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Research Article

Visfatin and 25-Hydroxy Vitamin D3 Levels Affect Coronary Collateral Circulation Development in Patients with Chronic Coronary Total Occlusion

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Abstract

Background:

CCC plays a vital role in the myocardial blood supply, especially for ischaemic myocardium. Evidence suggests that the visfatin and 25-(OH)D3 levels are related to the degree and incidence of vascular stenosis associated with coronary artery disease. However, few studies have evaluated the effect of visfatin and 25-(OH)D3 on CCC development in CTO patients. This study aimed to evaluate the relationship between the serum visfatin level and 25-hydroxy vitamin D3 [25-(OH)D3] and coronary collateral circulation (CCC) in patients with chronic total occlusion (CTO).

Methods:

A total of 189 patients with CTO confirmed by coronary angiography were included. CCC was graded from 0 to 3 according to the Rentrop-Cohen classification. Patients with grade 0 or 1 collateral development were included in the poor CCC group (n=82), whereas patients with grade 2 or 3 collateral development were included in the good CCC group (n=107). The serum visfatin and 25-(OH)D3 levels were measured by ELISA.

Results:

The visfatin level was significantly higher in the poor CCC group than in the good CCC group, and the 25-(OH)D3 level was significantly lower in the poor CCC group than in the good CCC group (P≤0.001).

Correlation analysis showed that the Rentrop grade was negatively correlated with the visfatin level (r=-0.692, P≤0.001) but positively correlated with the 25-(OH)D3 level (r=0.635, P≤0.001). Logistic regression analysis showed that the visfatin and 25-(OH)D3 levels were independent risk factors for CCC (OR=1.597, 95% CI: 1.3-1.961, P≤0.001 and OR=0.566, 95% CI: 0.444-0.722, P≤0.001, respectively). The visfatin and 25-(OH)D3 levels can effectively predict the CCC status.

Conclusion: The serum visfatin and 25-(OH)D3 levels are related to CCC development and are independent predictors of poor CCC.

Keywords: 25-hydroxyvitamin D3; coronary collateral circulation; chronic total occlusion

1.Introduction

Coronary collateral circulation (CCC) is crucial and is related to the prognosis of patients with coronary artery disease (CAD). Well-developed coronary collaterals can restore blood flow to ischaemic areas of the myocardium and protect myocardial tissue at risk [1]. Studies have shown that well-developed collateral circulation can limit the area of myocardial infarction, reduce arrhythmia, protect cardiac function, reduce mortality, and ultimately improve the cardiovascular prognosis [2-4]. Currently, there is no noninvasive technology to evaluate collateral function [2,5]. Therefore, the identification of circulating biomarkers to assess the advantages and disadvantages of CCC and exploration of its internal mechanism have important clinical significance.

Visfatin is an adipocyte factor that is produced in large quantities in visceral adipose tissue [6] and is additionally expressed within the heart, liver, muscle, placenta, lung, kidney and bone marrow[7].It has also been reported that visfatin plays a direct role in arterial remodeling[8].Therefore, the connection between visfatin and CCC has become an area of interest, but research in this area is still lacking.

Vitamin D is a steroid hormone produced by irradiating the skin with ultraviolet rays of the sun.It performs biological functions by binding to vitamin D receptors (VDRs) that are widely present in human tissues. VDRs have been confirmed on the surface of cardiomyocytes,smooth muscle cell, and sendothelial cells,and control the proliferation and differentiation of these cells[9,10].

Current studies have shown that vitamin D in an independent manner to promote human umbilical vein endothelial cells (HUVECs) proliferation and migration [11].Additionally, increases in the number of myeloid cells (MCs) [12] and regulation of the VEGF [13] signal transduction pathway affect arteriogenesis and angiogenesis. Although evidence exists to support a positive regulatory role of vitamin D in both arteriogenesis and angiogenesis, few reports have examined its relationship with CCC in patients with chronic total occlusion (CTO).

In this study, we explored the relationship among the serum visfatin level, serum 25-(OH)D3 level and CCC status in CTO patients.

2. Methods

2.1 Patient population

189 cases of coronary angiography confirmed that at least one coronary artery with 100% diameter stenosis was collected in the Department of Cardiology from November 2018 to February 2019[100 men and 89 women; mean age: 62±8 years].Patients were divided into a good CCC group (n= 107, Rentrop grade 2 or 3) and a poor CCC group (n=82, Rentrop grade 0 or 1) according to the Rentrop-Cohen classification of CCC on coronary angiography.

The exclusion criteria included recent acute coronary syndrome (<3 months), coronary artery bypass operation, heart failure, cardiomyopathy, valvular heart disease, previous revascularization history, peripheral vascular disease, chronic obstructive pulmonary disease, chronic kidney or liver disease, previous diagnosis of malignancy, concomitant inflammatory disease, metabolic disease related to vitamin D, and the use of medications containing vitamin D preparations within the previous 3 months. Coronary heart disease risk factors such as hypertension, diabetes, hyperlipidaemia, body mass index (BMI) and smoking history were recorded for all patients.

The study was approved by the hospital ethics committee, and informed consent of each patient was obtained.

2.2 Coronary angiography

Coronary angiography was performed using the Judkins method through radial artery puncture. CCC was graded according to the Rentrop-Cohen classification, as follows [14]: grade 0, no obvious collateral vessels (opaque area, no contrast medium filling in the distal infarction); grade 1, collateral perfusion of the coronary artery that did not reach the coronary artery of the pericardium; grade 2, partial perfusion of the subepithelial segment by CCC; and grade 3, perfusion of the coronary epicardial segment by CCC.

2.3 Biochemical measurements

Fasting venous blood was drawn from the enrolled patients on the day of coronary angiography. Routine laboratory parameters, including the troponin I (TnI), creatinine, total cholesterol (TC), triglyceride (TG), low-density lipoprotein cholesterol (LDL-C), and calcium levels, were immediately determined. The blood samples collected for 25-(OH)D3 and visfatin measurements were snap frozen and stored at -70°C until analysis. The serum concentrations of 25-(OH)D3 and visfatin were measured by the ELISA method with a Roche 601 (BISL kit, UK).

2.4 Statistical analysis

Statistical analysis was performed using the SPSS 23.0 statistical package (IBM Corporation, New York, NY, USA).

Continuous variables are expressed as the mean ± SD or median and 25th and 75th percentiles [(M (P25, P75)], categorical variables are expressed as percentages, and the chi-square test was used to assess the rational distribution hypothesis of continuous variables. Differences between two groups of normally distributed continuous variables were compared with a T-test, and differences between non-normally distributed continuous variables were assessed using the Mann-Whitney U test. Categorical data were compared with the chi-square test, and Spearman's correlation coefficient was used to assess the correlations between the serum 25-(OH)D3 and visfatin levels and the Rentrop grade. Binary logistic regression analysis was used to determine independent predictors of poor CCC development. A P value of 0.05 or less was considered to indicate statistical significance.

3. Results

3.1 Baseline patient characteristics

The 189 patients included in the study were included in the good CCC group (good collaterals, 107 patients, Rentrop grade 2 or 3) or poor CCC group (poor collaterals, 82 patients, Rentrop grade 0 or 1).

The baseline characteristics of patients with good CCC group and poor CCC group are shown in Table 1. Both groups were similar in age, gender, BMI, smoking history, hypertension, cholesterol and calcium levels (P>0.05). However, the proportion of patients with diabetes was higher in the poor CCC group than in the good CCC group (52.4% vs. 33.6%, P≤0.001). Furthermore, the LDL-C, TnI, and creatinine levels were obviously higher in the poor CCC group than in the good CCC group, as shown in Table 1

Variable	good(n=107)	Poor(n=82)	*P*
age （years）	63（56，68）	64（58，69）	0.078
Gender male n(%)	55（51.4）	45（54.9）	0.064
Hypertension n(%)	57（53.3）	54（65.9）	0.082
Diabetes n（%）	36（33.6）	43（52.4）	0.009
Smoking n（%）	55（51.4）	45（54.9）	0.635
BMI（kg/m^2）	26（25，29）	26（26，29）	0.204
LDL-C (mmol/l)	1.5(0.65,3.02)	2.6(2.03,3.02)	≤0.001
TG(mmol/l)	2.2（1.7，2.6）	2.03（1.58，2.08）	0.15
TC(mmol/l)	4.2（3.8，5.01）	4.3（3.9，4.8）	0.32
TNI(ng/ml)	0.08(0.05,0.16)	0.16(0.12,0.25)	≤0.001
Creatinine(mg/l)	66（50，90）	75（59，90）	0.034

25（OH）D_3 (ng/ml)	32(29,35)	25(21,28)	≦0.001
Calcium(mmol/l)	2.06（2.02，2.1）	2.06（2.03，2.1）	0.182
Visfatin(ng/ml)	10.3(9.2,12.8)	17.4(14.2,21.13)	≤0.001
Rentrop score (n)			
0		18	
1		64	
2	64		
3	43		

Table 1: Demographic properties and biochemical parameters of the good CCC group and the poor CCC group

CCC, coronary collateral circulation; BMI, Body mass index ; TnI:Troponin I;TC , Total cholesterol; TG, triglycerides; LDL-c, low-density lipoprotein cholesterol

3.2 Serum visfatin and 25-(OH)D3 levels in each patient group

The serum visfatin level was significantly higher in the poor CCC group than in the good CCC group (P≤0.001). The serum 25-(OH)D3 level was significantly lower in the poor CCC group than in the good CCC group (P≤0.001), as shown in Figure 1-2.

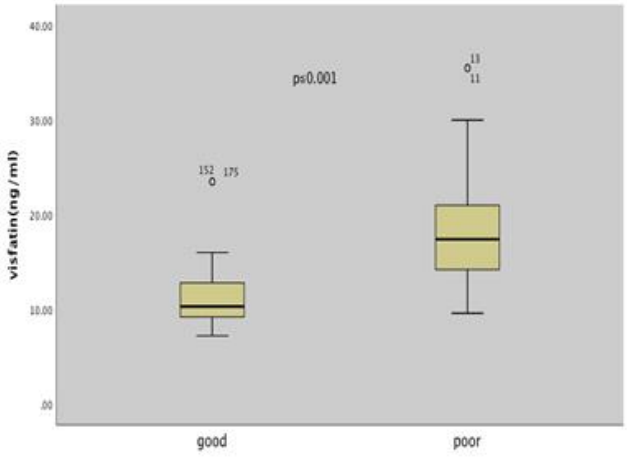

Figure1 Comparison of vistatin levels in good and poor CCC group

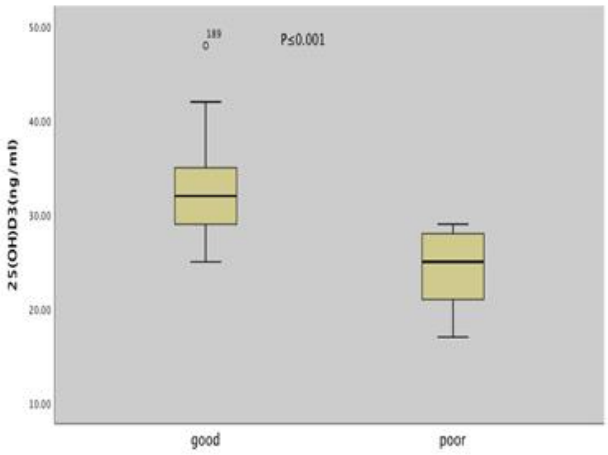

Figure 2. Comparison of 25(OH)D3 levels in good and poor CCC group

3.3 Relationship among the serum visfatin and 25-(OH)D3 levels and the Rentrop grade

Spearman correlation analysis showed a significant negative correlation between the visfatin level and the Rentrop grade (r=-0.692, P≤0.001) (Figure 3). A significant positive correlation was observed between the 25-(OH)D3 level and the Rentrop grade (r=0.689, P≤0.001) (Figure 4). Furthermore, an L-shaped relationship was observed between the 25-(OH)D3 and visfatin levels (Figure 5).

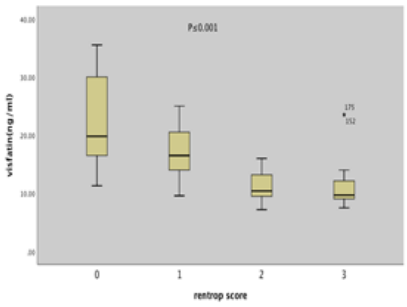

Figure3 Relation between serum visfatin levels and Rentrope Score

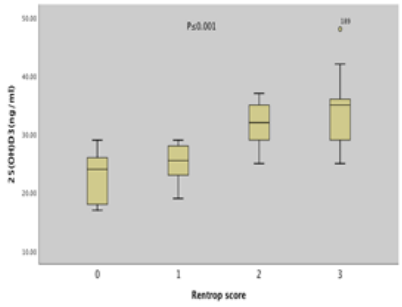

Figure 4. Relation between serum 25(OH)D3 levels and Rentrope Score

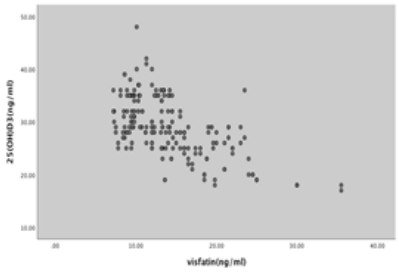

Figure5 Comparison of 25(OH)D levels and visfatin levels

3.4 Binary logistic regression analysis:

Binary logistic regression was performed between CCC development and 25-(OH) D3, visfatin, LDL-C and other factors. CCC development was used as the dependent variable, and all univariate analyses with P values less than 0.1 were included in the logistic regression equation as independent variables. Stepwise regression was used for multivariate analysis. The serum 25-(OH)D3, visfatin, and LDL-C levels and diabetes were independent risk factors for poor CCC development (Figure 6).

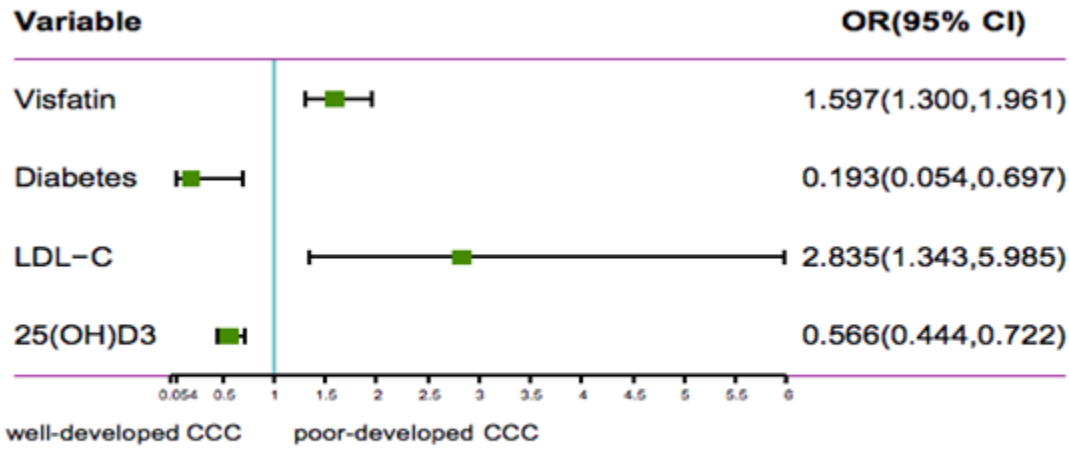

Figure 6 The influence of different variables on the development of CCC

Discussion

The present study revealed the following findings. (1) The visfatin level was significantly higher in CTO patients in the poor CCC group than in those in the good CCC group, and a positive correlation was observed between a high visfatin level and poorly developed CCC. A high visfatin level was an independent predictor of poor CCC in patients with CTO. (2) The 25-(OH)D3 level was significantly lower in CTO patients in the poor CCC group than in patients in the good CCC group. A low serum 25-(OH)D3 level was positively correlated with poorly developed CCC in CTO patients. It is worth noting that a low serum 25-(OH)D3 level was an independent predictor of poorly developed CCC in CTO patients. (3) An L-shaped relationship was observed between the serum 25-(OH)D3 and visfatin levels. (4) A high LDL-C level and a history of diabetes also contributed to poor CCC development in CTO patients.

Coronary collateral growth is thought to develop by the expansion of a pre-existing collateral network and the formation of new blood vessels [15,16]. Recent studies have found that the main factor affecting the development of coronary collateral vessels is the pressure gradient between segments located at the proximal and distal ends of the occlusion [17,18]. VEGF, EPCs, FGF, transforming growth factors (TGF-β), and nitric oxide (NO)-activated biological substances also play significant roles in both angiogenesis and arteriogenesis [19].

Visfatin is a known proinflammatory cytokine that plays an important role in many chronic inflammatory diseases, including atherosclerosis and

cardiovascular diseases.As a cytokine, visfatin can regulate the expression of key regulators of vascular remodelling, such as vascular endothelial growth factor (VEGF) [20], fibroblast growth factor 2 (FGF-2) [21] and matrix metalloproteinases (MMPs) [22].Recent studies shows that visfatin promotes angiogenesis through a VEGF-dependent mechanism in endothelial progenitor cells (EPCs) [23].Auguet et al. found that visfatin was highly expressed in unstable carotid plaques and coronary atherosclerotic plaques of lipomacrophages[24].In addition, it has recently been reported that extracellular visfatin is directly involved in vascular remodeling[8].However,the relationship between visfatin and coronary collateral circulation is still unclear.

This study reports, for the first time, the relationship between visfatin and CCC in CTO patents. The visfatin level was significantly higher in CTO patients in the poor CCC group than in patients in the good CCC group, which may be related to the following mechanisms: (1) Visfatin induces LOX-1 expression in vascular endothelial cells, which causes inflammation, leads to endothelial cell damage and dysfunction, and promotes the vicious cycle of atherosclerosis [25]. (2) Visfatin activates ERK 1/2 and NF-κB, resulting in increased inducible nitric oxide synthase (iNOS) production [26]. iNOS is a proinflammatory enzyme that can cause an imbalance in NO production, induce endothelial dysfunction, and ultimately lead to impaired angiogenesis. Therefore, the inflammatory effect of visfatin promotes endothelial dysfunction, which may result in poor CCC development. Visfatin also has a two-way regulatory effect in metabolic diseases. Further research is needed to understand the impact of visfatin in different situations and clinical conditions related to cardiovascular disease.

Recent studies have shown that vitamin D can regulate the growth of endothelial cells and smooth muscle[27].Even vitamin D deficiency is positively correlated with the degree of coronary vascular stenosis [28]. Vitamin D is also closely related to the degree of vascular endothelial dysfunction in patients with coronary heart disease [29]. However, the relationship between vitamin D and collateral circulation and the underlying mechanism are largely unclear.

In our study, the 25-(OH)D3 level was significantly higher in the good CCC group than in the poor CCC group. This is consistent with the research results reported by Dogan Y [30]. The following points may provide a reasonable explanation for the poor CCC development caused by vitamin D deficiency: (1) vitamin D promotes the adhesion of white blood cells, increases VEGF-A expression, and promotes the growth and migration of vascular smooth muscle cells [31,32]; therefore, vitamin D deficiency can lead to impaired CCC development; (2) vitamin D activates and induces NO production, mediates the VEGF signalling pathway, and stimulates the growth of coronary collaterals [33,34], leading to poor CCC development. Cianciolo et al. [35] confirmed that VDRs are also present on circulating EPCs. Vitamin D deficiency can not only reduce the number of circulating EPCs but also decrease their ability to proliferate and form blood vessels, leading to impaired CCC development.

Our study is also the first to show an L-shaped relationship between 25(OH)D3 and visfatin. In the absence of 25(OH)D3, when the visfatin level was less than 30 nmol/L, a negative correlation was observed between 25(OH)D3 and visfatin. However, when the 25(OH)D3 level was greater than 30 nmol/L, the visfatin level did not continue to decrease with increasing 25(OH)D3; in contrast, the level remained constant. This phenomenon may suggest that supplementation with vitamin D to the normal range will not lead to further declines in endolipids in patients with vitamin D deficiency. This may also provide another possible explanation for the recent results reported by Heike et al. [36], indicating an absence of cardiovascular benefits from vitamin supplementation (it is worth noting that in the intervention and control groups, the plasma 25(OH)D3 levels at baseline and 12 months after the intervention were higher than 50 nmol/L).

Our study found that the serum LDL-C level was significantly higher in the poor CCC group than in the good CCC group. Moreover, the 25-(OH)D3 level was negatively correlated with the LDL-C level, which is similar to the results reported by Song et al. [37].

High levels of LDL-C are a definite risk factor for coronary arteries, which can induce endothelial cell dysfunction and impair the growth of coronary collateral vessels[38,39].

In our study, the frequency of diabetes was significantly higher in the poor CCC group (52.4%) than in the good CCC group (33.6%), and diabetic patients had lower vitamin D levels than nondiabetic patients. Nisanci et al. [40] also found insufficient opening of the collateral circulation in diabetic patients by measuring the coronary wedge pressure. Studies have found that due to the severely impaired function of vascular endothelial cells in diabetic patients, the abilities of these cells for proliferation, adhesion, integration and eventually angiogenesis are significantly reduced [41].

In conclusion, visfatin and vitamin D seem to play important roles in the regulation of key mechanisms involved in CCC development.

Seasonal changes in vitamin D levels and sun exposure may affect the results of the study, so we recruited patients from November to February to minimize inter-individual variability. In addition, physical exercise is also one of the factors that affect the level of vitamin D and the development of collateral circulation. Although the patients we selected indicated that they did not participate in regular physical exercise, this could not completely eliminate the impact on the results of the study. This is the shortcoming of this study.

Conclusion

Our study shows that CTO patients with poor CCC development have lower serum 25-(OH)D3 and higher serum visfatin levels than CTO patients with good CCC development. Low serum 25-(OH)D3 and high serum visfatin levels are independent predictors of poor collateral circulation in CTO patients.

Data Availability

The analyzed data sets generated during the study are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

The ethics committee of China Aerospace Science and Industry Corporation 731 Hospital approved the present study, and all participants provided their written, informed consent.

Conflicts of interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

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Authors' contributions

X.-L. Ji wrote the manuscript. X.-L. Ji designs and performs experiments, analyzes data, interprets data. X.-L. Ji and S.-Q. Jin drafts experiments. Y.-X. Wang,Y.-M. Chen and J.-Zhang were responsible for ELISA and recorded date. All authors approved the manuscript.

References:

1. Meier P, Schirmer SH, Lansky AJ et al. (2013). The collateral circulation of the heart[J].*BMC Med*, 11:143.
2. Kim EK, Choi JH, Song YB et al. (2016). A protective role of early collateral blood flow in patients with ST-segment elevation myocardial infarction*[J].Am. Heart J*, 171:56-63.
3. Jamaiyar A, Juguilon C, Dong F et al. (2019). Cardioprotection during ischemia by coronary collateral growth[J].*Am J Physiol. Heart Circ Physiol*, 316:H1-H9.
4. Berry C, Balachandran KP, L'Allier PL et al. (2007). Importance of collateral circulation in coronary heart disease[J].*Eur Heart J*, 28:278-291.
5. Zimarino M, D'Andreamatteo M, Waksman R et al. (2014). The dynamics of the coronary collateral circulation[J].*Nat Rev Cardiol*, 11:191-197.
6. Dakroub, A., S, A. N., Younis, N., Bhagani, H et al. (2020). Visfatin: A possible role in cardiovasculo-metabolic disorders[J].Cells, ,9:1.
7. Samal, B., Sun, Y., Stearns, G et al. (1994). Cloning and characterization of the cDNA encoding a novel human pre-B-cell colony-enhancing factor[J].Molecular and Cellular Biology, 14:1431–1437.
8. Sun, X., Sun, B. L., Babicheva, A et al. (2020). Direct extracellular NAMPT involvement in pulmonary hypertension and vascular remodeling. transcriptional regulation by SOX and HIF-2alpha[J]. *American Journal of Respiratory Cell and Molecular Biology* 63:92–103.
9. Nibbelink KA, Tishkoff DX, Hershey SD et al. (2007).1,25(OH)2-vitamin D3 actions on cell proliferation, size, gene expression, and receptor localization, in the HL-1 cardiac myocyte[J].*J Steroid Biochem Mol Biol , 103*:533-537.
10. Tarcin O, Yavuz DG, Ozben B et al. (2009). Effect of vitamin D deficiency and replacement on endothelial function in asymptomatic subjects[J].*J Clin Endocrinol Metab*, 94:4023-4030.
11. Pittarella P, Squarzanti DF, Molinari C et al. (2015). NO-dependent proliferation and migration induced by vitamin D in HUVEC[J].*J Steroid Biochem Mol Biol*, 149:35-42.
12. Wong MS, Leisegang MS, Kruse C et al.Vitamin D promotes vascular regeneration[J].Circulation, 130:976-986.
13. Grundmann M, Haidar M, Placzko S et al, (2012).Vitamin D improves the angiogenic properties of endothelial progenitor cells[J]. Am J Physiol Cell Physiol 303:C954-62.
14. Yang ZK, Shen Y, Hu J et al. (2017).Impact of coronary collateral circulation on angiographic in-stent restenosis in patients with stable coronary artery disease and chronic total occlusion[J]. *Int J Cardiol*, 227:485-489.
15. Seiler C, Stoller M, Pitt B et al. (2013).The human coronary collateral circulation: development and clinical importance[J]. *Eur Heart J* 34:2674-82.
16. McEntegart MB, Badar AA, Ahmad FA et al. (2016).The collateral circulation of coronary chronic total occlusions[J].EuroIntervention 11:e1596-603.
17. Alsanjari O, Chouari T, Williams T et al. (2020). Hildick-Smith D Angiographically visible coronary artery collateral circulation improves prognosis in patients presenting with acute ST segment-elevation myocardial infarction[J].*Catheter Cardiovasc Interv* 96:528-533.
18. Carmeliet P, (2000).Mechanisms of angiogenesis and arteriogenesis[J].Nat Med 6:389-395.
19. Qin X, Qin L, Luo J et al. (2019). Correlation analysis between 25-hydroxyvitamin D3, vitamin B12 and vitamin C and endothelial function of patients with CHD, Exp Ther Med, 17:418-22.
20. Adya Raghu., Tan Bee K., Punn Anu et al. (2008). Visfatin induces human endothelial VEGF and MMP-2/9 production via MAPK and PI3K/Akt signalling pathways: novel insights into visfatin-induced angiogenesis[J].Cardiovasc Res, 78:356-365.
21. Bae, Y.-H., Bae, M.-K., Kim, S.-R et al. (2009).Upregulation of fibroblast growth factor-2 by visfatin that promotes endothelial angiogenesis.Biochemical and Biophysical Research Communications.
22. Dambala, K.,Vavilis, D.,Bili, E et al. (2017). Serum visfatin, vascular endothelial growth factor and matrix metalloproteinase-9 in women with polycystic ovary syndrome*[J].Gynecological Endocrinology*, 33:529-533.
23. Tsai, C. H., Liu, S. C., Chung, W. H et al. (2020). Visfatin increases VEGF-dependent angiogenesis of endothelial progenitor cells during osteoarthritis progression[J].Cells, 9:1315.
24. Auguet, T., Aragonès, G., Guiu-Jurado E et al. (2016). Adipo/cytokines in atherosclerotic secretomes: Increased visfatin levels in unstable carotid plaque[J]. *BMC Cardiovascular Disorders*, 16:149.
25. Stancel N, Chen C C, Ke L Y et al. (2016). Interplay between CRP, atherogenic LDL, and LOX-1 and its potential role in the pathogenesis of atherosclerosis[J].Clin Chem, 62:320-27.
26. T. Romacho, V. Azcutia, M. Vázquez-Bella et al., (2009). “Extracellular PBEF/NAMPT/visfatin activates pro-inflammatory signalling in human vascular smooth muscle cells through nicotinamide phosphoribosyltransferase activity[J].Diabetologia, 52:2455–2463.
27. Wu-Wong, J.R.; Nakane, M.; Ma, J et al. (2006).Effects of Vitamin D analogs on gene expression profiling in human coronary artery smooth muscle cells[J]. *Atherosclerosis*, 186:20–28.
28. Goleniewska B, Kacprzak M, Zielińska M. (2014). Vitamin D level and extent of coronary stenotic lesions in patients with first acute myocardial infarction,Cardiol J, 21:18-23.
29. Cianciolo G, La Manna G, Cappuccilli ML et al. (2011). VDR expression on circulating endothelial progenitor cells in dialysis patients is modulated by 25(OH)D serum levels and calcitriol therapy*[J].Blood Purif*, 32:161-173.
30. Dogan Y, Sarli B, Baktir AO et al. (2015).25-Hydroxy-vitamin D level may predict presence of coronary collaterals in patients with chronic coronary total occlusion[J]. *Postepy Kardiol Interwencyjnej*, 11:191-196.
31. Cardús A, Parisi E, Gallego C et al. (2006).1,25-Dihydroxyvitamin D3 stimulates vascular smooth muscle cell proliferation through a VEGF-mediated pathway[J].Kidney Int, 69:1377-84.
32. Grundmann M, Haidar M, Placzko Set al. (2012).Vitamin D improves the angiogenic properties of endothelial progenitor cells[J].Am J Physiol ,*Cell Physiol*, 303:C954-C62.

33. Dai X, Faber JE, (2010). Endothelial Nitric Oxide Synthase Deficiency Causes Collateral Vessel Rarefaction and Impairs Activation of a Cell Cycle Gene Network During Arteriogenesis[J].Circ Res, 106:1870-81.
34. Uberti F, Lattuada D, Morsanuto V et al. (2014).Vitamin D protects human endothelial cells from oxidative stress through the autophagic and survival pathways[J].J Clin Endocrinol Metab, 99:1367-74.
35. Cianciolo G, La Manna G, Cappuccilli ML et al. (2011).VDR expression on circulating endothelial progenitor cells in dialysis patients is modulated by 25(OH)D serum levels and calcitriol therapy[J].*Blood Purif*, 32:161-173.
36. Luttmann-Gibson Heike,Mora Samia,Camargo Carlos A et al. (2019). Serum 25-hydroxyvitamin D in the VITamin D and OmegA-3 TriaL (VITAL): Clinical and demographic characteristics associated with baseline and change with randomizedvitamin D treatment[J].*Contemp Clin Trials*, 87:105854.
37. Song Y, Wang L, Pittas AG et al. (2013). Blood 25-hydroxy vitamin D levels and incident type 2 diabetes: a meta-analysis of prospective studies[J].Diabetes Care, 36:1422-1428.
38. Shen Y,Chen S,Dai Y et al. (2019). Lipoprotein (a) interactions with cholesterol-containing lipids on angiographic coronary collateralization in type 2 diabetic patients with chronic total occlusion[J].*Cardiovasc Diabetol* 18:82.
39. Puri R, Nissen SE, Shao M et al. (2016). Non-HDL Cholesterol and Triglycerides: Implications for Coronary Atheroma Progression and Clinical Events[J].Arterioscler Thromb Vasc Biol 36:2220-2228.
40. NianciY, Sezer M, Umman B, Yilmaz E et al. (2002). Relationship between pressure-derived collateral blood flow and diabetes mellitus in patients with stable angina pectoris: a study based on coronary pressure measurement[J].*J Invasive Cardiol*, 14:.118-122.
41. Shen Y, Ding FH, Dai Y et al. (2018). Reduced coronary collateralization in type 2 diabetic patients with chronic total occlusion[J].*Cardiovasc Diabetol*, 17:26.

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