showed significantly increased proliferation, adhesion and migration rates, which led to a significantly higher intimal hyperplasia rate in diabetic patients. AronsonD [57] et al. found that with the progression of diabetes, advanced glycation end products (AGEs) accumulate in vascular tissues, which may lead to VSMC proliferation and the production of extracellular matrix, eventually leading to intimal hyperplasia and vascular restenosis. The domestic study found that [58] the vascular intimal thickness of diabetic nephropathy group and chronic glomerulonephritis group was compared within the same time of fistula application, and it was found that the former had a higher degree of intimal hyperplasia. Diabetes mellitus Shaanxi University of Traditional Chinese Medicine 2024 master degree thesis 6 Vascular damage itself can cause AVF dysfunction.

3.10 Hyperlipemia

Hyperlipidemia has a significant impact on intimal hyperplasia and vasomotor function [59]. ZhuB [60] et al. found in their study that hyperlipidemia can induce neointimal hyperplasia and VSMC proliferation. WangYC [61] divided the New Zealand rabbits undergoing AVF surgery at the same time into the high cholesterol diet group and the normal diet group. After 4 weeks, it was found that the intimal hyperplasia of AVF in the New Zealand rabbits with high cholesterol diet was significantly increased, and the collagen deposition was

rich. In the same experiment, the results of New Zealand rabbits with high cholesterol diet after stent implantation [62] and common carotid artery retrograde vein bypass [63] were consistent with the above, and hyperlipidemia can lead to obvious intimal hyperplasia and luminal stenosis. The same is true of the experimental results in the rat model of hyperlipidemia [64]. In patients with uremia, hyperlipidemia, a risk factor, increases the risk of AVF stenosis and intimal hyperplasia [65].

3.11 Smoking

Smoking can cause a series of diseases in the respiratory system, urinary system, cardiovascular and cerebrovascular system [66], and is an important risk factor for CKD [67]. A foreign retrospective analysis shows that there is a certain relationship between smoking population and CKD in the general population. Smoking can aggravate the progress of CKD to a certain extent [68] and damage renal function [69]. One study [70], which included 141 patients with AVF and conducted statistical analysis, suggested that smoking may increase the incidence of AVF thrombosis and vascular stenosis. In the process of chronic management of AVF complications, it is very important to control or even quit smoking for the long-term maintenance of AVF.

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