Original Article

The role of thoracic ultrasonography in the diagnosis of pulmonary embolism

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Abstract:

OBJECTİVES: The diagnosis of pulmonary embolism (PE) is still a problem especially at emergency units. The purpose of study was to determine the diagnostic accuracy of thoracic ultrasonography (TUS) in patients with PE.

METHODS: In this prospective study, 50 patients with suspected PE were evaluated in Department of Pulmonary Diseases of a Training and Reasearch Hospital between January 2010 and July 2011. At the begining, TUS was performed by a chest physician, subsequently for definitive diagnosis computed tomography pulmonary angiography were performed in all cases as a reference method. Other diagnostic procedures were examination of serum d-dimer levels, echocardiography, and venous doppler ultrasonography of the legs. Both chest physician and radiologist were blinded to the results of other diagnostic method. Diagnosis of PE was suggested if at least one typical pleural-based/subpleural wedge-shaped or round hypoechoic lesion with or without pleural effusion was reported by TUS. Presence of pure pleural effusion or normal sonographic findings were accepted as negative TUS for PE.

RESULTS: PE was diagnosed in 30 patients. It was shown that TUS was true positive in 27 patients and false positive in eight and true negative in 12 and false negative in three. Sensitivity, specificity, positive predictive value, negative predictive value, and diagnostic accuracy of TUS in diagnosis of PE for clinically suspected patients were 90%, 60%, 77.1%, 80%, and 78%, respectively.

CONCLUSİONS: TUS with a high sensitivity and diagnostic accuracy, is a noninvasive, widely available, cost-effective method which can be rapidly performed. A negative TUS study cannot rule out PE with certainty, but positive TUS findings with moderate/high suspicion for PE may prove a valuable tool in diagnosis of PE at bedside especially at emergency setting, for critically ill and immobile patients, facilitating immediate treatment decision.

Key words:

Chest ultrasonography, diagnosis, pulmonary embolism

Pulmonary embolism (PE) is a frequently underestimated, underdiagnosed, and undertreated disease.[1] The incidance of PE in the United States is 23 to 69 per 1,00,000.[2] Many deaths occur in hemodynamically unstable patients and the estimated mortality for inpatients with hemodynamic instability is between 15% and 25%.[3] Timely diagnosis of PE is crucial because prompt appropriate managment can decrease mortality but is often confounded by nonspecific clinical pesentation.[4]

Moreover, there is virtually no single noninvasive diagnostic test, which is sufficiently sensitive for the diagnosis in all suspected cases.[4] The combination of clinical probability, ventilation-perfusion lung scanning, and lower extremity sonography has simplified the diagnostic approach. Although lung scanning, computed tomography pulmonary angiography (CTPA), and pulmonary angiography are important for the diagnosis, unfortunately they are not widespread enough and cannot be reached at the emergency units, also time factor is important. So, PE remain undiagnosed especially at the emergency units in the majority of patients, suggesting the need for alternative, easy, and widespread bedside diagnostic approaches.[5,6]

These questiones were asked by Mathis years ago. "What's up in emergency rooms or in intensive care units? How can we manage diagnostic procedures like CT with patients in a state of shock? Can transthoracic ultrasound improve the diagnostic imaging of PE?".[7]

The detection of thromboembolic lesions of the lung by thoracic ultrasonography (TUS) was first described 40 years ago.[1,8,9] These early reports were overlooked for many years. Mathis *et al.*,[1,7,10,11] reported their results more recently. Also, Reissig *et al.*,[12] suggested that transthoracic sonography of the lung and pleura may serve as additional method in the diagnostic workup of suspected PE.

There are number of criteria which can be applied in the diagnosis of PE. The most charactheristic finding in PE is hypoechoic, pleural-based paranchymal alteration. Greater than 85 of these lesions are wedge-shaped.[1,13] They may also

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have rounded or polygonal configuration. A single hyperechoic structure localized at the center of the lesion which indicates the presence of air-filled bronchiole may be detected in 20% of the patients.[1,13,14] Pleural involvement in PE initially leads to localized fluid collection adjacent to the affected pulmonary region and may eventually develop into a basal pleura effusion.[1,13] Exploration of lesions by color Doppler imaging may provide additional diagnostic information. In pulmonary infarction, pulmonary arterial flow cannot be detected by color Doppler ultrasound, referred to as "consolidation with little perfusion".[13,15] A congested thromboembolic vessel may be visible called "vascular sign".[10,13,16] These described TUS findings support the diagnosis of PE, but in the absence of them PE cannot be ruled out.

The aim of this study was to determine the sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and diagnostic accuracy of TUS for diagnosing the PE in patients with moderate to high suspicion of pulmonary emboli. We also evaluated and compared the symptoms, risk factors, chest X-ray findings, and pulmonary arterial systolic pressure of PE positive and negative groups.

Methods

Study design

Between January 2010 and July 2011, a total of 50 consecutive patients with moderate or high clinical suspicion of PE evaluated in Department of Pulmonary Diseases of a Training and Reasearch Hospital in Istanbul were enrolled in this prospective study. The main inclusion criteria were clinical suspicion of PE under consideration of risk factors. The risk factors were the presence of malignancy, lower extremity fracture, obesity, congestive heart failure, postpartum period, and history of venous thromboembolism, operation, and PE. In the presence of risk factors for PE, the presence of unexplained dyspnea, tachypnea, pleuritic pain, and unexplained radiological findings and blood gas abnormalities are accepted as high clinical suspicion. In the presence of risk factors for PE, presence of dyspnea or hypoxemia which can be explained by conditions other than PE or the presence of unexplained dyspnea or hypoxemia without risk factors for PE are accepted as moderate clinical suspicion.

Written informed consent was obtained from all patients and study protocol was approved by the local ethics committee.

Multislice CTPA was used as the reference method in diagnosis of PE. CT-angiography examinations were interpreted by radiologists who do not know the result of TUS. And, also TUS was performed by an experienced chest physician who do not know the result of CTPA. In addition, echocardiography, duplex sonography of bilateral lower extremity veins, serum d-dimer level, and arterial blood gasses measurements were performed in all patients. CTPA and echocardiography were performed within 24 hours to reduce the time factor of thrombolysis. Duplex sonography of lower extremity veins was conducted within 2 days. Plasma d-dimer levels were measured by a quantitative enzyme-linked immunosorbent assay.

Thoracic ultrasound

In case of a clinically suspected PE, chest ultrasound was performed prior to other imaging procedures. The chest ultrasound was performed with the patient in a sitting position, arms raised and hands placed at the back of the head in order to extend the intercostal spaces and rotate the scapula outward. TUS was performed by an experienced chest physician who had completed a postgraduate thoracic ultrasonography course and trained by a radiologist. GE Logic 7 ultrasound device with 3.5 MHz convex probe was used for sonographic examination. If the patient had chest pain, the physician began the sonographic examination from the painful area and intercostal areas were systematically examined in six vertical lines which were paravertebral, midscapular, posterior axillary, midaxillary, anterior axillary, and midclavicular. Patients were divided into five groups based on the following criteria of sonographic findings: (1) Two or more characteristic wedge-shaped, triangular, or rounded pleura-based hypoechoic lesions with or without pleural effusion; (2) One characteristic wedge-shaped, triangular, or rounded pleura-based hypoechoic lesions with pleural effusion; (3) One charactheristic wedge-shaped, triangular, or rounded pleura-based hypoechoic lesions; (4) Nonspecific subpleural lesions more than 5 mm in size or a free pleural effusion alone; and (5) Normal sonographic findings. The diagnosis of PE was suggested if at least one or more typical pleural-based/subpleural hypoechoic lesion with or without pleural effusion were reported by TUS (Groups 1, 2, and 3). In the presence of nonspecific subpleural lesions more than 5 mm in size, pure-free pleural effusion or normal sonografic findings, the diagnosis of PE was not supposed (Groups 4 and 5). Sonographic image of the lesion was frozen then the longitudinal and transverse axes were measured. A characteristic, pleural-based, wedge-shaped, hypoechoic infarct area with central hyperechoic bronchiolar pattern was given in Figure 1.

Multislice CT angiography

Standard contrast material enhanced multidetector CT was performed by using a 64-section (Aquilion 64; Toshiba Medical Systems) CT scanner, and 0.5 mm sections of the entire chest were acquired for the diagnosis or exclusion of pulmonary embolism. The rotation time was 0.5 second, tube current was 300-350 mA, and tube voltage was 120 kV. Acquisitions were performed during a single breath hold that lasted for 10-12 seconds or less, depending on the scanner type. A total

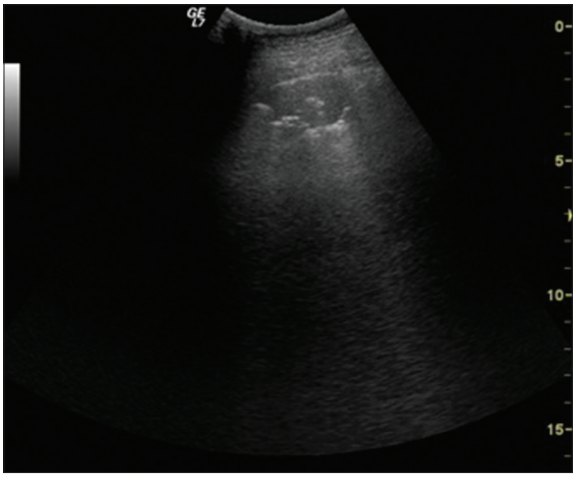

Figure 1: Pleural-based, wedge-shaped, hypoechoic infarct area with central hyperechoic bronchiolar pattern in the sonographic examination of a pulmonary embolism case

of 80-100 mL of contrast agent (Omnipaque 350, GE Healthcare, Irelad, Cork; or Iomeron 350, Pantheon, Italy) was injected into the antecubital vein with a intravenous catheter with integrated 3-way stop cock (Medicath, Gurgaron, India) at a rate of 4.0 mL/s. This injection was followed by a 40 mL saline bolus chaser, which was injected at a flow rate of 4.0 mL/s. Static pulmonary angiographic scanning was started after automated threshold enhancement detection in the main pulmonary artery. A threshold difference of 150 HU was selected for the start of a acquisition.

Data analysis

The diagnosis of pulmonary embolism was confirmed by the presence of at least one filling defect in the pulmonary artery. The static multidetector row CT scan was analyzed on a Vitrea workstation (Version 4.1.8.0; Vital Images).

The location of thrombus, the number and location of parenchymal lesions at CTPA in PE patients were recorded.

Statistical analysis

The sensitivity, specificity, PPV, NPV, and diagnostic value of the TUS in the diagnosis PE were calculated using the standard definitions. The data were statistically compared using Chi-square test and $P < 0.05$ was regarded as a significant difference. The SPSS for Windows software package version 17.0 (Chicago, Illinois, USA) was used for descriptive analyses and for the statistical analysis.

Results

In this prospective study, 50 patients with suspected PE were evaluated in the Department of Pulmonary Diseases between January 2010 and July 2011. Of these 50 patients, 27 (54%) were males and 23 (46%) were females. The mean age was found to be 54.1 ± 17.9 (max: 85;min: 19) years. Thirty-three (66%) of the included cases had chest pain, 30 (60%) had dyspnea, and 16 (32%) had hemoptysis at the admission. When the PE positive and negative groups were compared with each other, chest pain is significantly higher in PE positive group ($P < 0.05$). At least, one risk factor is present at 36 (72%) of the cases and as a risk factor deep venous thrombosis (DVT) is observed significantly higher in PE positive group ($P < 0.05$). Twenty-six (52%) of cases defined as high clinical suspicion, 24 (48%) as intermediate clinical suspicion. Thirty (60%) of the cases diagnosed as PE by CT angiography. Most common symptoms were chest pain and dyspnea in both PE positive and PE negative groups: 83.4% and 40%, 70% and 45%, respectively. Risk factors were determined in 76.7% of PE positive group and 65% in PE negative group. DVT of lower extremity was estabilished in 13 (43.4%) of PE positive group. Eleveted pulmonary artery systolic pressure (>36 mmHg) was determined with echocardiographic examination in 13 (43.4%) of PE positive group. The data of PE positive and negative groups and distribution of risk factors were given at Tables 1 and 2. Most common chest X-ray findings were consolidation (43.4%) in PE positive group and normal X-ray (50%) in PE negative group [Table 3].

Totally, 46 hypoechoic, triangular, or wedge-shaped, subpleural lesion and 18 pleural effusions were diagnosed sonographically in 30 PE positive cases (mean 2.14 lesions per patient; range: 1-4 lesions per patient). The average size of the lesions was 22.9 × 31.2 (min: 5 × 11; max: 49 × 60) mm. Ten patients (33.4%) had only one parenchymal lesion, whereas the remaining 20 (66.6%) patients had multiple lesions (parenchymal lesion(s) + pleural effusion). Majority of the lesions (73.4%) were determined in the posterior lower part of lungs also the majority of lesions (66.7%) were in the right lung. The distribution of lesions was given at Figure 2.

CTPA established thrombus at right lung in 14, at left lung in seven, and bilateral in 12 cases. Twelve thrombus was detected at right or left main pulmonary arteries, 23 at segmental level, and 10 at subsegmental level. Twenty-nine parenchymal lesions were detected and characteristics of the lesions were given at Table 4. Parenchymal window of thoracic CT and sonographic examination of pulmonary embolism case was

Table 1: Data of pulmonary embolism positive and pulmonary embolism negative groups

Findings	PE positive group *n* (%)	PE negative group *n* (%)	*P*
Hemoptysis	9 (30)	7 (35)	0.581
Dyspnea	21 (70)	9 (45)	0.082
Chest pain	25 (83.4)	8 (40)	0.002
Risk factors	23 (76.7)	13 (65)	0.368
Moderate risk	15 (50)	9 (45)	0.806
High risk	15 (50)	11 (55)	0.908
DVT	13 (43.4)	5 (25)	0.039
Eleveted PAPs	13 (43.4)	6 (30)	0.286

DVT = Deep venous thrombosis, PAPs = Pulmonary arterial systolic pressure, PE = Pulmonary embolism

Table 2: Distribution of risk factors in pulmonary embolism positive and pulmonary embolism negative groups

Risk factors	PE positive group *n* (%)	PE negative group *n* (%)	*P*
Malignancy	7 (23.4)	3 (15)	0.542
History of VTE	6 (20)	2 (10)	0.383
Fracture of lower extremity	3 (10)	1 (5)	0.559
History of operation	3 (10)	2 (10)	0.678
Postpartum	2 (6.7)	2 (10)	0.602
History of PE	2 (6.7)	2 (10)	0.602
Obesity	1 (3.4)	1 (5)	0.649
Congestive heart failure	-	2 (10)	0.048

PE = Pulmonary embolism, VTE = Venous thromboembolism

Table 3: Distribution of chest X-ray findings in pulmonary embolism positive and pulmonary embolism negative groups

Chest X-ray findings	PE positive group, *n* (%)	PE negative group, *n* (%)
Consolidation	13 (43.4)	4 (20)
Normal	2 (6.7)	10 (50)
Blunt sinus	6 (20)	2 (10)
Pleural effusion	4 (13.4)	2 (10)
Diaphragm elevation	2 (6.7)	1 (5)
Enlarged pulmonary artery	2 (6.7)	-
Oligemia	-	1 (5)
Lineer atelectasis	1 (3.4)	1 (5)

PE = Pulmonary embolism

given at Figure 3a and b. There were mean 1.0 (range: 0-3) lesions in CTPA per patient. In six (20%) of the PE positive cases although there is not any paranchymal lesion at CTPA, TUS examination detected sonographic parenchymal lesions of PE [Table 5].

Distribution of sonographic findings (Group 1-5) in PE positive and negative groups was given in Figure 4. TUS was true positive in 27 (54%), true negative in 12 (24%), false positive in eight (16%) and false negative in three (6%) cases. The sensitivity, specificity, NPV, PPV, and diagnostic value of TUS in clinically suspicious (moderate-high) PE cases were presented as 90%, 60%, 80%, 77.1%, and 78%, respectively.

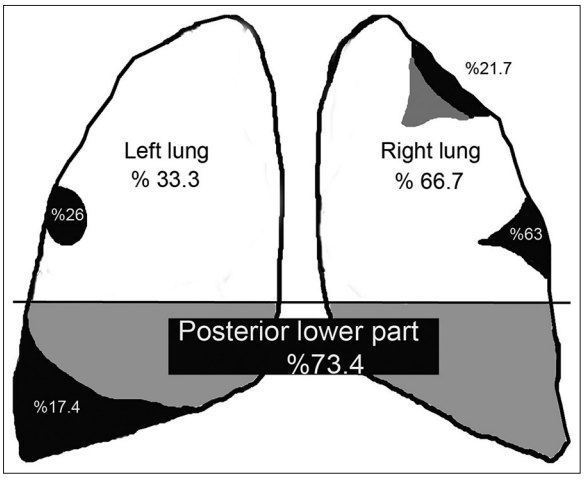

Figure 2: Distribution of lesions detected by transthoracic ultrasound

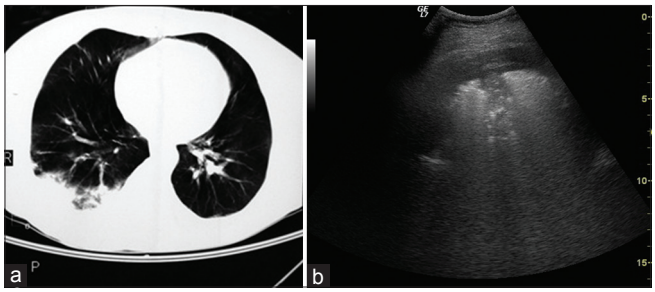

Figure 3: (a and b) Parenchymal window of thoracic computed tomography and sonographic examinations of pulmonary embolism case. Pleural-based, irregular-circumscribed, hypoechoic lesion with local pleural effusion at sonographic examination

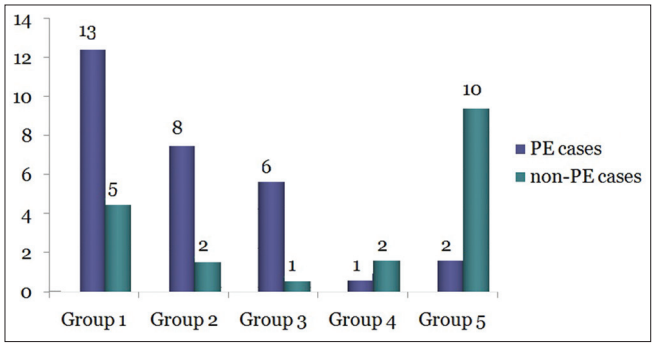

Figure 4: Distribution of groups based on sonographic findings in pulmonary embolism and nonpulmonary embolismcases

According to the TUS findings when Groups 1,2, and 3 were discussed, in the presence of two or more subpleural, characteristic, hypoechoic lesions (only Group 1) the sensitivity of TUS for diagnosing PE was 43.3%, specificity was 75%. Whereas in addition to two or more hypoechoic lesions (Group 1), one lesion together with localized pleural effusion (Group 2) were accepted as PE, the sensitivity increases to 70% and specificity decreases to 65%. If only one characteristic lesion without pleural effusion (Group 3) was also accepted as PE sensitivity increases significantly up to 90% but specificity decreases to 60% [Table 6]. Negative predictive value was 80%, if Groups 1, 2, and 3 were considered together, 59% if Groups 1 and 2 were considered and 46.8% if only Group 1 was taken into consideration.

Discussion

In this study, the aim was to determine the role of bedside TUS for diagnosing the PE in patients with moderate/high clinical suspicion of PE, we concluded that pathological lesions such as consolidation, atelectasis, and local pleural effusion can be identified by TUS easily and TUS is a safe, cheap, and available method for the early diagnosis and treatment decision of PE.

Table 4: Characteristics of paranchymal lesions of computed tomography pulmonary angiography

Lesion	*n*
Pleural-based lesion	8
Bilateral consolidation	5
Left-sided consolidation	5
Localized pleural effusion	3
Athelectasis	5
Right-sided consolidation	2
Graound glass opacity	1

CTPA = Computed tomography pulmonary angiography

Table 5: Type of the lesions identified by transthoracic ultrasound and computed tomography pulmonary angiography

Lesion type	Transthoracic ultrasound *n* (%)	CTPA *n* (%)
Wedge-shaped lesions	29 (63.0)	12 (41.4)
Rounded lesions	12 (26.0)	5 (17.2)
Other parenchymal lesions	5 (10.8)	6 (20.7)
Localized pleural effusion	10 (21.7)	3 (10.3)
Basal pleural effusion	8 (17.4)	3 (10.3)

CTPA = Computed tomography pulmonary angiography

Table 6: The distribution of pulmonary embolism cases into the three transthoracic ultrasound groups evaluated in favor of pulmonary embolism and sensitivity, specificity, and diagnostic value for each group

Group	*n*	Sensitivity (%)	Specificity (%)	NPV (%)	PPV (%)	Diagnostic value (%)
Group 1	13	43.3	75	46.8	72.2	56
Group 1+2	21	70.0	65	59.0	75.0	68
Group 1+2+3	27	90.0	60	80.0	77.1	78

NPV = Negative predictive value, PE = Pulmonary embolism, PPV = Positive predictive value, TUS = Transthoracic ultrasound

Despite the new improvements in technology such as multislice CTPA, since it is costly and cannot be available at every medical center also it is associated with potentially harmful radiation and application of contrast medium, diagnosing PE still remains a significant medical problem especially at the emergency departments. On the contrary, accurate diagnosis and early treatment of PE is important and potantially life-saving.[13] The decision about the PE suspected cases need to be made in real time and the time for making the decision is short.

Thromboembolic occlusion of pulmonary artery causes intraalveolar hemorrhage, necrosis, atelectasis due to loss of surfactant, increased permeability because of mediator secretion and alveolar edema and these changes occur mostly in the subpleural area of lung periphery. These patological situations, whether or not pleural effusion is present, provide an ultrasonographic window. The early formation of these lesions within minutes, makes it possible to be identified with ultrasound in the early period.[13,17]

In our study, TUS detected 64 lesions (mean: 2.14 lesions per patient), whereas CTPA detected 29 parenchymal lesions (mean: 1.0 lesions per patient) in 30 PE patients. Also in six (20%) cases although there is not any paranchymal lesion at CTPA, TUS examination detected sonographic parenchymal lesions of PE. But CTPA detected PE in three patients which TUS was negative. Pfeil *et al.*,[18] reported 73 parenchymal findings (1.60 ± 1.58 [right] 1.60 ± 2.07 [left] lesions per patient) by TUS and 149 parenchymal findings (3.20 ± 2.39 [right] 2.30 ± 1.77 [left] lesions per patient) by CTPA in 10 PE patients of 33. In their study, CTPA visualized PE in three patients with a negative result from TUS and TUS visualized PE in seven patients with a negative result from CTPA.[18] Reissig *et al.*,[1] detected 91 peripheral lesions (2.6 lesions per patient) by TUS in 69 patients in which 44 had experienced PEs. Nine patients without lesions shown on TUS had central PEs detected by CT.[1] In the study of Mathis *et al.*,[2] in 144 of 194 patients TUS demonstrated 333 lesions (2.3 lesions per patient), while CTPA showed 215 peripheral parenhymal lesions (1.5 lesions per patients). Reissig *et al.*,[19] demonstrated 74 characteristic parenchymal lesions with TUS and 56 parenchymal lesions with CT at 39 PE cases. Better resolution of sonography in the subpleural region may explain the larger number of parenchymal lesions detected by TUS than CTPA.[2,17] Also if the time for CTPA examination exeeds 48 hours the parenchymal lesions may disappear. An embolic occlusion of a pulmonary artery initially leads to intraalveolar hemorrhage without necrosis on the first 2 days of PE. This hemorrhage may result in a complete pulmonary infarction with necrosis of alveolar walls in about 15% of all infarctions. This necrosis of the alvolar walls begins usually after 2 days. Infarction mostly remains incomplete and disappear completely within 2-4 days in healthy lungs. So after this period of time, it may not be possible to detect the incomplete infarcts which are the most common type.[17]

The average size of the lesions was 22.9 × 31.2 (min: 5 × 11; max: 49 × 60) mm in the present study. Reissig *et al.*,[1] informed that parenchymal lesions detected by TUS had an average size of 13.8 × 10.6 mm, also Mathis *et al.*,[2] reported the average size of ultrasound lesions 15.5 × 12.4 mm. While the lesions on CTPA were 19.9 × 16.9 mm in size, the size of lesions detected by TUS was greater than reported at the literature.

In this study, majority of the lesions (73.4%) were determined in the posterior lower part of lungs also the majority of lesions (66.7%) were in the right lung. Also in our study, there were no lesions in the anterior parts of the lungs. Mathis *et al.*,[2] reported that the majority (66%) of lesions were located in the posterior basal segments of lung. Pfeil *et al.*,[18] also demonstrated the main paranchymal lesions for both TUS and CTPA were located in the lower lobe. Because of the hemodynamic properties, peripheral hemorrhages and incomplete infarctions (without necrosis) mainly occur in the lower lobes of the lung.[2,19,20] Since the pulmonary arteries have a large axial trunk that branches off at an angle and terminates in the posterior basal segment, PE lesions have a pleural base mainly placed in the lower lobes.[2,20] The lower lobes are easily viewed by TUS, while the upper lobes can only be inspected by an experienced investigator.[2]

The majority of lesions were wedge-shaped both in TUS (63%) and CTPA (48.7%), localized pleural effusion was detected also in in our study. Pfeil *et al.*,[18] reported the major parenchymal lesions as localized pleural effusion, polygonal lesion, and wedge-shaped lesion in order. Reissig *et al.*,[19] also noted the most common parenchymal TUS findings as wedge-shaped or rounded hypoechoic lesions. Mathis *et al.*,[2] reported that sonographic morphology was mainly triangular toward the hilum of lung in 58% and rounded or mixed in 42%.

In the literature, the sensitivity, specificity, and accuracy of TUS for diagnosing PE is 74%-80%, 92%-95%, and 84%, respectively.[1,2,17] Reissig *et al.*,[1] reported the sensitivity, specificity, PPV, NPV, and accuracy of TUS as 80%, 92%, 95%, 72%, and 84%, respectively. In the study of Mathis *et al.*,[2] the sensitivity, specificty, PPV, NPV, and accuracy were presented as 74%, 95%, 95%, 75%, 84%, respectively. Pfeil *et al.*,[18] reported the sensitivity of TUS for detecting PE 70% and specificity 69.6% in a recent study. Also, NPV and PPV were 84.25% and 50%, respectively. In our study, the sensitivity, specificity, NPV, PPV, and diagnostic value of TUS in clinically suspicious (moderate-high) PE cases are presented as 90%, 60%, 80%, 77.1%, and 78%, respectively. These findings are slightly lower than the previously published values for TUS in the diagnosis of PE, except the the study of Pfeil *et al.*, Since only Pfeil *et al.*,[18] was compared, all the PE patients' TUS findings with CTPA, the specificity given in their study were close to the specificity in our study. The sensitivity of multidetector CTPA for detecting PE varies between 83% and 100%, whereas the specificity lies between 89% and 96%, respectively.[17,21,22]

Multislice CTPA was used as the reference method in the diagnosis of PE in our study. Reissig *et al.*,[1] and Mathis *et al.*,[2] used CT or CTPA in some of the cases but in others they used the combination of other diagnostic methods such as clinical findings, d-dimer levels, dupplex sonography of legs, and echocardiography. Pfeil *et al.*, compared the TUS findings with CTPA findings of 10 (30.3%) PE established of 33 patients. This makes the present study different from the previous ones, except the study of Pfeil *et al.*[18]

Altough the number of patients enrolled in the study is low and future prospective studies are warranted, the data suggest that TUS presents a reliable screening technique for diagnosing PEs with a high sensitivity but considerably low specificity.

TUS with a high sensitivity and diagnostic accuracy, is a noninvasive, widely available, cost-effective method which can be rapidly performed. A negative TUS study cannot rule out PE with certainty, but positive TUS findings may prove a valuable tool in the diagnosis of PE at bedside especially at emergency setting, facilitating immediate treatment decision. TUS may be useful in the diagnosis of PE as a screening method in an emergency-based situation especially for critically ill and immobile patients, that allows initiation of anticoagulation, but the diagnosis of PE needs additional confirmation.

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References

1. Reissig A, Heyne JP, Kroegel C. Sonography of lung and pleura in pulmonary embolism: Sonomorphologic characterization and comparison with spiral CT scanning. Chest 2001;120:1977-83.
2. Mathis G, Blank W, Reissig A, Lechleitner P, Reub J, Schuler A, *et al*. Thoracic ultrasound for diagnosing pulmonary embolism: A prospective multicenter study of 352 patients. Chest 2005;128:1531-38.
3. Marshall PS, Matthews KS, Siegel MD. Diagnosis and management of life-threatening pulmonary embolism. J Intensive Care Med 2011.
4. Mastruzzo C, Perracchio G, Poidomani G, Romano M, Crimi N, Vancheri C. Subsegmental pulmonary embolism: Value of thoracic ultrasound for diagnosis and follow-up. Inter Med 2008; 47:1415-7.
5. Mohn K, Quiot JJ, Nonent M, Lacut K, Le Gall G, Couturaud F, *et al*. Transthoracic sonography of the lung and pleura in view of a suspected pulmonary embolism: A Pilot Study. J Ultrasound Med 2003;22:673-8.
6. Ginsberg JS. Drug therapy: Management of venous thromboembolism. N Engl J Med 1996;335:1816-28.
7. Mathis G. Ultrasound diagnosis of pulmonary embolism. Eur J Ultrasound 1996; 3:153-60.
8. Joyner CR Jr, Miller LD, Dudrick SJ, Eskin DJ, Knight DH. Reflected ultrasound in detection of pulmonary embolism. Trans Assoc Am Physicians 1966; 79:262-77.
9. Miller LD, Joyner CR Jr, Dudrick SJ, Eskin DJ. Clinical use of ultrasound in the early diagnosis of pulmonary embolism. Ann Surg 1967;166:381-93.
10. Mathis G, Metzler J, Fussenegger D, Sutterlütti G, Feurstein M, Fritzsche H. Sonographic observation of pulmonary infarction and early infarctions by pulmonary embolism. Eur Heart J 1993;14:804-8.
11. Mathis G, Bitschnau R, Gehmacher O, Scheier M, Kopf A, Schwarzler B, *et al*. Chest ultrasound in diagnosis of pulmonary embolism in comparison to helical CT. Ultraschall Med 1999;20:54-9.
12. Reissig A, Heyne JP, Kroegel C. Diagnosis of pulmonary embolism by transthoracic sonography. Sono-morphologic characterization of pulmonary lesions and comparison with spiral computed tomography. Dtsch Med Wochenschr 2000;125:1487-91.
13. Reissig A, Kroegel C. Transthoracic ultrasound of lung and pleura in the diagnosis of pulmonary embolism: A novel non-invasive bedside approach. Respiration 2003;70:441-52.
14. Mathis G, Dirschmid K. Pulmonary infarction: Sonographic appearance with pathologic correlation. Eur J Radiol 1993;17:170-4.
15. Yang PC. Color doppler ultrasound of pulmonary consolidation. Eur J Ultrasound 1996;3:169-78.
16. Mathis G. Vascular lung consolidations: Pulmonary embolism and pulmonary infarction. In: Mathis G, editor. Chest Sonography. 2nd ed. Berlin: Springer-Verlag; 2008. p. 72-87.
17. Reissig A, Kroegel C. Diagnosis of pulmonary embolism and pneumonia using transthoracic sonography: Pulmonary embolism. In: Bolliger CT, Herth FJF, Mayo PH, Miyazawa T, Beamis JF, editors. Clinical Chest Ultrasound from the ICU to the Bronchoscopy Suite. Basel: Karger; 2009. p. 43-50.
18. Pfeil A, Reissig A, Heyne JP, Wolf G, Kaiser WA, Kroegel C, *et al*. Transthoracic sonography in comparison to multislice computed tomography in detection of peripheral pulmonary embolism. Lung 2010;188:43-50.
19. Reissig A, Heyne JP, Kroegel C. Ancillary lung parenchymal findings at spiral CT scanning in pulmonary embolism. Relationship to chest sonography. Eur J Radiol 2004;49:250-7.
20. Corrin B. Pathology of Lungs. London, UK: Churchill Livingstone; 2000. p. 365-9.
21. Winer-Muram HT, Rydberg J, Johnson MS, Traver RD, Williams MD, Shah H, *et al*. Suspected acute pulmonary embolism: Evaluation with multi-detector row CT versus digital subtraction pulmonary angiography. Radiology 2004;233:806-15.
22. Stein PD, Fowler SE, Goodman LR, Gottschalk A, Hales CA, Hull RD, *et al*. Multidetector computed tomography for acute pulmonary embolism. N Engl J Med 2006;354:2317-27.

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