

The value of CT pulmonary angiography to the diagnosis of right ventricular dysfunction due to acute pulmonary embolism: compared with ultrasonographic cardiography[☆]

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Abstract

Objective: To analyze the value of CT pulmonary angiography (CTPA) in assessing right ventricular dysfunction (RVD) after acute pulmonary embolism. **Methods:** Thirty-six patients with CTPA-confirmed PE who underwent ultrasonic cardiography (UCG) within the ensuing 24 hours were retrospectively reviewed. According to the severity of the disease, the patients were divided into the massive PE group (24 cases) and non-massive PE group (12 cases) respectively. CT scans were analyzed for findings suggestive of RVD. Scans were considered positive for RVD if the right ventricle was dilated ($RV_d/LV_d > 1$) or if the interventricular septum was straightened or deviated towards the left ventricle. Results were then compared with the results of UCG to estimate the value of CTPA in detecting RVD associated with PE. **Results:** In all cases, compared with UCG, the diagnostic sensitivity, specificity, positive likelihood ratio, negative likelihood ratio, positive predictive value, and negative predictive value of CTPA was 84.61%, 78.26%, 3.892, 0.197, 68.75% and 90% respectively. Kappa value was 0.60, which suggested moderate agreement between CTPA and UCG in the whole level. In the massive PE group, the diagnostic sensitivity, specificity, positive likelihood ratio, negative likelihood ratio, positive predictive value, negative predictive value of CTPA was 84.61%, 72.73%, 3.103, 0.212, 78.57% and 80% respectively. Kappa value was 0.58, which suggested moderate agreement between CTPA and UCG in the massive PE group. In the non-massive PE group, the diagnostic specificity of CTPA was 83.33%. By statistics, the value of RV_d/LV_d had significant difference between the massive PE and the non-massive PE group. **Conclusion:** CTPA can reliably detect RVD through the evaluation of cardiac morphology. However, this result requires confirmation using a larger prospective cohort study.

Key words: pulmonary embolism; tomography; X-ray computed; angiography

INTRODUCTION

Pulmonary embolism (PE) is a common and death-

threatening disease. Mortality is thought to be caused in part by acute pulmonary arterial hypertension caused by PE, which initially results in right ventricular dysfunction (RVD), and may progress to right ventricular failure and circulatory collapse^[1]. Patients with RVD after PE have a higher mortality rate than those with normal right ventricular function even if hemodynamically stable at presentation^[2-4]. Therefore, early detection of RVD and prompt thrombolysis therapy in

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patients with PE is very important for their prognosis.

Ultrasonic cardiography(UCG) is recommended as a first-line examination to diagnose the signs of RVD. However, computed tomographic pulmonary angiography(CTPA) has progressively been established as the frontline imaging model for the diagnosis of PE. CTPA can also depict the ventricular size and the position of the interventricular septum, allowing for the evaluation of RVD^[5].

The primary goal of this investigation was to determine the diagnostic value of CTPA to RVD compared with the results of UCG.

MATERIALS AND METHODS

Patients

Thirty-six cases of CTPA-confirmed pulmonary embolism presented between June 2002 and December 2004 in Beijing Chaoyang Hospital were retrospectively analyzed. In all cases UCG was done within 24 hours of the initial CTPA. The 36 patients included 23 men and 13 women(mean age 58.38 years; range from 29 to 73 years). According to the clinics and pulmonary artery obstructive area, the cases were divided into the massive pulmonary embolism group(MPE) and the non-massive pulmonary embolism group(NMPE). Massive pulmonary embolism group consisted of shock and/or hypotension(defined as a systolic blood pressure < 90 mmHg or a pressure drop of =40 mmHg for > 15 min if not caused by new-onset arrhythmia, hypovolemia or sepsis)^[6], or the obstructed arteries=2 lobes or=7 segments. Otherwise non-massive pulmonary embolism was diagnosed. After grouping, there were 24 cases in the MPE group(15 male and 9 female, 29-73 years, average age 60.25 years) and 12 cases in the NMPE group (8 male and 4 female, 35-71 years, average age 59.5 years).

CTPA

Spiral CTPA was performed using a GE Highspeed spiral CT scanner(General Electron, dual-row detector) during intravenous administration of 80-100 ml of Omnipaque(Nycomed) at 3.5 to 4 ml/sec. Imaging was performed after 12-15-second delay using 2-mm collimation and 0.7-s gantry rotation time. Subsequently, 1-mm reconstructions were performed and used for the final interpretation, which was done on the workstation. CTPA was considered positive for RVD if the RV was dilated and/or the interventricular septum was deviated towards the LV^[5,7,8](Fig. 1,2). The widths of the ventricular cavities were assessed on a single axial image obtained at the plane of maximal visualization of the ventricular cavities. The right ventricle was considered dilated if the cavity was wider than the left ventricular cavity along the short axis. The interventricular septum

was considered deviated towards the left ventricle if the left ventricular border was convex towards the left ventricle.

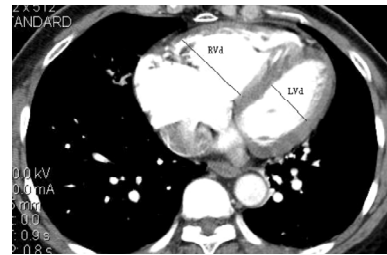


Fig. 1 Massive pulmonary embolism. The right ventricle was larger than left ventricle($RV_d/LV_d > 1$). The interventricular septum was straightened.

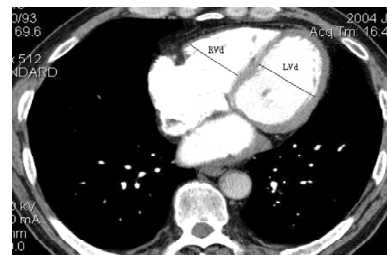


Fig. 2 Non-massive pulmonary embolism. The right ventricle was smaller than left ventricle($RV_d/LV_d < 1$). The interventricular septum was normal.

Ultrasonographic cardiography

Two-dimensional transthoracic UCG was performed using parasternal short axis, parasternal long axis, subcostal short axis, and apical four-chamber views. UCG was considered positive for RVD if any of the following characteristic findings were present: RV dilatation, RV hypokinesis, interventricular septal flattening, or paradoxical motion of the interventricular septum^[5,9].

Statistical analysis

The diagnostic sensitivity, specificity, positive likelihood ratio, negative likelihood ratio, positive predictive value, negative predictive value of CTPA was calculated according to the results of UCG. Kappa analysis was used to assess the diagnostic consistency between CTPA and UCG. Using the non-parametric Mann-Whitney Test, we calculated the RV_d/LV_d difference of CTPA and UCG between the MPE and NMPE group respectively. A *P* value less than 0.05 was considered statistically significant. Statistical analysis was performed with a statistical software system SPSS 13.0.

RESULTS

The number of cases that was positive and negative for RVD on CTPA and UCG was listed in Table 1-3 separately according to different group: Table. 1 showed all cases, Table 2 showed the MPE group, and Table 3

the NMPE group. The diagnostic values of CTPA to RVD were listed behind the corresponding table. The RV_d/LV_d value of the MPE and NMPE group was listed in Table 4.

Table 1 Results of 36 cases of pulmonary embolism

CTPA	UCG		TOTAL
	+	-	
+	11	5	16
-	2	18	20
TOTAL	13	23	36

Compared with UCG, the diagnostic sensitivity, specificity, positive likelihood ratio, negative likelihood ratio, positive predictive value, and negative predictive value of CTPA was 84.61%, 78.26%, 3.892, 0.197, 68.75% and 90% respectively. Kappa value was 0.60.

Table 2 Results of 24cases of massive pulmonary embolism

CTPA	UCG		TOTAL
	+	-	
+	11	3	14
-	2	8	10
TOTAL	13	11	24

Compared with UCG, the diagnostic sensitivity, specificity, positive likelihood ratio, negative likelihood ratio, positive predictive value, and negative predictive value of CTPA was 84.61%, 72.73%, 3.103, 0.212, 78.57% and 80% respectively. Kappa value was 0.58.

Table 3 Results of 12 cases of non-massive pulmonary embolism

CTPA	UCG		TOTAL
	+	-	
+	0	2	2
-	0	10	10
TOTAL	0	12	12

Compared with UCG, the diagnostic specificity was 83.33%.

Table 4 RV_d/LV_d of the MPE and NMPE group

GROUP	RV_d/LV_d	
	CTPA	UCG
MPE	1.38	1.11
NMPE	0.87	0.76

Of CTPA and UCG, there was significant difference for RV_d/LV_d between the MPE and NMPE group ($P = 0.000$).

DISCUSSION

Mortality in untreated pulmonary embolism is approximately 30%, but with adequate treatment, it can be reduced to 2-8%. Mortality is usually due to circulatory failure from right ventricular dysfunction and failure, which always happens within the first hours of onset.

The obstruction of pulmonary arteries, release of vasoconstricting agents, and hypoxemia together increase pulmonary vascular resistance and pulmonary arterial pressure. Sudden increase in RV afterload results in elevated right ventricular wall tension, RV dilatation, and eventually RVD. Dilatation of the right ventricle causes the interventricular septum to shift

towards the left ventricle, which results in decreased left ventricular diastolic volume. Right ventricular contractile dysfunction and acute tricuspid regurgitation cause decreased output from the right ventricle, also contributing to underfilling of the left ventricle. Underfilling of the LV results in decreased cardiac output, systemic blood pressure and perfusion, which result in ischemia of the right ventricular myocardium. This produces worsening of RVD and the potential for a vicious cycle of ventricular hypoxia, right ventricular dilatation, and decreased cardiac output progressing to cardiogenic shock and circulatory collapse^[2,9,10].

Although ultrasonographic cardiography can not diagnose or exclude the diagnosis of PE in most cases, it has long been taken as a rapid, practical and sensitive model for the evaluation of RVD associated with PE. However, UCG is not routinely performed in all patients with PE. It also is an operator-dependent model, and certain patient characteristics such as obesity and respiratory distress may not permit an optimal study.

CTPA provides a means for direct visualization of pulmonary artery thrombi, as well as simultaneous assessment of the lung parenchyma, pleura, and great vessels. Increasingly available multidetector-row CT scanners have shortened the examination time, improved temporal resolution, allowed visualization of segmental and subsegmental vessels and evaluation of RVD.

Using the diagnostic standard of previous investigators^[5,7,8], we found that in the 36 cases of PE, the diagnostic sensitivity and specificity of CTPA to RVD compared with UCG was 84.61% and 78.62% respectively. Contractor *et al.* correlated CTPA findings of RVD with the results of UCG, and reported that CTPA had moderate sensitivity (78%) and high specificity (100%) for detection of RVD^[5]. Quiroz *et al.*^[11] also suggested that RVD could be diagnosed using CTPA. However, the investigators used CT-reconstructed imaging, which is time consuming and as not applicable in daily practice in an emergency department.

In the massive PE group, the diagnostic sensitivity and specificity of CTPA was 84.61% and 72.73% respectively. Lim *et al.*^[12] studied 14 cases of massive PE using the same diagnostic standard, and reported the diagnostic sensitivity of 91.6, specificity 100%, positive predictive value 100%, negative predictive value 67%. Our results were lower than theirs, maybe due to the different severity of the studying cases. However, the Kappa value=0.60 and 0.58 in all PE cases and the massive PE cases indicated moderate diagnostic agreement between CTPA and UCG, which suggested the acceptable diagnostic value of CTPA to RVD.

In our study, we found significant difference ($P = 0.000$) of the RV_d/LV_d value between the massive PE

group(1.38) and the non-massive PE group(0.87). Reid *et al* reported that in 7 cases of massive PE cases, the RV_d/LV_d was larger than 1.5, which was similar to our results. Wintersperger *et al.*^[13] also found that there was significant difference of the RV_d/LV_d value between the severe and non-severe PE cases.

We agree that the axial CTPA imaging can not accurately reflect the size of both ventricles and the position of the interventricular septum just like UCG. However, it is simple, rapid and comparably reliable for the evaluation of RVD, as our results show. However, there were comparably few cases in our study, so the diagnostic value of CTPA to RVD requires confirmation using a larger prospective cohort study.

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