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Carotid Arteriosclerosis and Function Changes of Endothelial Cells in Senile Diabetic under Microscope

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Abstract: With the development of an aging society, the elderly will not only get diabetes, but also face osteoporosis, arteriosclerosis (coronary artery disease, peripheral artery disease, cerebrovascular disease), malignant tumors, various organs transsexual. Therefore, the causes of diabetes, carotid atherosclerosis, and other diseases frequently encountered by the elderly are further studied to explore solutions. In this paper, the age-related diabetic carotid artery atherosclerosis and the functional changes of endothelial cells in the microscopic area were determined. The side wall WSS after carotid stenosis recovery was 0.11±0.02 Pa, and the control group WSS was 0.05±0.07 Pa. As a result, from a genetic point of view, genetic polymorphisms can determine the differences between different individuals in the intensity of inflammatory response (such as Aβ and lipid deposition), and ultimately affect the severity and prognosis of the disease. Changes in the volume of GM and cerebral cortex caused by type 2 diabetes cause problems with thinking and memory, making patients more prone to cognitive impairment.

1. Introduction

Diabetes can cause vascular complications such as blood vessels in the lower extremities, and mortality and obstacle rates will also increase (diabetic feet, etc.). Middle-aged and elderly people become more vulnerable to chronic diseases due to poor physical function, low physical activity, and low immunity. In order to better help the elderly to prevent and improve their physical fitness, it is very necessary to establish a quality of life and a disease prediction model for the elderly. The formation and stenosis of carotid Atorom plaques not only show intracranial or systemic Atorom's arteriosclerosis, but also may be a direct cause of cognitive decline. Cerebral hemodynamics caused by severe carotid stenosis are two important mechanisms of cognitive impairment caused by asymptomatic carotid stenosis.

It is important to observe the functional changes of carotid atherosclerosis and endothelial cells in elderly diabetic patients under a microscope, and to manage and control the related factors. It can

strengthen the relationship between diabetes and mild cognitive impairment, conduct cognition and research, reduce the damage caused by diabetes, improve the quality of daily life, and benefit patients.

Carotid atherosclerosis can cause lesions inside blood vessels, further destroy the blood-brain barrier, and impair permeability. Jain believes that diabetes is a well-known risk factor for carotid atherosclerosis. However, screening all diabetics by carotid ultrasound is both troublesome and cost-effective. Finding anthropometric methods related to the severity of the disease has been a tireless effort. He proposed to use the calf waist circumference as a better marker of carotid atherosclerosis because it is cost-effective and non-invasive, and therefore better than traditional measurement methods. This is a cross-sectional observation study. He included 100 patients, recording detailed medical history, clinical examination, biochemical indicators and anthropometry. He uses a high-resolution USG system with a 10 MHz linear transducer to measure carotid atherosclerosis. The carotid intima-media thickness (CIMT) was measured at 3 points on the distal wall of the distal CCA and 1 cm proximal to the carotid balloon. The average of six measurements from the right CCA and left CCA was used [1]. Although his research is more feasible in theory, it is more difficult to operate. In order to study the various risk factors for cognitive decline in the elderly, Kawasaki conducted a community-based cohort study conducted in Tokyo on 494 of the oldest comprehensive health surveys in Tokyo that were over 85 years old and had not been diagnosed with dementia. The subject of the disease was studied. He assessed cognitive function at baseline and at 3-year follow-up using a small mental state examination (MMSE), and used ultrasound examination to measure carotid plaque. In a cross-sectional analysis using Tobit regression, individuals with a high carotid plaque score (⩾5.0) had an MMSE score 1.08 points lower than the MMSE score without plaque. Individuals with the highest CMV IgG titer had an MMSE score that was 1.47 points lower than those in the lowest quartile [2]. His research ideas are correct, but not rigorous enough. Wang believes that there is limited epidemiological data on carotid artery disease in a large sample of the national representative population. He aims to assess the prevalence and related risk factors of CA found by carotid ultrasound based on a national survey in China. 107095 residents from the National Stroke Prevention Project in China, aged ⩾40 years, received carotid ultrasound. He excluded patients undergoing carotid endarterectomy or carotid stenting and patients with stroke or coronary heart disease. The analysis included data from 84,880 participants. CA is defined as intima-media thickness (IMT) ⩾ 1 mm or the presence of plaque. Among 84,880 participants, males accounted for 46.4%, with an average age of 60.7 ± 10.3 years. Overall, the standardized prevalence of CA is 36.2%, which increases with age and is higher in men than women. Approximately 26.5% of participants had increased IMT, while 13.9% of patients developed plaque. IMT increases, the presence of plaques and stenosis participants are related to age, and CA is very common in the middle-aged and elderly population [3]. His research is more comprehensive, but not feasible.

In this article, by recording the changes in cells by observing endothelial cells, studying the pathogenesis of senile diabetes based on these changes, and considering the relationship between endothelial cell function and the pathogenesis of ischemic stroke and carotid atherosclerosis, you can Infer that there is a close relationship between the two. It provides some scientific evidence that can reduce the risk of diabetes in the elderly and promote their health and quality of life.

2. Carotid Atherosclerosis and Endothelial Cell Function in Elderly Diabetic

2.1. Senile Diabetes

Diabetes can include four types: pathological physiology, age of onset, sequelae, and

complications. Type 1, type 2, special type, and gestational diabetes are the most common. Type 2 diabetes is the most common type. Diabetes was a concept introduced in the 1960s, which means that pancreatic islet cells have insulin-related resistance, and the blood glucose level has not reached the diagnostic criteria for diabetes. In addition, high blood sugar usually causes changes in the physiology and pathology of the precursors of type 2 diabetes. Most pathologies (about 80%) of people with pre-diabetes are unclear. If you do not change your lifestyle, 15 to 30% will have the risk of type 2 diabetes within 5 years [4].

MCI is a dynamically developing disease, its cause is difficult to determine, the degree of damage, severity and direction of development, and the rate of disease progression are uncertain. Compared with normal elderly people who match their age and education level, depending on the etiology and damaged parts of the brain, there are cognitive dysfunctions, such as executive function. The element of the concept is that cognitive function has declined, but the basic ability to live is normal. Diabetes and chronic chronic hyperglycemia cause a variety of diabetic vascular complications, including cardiovascular, cerebrovascular and leg vascular disease, diabetic nephropathy, fundus disease, nervous system damage, peripheral neuropathy, etc., all of which constitute a huge human health Threat [5].

The prevalence of diabetes varies with age and increases with age. Different people have different diabetes epidemics. Due to the long history of diabetes, many complications, complex symptoms, and vital organs throughout the body may occur alone. Similarly, different combinations may appear before or after or simultaneously. Compared with young diabetic patients, the risk of multi-drug therapy, dysfunction and general aging conquest (including cognitive impairment, depression, urinary incontinence, falls and persistent pain) is significantly increased [6].

At present, there is no "gold standard" for clinical diagnosis and cognitive impairment, which mainly depends on clinical symptoms and examination of each neuropsychological table. Neuropsychological testing is a popular and effective method for detecting cognitive functions to confirm the interaction between the brain and actions, and can functionally locate cognitive processes. In this study, the decline in cognitive function was accurately identified, thereby identifying possible interventions. It aims to achieve, improve the quality of life and delay the development of medical conditions. The call body is the largest connecting fiber in the cerebral hemisphere, and connects the left and right cerebral hemispheres. It is the final storage area of the memory, which can process and process information from all directions, and is related to long-term and long-term storage. Any lesion that disrupts the cortical neural circuit can lead to memory impairment, leading to high cortical dysfunction and mental decline. At present, few studies have analyzed the relationship between callus atrophy and cognitive function by quantitatively measuring the MRI call of elderly diabetic patients [7].

2.2. Carotid Atherosclerosis

Atherosclerosis is the main cause of coronary arteriosclerosis. It causes the arterial intima to become the earliest fatigue, and thickens the intima during the occurrence and development of AS, which leads to further plaque formation. The carotid artery is an important organ channel connecting the heart and brain, so it may be related to the coronary artery and stroke. Cys-c is considered to be a basic protein with a molecular weight of 14kDa, is a rare element with good supercysteine processe inhibitor properties, and is present in the nuclear cell body SOM line. Catechins need to regulate self-stabilizing proteins because of their different evolutionary history, structure, matrix characteristics, and biological characteristics [8]. Cysteine itself is the target of protein decomposition and hydrolysis, so serine proteases and asparaginate catechin D are therefore inert. In Cys-c, there are many biological characteristics, from the control of normal tissue

processes such as cell proliferation, growth, bone regeneration, and cell differentiation to the control of pathogenic processes such as infection and inflammation. Causes tumor metastasis and neurodegenerative diseases. Cys-c is present in specific tissues and body fluids. The level of Cys-c helps as an adjunct treatment for a range of diseases [9].Changes in its concentration are related to various diseases such as nephritis, tumors, glucocorticosteroid therapy, and thyroid function. As with normal aging, the formula for calculating the insulin resistance index in the steady-state model is as follows:

$$\text{HOMA-IR} = \text{FINS(mU/L)} \times \text{FPG(mmol/L)}/22.5 \tag{1}$$

For the Mises yield condition, such a directional enhancement model is as follows.

$$\bar{\sigma} = \sqrt{\frac{3}{2} S_{ij} S_{ij}} = \psi(\xi) \tag{2}$$

In the equation, $\bar{\sigma}$ is the equivalent stress, S_{ij} is the bias tensor, and ξ is the scalar internal variable.

The formula for carotid atherosclerosis concentration change is as follows.

$$\tau = \frac{16T}{\pi(D^2 - d^2)(D + d)} \tag{3}$$

$$\sigma eq = \sigma true = \frac{F}{A} = \frac{F}{A_{0l}^{l_0}} = \sigma(1 + \varepsilon) \tag{4}$$

In the formula, τ is the shear stress and T is the torque.

Carotid atherosclerosis is a typical feature of systemic atherosclerosis, and the degree of sclerosis is related to age. In other words, the older you are, the more likely you are to have arteriosclerosis, usually forming spots on your face, which appear and become harder, forming a thrombus [10]. There are many risk factors for CAS, which are caused by high hygroscopicity. Hyperthrombosis and diabetes are closely related to genetic diseases, which indicates that the occurrence and progression of CAS, obesity is the main reason. The spread of the disease is wide, so the disease needs to be treated in time.

2.3. Endothelial Cells

Physiologically, vascular endothelial cells have barrier function, information transmission function, endocrine function and so on. Endothelial cell dysfunction is the main pathophysiological change that initially caused atherosclerosis. Due to changes in endothelial cell function and morphology, changes in vascular permeability, the sinking of harmful metabolites, decreased vascular barrier function, lipid and other substances are easily precipitated in the subendothelial space and atherosclerosis in the formation of spots in atherosclerosis In the process of sclerosis, after endothelial cells and smooth muscle cells are damaged, chronic inflammatory hyperplasia occurs in local areas of blood vessels. NO is the strongest endothelial vascular factor, ET is the strongest endothelial source of vasoconstrictor, and is released and diffused into surrounding tissue cells, mediating smooth muscle relaxation, and adhesion and aggregation of white blood cells and platelets [11]. It exerts cardiovascular protection by preventing smooth muscle cell proliferation and the expression of viscous molecules. NO is considered to be the most important protective factor of AS. The expression and activity of eNOS determine the production of NO. In the case of diabetes, endothelium-dependent vasodilation is impaired, such as hyperglycemia, alternation of fat, increased secretion of IR and inflammatory factors, decreased expression or activity of eNOS during T2DM can cause endothelial dysfunction, induced and endothelial Dysfunction is considered

to be an important pathogenic structure of diabetic vascular complications [12].

The formation of blood vessels is through the propagation and proliferation of bacteria, mainly accompanied by the decomposition of the extracellular matrix of vascular endothelial cells. The proliferation and movement of vascular endothelial cells, the formation of blood sample structures and networks are controlled by complex molecular cells and machinery. As part of repairing certain physical and tissue damages, cells when the growth rate of tissue cells is higher than the growth rate of the vascular system, tissue cells will increase or repair the relative hypoxia of tissue cells. This hypoxia phenomenon stimulates angiogenesis factors and triggers the angiogenesis process by activating tissue cells. When new blood vessels are produced, the consumption of tissue oxygen is reduced, various angiogenic factors are significantly reduced, and the blood vessel production process is terminated. Abnormal fat metabolism, oxidative stress, and endothelial dysfunction are important risk factors for cardiovascular disease, and hyperlipidemia is an independent risk factor for arteriosclerosis. Patients with hyperlipidemia often indicate clinical symptoms of vascular endothelial dysfunction, and are associated with the release of various inflammatory factors. Further accelerating the onset and development of arteriosclerosis and cardiovascular disease may bring risks. Abnormal endothelial function is not only an important initial stage of arteriosclerosis, but also an important factor for its progress. Patients with hyperlipidemia may have abnormal vascular endothelial function before atherosclerosis. At the same time, endothelial dysfunction is an important process of atherosclerosis. It is believed that endothelial dysfunction can be used to predict the future of healthy individuals and early patients with cardiovascular disease. It has been identified as the ultimate risk factor for cardiovascular disease.

3. Carotid Atherosclerosis Endothelial Cell Experiment

3.1. Experimental Instruments and Reagents

The main instruments and experimental reagents are shown in Table 1 and Table 2.

Table 1. Experimental instruments

Serial number	Instrument name
1	Ultra-clean workbench
2	5%CO2 constant temperature incubator
3	Cryogenic centrifuge
4	Inverted phase contrast microscope
5	550 microplate reader

Table 2. Main reagents

Serial number	Reagent name
1	Collagenase type I
2	Trypsin
3	EDTA
4	M199
5	Percoll
6	FCS
7	Gelatin
8	mRNA extraction kit
9	Reverse transcription kit
10	TaqDNA polymerase
11	Methylene/acrylamide
12	Coomassie

3.2. Cell Culture

The umbilical cord ECV304 endothelial cell line was recovered, inoculated in a 50ml culture flask to grow it, and passed on to offspring. If passed down from generation to generation, the medium should be discarded first, washed twice with pre-warmed PBS solution, added with digestion solution, smoothed with endothelial cells, and then digested at room temperature. Endothelial cells fuse under the microscope and become round. If the cell gap becomes larger, immediately rotate the flask, discard the digestion solution, wash twice with PBS solution and add fresh medium. Gently blow the endothelial cells to adjust the density of the endothelial cells and press them into the culture flask. The liquid is changed every 2 days for 2-3 generations and then used in the experiment.

3.3. Isolation and Purification of Endothelial Cells

The lateral tissue of the cancer except the tumor envelope was cut, the tumor tissue was cut into pieces of tissue, 0.2% type I coenzyme (Type I, Sigma) preheated to 37℃ was added, and digested at 37℃. It vibrates violently every five minutes. The filtered cell suspension was centrifuged in serum-free DEM solution (500g×5 minutes) and washed twice. After 24 hours, the parietal cells were washed with serum-free DEM solution, and the endothelial cells were replaced to make a complete medium. Replace the solution every 2-3 days.

After culturing for 5 to 7 days, the typical form of endothelial cells and the clones of cells with growth characteristics were selected using a phase contrast microscope, and the suspension cells were washed three times with serum-free DMEM. After separation and purification, the vascular endothelial cells of liver cancer multiply rapidly in the primary culture. With the passage of the cells, the proliferation rate gradually decreases and the shape becomes multilateral. The cells showed "monolayer growth" and "impeded contact", showing a significant dependence on ECGS.

3.4. Statistical Analysis

For statistical processing, the SPSS 19.0 program was used. The program was restored to the narrow side. The wall of the initial carotid band had shear resistance. The OSI seismic factor and the pressure gradient of the tubular wall indicated the mean ± standard deviation ($X \pm S$). For the statistical difference between the narrower recovery group and the smaller healthy group, $p < 0.05$ considered the difference between the two groups to be statistically significant.

4. Analysis of Protein Changes in Elderly Diabetic Patients

4.1. Comparative Analysis of BMD in Diabetic Patients

As shown in Table 3, there is no statistically significant difference in age, FBG, Ca, P and 25-oh-vd ($P>0.05$). The level is usually within a limited range. Compared with normal bone mass group, the history of osteoporosis group and bone mass reduction group was significantly prolonged, and HbA1c level was significantly increased ($P<0.05$ or $P<0.01$). HbA1c was also higher than the bone mass reduction group ($P<0.05$ or $P<0.01$). Bone mass reduction and osteoporosis, BMI is lower than normal bone mass group ($P<0.01$). The PTH level of osteoporosis group was higher than that of bone loss group and normal bone group ($P<0.05$). Compared with the normal bone mass group, the BMD values of bone neck, triangulation, major trochanter and 12-4 of osteoporosis group and osteoporosis group were all reduced ($P<0.01$), BMD value of all parts, The group was also lower than the bone mass reduction group ($P<0.05$).

Table 3. Comparative analysis of BMD of each group

Grouping	Normal bone mass group	Bone mass reduction group	Osteoporosis group	F value	P value
Age	70.9±7.1	72.6±6.9	72.5±7.8	0.738	0.462
BMI	25.6	23.8	20.5	22.432	0
FBG	8.84	8.62	7.59	2.235	0
25-OH-VD	17.258	15.429	12.356	2.044	0
PTH	66.214	60.159	57.471	3.103	0
Ca	2.16	2.44	1.52	1.055	0.214
P	1.23	1.29	1.14	2.014	0.114
Femoral neck	0.845	0.796	0.885	11.023	0
Triangle	0.824	0.785	0.699	17.026	0
Big rotor	0.952	0.756	0.688	13.524	0
Lumbar spine	1.236	1.355	1.023	13.258	0

The glycated hemoglobin level in the test group decreased by 0.87%, which was significantly higher than the baseline value (P<0.01). Compared with the glycosylated hemoglobin group, it was reduced by 0.7%, and it was significantly improved compared with the baseline value (P<0.01). From the baseline value, the degree of reduction in fasting blood glucose in the experimental group and the control group is not yet clear. This is the degree of reduction in the two groups (0.8mmol/L vs 0.25mmol/L), which is not statistically significant. After the meal, blood glucose increased by 1.43mmol/L and blood glucose decreased by 1.02mmol/L. The difference between the two groups was statistically significant (P=0.04). Compared with the reference value, the triglyceride in the test group decreased by 0.45mmol/L, which was statistically significant (P<0.05), and decreased by 0.22mmol/L compared with the control group. There was no significant difference between the two groups, and other biochemical indicators were not statistically different from the previous differences. Brain aging is a degenerative change caused by neural origin during the aging process of the human body. Normal elderly people also cause some aspects of cognitive decline. Among them, the cognitive impairment of diabetic patients is the result of diabetes and the elderly. The knowledge function is obviously reduced. As one of the factors that affect the cognitive function of age, with age, the cognitive impairment of diabetic patients becomes more obvious.

4.2. Analysis of Related Factors in Patients with Carotid Atherosclerosis

(1) GA control level analysis

As shown in Table 4, the number of men and women with relatively good control is 42 and 46, the number of men and women with general control is 35 and 27, and the number of men and women with poor control is 70 and 158. The average ages were 59.7±4.11, 57.4±3.8, and 59.7±5.1, and there was no difference in gender composition and age between the three groups (P>0.051). The carotid atherosclerosis rate in the well-controlled GA group, the general control group, and the poorly controlled group was 60.7%, 72.9%, and 85.3%, respectively.

Table 4. Carotid atherosclerosis rate at GA level

Variable	Well-controlled group	Control general group	Poorly controlled group	P value
Men and women	42/46	35/27	70/158	0.297
Age	59.7±4.11	57.4±3.8	59.7±5.1	>0.051
BMI	29.14	27.19	21.02	0
GA	16.88	20.99	29.58	0
Carotid atherosclerosis	58	47	101	0.016
CIMT	0.087	0.086	0.11	0.021

(2) Wall shear stress

As shown in Figure 1, the numerical simulation of the finite element arithmetic system ANSYS SCFX 16.0 shows the wall shear stress changes in the region of interest, while the numerical simulation shows the changes of the non-contralateral WSS after 3D reconstruction of the carotid artery. The average WSS of the systolic phase of the outer wall of the healthy carotid artery in the initial stage of the patient's arterial phase was 0.56±0.38 Pa. After the affected carotid stenosis was partially restored, the average time of the lateral artery phase was restored. The WSS was 0.23±0.17 Pa, p<0.01, and the systolic WSS after the recovery of the healthy side and the affected side was statistically significant. The side wall WSS after carotid stenosis recovery was 0.11±0.02 Pa, and the control group WSS was 0.05±0.07 Pa, p<0.01, with statistical significance. Carotid atherosclerosis is now considered to be a chronic, progressive inflammatory lesion of the blood vessel wall. The narrowing of the itchy appearance of the carotid artery in the neck often occurs in the outer wall of the carotid artery bifurcation, and may be related to the complex and well-changing blood flow pattern in this area. can. Among them, lipid abnormality has been identified as a risk factor for AS, and the relationship between LDL-c and AS is particularly clear. LDL-c can start the deposition of cholesterol on the arterial wall by combining aminoglycans and glycoproteins on the arterial wall, causing the destruction of endothelial function, etc., and eventually aggravating the formation of AS.

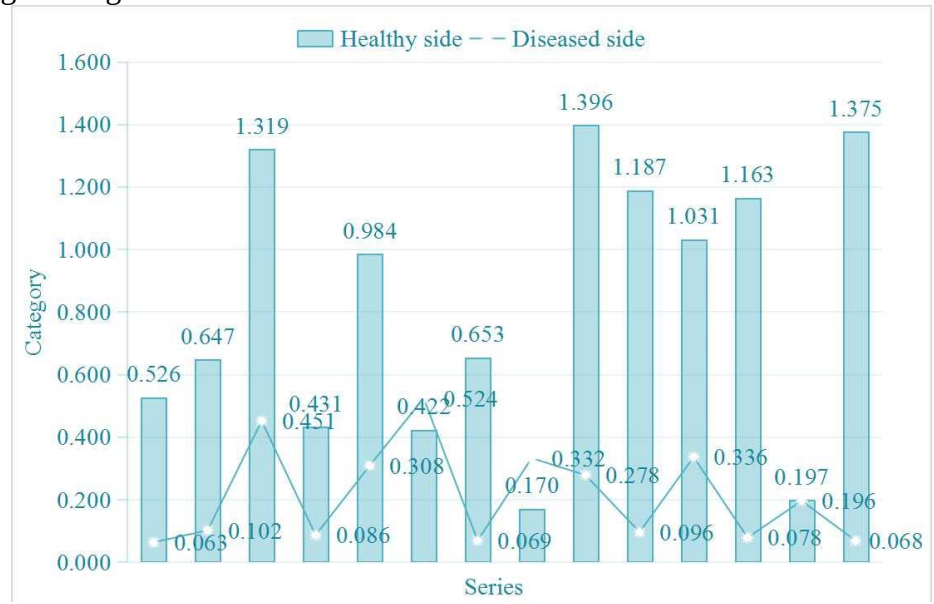

Figure 1. The different effects of wall shear stress on the healthy side and the diseased side

(3) The relationship between carotid block and coronary artery branch number and angiography score

As shown in Figure 2, the correlation coefficient between carotid block score and coronary artery lesion score is 0.60 (P<0.001), and the correlation coefficient between carotid block score and coronary angiography score is 0.28 (P<0.01).

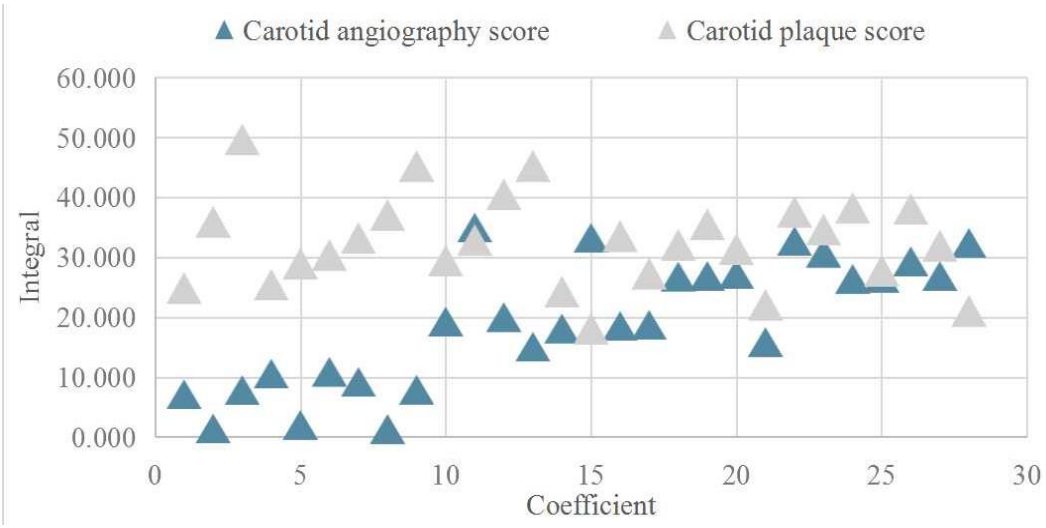

Figure 2. Correlation between carotid plaque score and coronary angiography score

4.3. Analysis of Surface Changes of T3A Cells and LSEC Endothelial Cells

As shown in Figure 3, the morphology of HCC vascular endothelial cells is significantly different from the morphology of human cave endothelial cells that primarily represent TNF receptor P75 (TNRII). In normal sinusoidal endothelial cells, TNF receptor P55 (TNRI) was compared with sinusoidal endothelial cell TNF receptor P55 (TNRI). The molecules attached to the cell surface are ICAM1, ICAM-3, VLA4, CD31 and CD34. Compared with HCC endothelial cells, CD18 is greatly reduced.

Figure 3. Expression of various phenotypic molecules in human hepatocellular carcinoma endothelial cells

As shown in Figure 4, low molecular weight glycerol can promote the secretion of NO from endothelial cells. If the concentration of low molecular weight glycerol increases, the promoting effect will increase, showing a positive correlation. In addition, at 5IU/mL, low molecular weight glycerides have a strong effect on promoting NO secretion from endothelial cells, but a

concentration of 8IU/ml prevents NO secretion from endothelial cells. Low molecular weight glycerin has always inhibited the production of ET, and the inhibition increased with the increase of the low molecular weight glycerin concentration, showing a negative correlation. Low-molecular-weight liver hinders MDA production at the culture site. However, low-molecular-weight liver concentration has nothing to do with MDA production, and P>0.05 has no statistical validity. The effect of MDA in low molecular weight liver suppression culture does not change with the concentration of low molecular weight liver elements. Molecular glycerol has a two-in-one regulation effect on the secretion of NO endothelial cells. Low concentration promotes NO secretion, and high concentration inhibits NO secretion. However, low-molecular-weight glycerol usually inhibits the production of ET and MDA.

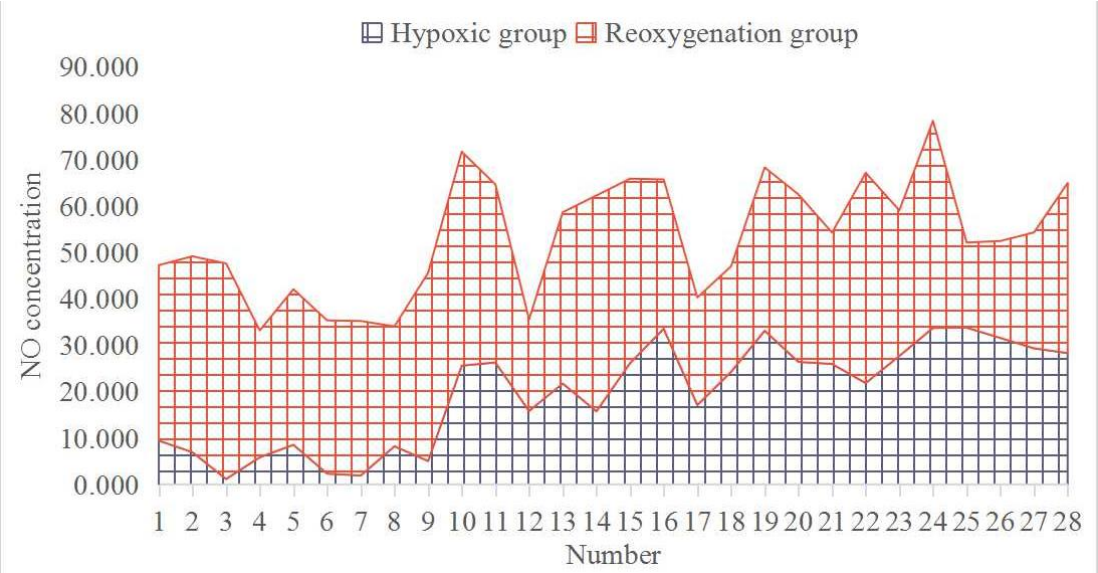

Figure 4. Comparison of hypoxia group and reoxygenation group

Cell membrane particles from different sources have different mechanisms that may affect the function of endothelial cells. In addition, the particle surface types of endothelial cell membranes, patient demographic data, risk factors and laboratory test results were statistically analyzed. As a result, the levels of CD144+CD41a-EMPs were positively correlated with graduation severity (r=0.750, p=0.020). However, under the OCS classification, the remaining epidermal endothelial cell membrane particle levels were not related to the stroke subgroup. In response to different concentrations of H2O2, the two endothelial cells showed a dose-dependent cytotoxic effect, but the mortality rate of LSEC was significantly higher than that of T3A cells, and the difference between the two cytostatics was very significant, indicating that T3A cells have resistance the ability of hydrogen peroxide.

5. Conclusion

This article mainly studies the changes of diabetic arteriosclerosis and endothelial cell function under the microscope. The TLR4 ligand MyD88, TIRAP and the negative regulator TOLLLIP-TLR4 pathway are closely related to all genetic polymorphisms, and the inflammatory response and gene polymorphism are closely related to genetic levels. Ability, the final determinant Aβ and the difference in the intensity of inflammation caused by lipids, will ultimately affect the severity and prognosis of the disease.

As the selected critical point of the occurrence probability of diabetes in the elderly changes, the

reliability index of the prediction result also changes. According to the situation in different regions, comprehensively consider various factors, select the probability points of diabetes examination suitable for the elderly population with diabetes risk, and then take effective countermeasures to reduce the diabetes risk of the elderly.

Diabetes may cause progressive destruction of the brain's nervous system. People with diabetes may suffer from cognitive dysfunctions such as MCI and dementia. People with type 2 diabetes have deficiencies in learning and memory. Type 2 diabetes has obvious effects on muscles, emotions, and language. In the early stages, neural necrosis caused by hypoglycemia may cause changes in the cerebral cortex, which leads to type 2 diabetes. GM causes changes in the cerebral cortex that cause volume changes, thinking, and memory. People with problems will make more patients cope with difficulties due to the onset of cognitive impairment.

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Data Availability

Data sharing is not applicable to this article as no new data were created or analysed in this study.

Conflict of Interest

The author states that this article has no conflict of interest.

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