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Multiple regression-based correlation between abdominal aortic calcification and paravertebral muscle degeneration

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ABSTRACT

Objective, to investigate the correlation between abdominal aortic calcification and paravertebral muscle degeneration, and to explore possible common risk factors for both. Methods, all patients with lumbar spinal stenosis admitted to Hospital X for MC and CT examination from 2016 to 2024 were selected, and through screening and exclusion, a total of 352 patients with LSS were included in the study, which consisted of 202 males and 150 females aged 40-80 years, with a mean of 63.24 years. The degree of paraspinal muscle degeneration in lumbar MRI, the degree of abdominal aortic calcification in lumbar CT scanning, as well as the patient's age, duration of LSS, glomerular filtration rate and other indicators were counted, and the distribution characteristics of abdominal aortic calcification and its correlation with paraspinal muscle degeneration were analyzed by the method of multiple regression. Results, of the 352 patients with LSS who were included to meet the criteria, the calcification group (151, 42.90%) and the non-calcification group (201, 57.10%). Mild, moderate and severe paravertebral muscle degeneration accounted for 56.53%, 28.69% and 14.77%, respectively. The AACS in patients with mild PD degeneration stage, moderate PD degeneration stage and severe PD degeneration, all showed a gradual increasing trend with age (P<0.001). Regression results showed that age, paravertebral muscle degeneration and eGFR were risk factors for AAC in patients with LSS. Conclusion, there was a significant correlation between abdominal aortic atherosclerotic calcification and paravertebral muscle degeneration (P<0.001), and the degree of PD degeneration can be used as an effective indicator for early warning of the occurrence of AAC in patients with LSS.

Keywords: abdominal aortic calcification; paravertebral muscle degeneration; LSS patients; lg eGFR; multiple regression

1. Introduction

Abdominal aortic aneurysm (AAA) is a common vascular disease, of which the prevalence in men ranges from 1.9% to 18.5%. Despite its insidious onset, the lethality of AAA rupture ranges from 80% to 95% [1-2]. The main predictor of AAA rupture has previously been the maximum diameter of the AAA (DMax),

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with other risk factors including aneurysm dilatation rate, surgical history, uncontrolled hypertension, and smoking history [3-4]. However, logistic regression prediction models based on maximum diameter and gender have only 60% sensitivity and 77% specificity (AUC 0.67) for predicting the risk of rupture [5-6]. Several studies have reported other potential risk factors for rupture, such as serologic markers or aortic atherosclerosis, but there is no ideal index for clinical application. Poor performance in predicting rupture may affect the choice of patient treatment plan and is detrimental to the improvement of patient prognosis [7-8]. Therefore, there is an urgent need to develop new predictors to improve the accuracy of AAA rupture prediction. Previous clinical studies have reported that calcification may be a contributing factor to AAA rupture, but this conclusion remains controversial [9-10].

On the other hand, spinal stability depends on the overall regulation of active subsystems, passive subsystems and neural subsystems [11-12]. As a component of the active subsystems, the role of paravertebral muscles in maintaining spinal stability cannot be ignored [13]. In recent years, the results of many studies have shown that decreased paraspinal muscle mass and increased fat content are closely related to the occurrence of various lumbar spine diseases. The role of paravertebral muscles has received more and more attention from scholars, but only a few studies have reported the characteristics and changing patterns of paravertebral muscles in healthy individuals [14-15]. Whether calcification of the abdominal aorta affects paraspinal muscle degeneration and how the mechanism of the effect occurs is well worth studying.

In this paper, the correlation between abdominal aortic calcification and paravertebral muscle degeneration was explored by retrospectively analyzing all patients admitted to Hospital X for lumbar spinal stenosis (LSS) who underwent MC and CT from 2016 to 2024. Abdominal aortic calcification was defined according to the type of abdominal aortic calcification on the basis of CT scanning, and was categorized into unreached abdominal aortic calcification (grade 0), mild (grade 1), moderate (grade 2), and severe (grade 3) AAC groups. The degree of paravertebral muscle degeneration was analyzed according to the Pfirrmann grading criteria, defining that patients with the highest grade of IV or V in each segment of the paravertebral muscle were identified as having paravertebral muscle degeneration. Through statistical methods such as multiple regression, the risk factors for AAC in LSS patients were explored, providing new ideas and diagnostic methods for controlling or delaying AAC.

2. Information and methodology

Lumbar spinal stenosis (LSS), as a kind of lumbar degenerative disease, is a commonly occurring chronic disease. Its population incidence can reach 50%-90%, which has become an important factor troubling people's lives, and thus research on the mechanism of LSS has become a hot topic of concern. Paravertebral degeneration (PD) is an important causative factor of LSS, and reduced blood supply causing nutrient supply has been confirmed to be associated with paraspinal degeneration. As the origin of segmental vasculature in the lumbar spine, abdominal aortic calcification (AAC) would affect the blood supply to the intervertebral discs and may be associated with paravertebral muscle degeneration.

2.1. Clinical information

2.1.1. Imaging:

1) CT examination. A GE Hispeed CT/i scanner with a layer thickness of 5 mm and a layer spacing of 0 mm was used to scan parallel to the vertebral body.

2) MR examination. A GE Signa CV/i 1.5T MRI scanner was applied, and lumbar sagittal T2WI scans as well as cross-sectional T2WI scans were performed. The corresponding scanning parameters were as follows:

(1) TR/TE, 2000ms/200ms. Layer thickness/layer spacing, 5mm/0.5mm.

(2) Matrix, 620/256. Number of excitations, 5. Variable bandwidth, 30.5kHz.

(3) Sagittal field of view, 32×32. Cross-sectional field of view, 20×20.

(4) Scanning software, No Phase Wrap, Variable Bandwidth, Tailored RF, etc.

2.1.2. Imaging analysis:

1) Abdominal aortic calcification (AAC). The types of abdominal aortic calcification were defined based on serial CT scans as follows.

(1) Grade 0, unreached abdominal aortic calcification.

(2) Grade 1, the largest calcification foci are detected on CT scanning and involve less than or equal to 1/3 of the circumference of the abdominal aorta.

(3) Grade 2, the largest calcification foci involve less than or equal to 2/3 or more than 1/3 of the circumference of the abdominal aorta.

(4) Grade 3, the largest calcification foci involve the abdominal aorta circumference greater than 2/3.

2) Paravertebral degeneration (PD). The degree of paravertebral muscle degeneration was analyzed according to the Pfirrmann grading standard, which is shown in Table 1. Paravertebral muscle degeneration was recognized when the highest grade of each segment of the paravertebral muscle of the patient was defined as grade IV or grade V.

Table 1. Pfirrmann grading standard

Classification	Intervertebral disc structure	Boundary between the annulus fibrosus and the nucleus pulposus	Signal strength	Disc height
Level I	Even, white and bright	Clear	High or medium signal	Normal
Level II	Unevenness	Clear	High or medium signal	Normal
Level III	Uneven, gray	Unclear	Medium signal	Normal or slightly reduced
Level IV	Uneven, gray to black	Forfeit	Medium to low signal	Normal or moderate reduced
Level V	Uneven, black	Forfeit	Low signal	Vertebral space collapse

2.1.3. Information collection. All patients with lumbar spinal stenosis (LSS) admitted to X hospital for MC and CT examination from 2016-2024 were selected, excluding those with clear pathological changes such as tumors, inflammation, etc., a total of 1,035 cases, 592 males and 443 females, aged 18-80 years old, average 50.2 years old.

The diagnostic criteria for lumbar spinal stenosis (LSS) were referred to the diagnostic criteria for lumbar spinal stenosis provided in the Guidelines for the Prevention and Treatment of Low Back Pain, which was developed by the Ministry of Health's Family Medicine Training Center in 2007.

1) A history of chronic low back pain, with some patients having a history of trauma.

2) Long-term recurrent low back pain and intermittent trekking.

3) Restriction of lumbar back extension and pain.

4) Radicular symptoms in the lower limbs, mostly bilateral, obvious when walking.

5) Straight leg raising test is positive or negative.

6) Weakness of tendon reflexes of the lower limbs, mostly heel reflexes, knee reflexes may be normal.

7) Lumbar X-ray can clearly show that the sagittal diameter of the spinal canal is narrower than normal, and its absolute value is mostly less than 15mm.

8) CT or myelography showing a cross-sectional area of the paraspinal muscles <100 mm^2 and a nerve root canal fossa <5 m. Vertebrograms may show a typical "wasp-waist" defect.

Inclusion criteria for LSS patients:

1) Age >40 years.

2) Central lumbar spinal stenosis with degenerative nature on CT or MRI.
3) Fulfillment of diagnostic criteria and clinical diagnosis of degenerative by an orthopedic surgeon. The diagnostic basis included history review, physical examination, and MRI imaging of the anatomical site of stenosis.
4) CT and MRI films were complete.

Study exclusion criteria:

1) Previous history of spinal surgery.
2) Patients with comorbid acute spinal disease (e.g., disc herniation).
3) Specific low back pain.
4) Spinal cord infection.
5) Primary or metastatic spinal tumor.
6) Other musculoskeletal disorders that cause walking dysfunction (e.g., severe osteoarthritis of the hip, osteoarthritis of the knee).
7) Polyneuropathy, peripheral arterial disease.
8) Other specific comorbidities, including any limiting factors that may affect walking, such as coronary artery disease, chronic obstructive pulmonary emphysema, etc.
9) Those with incomplete imaging data.
10) Other conditions affecting the study.

Hospitalization number, gender, age, height, weight, occupation, duration of low back pain, walking distance, ODI, and VAS were also collected. The basic information collected from the patients and the information of X-rays and MRI films were stored in the electronic medical record system.

2.2. Research methodology

2.2.1. Laboratory tests. Fasting venous blood levels of hemoglobin, neutrophil count, platelets, triglycerides, total cholesterol, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, uric acid, creatinine, albumin, ultrasensitive C-reactive protein, corrected calcium, phosphorus, alkaline phosphatase, and whole-segment parathyroid hormone were recorded in all study subjects. In this case, corrected calcium level = calcium + [40 - albumin (g/L)] × 0.8.

2.2.2. Detection of serum CD146. Early morning fasting blood samples were retained from all study subjects and placed at room temperature for 0.5 h. The samples were centrifuged at 5000 g for 15 min using a centrifuge with a constant temperature of 4°C, and the upper layer of serum was placed at -80°C for freezing. CD146 was detected by sandwich enzyme-linked immunosorbent assay (ELISA), and the kit was purchased from R&D (item DY921-06).

2.2.3. AAC assessment and grouping. In this study, abdominal CT was used to assess AAC, which was defined as the presence of plaques with a density greater than 130 HU in the region of the abdominal aortic alignment. The degree of circumferential angle corresponding to aortic calcification in each cross-section of CT was measured, and the maximum circumferential angle was defined as the angle of vascular calcification, and a calcification angle of $\geqslant 90°$ was defined as severe AAC.All patients with LSS were classified into AAC and non-AAC groups according to the occurrence of AAC, and then the AAC group was further classified into severe AAC and non-severe AAC groups.

2.3 Statistical methods

SPSS 22.0 software was applied for statistical analysis, and continuous variables obeying normal distribution were expressed as mean ± standard deviation, and the t test was adopted for comparison between groups, and continuous variables not obeying normal distribution were expressed as median (quartile), and the rank sum test was adopted for comparison between groups.

Count data were expressed as $[n(\%)]$, and the chi-square test or Fisher's exact probability method was

used for comparison between groups. Spearman correlation analysis was used to assess the correlation between the two indicators of AAC and PD in patients with LSS. One-way logistic regression was used for initial screening of risk factors, and variables with $P<0.10$ were included in multivariate logistic regression.

The Box-Tidwell method was used to test for the existence of a linear relationship between the continuous independent variables and the logit transformed values of the dependent variable, and to test for the existence of covariance between the independent variables. The predictive value of different indicators for AAC and severe AAC was assessed by plotting the subjects' work characteristics (ROC) curves. $p<0.05$ was taken as statistically significant difference.

3. Results

3.1. Basic Situation Analysis

3.1.1. General situation characteristics. The detailed distribution of the demographic characteristics of the participants is shown in Table 2.1035 patients were screened and a total of 352 patients with LSS were included in the study, consisting of 202 males and 150 females, aged 40-80 years, with a mean of 63.24 years.

Table 2. Demographic characteristics of the participants

Variable	Mean (SD)Median(Q1-Q3)/N(%)
Age	63.24 ± 13.25
<65 Year	251 (71.31%)
⩾65 Year	101 (28.69%)
Sex	352 (100%)
Male	202 (57.39%)
Female	150 (42.61%)
Educational level	
Below ninth grade	51 (14.49%)
High school graduation	102 (28.98%)
College degree or above	199 (48.86%)
Graduate student	27 (7.67%)
Blood calcium content (mg/d1)	9.12±0.45
Systolic blood pressure (mmHg)	128.24±18.65
Diastolic blood pressure (mmHg)	71.35±13.22
High density lipoprotein (mmol/L)	1.45±0.41
Triglyceride (mg/d1)	100.05(67.82-148.25)
Low density lipoprotein (mg/dl)	114.6(87.40-135.42)
Urinary creatinine (mg/dl)	108.73 ± 74.32
Proteinuria (mg/24h)	52.83 ± 278.46
Apolipoprotein B(g/1)	0.92 ± 0.38
Folic acid (nmol/L)	1268.41 ± 591.83

3.1.2. Calcification of the abdominal aorta. Table 3 demonstrates the median abdominal aortic calcification score (AAC) and interquartile range 0 (0.0, 2.0) of the participants. Based on the definition of abdominal aortic calcification score, the participants were divided into a calcified group (AAC greater than 0) and a non-calcified group (AAC equal to 0) containing 151 and 201 participants, respectively, with a weighted percentage of 42.90% and 57.10%, in that order.

Table 3. Study subjects abdominal aortic vascular calcification

Variable	Number of cases/Median	Mean (SD)Median(Q1-Q3)/N(%)
Abdominal aortic calcification Score (AAC24)	0	(0.0, 2.0)
Abdominal aortic calcification group	352	100%
Abdominal aortic calcification group (AAC)	151	42.90%
Abdominal aorta without calcification group (Non-AAC)	201	57.10%

3.1.3. Paravertebral muscle degeneration status. Table 4 exhibits the paraspinal degeneration (PD) status of the participants, 352 participants were categorized according to the Pfirrmann grading criteria for paraspinal degeneration status, with the number of Level I-Level V distributions being 42, 73, 84, 101, and 52, respectively. Levels I-III were defined as mild paravertebral muscle degeneration, Level IV as moderate paravertebral muscle degeneration, and Level V as severe paravertebral muscle degeneration. The numbers of the three groups were 199, 101, and 52, respectively, and their weighted proportions were 56.53%, 28.69%, and 14.77%, in that order.

Table 4. Status of paravertebral muscle degeneration (PD) in the patient

Classification	Mean	Mean (SD)Median(Q1-Q3)/N(%)	Classification
Level I	42	11.93%	199 (56.53%)
Level II	73	20.74%%	
Level III	84	23.86%	
Level IV	101	28.69%	101 (28.69%)
Level V	52	14.77%	52 (14.77%)

3.2. AAC distribution of variables in LSS patients

3.2.1. Age and AAC distribution characteristics of LSS patients. The distribution of AACS (median, interquartile spacing) in different age groups is shown in Figure 1, where it can be seen that the median AACS in young (40-45) LSS patients was 2 (interquartile spacing 0.5-5). The median AACS was 5 (interquartile spacing 0.5-10) in the middle-aged group (46-59), 7 (interquartile spacing 1-13) in the young-aged group (60-74), and 8 (interquartile spacing 2-15) in the old-aged group (75-80). There was a trend of progressively higher AACS with increasing age ($p<0.001$).

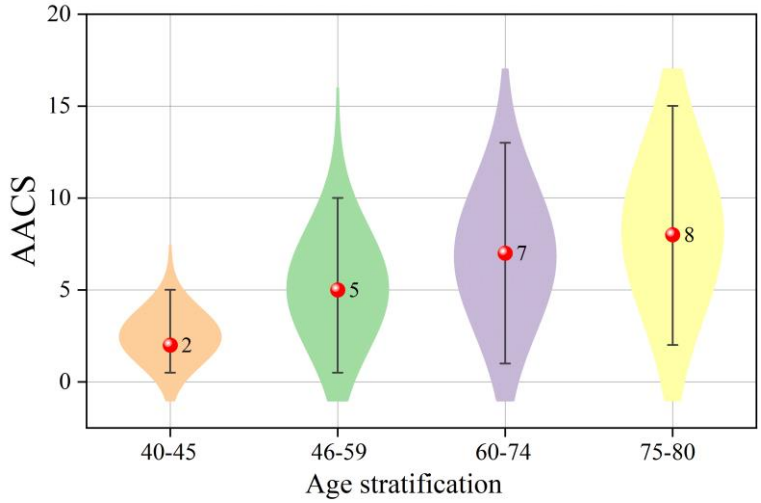

Fig. 1. AACS in different age groups

The results of AAC distribution (%) in different age groups are shown in Figure 2, the prevalence of AAC was 36.5% in the young group, 62.6% in the middle-aged group, and as high as 83.6% and 96.9% in the young-aged and old-aged groups of LSS patients. The prevalence of AAC gradually increased with age ($P<0.001$). In AAC, mild calcification was predominant in young patients and rarely severe calcification (only 1.7%). Mild and moderate calcification predominated in middle-aged and young elderly patients, and moderate calcification predominated in old elderly patients (56.2%), accompanied by severe calcification in 13.2%.

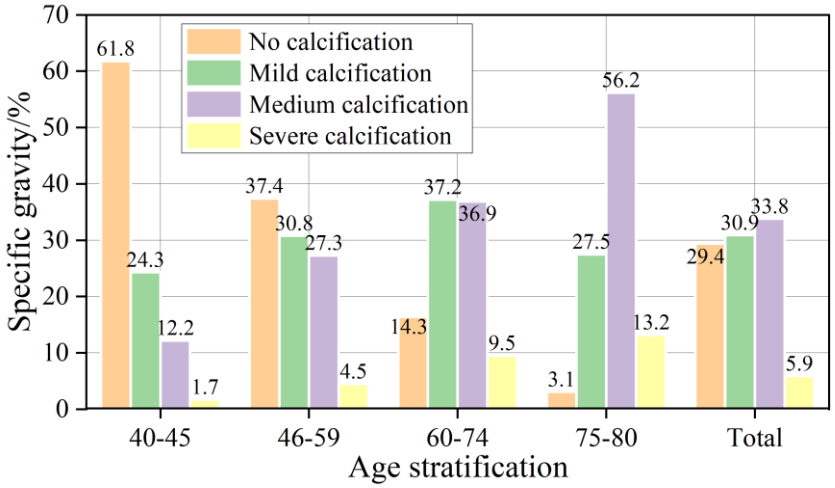

Fig. 2. AAC distribution by age group (%)

3.2.2. Characteristics of AAC distribution in patients with PD and LSS. The distribution of AACS (median, interquartile spacing) in patients with LSS between different degrees of paravertebral degeneration (PD) is shown in Figure 3. The median AACS was 1 (interquartile spacing 0-2) in patients with mild PD, 3 (interquartile spacing 0-7) in patients with moderate PD, and 4 (interquartile spacing 1-7) in patients with severe PD. It was found that the AACS tended to increase progressively with the degree of paravertebral muscle degeneration (P<0.001).

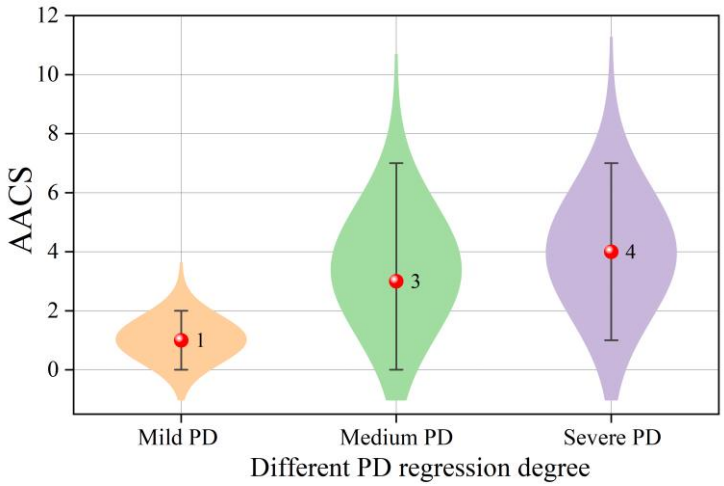

Fig. 3. AACS in different degrees of PD degeneration

The distribution (%) of AAC between different degrees of paravertebral degeneration (PD) is shown in Fig. 4. 47.2% of the patients had developed AAC in mild PD. 64.5% of the patients had AAC in moderate PD stage, and the prevalence of AAC in patients with severe PD was as high as 78.2%. The prevalence of AAC gradually increased with increasing PD degeneration (P<0.001). In AAC, patients with mild PD stage were predominantly mildly calcified (32.5%) without severe calcification. Patients with moderate PD stage had predominantly mild and moderate calcifications (29.8% and 30.3%, respectively). Patients with severe PD stage also had predominantly mild and moderate calcifications, 33.1% and 35.2%, respectively, but were accompanied by 9.9% severe calcifications (P<0.001).

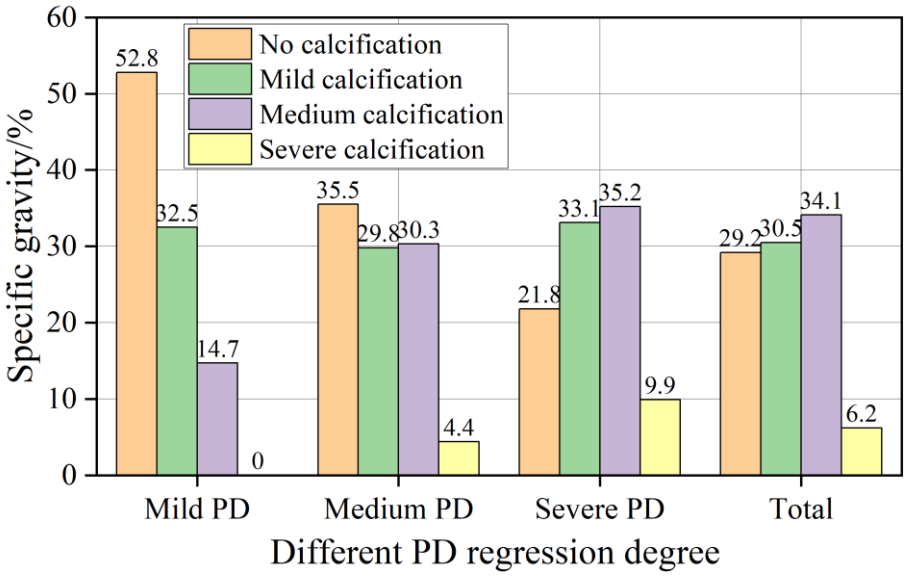

Fig. 4. AAC distribution among different PD (%)

3.2.3. Age, PD and AAC distribution characteristics of LSS patients. The results of AACS distribution (median, interquartile spacing) of LSS patients with different ages and degrees of PD degeneration are shown in Figure 5. It can be seen that the AACS of patients with mild PD regression stage, moderate PD regression stage and severe PD regression all showed a gradual increasing trend with age (P<0.001). The median AACS of young elderly (60-74) and old elderly (75-80) LSS patients with mild PD stage also reached 1.5 and 4, respectively.

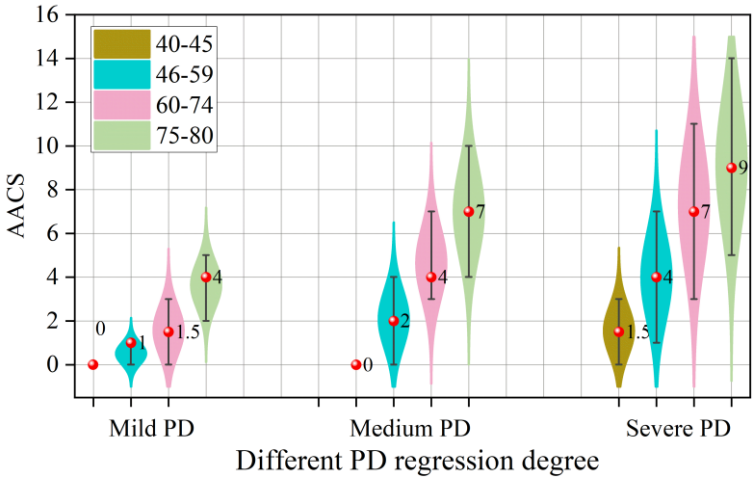

Fig. 5. AACS distribution in LSS patients with different ages and degrees of PD degeneration

The results of the distribution of AAC prevalence (%) by age and degree of PD regression are shown in Figure 6, where the prevalence of AAC in patients with mild PD regression stage reached 27.8% in the young group, and the prevalence of AAC in patients with mild PD regression stage in the middle-aged and the young-aged old was 34.5% and 58.3%, respectively (P=0.02<0.05). The prevalence of AAC in old elderly patients was more than 90% in all PD regression stages.

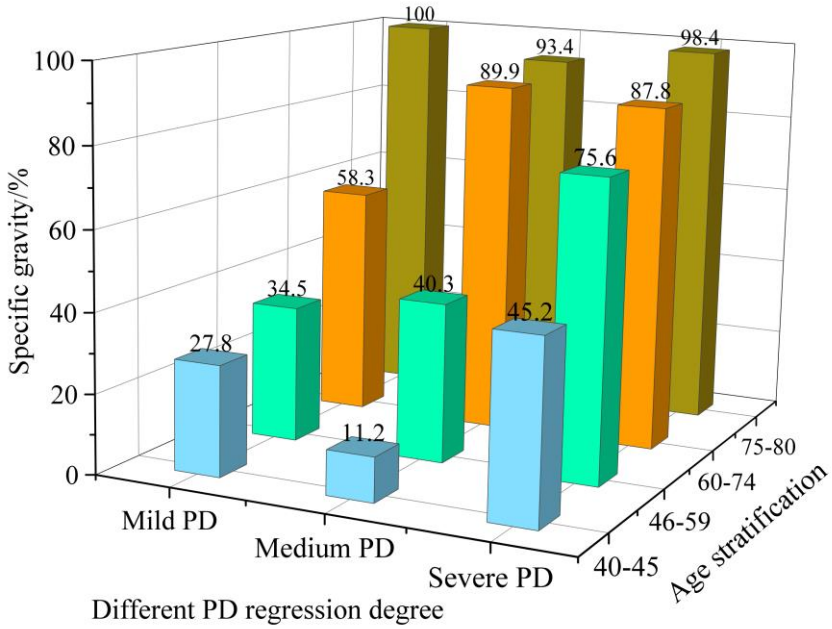

Fig. 6. AAC prevalence (%) distribution at different ages and degrees of PD degeneration

3.3. Multiple regression analysis of AAC

To analyze the factors affecting AAC, multivariate logistic regression analysis was performed with moderate-to-severe AAC (0=no/mild calcification, 1=moderate-to-severe calcification) as the dependent variable. Firstly, the first univariate logistic regression analysis was performed, and the included independent variables included age, duration of LSS, DBP, hemoglobin, phosphorus, total cholesterol, HDL, LDL, urea, creatinine, the degree of PD regression (1=mild stage of regression, 2=moderate stage of regression, 3=severe stage of regression) and lg eGFR (glomerular filtration rate). Multiple regression analysis was then performed based on stepwise regression method and the results are shown in Table 5. The results showed that age, moderate PD regression stage (vs. mild PD stage), severe PD (vs. mild PD stage) and lg eGFR were risk factors for AAC in patients with LSS.

Table 5. Logistic multiple regression analysis of AAC in LSS patients

	Univariate			Multivariate		
	OR	95%CL	P	OR	95%CL	P
Model 1	Adjust R2=0.272					
Age	1.085	1.052-1.087	<0.001	1.083	1.049-1.083	<0.001
Degree of degeneration of PD						
Medium PD vs. Mild PD	2.823	1.431-5.085	0.002	3.056	1.115-8.231	0.021
Severe PD vs. Mild PD	4.522	2.569-8.535	<0.001	6.424	2.635-15.986	<0.001
Model 2	Adjust R2=0.265					
Age	1.052	1.056-1.087	<0.001	1.056	1.051-1.084	<0.001
lg eGFR	0.451	0.311-0.682	<0.001	0.342	0.179-0.528	<0.001

Note: Dependent variables, 0=no/mild calcification, 1=moderate to severe calcification.

Independent variables, including age, sex (1=male, 2=female), duration of LSS, DBP, hemoglobin, phosphorus, total cholesterol, HDL, LDL, degree of PD regression (1=mild regression stage, 2=moderate regression stage, 3=severe regression stage) and lg eGFR.

The independent variables for Model 1 and Model 2 were incorporated into the degree of PD regression

(1=mild regression phase, 2=moderate regression phase, 3=severe regression phase) and lg eGFR, respectively.

4. Discussion

The paravertebral muscles play an important role in maintaining stability and motion of the lumbar spine, respectively. The multifidus muscle facilitates lumbar extension and is also involved in joint control at each lumbar segment. The multifidus is located in the deeper part of the erector spinae muscle and is the only muscle fiber in the lumbosacral region. It is closely connected to the lumbar vertebrae, and when the spine is out of balance, it pre-contracts, which can increase the tension of 1-3 lumbar vertebral segments and maintain a normal spinal line of force, which reduces vertebral movement, increases spinal stability, and maintains physiological anterior curvature of the lumbar vertebrae. This is mainly related to the muscle fiber composition of the multifidus muscle, and it has been shown that patients with spinal degeneration with sagittal imbalance have increased intrinsic contractile muscle disorders (i.e., impaired specific force production) compared to those without deformity, resulting in dysfunctional intrinsic force muscle segment length characteristics [16]. The muscle fibers of skeletal muscle are generally divided into two types: type I and type II muscle fibers, which are distinguished from each other by the fact that these two types of muscle fibers are composed of a different number of muscle fibers, but will not contain only one type of muscle fiber. The blood supply to the lumbar spine is segmental, and the feeder arteries of each segment traffic with each other through small anastomotic branches in the longitudinal ligament, paravertebral muscles, notochord, and dura mater. Literature [17] has also demonstrated a significant correlation between increased fatty infiltration of the multifidus muscle and ODI scores, and that keeping the paravertebral muscles healthy improves surgical planning, improves rehabilitation outcomes, and reduces postoperative disability. The results of this paper suggest that decreased blood supply is an important cause of paravertebral muscle (PD) degeneration, and abdominal aortic calcification (ACC), as a hallmark of abdominal aortic atherosclerosis, severely affects the blood supply to the lumbar segmental vasculature. The incidence of both increases with age.

It has been shown that the expression in intervertebral disc cells is relatively high, with mean fold changes of 6.9, 10.0, and 6.3 fold for AFC, CEPC, and NPC, respectively, relative to autologous OB, and 2.3, 3.4, and 3.2 fold for AFC, CEPC, and NPC, respectively, relative to autologous MSCs [18]. Therefore, when nutrient supply is decreased, metabolite elimination is also impeded. As a result, the intervertebral disc is then in an acidic environment and the cells continue to produce matrix metalloproteinases. And this enzyme degrades the macromolecules in the matrix within the intervertebral disc, which leads to the destruction of the disc structure. In this study, a systematic statistical analysis of a sample of patients with LSS was performed, and a statistically significant correlation between abdominal aortic atherosclerosis (calcification) and paravertebral muscle degeneration was verified. And the strongest correlation between the two was found in patients in the lower age group. This suggests that active clinical prevention and treatment of atherosclerosis may be a way to prevent and reduce the degeneration of the paravertebral muscles, thereby reducing the incidence of low back pain.

5. Conclusion

Paravertebral muscle degeneration, eGFR and age have an important influence on the occurrence of AAC in patients with LSS, and the level of paravertebral muscle degeneration can be used as an important reference index for predicting AAC, and the two are highly correlated ($P<0.001$), and the delaying of the level of paravertebral muscle degeneration in an appropriate way is clinically effective in delaying the occurrence of AAC in patients with LSS. However, because abdominal aortic calcification is a sign of the progressive stage of abdominal aortic atherosclerosis, not all abdominal aortic atherosclerosis forms abdominal aortic calcification, and there is no effective way to noninvasively determine the early stage of abdominal aortic atherosclerosis with the current examination methods. Moreover, this study only

collected cases from one hospital, which is a small sample, so the results have their limitations. Prolonging the observation period, multicenter collaborative studies, and searching for more effective methods of evaluating lesions will lead to more general conclusions.

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