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Ratio of Vascular Pedicle Width and Thoracic Diameter to Differentiate Cardiogenic and Non-Cardiogenic Pulmonary Edema

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Abstract

Background: Excess intravascular volume evaluation is essential in the intensive care unit (ICU); however, clinical information to differentiate cardiogenic and non-cardiogenic pulmonary edema has been proven ineffective. Thus, this study aimed to distinguish cardiogenic from non-cardiogenic pulmonary edema using the ratio of vascular pedicle width (VPW) to thoracic diameter (VPTR).

Methods: This cross-sectional study was conducted based on secondary data from chest radiographs of 100 patients with clinical symptoms of pulmonary edema in the ICU from January 2013 to December 2015. Cardiogenic and non-cardiogenic pulmonary edema were distinguished using VPW and cardiothoracic ratio measurements (CTR). VPTR was measured to differentiate between the two types of pulmonary edema, and the cut-off value was obtained using a receiver operating characteristic curve.

Results: This study revealed a prevalence of 21% and 79% for cardiogenic and non-cardiogenic pulmonary edema, respectively. A VPTR cut-off value of 25.1% with a sensitivity of 90% and specificity of 86%, may distinguish cardiogenic from non-cardiogenic pulmonary edema.

Conclusions: VPTR is an alternative method to differentiate between cardiogenic and non-cardiogenic pulmonary edema, and this ratio measurement is useful in cases where radiograph films are not standardized.

Keywords: blood vessels, critical illness, diagnostic imaging, intensive care units, pulmonary edema, radiography

INTRODUCTION

Failure to promptly determine excess intravascular volume in the intensive care unit (ICU) has been associated with increased mortality, in-hospital stay duration, and multi-system organ dysfunction.[1,2] Accurate intravascular volume status measurement in patients with critical illness remained one of the most challenging tasks in the ICU, and forecasting patients' hemodynamic condition solely based on clinical information was not proven very successful.[3] Lung edema is one of the most often encountered excess intravascular volume manifestations in the ICU.

Lung edema is classified into two categories according to its etiology: cardiogenic and non-cardiogenic. Differentiating between the various types of pulmonary edema is important because their management varies and cardiogenic edema requires ICU management. More rapid detection of each form of pulmonary edema would aid in treating patients with critical illness in the emergency unit because of the limited capacity of the hospital's ICU, thereby reducing the ICU admission requirements for patients. A decreased death rate has been related to more targeted hemodynamic therapy for the specific kind of pulmonary edema.[4] Excess intravascular volume is usually caused by acute kidney injury (AKI) in the ICU. In Indonesia, the incidence of AKI is approximately 41–43% and mortality rates are approximately 58–77%.[5,6] Delayed diagnoses and treatment of hypervolemia can result in complications, including multiple organ system failure, longer hospitalization, length of ICU stays, and even death.[7]

Predicting intravascular volume status in patients clinically suspected of pulmonary edema has several approaches, including non-invasive procedures, such as brain-type natriuretic peptide, echocardiography, and lung ultrasound, and invasive procedures, such as transpulmonary thermodilution and pulmonary artery occlusion pressure (PAOP) catheterization.[8,9] Numerous studies have cited PAOP catheterization as the gold standard for measuring intravascular volume status due

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to its excellent accuracy. The North American-European Consensus Committee criterion of PAOP of <18 cm H_2O to detect non-cardiogenic pulmonary edema has an 82% sensitivity and 76% specificity.[10] However, the test is intrusive, operator-dependent, costly, and must be performed in specialized places, such as the ICU. Additionally, numerous risks related to the procedure include insertion site hematoma, pneumothorax, arrhythmia, and infection.[1]

Measuring vascular pedicle width (VPW) in conventional chest radiographs could be used as an alternative to differentiate between cardiogenic and non-cardiogenic pulmonary edema due to excess intravascular volume. Not only does this reduce costs and hazards, but it can also be performed outside of the ICU and is relatively fast. However, not every hospital is equipped to digitally measure VPW. VPW measurement discrepancies could arise due to the unstandardized magnification utilized in each film. Therefore, the vascular pedicle-thoracic ratio (VPTR) could be the solution to the unstandardized magnification of films in hospitals not equipped with digital radiography. This study aimed to investigate the cut-off value of VPTR on conventional chest radiographs to differentiate cardiogenic and non-cardiogenic pulmonary edema.

METHODS

This study was approved by the Research Ethics Committee of the Faculty of Medicine, Universitas Indonesia. The study did not include any patient identities or personal information.

This cross-sectional study was conducted based on secondary data from adult patients with critical illness in the ICU of Dr. Cipto Mangunkusumo National Central General Hospital. Secondary data were gathered from February 2012 to December 2015. Random sampling was conducted regardless of sex from December 2015 and backward. All chest x-ray measurements were conducted using secondary data obtained from Picture archiving and communication system (PACS) Infinitt Healthcare software (Seoul, South Korea). All x-ray machines during the study period have passed a routine standard calibration following the hospital's accreditation standard. The sample size in this study was calculated based on a previous study by Kwok *et al*., wherein the prevalence of cardiogenic pulmonary edema in a Hong Kong emergency department is at 40.7% in 2004–2005.[11]

This study included 100 participants. The inclusion criteria for the participants were: adult patients (aged ≥18 years), clinically diagnosed with pulmonary edema, anteroposterior (AP) chest films that met standard reading criteria (e.g., adequate inspiration), and native Indonesians. Standard reading criteria were referred to the hospital's standard operating procedure.[12] Participants with signs of mediastinum pathology (aortic dissection, tumors, lymphadenopathy, or pneumothorax), a history of mediastinum, heart, or lung surgery or radiotherapy, a massive pleural effusion covering the left or right heart borders, normal chest radiographs, or a rotation of >15° on chest x-ray film were excluded from this study.

Clinical signs of pulmonary edema of participants were checked by the anesthesiologist and written in the electronic medical record. AP chest radiographs were obtained in the ICU with portable chest x-rays, with patients in the supine position. Cardiogenic and non-cardiogenic pulmonary edema was differentiated and confirmed in the hospital based on specific chest radiographic features VPW of >70 mm and cardiothoracic ratio (CTR) of >0.55, or VPW of >70 mm for cardiogenic pulmonary edema while non-cardiogenic pulmonary edema was categorized based on VPW of <70 mm alone.

VPW, CTR, and VPTR measurement methods were obtained in reference to the previous studies.[3,13] The VPW value was visually measured in millimeters from a point closest to the left subclavian artery's left border to the right superior vena cava's outermost side, where it crosses with the main right bronchus. The measurement was performed by drawing a vertical line on both sites and measuring the horizontal distance between the two vertical lines using the tools included in the PACS. The CTR value was determined by tracing the longest horizontal line on the heart boundaries and chest cavity using digital measurement tools from the PACS software. VPTR was manually calculated by dividing VPW by the chest cavity's largest diameter. Additionally, expert radiologists remeasured the data to produce a more reliable result by taking the average of the two readings. The techniques for calculating VPW, CTR, and VPTR from AP chest radiographs are detailed in Figure 1. Figures 2, 3, and 4 exhibit examples of measurements obtained during this study.

Data analysis

The data were statistically analyzed using IBM Statistical Package for the Social Sciences (SPSS) version 17.0 software (Armonk, NY, USA). The Kolmogorov-Smirnov tests were used to determine the normality of data distributions for samples of >50. Next, the mean and standard deviation of quantitative data with a normal distribution were determined. Conversely, the median and range were recorded for data without a normal distribution. Finally, the cut-off value for the VPTR was determined using the receiver operating characteristic (ROC) curve, which allows for simultaneous sensitivity and specificity measurements.

VPW, thoracic diameter, and VPTR in this study were remeasured by two raters. The Intra-class correlation coefficient (ICC) between the two raters for VPW, thoracic diameter, and VPTR was analyzed using IBM SPSS version 25.0 software (Armonk, NY, USA) and calculated using a two-way mixed model and absolute agreement type. Rater

1 was a cardiothoracic radiologist consultant with >10-year experience, and Rater 2 was a senior radiology resident with prior training in VPTR measurement. ICC was calculated using measurement data from all 100 participants (males and females). ICC was tested to determine the need for additional training to standardize measurement if utilized by someone else, as well as the accuracy of manual measurement between two raters.

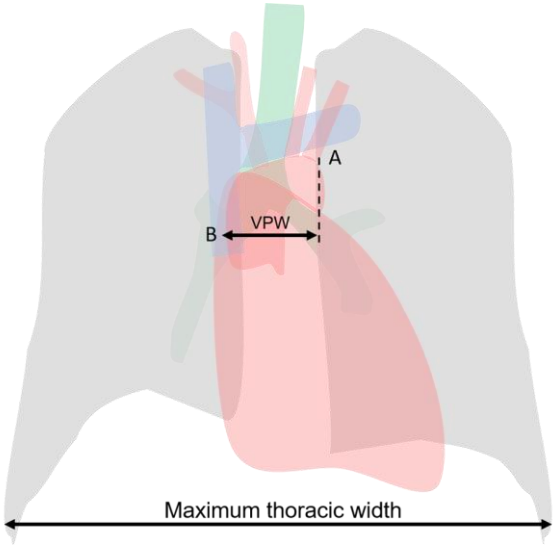

FIGURE 1. The landmarks for measuring VPW on a standard chest radiograph are illustrated. The term "Point A" refers to the location of the left subclavian artery's origin in the aortic arch. The superior vena cava and the right main bronchus intersect at point B. VPTR can be calculated by dividing VPW by the maximum thoracic width.

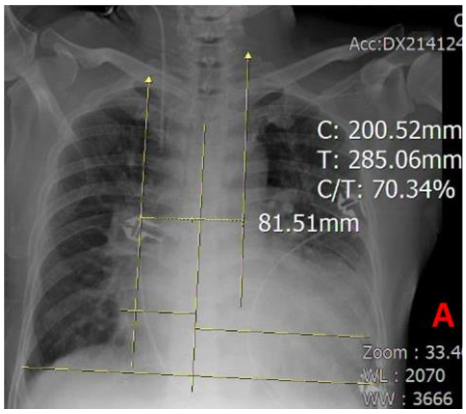

FIGURE 2. Example of VPW, CTR, and VPTR measurements in conventional AP supine chest radiographs of patients with clinical symptoms of lung edema. (A) A 63-year-old male patient. VPW measurement is 81.51 mm, the cardiac diameter is 200.52 mm, and the thoracic diameter is 285.06 mm, hence CTR is 70.34%, this case is a cardiogenic lung edema, with VPTR of 28.6%.

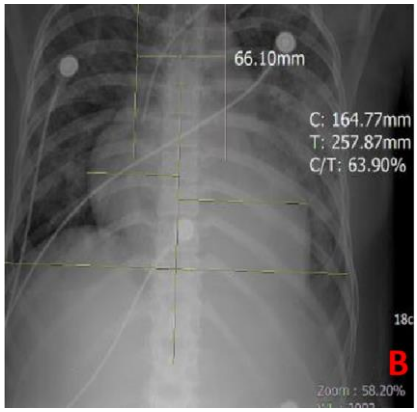

FIGURE 3. (B) A 30-year-old female patient. VPW measurement is 66.1 mm, the cardiac diameter is 167.77 mm, and thoracic diameter is 257.87 mm, hence CTR is 63.90%, this case is a non-cardiogenic lung edema, with VPTR of 25.6%.

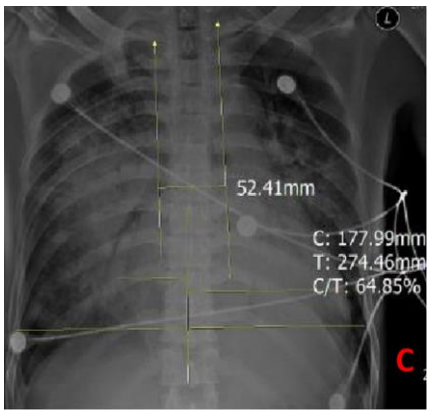

FIGURE 4. (C) A 37-year-old male patient. VPW measurement is 52.41 mm, the cardiac diameter is 177.99 mm, and the thoracic diameter is 274.46 mm, hence CTR is 64.85%, this case is a non-cardiogenic lung edema, with VPTR of 19.1%.

RESULTS

The study includes 100 out of 106 people after excluding participants based on the exclusion criteria. Six patients were excluded from the study because they were under the age of 18 (2), had a history of cardiac surgery (3), or demonstrated symptoms of a mediastinum tumor (1). All 100 participants who meet the inclusion criteria would undergo VPW, CTR, and VPTR measurement. Of the participants, 42% are males and 58% are females. The participants were not normally distributed in age and had a median age of 50.5 years (19–83). Table 1 summarizes the participant characteristics.

The diameters of the thorax or chest cavity, CTR, and VPTR on AP conventional chest x-rays were normally distributed in this study using the Kolmogorov-Smirnov test. In comparison, the VPW value did not follow a normal distribution. The median VPW value is 62.5 mm (46.8–89.4 mm), whereas the mean thorax diameter is 267.3 ± 21.5 mm; the mean CTR value is 59.9% ± 6.3%, while the mean

VPTR value is 23.7% ± 3.2%. The characteristics (VPW, thorax diameter, CTR, and VPTR) were further summarized in Table 2. Cardiogenic and non-cardiogenic pulmonary edema affected 21% and 79% of patients, respectively.

The VPTR cut-off value of 25.1% was determined using the ROC curve and boxplot (>25.1% for cardiogenic lung edema and ≤25.1% for non-cardiogenic lung edema). The sensitivity was 90.5% and the specificity was 86.1%. Table 3 contains a two-by-two matrix summarizing the VPTR cut-off value to distinguish cardiogenic and non-cardiogenic pulmonary edema.

TABLE 1. The distribution of participant characteristics

Characteristics	Frequency (N)	Percentage (%)
Sex		
Males	42	42
Females	58	58
Lung edema		
Cardiogenic	21	21
Non-cardiogenic	79	79

Data normality was calculated using the Kolmogorov-Smirnov test. Participants included are native Indonesian adults (aged ≥18 years), clinically diagnosed with pulmonary edema with AP chest films that met the standard reading criteria.

TABLE 2. Values of parameters (VPW, diameter of the thorax, CTR, and VPTR) in the AP conventional chest radiograph

Parameter	Mean ± SD	Median	Min–Max
VPW (mm)	63.1 ± 8.7	62.5	46.8–89.4
Diameter of the thorax (mm)	267.3 ± 21.5	265.2	224.0–318.1
CTR (%)	59.9 ± 6.3	58.9	44.3–80.4
VPTR (%)	23.7 ± 3.2	23.4	17.7–31.6

SD = standard deviation; min= minimum; max= maximum.

TABLE 3. Sensitivity and specificity of VPTR to differentiate cardiogenic and non-cardiogenic pulmonary edema

VPTR cutoff value	Pulmonary Edema		Total
	Cardiogenic	Non-cardiogenic	
Positive (>25.1)	19	11	30
Negative (≤25.1)	2	68	70
Total	21	79	100

VPTR cut-off value of >25.1% for cardiogenic pulmonary edema and ≤25.1 for non-cardiogenic pulmonary edema. Sensitivity: 90.5%; Specificity: 86.1%.

TABLE 4. Interobserver reliability and agreement

Parameter	ICC (95% CI)	*p*
VPW	0.73 (0.07–0.89)	<**0.001**
Thoracic diameter	0.82 (0.74–0.88)	<**0.001**
VPTR	0.36 (0.06–0.56)	**0.012**

The ICC for VPW, thoracic diameter, and VPTR are 0.73 ($p < 0.05$), 0.82 ($p < 0.05$), and 0.36 ($p < 0.05$), respectively (Table 4). Each rater separately assessed the quantitative aspects of the chest radiograph. The VPTR cut-off value is >25.1 mm in the case of cardiogenic lung edema and ≤25.1 mm in the case of non-cardiogenic lung edema.

DISCUSSION

This study used the AP chest radiograph to measure the VPTR to evaluate the cut-off point and sensitivity and specificity to distinguish cardiogenic and non-cardiogenic pulmonary edema. VPTR would be useful in a resource-limited setting where digital radiography is not available and plain radiograph magnification varies, not allowing for accurate VPW measurements. Studies examining the link between VPW and pulmonary edema remained uncommon, and to the author's knowledge, no studies have examined the association between VPTR and pulmonary edema.

No correlation was found between sex and the occurrence of pulmonary edema; hence, the fact that females are more than males in the current study is controversial. The median VPW and mean VPTR are larger compared to normal healthy participants in reference to the study conducted by Zunera *et al*.[13] The incidence of non-cardiogenic pulmonary edema in the center was also higher within the study period. The low ICC for VPTR measurement may be caused by the combined differences in VPW and thoracic diameter measurements, as well as differences in experience between raters in measuring VPW and thoracic diameter. Additional training should be conducted to familiarize radiologists with the VPTR measurement technique if it is practiced in the future.

Based on the ROC curve, manual calculations using a 2 × 2 table provided a relative sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and accuracy of 90.5%, 86.1%, 63.3%, 97.1%, and 87%, respectively, for differentiating cardiogenic and non-cardiogenic pulmonary edema with a VPTR cut-off point of 25.1%. This investigation did not significantly differ from those of Wichansawakul *et al*. in terms of the sensitivity and specificity of the VPW value.[14] However, Wichansawakul *et al*. conducted prospective cohort research on patients in the ICU in Thailand, and their findings slightly differed from Farshidpanah *et al*. and Thomason *et al*., probably due to variances in the participants' ethnicity.[3,15]

Milne *et al*. published a study in 1984 on the quantitative estimation of intravascular volume status using VPW.[16] The VPW is comprised of the mediastinal silhouette of major vessels, such as the superior vena cava, azygos vein, thoracic aorta, and left subclavian artery, on conventional chest radiographs. Understanding that the VPW's left

border is comprised of veins while the right border is made up of arteries is fundamental. This is because any changes in VPW size caused by increased intravascular volume are primarily attributable to greater venous compliance rather than the arteries.[16]

The magnification parameters of the cardiac silhouette may affect the accuracy of the VPW reading. The VPTR is measured similarly to the CTR on a chest radiograph. The ratio of VPW to thoracic diameter was developed to eliminate the magnification problem in chest radiographs when film sizes and magnifications are inconsistent. Measuring the relative size of an organ to another variable in a chest radiograph has been documented to be useful since 1919 by Danzer *et al*., who pioneered the use of the CTR measurement technique.[17] This technique is advantageous because the relative ratio is not reliant on the magnification scale of the silhouette of the other organs, as both organs have a linear proportion in magnification, thereby eliminating any magnification disparities. VPTR is designed to assist radiologists at facilities lacking a digital system in predicting the kind of pulmonary edema based solely on conventional chest radiographs.

Several variables could negatively affect the study outcomes. A rotational or asymmetrical image and insufficient inspiration of the chest radiograph are two examples. Patients whose chest radiographs are taken while their chest is tilted to the right or left may have increased VPW values, and patients with insufficient inspiration may have an increased heart diameter on an AP chest radiograph. Milne *et al*.[16] reported on both of these aspects, stating that participants tilting to the left or right every 15° could increase the VPW score by as much as 6% and insufficient inspiration has minimal effect on VPW measurement.[16]

Obtaining the ratio of size between two objects has been previously used, such as calculating CTR. Since 1919, CTR was developed to easily determine cardiac enlargement.[18] CTR remained widely used today despite having a weak correlation to true chamber size obtained from cardiac MRI. This may be because CTR measurement is relatively affordable and practical, and conventional chest radiography is widely available.[19] However, a CTR value of >0.55 suggests a true heart chamber enlargement and has an excellent interobserver agreement between raters.[19]

Zunera *et al*. first mentioned VPTR. According to the study, the normal VPW in an erect chest PA radiograph is 48 ± 5.5 mm, and the VPTR is 17.2% ± 17%. Additionally, it indicated that the average VPW and VPTR readings were 10% higher in an AP projection compared to an erect PA projection.[13] This difference in magnification due to positioning could affect the physician's VPW measurement to distinguish cardiogenic from non-cardiogenic edema.

Several studies have been conducted to determine the VPW value in patients with critical illness in the ICU. For example, Farshidpanah *et al*.[3] found no significant difference in VPW measurement between a radiologist and a non-radiologist with prior training in diagnosing lung edema. The study enrolled 80 patients in the ICU and used a reference value of VPW of >70 mm as the cut-off for cardiogenic and non-cardiogenic pulmonary edema, respectively. This cut-off point has sensitivity, specificity, PPV, NPV, and accuracy of 55%, 88%, 81%, 69%, and 73%, respectively, for diagnosing cardiogenic lung edema.[3]

This study is comparable to that of Ely *et al*., who assessed the intravascular volume status of 100 patients in the ICU with and without pulmonary edema.[20] Ely *et al*. then determined that a VPW value of >70 mm and a CTR value of >55% was the most significant discriminator for predicting a PAOP of >18 mmHg.[20] Additionally, the study uses a prospective cohort and 100 samples (similar to our study). Based on chest x-ray alone, these parameters could result in a likelihood ratio greater than three, thereby increasing the diagnostic accuracy of cardiogenic lung edema to 70%.[21] Measuring both a VPW of >70 mm and CTR of >0.55 results in a sensitivity, specificity, PPV, and NPV of 46%, 85%, 65%, and 70%, respectively.[20,21] Numerous other investigations have indicated that a VPW of >68 mm is associated with hydrostatic or cardiogenic pulmonary edema.[15,22–24] Additionally, Thomason *et al*. used both CTR of >0.52 and VPW of >63 mm to indicate cardiogenic pulmonary edema, finding that combining the two criteria improves diagnosis accuracy by up to 73% than VPW or CTR alone.[15]

This study has various limitations that should be highlighted. First, this study could not validate the diagnosis of pulmonary edema with PAOP, which is the gold standard for distinguishing cardiogenic from non-cardiogenic lung edema. This is because PAOP assessment is not a standard operation in the hospital's ICU due to its high cost.

CONCLUSIONS

VPTR can distinguish between cardiogenic and non-cardiogenic causes of pulmonary edema. Additional research showed a cut-off value of 25.1% (sensitivity: 90.5%; specificity: 86.1%) to differentiate between the two etiologies. VPTR can be beneficial in healthcare facilities that continue to employ analog radiography techniques, which are usually unable to accurately determine absolute values due to magnification problems. Further studies are needed to validate the diagnostic performance of VPTR with PAOP to differentiate cardiogenic and non-cardiogenic pulmonary edema.

CONFLICT OF INTEREST

The author(s) declare no competing interests.

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