

Branches of the brachiocephalic was an independent risk factor for hemorrhagic pulmonary sheath (HPS) patients with acute Stanford A aortic dissection

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Abstract

Background: AAD refers to the blood flow into the middle membrane through the intimal rupture of the aorta. Hemorrhagic pulmonary sheath (HPS) is a common complication of Stanford-A AAD. The risk factors of HPS are remaining unclear

Methods: In this study, we have probed the potential risk factors of HPS patients with acute Stanford A aortic dissection. 18 HPS patients with acute Stanford A aortic dissection were selected as the case group. The age difference ± 5 years and the same sex are set as the matching principles. 36 patients with acute Stanford-A type AD who did not detect HPS in the same period were matched according to the ratio of 1:2. Demographic data, treatment methods, AD-related disease history, clinical symptoms and Charlson comorbidity index (CCI) values of each patient were collected. Meanwhile, the values of the maximum diameter of ascending aorta (mm), aortic dissection range, and the main branch of the aorta, pleural effusion/blood, and pericardial effusion/blood were measured by two experienced cardiovascular radiological physicians. Univariate and multivariate conditional logistic regression analysis was used in this study.

Results: CCI value and the branches of the brachiocephalic in the case group were significantly higher than those in the control group ($p < 0.05$). Univariate conditional logistic regression analysis showed CCI and branches of the brachiocephalic were associated with HPS. Multivariate conditional logistic regression analysis suggested that branches of the brachiocephalic were an independent risk factor for HPS (OR=7.02, 95%CI=1.28-38.62, $p=0.025$).

Conclusions: Branches of the brachiocephalic were an independent risk factor for HPS.

Background

Acute aortic syndrome (AAS) is a serious, life-threatening aortic syndrome, which mainly includes acute aortic dissection (AAD), aortic penetrating ulcer, intra-aortic hematoma, and traumatic aortic injury [1-2]. AAD refers to the blood flow into the middle membrane through the intimal rupture of the aorta, which results in the separation of the inner and outer membranes. The incidence rate of AAD in population is about 2.6-3.5/100000/year. [3] Meanwhile, about 85%-95% of AAS patients could be diagnosed with AAD [3]. AAD has various clinical features, including acute onset, rapid progress, complications, and high mortality. About 20% of patients die within one hour of onset [4-5]. Current medical anatomical classification methods for AAD include DeBakey typing and Stanford type. The Stanford typing method is closely related to the treatment method. Therefore, it is more commonly used [6]. The Stanford typing method divides AAD into Stanford-A and Stanford-B. Stanford-A accounts for approximately 75% of AAD. Meanwhile, the diagnosis of AAD mainly depends on the imaging examination. CT examination as one of the most important imaging method has been widely used in the diagnosis of AAD [6, 7]. Multi-detector computed tomography (MDCT) has high imaging speed and high resolution. Computed tomographic angiography (CTA) has high sensitivity and specificity for AAD diagnosis. The sensitivity and specificity

of both methods for AAS diagnosis are close to 100% [8-10]. The mechanism of AAD has not been clarified. Studies have shown that genetic or metabolic abnormalities may cause degeneration and/or cystic necrosis of collagen fibers and elastic fibers in the aortic wall, which would further result in the separation of the aortic intima and adventitia in the proximal and/or distal aorta [11-13]. Meanwhile, studies have shown that multiple acquired factors can affect the occurrence and development of AAD. These factors include Hypertension, trauma, infection, iatrogenic injury, dyslipidemia, atherosclerosis, pregnancy, smoking, stroke, drugs, chronic renal insufficiency, autoimmune diseases, chronic obstructive pulmonary disease, etc [6]. Approximately 77% of AAD patients are associated with hypertension. Therefore, hypertension is considered to be the most important risk factor [12]. Hypertension can lead to a variety of emergency reactions, including increased arterial wall pressure, the release of matrix metalloproteinases and various cytokines, and excessive degradation of the extracellular matrix. Therefore, it ultimately leads to intimal injury and aortic wall degeneration [13].

Hemorrhagic pulmonary sheath (HPS) is a common connective tissue with a sheath structure in which the blood of the aortic dissection breaks into the central pulmonary artery and the aortic root. Previous reports have suggested that HPS is a common complication of Stanford-A AAD [14]. Due to the poor evidence, previous studies used different terms to describe this complication such as intrahepatic hematoma [15, 16], pulmonary sphincter hematoma [17], blood infiltration of the aorta and pulmonary vascular adventitia [18], and hemorrhage along the pulmonary sheath [19], extraluminal perivascular hemorrhage [20], and hemorrhage along the pulmonary artery [21-23]. The results of HPS in chest radiographs examination varies according to the range of bleeding (normal to double lung flaky infiltration) [24-25]. Sueyoshi et al. retrospectively analyzed 232 cases of Stanford-A AD patients with extravasation of blood along the pulmonary artery [23]. The results suggested that 21 cases (9.1%) of extravasated blood along the pulmonary artery, double-chamber aortic dissection, and blood in the alveoli are associated with poor prognosis. The authors speculate that the poor prognosis is due to high pressure around the pulmonary artery and massive exudation of blood around the pulmonary artery, which leads to alveolar hemorrhage and reduced pulmonary circulation [23].

In word wild, there are few studies about the incidence of HPS. More importantly, the risk factors of HPS are remaining unclear. In this study, we have carried out a case-control study. Traditional AAD risk factors (age, gender, hypertension, etc.), CCI and MDCT characteristics (upper aortic maximal diameter, aortic dissection range, main aortic vascular involvement, pleural effusion/blood, pericardium) have been included to study the relationships with HPS in this study. Risk factors for HPS have been screened. The information in this study would be helpful for future detection and treatment of HPS.

Methods

Group

This study retrospectively analysis 188 patients who were diagnosed with acute Stanford-A AD by chest and abdomen CT scan and CTA examination in The Eighth Affiliated Hospital of Sun Yat-sen University

between January 2011 and January 2016. 18 HPS patients were finally included based on the results of the CT scan image and CTA image. 36 acute Stanford-A aortic dissection without HPS were included and marked as the control group. Patients in the control group should satisfy the following criteria: age difference in ± 5 years, same gender, and paired with HPS patients according to the similar visiting time. Exclusion criteria complied with the following conditions: patients with chronic aortic dissection; patients with aortic dissection stenting; patients with a simple aortic aneurysm; patients with severe lesions (trauma); poor quality of CT images.

Clinical information

The data of the control group and the case group were collected: 1) baseline data (age, gender), 2) AD-related diseases information (history of hypertension, history of diabetes, history of connective tissue disease, history of chronic lung disease, history of chronic cardiovascular disease, chronic History of kidney disease, history of hyperlipidemia. 3) Clinical symptoms and clinical signs associated with AD: chest and back pain, syncope, sudden dyspnea, cough, hemoptysis or blood stasis, nervous system symptoms, shortness of breath, tachycardia, decreased blood pressure, myocardial infarction or infarction, heart failure, renal failure Abnormal liver function. 4) Treatment information (surgical treatment or conservative treatment). Charlson comorbidity index (CCI) was calculated based on the information of AD-related diseases. Table S1 showed detailed information for scoring criteria.

MDCT scanning method and CT image analysis

A 64-slice spiral CT (Aquilion TSX-101A, Canon Medical Systems (China) Co., Ltd.) was used to perform a three-phase (plain, arterial, venous) scan. The patient was placed in a supine position. Scanning was ranged from chest inlet to groin level. Scanning parameters were listed as following: tube voltage 120kV, automatic tube current, volume data layer thickness 1mm, layer spacing 0.8mm, rotation time 0.5s/Rot, pitch 0.828, collimator width 64×0.5mm. The matrix was 512×512. The window width was set to 400HU, the window level was set to 40HU. The soft tissue reconstruction algorithm was used. The thick layer transverse image reconstruction layer had a thickness of 3 mm and a layer spacing of 3 mm. After the flat scan, a high-pressure syringe (meDRAD) was used to inject 80 ml iopromide (300 mg /ml, Bayer HealthCare Co., Ltd.) through the right elbow vein (3.5 ml/s). After the injection, 40 ml of physiological saline was added at the same rate to perform flushing. The contrast agent automatic tracking technology is used to trigger the arterial phase scan. Firstly, a layer is pre-scanned on the diaphragm plane as the detection plane. The region of the aortic cavity is placed to trigger the region of interest to monitor the change of CT value in the aortic cavity. The scanning interval was 1 s. The auto-trigger CT threshold was set to 150HU. When the CT value threshold of the interested region reached 150HU, the delay 6s automatically started scanning to acquire the arterial phase image. The venous phase was scanned 65 seconds after the contrast injection. The scanning range and scanning parameters were the same as those of the arterial phase. Acute Stanford-A aortic dissection refers to dissection that separates the intima of the ascending aorta from the adventitia [26]. Meanwhile, the onset time is <14 days [27]. The HPS diagnostic criteria were listed as following: 1) The CT scan image had a circular, crescent-shaped

high-density shadow around the main pulmonary artery and/or pulmonary artery trunk or branch vessel wall. 2) Enhanced CT images showed that ring-shaped, crescent-shaped shadows were low-density soft tissue shadows without enhancement. Aortic branch vascular involvement is defined as the following: 1) The torn inner membrane entered into the intima of the branch vessel. The branch vessel was supplied by the true cavity and the false lumen. 2) The inner membrane of the branch vessel was completely torn. The blood supply of the branch vessel was completely from the false lumen. 3) The tearing inner membrane did not enter the branch vessel. The blood supply of the branch vessel was derived from true lumen with branch vessels. Any combination of dissection involving the brachiocephalic or left common carotid artery or left subclavian artery or brachiocephalic trunk left subclavian artery and left common carotid artery was defined as involvement of the brachial vascular branch. Any combination of dissection involving the celiac trunk or superior mesenteric artery or left or right renal artery or celiac trunk, superior mesenteric artery, left renal artery, and the right renal artery was defined as abdominal branch vessel involvement. Chest or pericardial exudate CT value > 20HU was defined as chest or pericardial hemorrhage [28]. Definitions of hypertension, heart disease, and dyslipidemia were referred to published guidelines [29, 30].

Statistics

Data analysis was performed using the SPSS 20.0 software package (Version 16, IBM, USA). Quantitative data with normal distribution were expressed as mean and standard deviation. Quantitative data with non-normal distribution were expressed by median and quartile. Categorical variables were described by percentage. The nonparametric rank-sum test (Wilcoxon method) was used to compare the difference of the age the maximum diameter of the ascending aorta, and the CCI index between the case group and the control group. χ^2 test and Fisher exact probability method were used to calculate the differences of gender, CCI, aortic dissection, aortic main branch vascular involvement, pleural effusion/blood, pericardial effusion/blood, hypertension, basic disease history, clinical symptoms, clinical Signs between the case group and the control group. Before the single factor logistic regression analysis, the variables were assigned (Table S2). The single factor regression analysis of 1:2 matching data was performed to calculate the P-values, odds ratio (OR) and odds ratio (OR) with 95% CI of age, gender, maximal diameter of ascending aorta, CCI, the extent of aortic dissection, the involvement of major branches of the aorta, pleural effusion/blood, pericardial effusion/blood, hypertension. Before the multiple factor logistic regression analysis, the variables were assigned (Table S3). All factors with $p < 0.1$ were included in a 1:2 matched multivariate conditional logistic regression analysis (fitting using Cox in survival analysis) to screen for risk factors for HPS. The odds (OR) was used as an approximate estimate of relative risk (RR). $OR > 1$ was an independent factor, and $OR < 1$ is an independent protective factor. In the single-factor analysis test, the test level was $\alpha = 0.1$. In a multivariate analysis test, the test level was $\alpha = 0.05$. All p values represent the two-sided probability.

Results

Basic information

In this study, a total of 188 patients, including 136 males (72.3%) and 52 females (27.7%) with acute Stanford-A AD were enrolled. The male to female ratio of total patients was 2.61:1, with an age range of 29-78 years and an average age of 59 years. There were 18 Stanford-A AD patients with HPS (case group), 13 males and 5 females. The male to female ratio of those patients was 2.6:1. The youngest was 34 years old, and the oldest was 74 years old. The average age was 60.0 years. In the control group, members were matched with 2:1 for each HPS in the case group (age \leq 5 year and the same gender). A total of 36 Stanford-A AD patients without HPS were included in the control group (26 males and 10 females, aged 29-78 years). The average age was 60.0 years old. There was no significant difference between the two groups ($p>0.05$). The detailed information of patients was listed in Table 1 and Table 2. Moreover, we have compared the clinical information between the control group and the case group. In the case group, there are 16 patients with hypertension (88.9%), 3 patients with diabetes (16.7%), 5 patients with chronic cardiovascular and cerebrovascular diseases (27.8%), 2 patients with chronic lung disease (11.1%), 4 patients with moderate/severe of renal damage (22.2%), 3 patients with connective tissue disease (16.7%), 4 patients with peripheral vascular disease (22.2%), 2 patients with mild liver damage (11.1%), and one patient with tumor (5.6 %). Meanwhile, in the control group, there are 34 patients with hypertension (94.4%), 7 patients with diabetes (19.4%), 12 patients with chronic cardiovascular and cerebrovascular diseases (33.3%), 4 patients with chronic lung disease (11.1%), 7 patients with severe renal damage (19.4%), 4 patients with connective tissue disease (11.1%), 7 patients with peripheral vascular disease (19.4%), 3 patients with mild liver damage (8.3%), and one patient with tumor history (2.8%). There are no significant differences of hypertension, diabetes, chronic cardiovascular and cerebrovascular diseases, chronic lung disease, moderate/severe renal damage, connective tissue disease, peripheral vascular disease, mild liver damage, and tumor history between case group and control group ($p>0.05$). The detailed information has been presented in Table 3. Moreover, we have further compared the Charlson comorbidity index (CCI) between the two groups (Table 4). The results suggested that significant differences could be detected in both groups. In the case group, the median CCI was 2, the 25th percentile was 1; the 75th percentile was 3. Meanwhile, in the control group, the median CCI was 1, the 25th percentile was 1; the 75th percentile was 1.5 (Table 4). There were statistical differences between the two groups in the CCI index rank-sum test and CCI score distribution chi-square test ($P<0.05$).

Clinical information

In this study, we have further compared the clinical symptoms, clinical signs, and MDCT features between the two groups. In clinical symptoms study, we have compared six indexes, including chest pain, syncope, sudden breathing difficulty, hemoptysis or blood stasis, cough, and shortness of breath. There are no significant differences could be harvested ($P>0.05$) (Table 5). In clinical signs study, we have studied tachycardia, blood pressure drop, myocardial infarction or ischemia, renal failure, and abnormal liver function. The results indicated that no differences between these six indexes could be detected between the two groups ($P>0.05$) (Table 6). Moreover, we have studied the MDCT features between the two groups (Table 7). The results revealed that no differences could be harvested in maximum diameter of ascending aorta (mm), aortic dissection range, pleural effusion/blood, and pericardial effusion/blood. CT images

with representative case presentations were shown in Figure 1 and Figure 2. However, patients number related to branches of the brachiocephalic and ventral branch in case group was significantly higher than that in the control group ($P < 0.001$). This result indicated that the main branch of the aorta was an important indicator for HPS patients in the case group.

Univariate logistic regression analysis

In this study, HPS was used as the dependent variable Y. Meanwhile, all other factors were used as the independent variable X. We performed the univariate logistic regression analysis. The results suggested that no relationships could be retrieved between age, gender, hypertension and HPS ($P > 0.05$). However, the CCI value in the case group was lower than that in the control group ($P = 0.077$). Moreover, we had also calculated the relationship between other MDCT features and HPS. The results suggested that close relationships could only be detected between branches of the brachiocephalic and HPS (Table 8). The results mentioned above revealed that branches of the brachiocephalic and CCI value were two risk factors for HPS patients ($P < 0.1$).

Multivariate logistic regression analysis of HPS

Based on the results of univariate logistic regression analysis, two variables with $p < 0.1$ (CCI, branches of the brachiocephalic) were selected for multivariate logistic regression analysis. To avoid the false negative variables in multivariate analysis, $P < 0.1$ is considered to be related to HPS in multivariate logistic regression analysis. Enter method was used in multivariate logistic regression analysis. HPS was considered as the dependent variable with a value of 1. Meanwhile, no HPS as the dependent variable assigned a value of 0. Multi-factor conditional logistic regression analysis was carried out with a 1:2 match ratio (case group: control group) at the test level of $\alpha = 0.05$. Multivariate logistic regression analysis of variable names and assignment instructions were listed in Table S3. The results suggested that branches of the brachiocephalic are an independent risk factor for HPS (OR=7.02, 95%CI=1.28-38.62, $p = 0.025$) (Table 9).

Discussion

In this study, the average age of 188 patients with acute Stanford-A AD was 59.0 years old. Although the onset age of acute AD patients continued to increase with the aging of cardiovascular and cerebrovascular diseases and the aging of the population, the overall average age did not change much. The minimum age of this group of patients was 29 years old, and the maximum age was 78 years old (Average age=59.0 years). This is consistent with previous research results (64 ± 14.1 years) [14]. Men are more likely to develop acute Stanford-A AD than women. In this study, Male to female ratio was 2.61:1. Males accounted for 72.3% of all patients. The international related acute AD database (IRAD) had reported that male acute Stanford-A AD patients accounted for 2/3 of all acute Stanford-A AD patients. The results in this study were adopted with the literature reports [31]. It is speculated that the high incidence rate of acute Stanford-A AD in the male may be related to the bad habits of men such as smoking and drinking. Moreover, the clinical manifestations of patients with AAD are diverse. The

literature reported that sudden chest pain or back pain (96%) is the most common clinical symptom. The pain is continuous tearing, piercing, and knife cutting [32]. IRAD had studied a large sample size of AAD. The work (2932 cases) suggested that 85% of type A AAD clinical symptoms were chest pain [14]. In this study, 53 patients presented with chest pain (98.1%), which was higher than the results reported, which may be caused by the small sample size.

Multiple risk factors could be related to AD, including hypertension, trauma, dyslipidemia, iatrogenic injury, pregnancy, drugs, smoking, chronic renal insufficiency, chronic obstructive pulmonary disease, stroke, Marfan syndrome, etc [5, 6, 14]. Hypertension is the most important risk factor for AD (77% of AD patients with hypertension) [14]. Hypertension could lead to changes in arterial hemodynamics, increased aortic insufficiency, and increased pressure in the arterial wall. Meanwhile, hypertension could promote the release of matrix metalloproteinases and inflammatory factors, causing excessive degradation of the extracellular matrix and altering the morphology of collagen fibers in the aortic wall. In this study, 52 patients (96.3%) with Stanford-A AD had a history of hypertension, which was higher than the results of previous studies (70%) [33]. We speculated that it could be related to the high incidence of hypertension in China. Meanwhile, a variety of underlying diseases can affect the development of AD, such as chronic renal insufficiency, chronic obstructive pulmonary disease, infectious vascular disease, and so on. In this study, the CCI score was used to reflect the overall underlying disease status. The CCI index of the case group was higher than that of the control group. There was a statistically significant difference between the two groups. Univariate analysis showed that the CCI score was one of the risk factors for HPS. However, CCI was not an independent risk factor for HPS.

The formation and development of aortic dissection are associated with increased arterial blood flow pressure. Muta et al. suggested that pulsating blood flow pressure could repeatedly act on the edge of AD dissection, leading to AD expansion. Therefore, pulsating blood flow pressure is the most important factor affecting AD [34]. In this study, CCI and branches of the brachiocephalic were included in multivariate analysis. Hypertension and the maximum diameter of the ascending aorta had not been included in this study. Previous work indicated that aortic wall tension was increased when the blood pressure was increased. The higher the blood pressure of AD patients, the more likely the aortic rupture occurs. However, in B-type AD patients, there is no evidence about the relationships between higher blood pressure and aortic rupture based on the multi-factor analysis [35]. Although hypertension was the cause of AD, it was not necessarily a risk factor for AD rupture. We speculated that the fluctuations in blood pressure may have a greater impact on dissection rupture. Theoretically, the larger the aortic diameter, the more easily the aortic dissection was broken. However, the CT three-dimensional image study of AD patients showed that the higher ratio of the maximum diameter of the aortic dissection to the length of the dissection, the greater the risk of interlayer rupture [36]. In this study, no significant differences in the maximum diameter of the ascending aorta and the extent of involvement of the aortic dissection could be observed between the case group and the control group. The ratio of the maximum diameter of the aortic dissection to the length of the dissection had not been included in the study. Therefore, the correlation between the maximum diameter of the ascending aorta and HPS remains to be further studied.

The proportion of patients with involvement of branches of the brachiocephalic in the case group was higher than that in the control group (33.30% for the case group and 8.33% for the control group). There was a statistically significant difference between the two groups ($p < 0.001$). Univariate analysis showed CCI and branches of the brachiocephalic were closely related to HPS. Multivariate analysis showed that the involvement of branches of the brachiocephalic was an independent risk factor for HPS compared with the involvement of the ventral branch. Chang et al. showed that the degree of involvement of the main branches of the aorta can indirectly reflect the intracavitary tension and can indicate the risk of AD [37]. The involvement of the branches of the brachiocephalic may change the intra-orbital tension which could further cause increased blood flow impact and rupture of the ascending aorta. The results of this study showed that the risk of HPS involvement in the branches of the brachiocephalic was 7.02 times higher than that of the ventral branch, which may be due to the highest anatomical location of the brachiocephalic artery to ascending aorta. However, the relationships between the branches of the brachiocephalic number, brachiocephalic artery, left common carotid artery, left subclavian artery, and the HPS has not been fully demonstrated. Therefore, further study was necessary to conduct in related areas.

Conclusions

In summary, univariate analysis showed that CCI and branches of the brachiocephalic were the two major risk factors for HPS associated with acute Stanford-A AD. Meanwhile, multivariate analysis revealed that branches of the brachiocephalic were an independent risk factor for HPS.

Abbreviations

HPS: hemorrhagic pulmonary sheath; CCI: Charlson comorbidity index; AAS: Acute aortic syndrome; AAD: acute aortic dissection; MDCT: Multi-detector computed tomography; CTA: Computed tomographic angiography;

Declarations

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Authors' contributions

Qiuxia Xie: Study design, concept, writing; Haoling Qin and Ling Lin: Data collection, data analysis; Jian Guan, Xuhui Zhou: Corresponding author; Xuhui Zhou: Review the data organization; Jian Guan: Review the data management. All authors read and approved the final manuscript.

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Availability of data and materials

The datasets generated during the present study are not publicly available, because detailed clinical information of each participant was included in these materials.

Ethics approval and consent to participate

The study design was approved by the Ethics Committee of the Eighth Affiliated Hospital of Sun Yat-sen University (No: SunYat-sen-2011-KY-EAH-032; Aug. 8, 2011). Written informed consent was obtained from all participants. All procedures performed in studies involving human participants were following the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. Informed consent was obtained from all individual participants included in the study.

Consent for publication

Not applicable.

Competing interests

All authors declare no conflict of interest.

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Tables

Table 1 Gender composition of the case group and control group

Gender	Total (N=54)	Case (N=18)	Control (N=36)	P-value
Male	39	13	26	1.00
Female	15	5	10	

Table 2 Age composition of the case group and control group

Age	Total (N=54)	Case (N=18)	Control (N=36)	P-value
Year	60.0*	60.0	59.5	0.762
	(54.0,67.0)#	(55.0,70.0)	(53.5,65.0)	

* Median value; # (The first quartile value, the third quartile value)

Table 3 Clinical information of the case group and control group

Diseases	Total (N=54)	Case (N=18)	Control (N=36)	χ^2 value	P- value
Hypertension	50 (92.6%)	16 (88.9%)	34 (94.4%)	1.607	0.205
Diabetes Mellitus	10 (18.5%)	3 (16.7%)	7(19.4%)	0.136	0.713
Chronic cardiovascular and cerebrovascular diseases	17 (31.5%)	5 (27.8%)	12 (33.3%)	0.590	0.443
Chronic pulmonary disease	6 (11.1%)	2 (11.1%)	4 (11.1%)	-	1.000
Moderate/moderate kidney damage	11 (20.37%)	4 (22.2%)	7 (19.4%)	0.276	0.599
Connective tissue diseases	7 (13.0%)	3 (16.7%)	4 (11.1%)	1.495	0.221
Peripheral vascular diseases	11 (20.4%)	4 (22.2%)	7 (19.4%)	0.276	0.599
Mild liver damage	5 (9.3%)	2 (11.1%)	3 (8.3%)	0.235	0.628
Cancer	2 (3.7%)	1 (5.6%)	1 (2.8%)	0.974	0.324

Table 4 Charlson comorbidity index (CCI) comparison between the case group and the control group

CCI	case group	control group	Z/c ² value	P-value
Median	2* (1, 3)#	1 (1, 1.5)*	1.062	0.043
0	3 (16.67)	8 (22.2)	39.994	<0.001
1	5 (27.78)	19 (52.78)		
2	4 (22.22)	6 (16.67)		
3	6 (33.33)	1 (2.78)		
4	0 (0.00)	2 (5.56)		

* Median value; # (The first quartile value, the third quartile value)

Table 5 Clinical symptoms comparison between the case group and the control group

Clinical symptom	Total (N=54)	Case (N=18)	Control (N=36)	P-value
Chest pain	53 (98.15%)	18 (100%)	35 (97.22%)	0.320
Syncope	5 (9.26%)	2 (11.11%)	3 (8.33%)	
Sudden breathing difficulty	3 (5.56%)	2 (11.11%)	1 (2.78%)	
Hemoptysis or blood stasis	2 (3.70%)	1 (5.56%)	1 (2.78%)	
Cough	2 (3.70%)	1 (5.56%)	1 (2.78%)	
Shortness of breath	5 (9.26%)	2 (11.11%)	3 (8.33%)	

Table 6 clinical signs comparison between the case group and control group

Clinical signs	Total (N=54)	Case (N=18)	Control (N=36)	P-value
Tachycardia	5 (9.26%)	2 (11.11%)	3 (8.33%)	0.303
Blood pressure drop	1 (2.78%)	0 (0.00%)	1 (2.78%)	
Myocardial infarction or ischemia	4 (7.41%)	1 (5.56%)	3 (8.33%)	
Renal Failure	8 (14.81%)	3 (16.67%)	5 (13.89%)	
Abnormal liver function	2 (3.70%)	1 (5.56%)	1 (2.78%)	

Table 7 MDCT features comparison between the case group and control group

MDCT features	Subgroup	Case (N=18)	Control (N=36)	P- value
Maximum diameter of ascending aorta (mm)	-	50.5* (44.0, 68.0)#	49.0* (38.0, 85.0) #	0.321
Aortic dissection range	Ascending aorta	2 (11.11)	6 (16.67)	0.367
	Thoracic aorta	5 (27.78)	8 (22.22)	
	Lower abdomen aorta	11 (61.11)	22 (61.11)	
Main branch of aorta	No	5 (27.78)	14 (38.89)	<0.001
	Branches of the brachiocephalus	6 (33.33)	3 (8.33)	
	Ventral branch	7 (38.89)	19 (52.78)	
Pleural effusion/blood	No	9 (50.00)	16 (44.44)	0.204
	Pleural blood	0 (0.00)	1 (2.78)	
	Pleural effusion	9 (50.00)	19 (52.78)	
Pericardial effusion/blood	No	2 (11.11)	5 (13.89)	0.268
	Pericardial blood	12 (66.67)	20 (55.56)	
	Pericardial effusion	4 (22.22)	11 (30.55)	

* Median value; # (The first quartile value, the third quartile value)

Table 8 Univariate logistic regression analysis of MDCT features and HPS

Item	Subgroup	B	S.E,	Walds	df	P	OR	95%CI
Age	-	0.006	0.027	0.054	1	0.817	1.006	0.954-1.061
Gender	-	0.000	0.645	0.000	1	1.000	1.000	0.283-3.537
CCI	-	0.499	0.282	3.119	1	0.077*	1.647	0.947-2.864
Hypertension	-	-0.169	0.706	0.057	1	0.811	0.845	0.212-3.372
Maximum diameter of ascending aorta (mm)	-	0.005	0.032	0.023	1	0.879	1.005	0.943-1.070
Main branch of aorta	Brachiocephalus	1.838	0.830	4.910	1	0.027*	6.286	1.236-31.956
	No	0.357	0.692	0.266	1	0.606	1.429	0.368-5.548
Aortic dissection range	Ascending aorta	1.504	1.208	1.551	1	0.213	4.499	0.422-47.979
	Thoracic aorta	1.099	1.141	0.926	1	0.336	3.000	0.320-28.098
Pleural effusion/blood	Pleural effusion	-0.172	0.581	0.088	1	0.767	0.842	0.270-2.629
	Pleural blood	-12.79	797.1	0.000	1	0.987	<0.001	<0.001->999.999
Pericardial effusion/blood	Pericardial effusion	0.598	1.241	0.232	1	0.630	3.250	0.340-31.073
	Pericardial blood	1.179	1.152	1.047	1	0.306	1.818	0.160-20.713

Table 9 Multivariate logistic regression analysis of MDCT features and HPS

Item	B	S.E,	Walds	df	P	OR	95%CI
Age	-0.018	0.033	0.280	1	0.597	0.982	0.920-1.049
Gender	0.362	0.846	0.183	1	0.669	1.436	0.274-7.536
CCI	0.591	0.318	3.461	1	0.063	1.805	0.969-3.364
Main branch of aorta	-	-	5.038	2	0.081	-	-
Without main branch of aorta	0.658	0.763	0.744	1	0.388	1.932	0.433-8.621
Branches of the brachiocephalus	2.038	0.909	5.030	1	0.025#	7.02*	1.28-38.62

After adjusting for CCI, multivariate analysis showed that the branch of brachiocephalus in case group was different from that in the control group.* The risk of HPS is greater in the involvement of branches of the brachiocephalus than that in the ventral branch (OR=7.02).

Figures

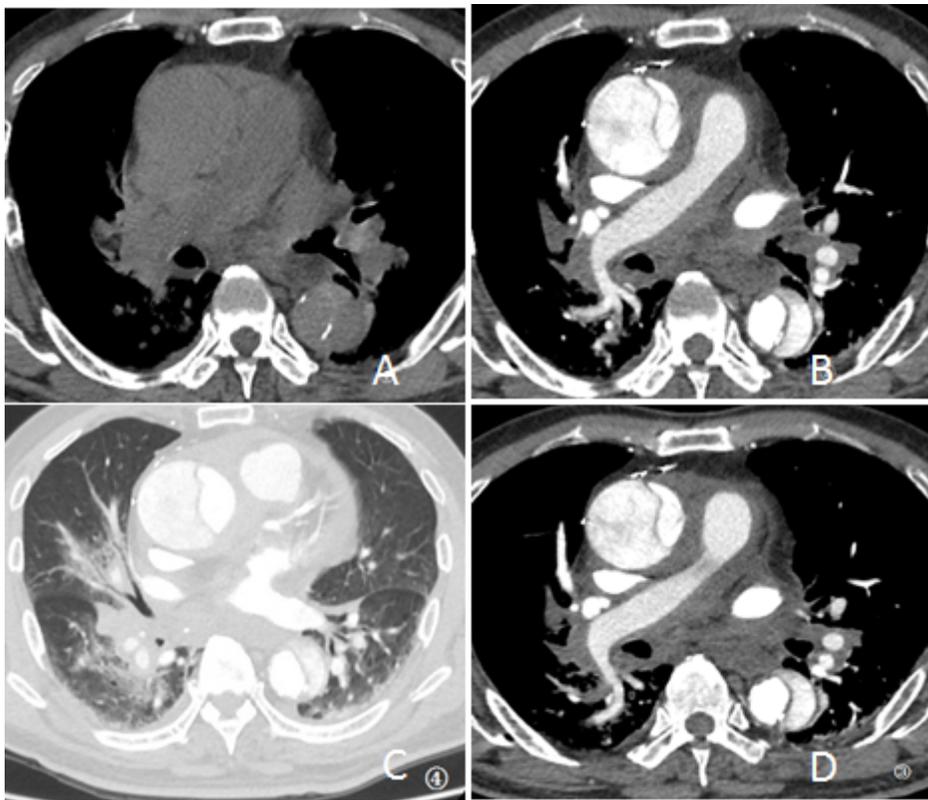


Figure 1

CT image of acute aortic dissection with type II hemorrhagic pulmonary sheath (74 years old, male) A) CT plain scan images showed high-density circular images of the right pulmonary artery. B) CTA images showed no enhancement in the ring-shaped soft high-density area of the right pulmonary artery, and HPS caused stenosis of the right pulmonary artery, the degree of stenosis was <50%. C) HPS is distributed

through the hilar area along the branches of the right pulmonary artery and the bilateral pulmonary artery hilar area. D) Lung glass shadow around the right bronchial vascular sheath.

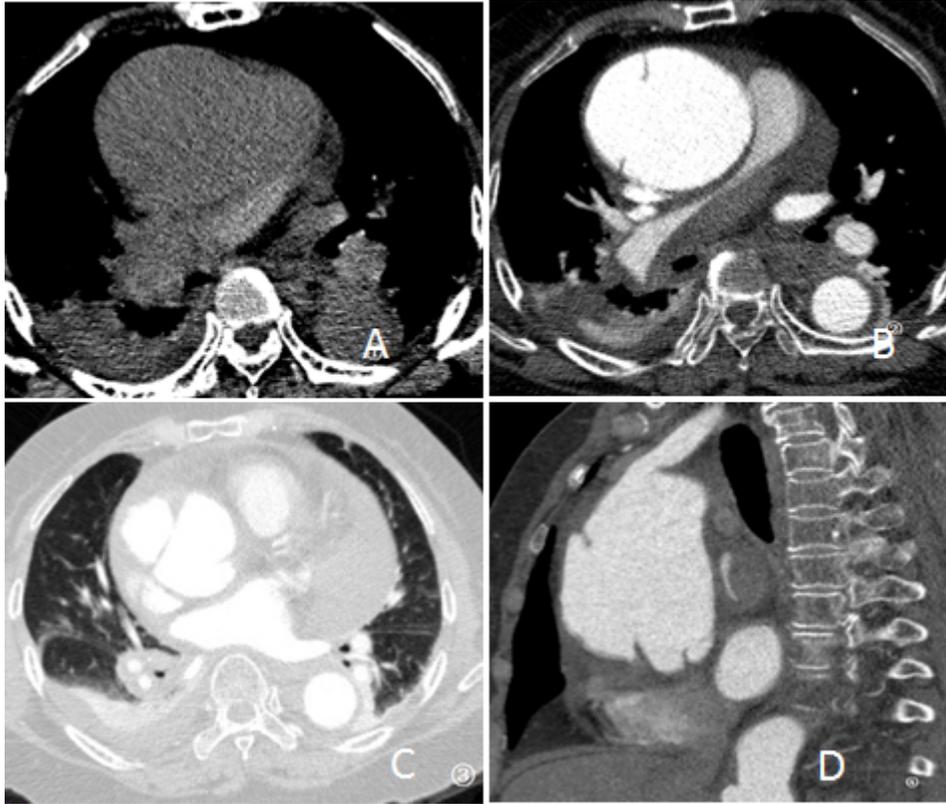


Figure 2

CT image of acute aortic dissection with type II hemorrhagic pulmonary sheath (74 years old, female) A) CT plain scan image shows a crescent-shaped high-density shadow of the right pulmonary artery. B) CTA images showed that there was no enhancement in the high-density area of the right pulmonary artery. HPS caused the right pulmonary artery to stenosis with a degree of stenosis > 50%. C) The lungs around the affected right pulmonary artery branch are clear. D) CTA sagittal images showed that HPS caused severe stenosis of the right pulmonary artery, the degree of stenosis was > 50%.

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