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ORIGINAL PAPER

Is there any diagnostic value of anteroposterior chest radiography in predicting cardiac chamber enlargement?

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Abstract

The anteroposterior (AP) portable chest radiograph is routinely performed to evaluate cardiopulmonary status, however heart size can be misrepresented by inherent technical factors. Our aim was to determine diagnostic accuracy of cardiothoracic ratio (CTR) on AP chest radiographs relative to echocardiography, as well as relative to axial computed tomography (CT) and frontal CT scout images in predicting cardiac chamber enlargement. 200 subjects with both chest CT and AP chest radiograph within 1 month were retrospectively identified. Patients with pericardial effusion or obscured heart borders were excluded. 130 of these subjects had also undergone echocardiography. Transverse diameters of the heart and thorax were used to calculate CTRs on AP chest radiograph, scout CT, and axial CT images. A second reader was used to verify measurement accuracy and reproducibility. Statistical analysis of CTRs for AP chest radiograph, CT scout, and axial CT images were calculated using echocardiography as gold standard. AP chest radiographs had higher CTR values than axial and scout CT images (by 0.075, $p < 0.001$), larger measured heart diameters by approximately 3 cm ($p < 0.001$), and larger thoracic diameters by approximately 2 cm ($p < 0.001$). CTRs on AP chest radiographs calculated with a cutoff of 0.50 had sensitivity of 86% and specificity of 32%. Sensitivity and specificity were 61% and 66% respectively when using a cutoff of 0.55, and 34% and 92% respectively when using a cutoff of 0.60. A CTR of 60% is more appropriate than 50–55% when evaluating an AP chest radiograph for cardiac chamber enlargement due to its much higher specificity.

Keywords Cardiothoracic ratio · Anteroposterior radiograph · AP radiograph · Computed tomography · CT scout image · Axial CT

Introduction

The portable anteroposterior (AP) chest radiograph is commonly used in clinical practice for routine evaluation of ICU patients and other inpatients, sometimes on a daily basis. The main rationale to obtain portable AP chest radiographs rather than posteroanterior (PA) radiographs is that many patients have limited mobility and cannot be properly positioned for PA chest radiography. Bedside AP chest radiography can be a helpful means of daily cardiopulmonary and hemodynamic evaluation for these patients, and can assist in monitoring the position of lines and tubes.

AP and PA radiographic techniques are substantially different from one another, and the differences in the resulting images can be easily misinterpreted if these differences are not taken into account.

For example, the evaluation of heart size can be misinterpreted due to magnification artifact inherent to the AP technique. Despite the frequent utilization of AP radiographs in clinical practice, the accuracy of predicting cardiac chamber enlargement from the cardiothoracic ratio (CTR) has not been investigated in the context of AP chest radiography.

Our aim was to investigate its diagnostic accuracy relative to CTR values calculated from axial computed tomography (CT) images, frontal view CT scout images and echocardiography in predicting cardiac chamber enlargement.

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Materials and methods

The study protocol was approved by the institutional review board.

Study population and recruitment

A study group of 200 random subjects was created, consisting of patients who had had both a portable AP chest radiograph and a thoracic CT.

The subjects were identified utilizing electronic search and filtering functions of the currently used picture achieving and communication system (PACS). Patients were eligible for selection if they had chest CT and AP radiographs acquired during the same admission or with a 30 day interval between them. Corresponding echocardiography data was subsequently retrieved from the electronic medical record of patients if it was available.

Only adult patients (greater than 18 years of age) were selected for this review. The study had no specific intention regarding the racial and ethnic distribution of the subjects. The racial and ethnic distribution of the subjects was a natural reflection of the regional patient referral base of the greater Rochester area and the state of New York.

Study activities

This was a retrospective review of existing data from January 2017 to May 2017. The data had been already collected and stored on the PACS (philips primordial) and the electronic medical record system (EPIC).

Specific data points which were obtained include the transverse diameter of the heart and the maximal transverse diameter of the thorax as demonstrated on AP radiographs (Fig. 1), on axial CT images (Fig. 2), and on CT scout images with a frontal view (Figs. 3, 4). We selected the closest echocardiography to the radiographic exam to ensure that the AP chest radiograph reflected the patients' true cardiopulmonary status as documented by echocardiography.

The study team was blinded to the clinical data and the echocardiographic results recorded for purposes of the study. Two radiologists generated the study population through the daily work lists. Two readers who were blinded to the clinical information made the measurements on the CTs and the chest radiographs. Another independent investigator recorded the echocardiography data points who was blinded to the rest of the data set and rest of the clinical information. The statistical calculation was performed by another investigator who was not involved in data collection.

Although one could argue that cases of left atrial enlargement should be excluded because the effect of left atrial enlargement on the CTR might be diminished, we did not find this to be confirmed in the literature. Therefore cases in which echocardiography showed enlargement of one or more cardiac chambers to be enlarged (including isolated left atrial enlargement) were included.

Echocardiography technique

The Echocardiography Laboratory at the University of Rochester Medical Center (URMC) is fully approved by the Intersocietal Accreditation Commission (IAC) for transthoracic, stress and transesophageal echocardiography. All echocardiographic images are stored as cine-loops of at least

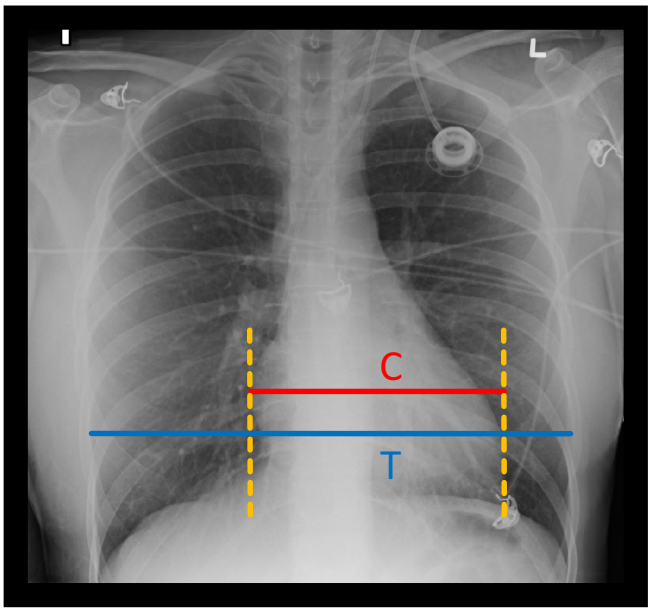

Fig. 1 CXR depicting the method of measuring the maximum transverse cardiac diameter (C) and the transverse thoracic diameter (T)

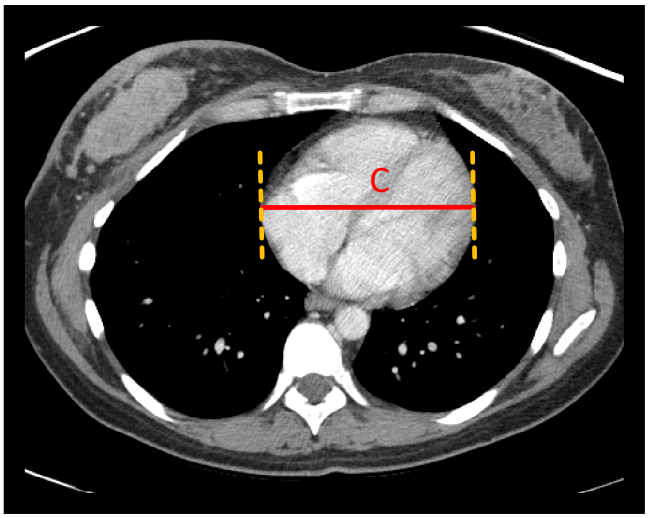

Fig. 2 Axial CT image depicting the method of measuring the maximum transverse cardiac diameter (C)

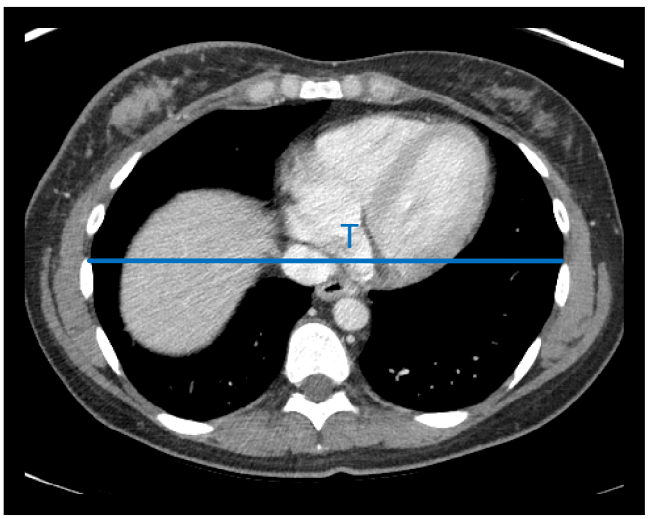

Fig. 3 Axial CT image depicting the method of measuring the maximum transverse thoracic diameter (T)

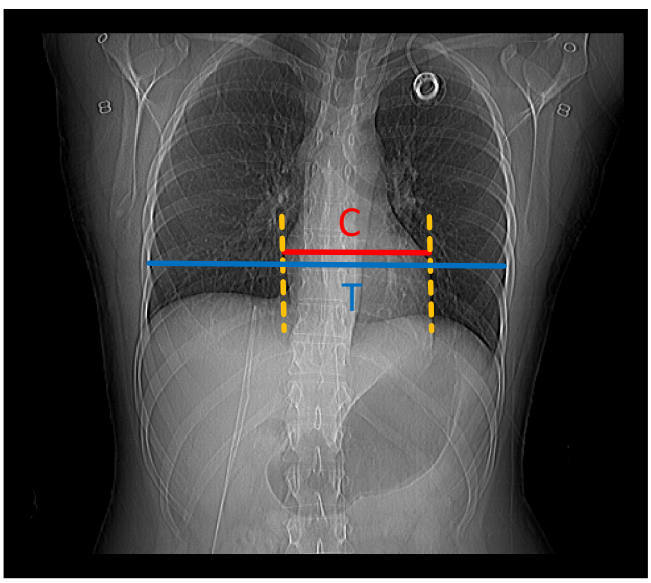

Fig. 4 Frontal CT scout image depicting the method of measuring the maximum transverse cardiac diameter (C) and the transverse thoracic diameter (T)

3-beats in duration with a frame rate of at least 30 Hz and in "raw" image format, which allows for complex post-capture image processing on an analysis workstation as if the imaging were still live on the ultrasound machine (EchoPac PC, General Electric Corporation, Milwaukee, WI). Accurate quantification is a primary goal of the URMC echo lab, so echo contrast is used in approximately 65% of transthoracic and stress echo cases to improve the delineation of endocardial boarders. Left ventricular (LV) diastolic and systolic volumes are measured using the modified Simpson's method from an average of 3 views (usually the apical 4C, 2C, and 3C views, but the parasternal long-axis (PLAX) might be substituted for the A3C view and the subcostal 4C might be substituted for the apical 4C, if needed due to limited image quality). The 3-view end-diastolic LV volume averages are then height and body surface area (BSA) indexed to determine whether or not enlargement is present. The presence of left ventricular hypertrophy (LVH) is determined from the absolute value of LV mass (calculated from the measured LV diameter and wall thickness) and the LVH type is determined from the wall thickness to cavity diameter ratio (concentric vs. eccentric LVH). End-systolic left atrial volume is also measured using the modified Simpson's rule from an average of 3-views (typically the A4C, A2C and A3C views, but the PLAX and SC4C might be used as for LV volume measurements). The 3-view LA volume averages are then height and BSA indexed to determine whether or not enlargement is present. It is difficult to measure right ventricular (RV) volume using echocardiography, so the diameter of the right heart is used as a surrogate (measured from the 4C or SC4C). RV size is also determined qualitatively by comparison to the left heart with knowledge of the LV volume and by inspection of the ventricular septum for diastolic flattening (so called "D-sign"). End-systolic right atrial (RA) volume is measured using Simpson's rule from the A4C view (often RV-centric angulation), RV inflow and SC4C views. The RA volume averages are then height and BSA indexed to determine whether or not enlargement is present. When determining size: The left atrial and RA volumes are calculated at end-systole. The RV diameter and LV volume are calculated at end-diastole. Both of these correspond to the max filling volume (the max volume for atria occurs right before the atrioventricular valves open, and the max volume for the ventricles occurs right before the outflow valves open). Gender is used for LV mass interpretation, but not for volume/size determination, as that is output determined (i.e., the heart needs to fit the body size).

Data analysis

Categorical variables (including the presence or absence of cardiomegaly or cardiac chamber enlargement) in this study were compared using Chi square analysis. Continuous variables (including measurements of cardiac diameter, thoracic diameter, and calculated CTR values) were compared between each of the CT and radiographic modalities using paired *t* test. Measurements between different readers were also compared using a paired *t* test. A $p < 0.05$ was considered statistically significant. A commercially available licensed software was utilized for the statistical calculations.

Results

CTR values calculated from scout CT images and axial CT images were not statistically different. CTR values calculated from chest radiograph were 0.075 greater than for CTR values calculated from CT images (including both scout and axial calculated separately, $p < 0.001$). Heart diameter as measured on chest radiograph was approximately 3 cm greater than diameters measured on both scout and axial CT images, with $p < 0.001$. Thoracic diameters measured on chest radiograph were approximately 2 cm greater than those measured on both scout and axial CT images, with $p < 0.001$ (Table 1). Similar results were demonstrated when the same values were calculated from measurements made by a second reader (Table 2). Heart and thorax diameters as measured on chest radiograph were not statistically different between the two readers.

Table 1 Comparison of cardiothoracic ratio, heart diameter, and thorax diameter between chest radiograph, scout CT, and axial CT for the first reader (200 patients)

	Comparison	Mean difference	p-value
Cardiothoracic ratio	Chest radiograph to scout CT image	0.08	< 0.001
	Chest radiograph to axial CT image	0.08	< 0.001
	Scout CT to axial CT	−0.001	0.65
Heart diameter	Chest radiograph to scout CT image	31.1 mm	< 0.001
	Chest radiograph to axial CT image	32.5 mm	< 0.001
	Scout CT to axial CT	1.5 mm	0.02
Thoracic diameter	Chest radiograph to scout CT image	17.5 mm	< 0.001
	Chest radiograph to axial CT image	21.0 mm	< 0.001
	Scout CT to axial CT	3.5 mm	< 0.001

Table 2 Comparison of cardiothoracic ratio, heart diameter, and thorax diameter between chest radiograph, scout CT, and axial CT for the second reader (98 patients)

	Comparison	Mean difference	p-value
Cardiothoracic ratio	Chest radiograph to scout CT image	0.07	< 0.001
	Chest radiograph to axial CT image	0.07	< 0.001
	Scout CT to axial CT	0.004	0.28
Heart diameter	Chest radiograph to scout CT image	27.9 mm	< 0.001
	Chest radiograph to axial CT image	31.9 mm	< 0.001
	Scout CT to axial CT	4.1 mm	< 0.001
Thoracic diameter	Chest radiograph to scout CT image	17.5 mm	< 0.001
	Chest radiograph to axial CT image	22.6 mm	< 0.001
	Scout CT to axial CT	5.1 mm	< 0.001

Among the subgroup of 130 patients who had echocardiographic evaluation, 80 had at least one chamber enlargement on echocardiography. It may be noteworthy that of those with one or more cardiac chambers shown to be enlarged on echocardiography, 25 had isolated left atrial enlargement. CTR values were calculated from AP chest radiograph measurements. Using echocardiography as a gold standard for whether there was truly cardiac chamber enlargement, various diagnostic test evaluation metrics were calculated including sensitivity and specificity, positive and negative likelihood ratios, and positive and negative predictive values. This was first performed using a ratio of 0.50 as a cutoff, and was repeated using a CTR cutoff of 0.55, 0.60, and 0.65 (Table 3; Figs. 5, 6).

Similar data and comparisons were also performed using frontal scout images and axial images from chest CT studies, also using cutoffs of 0.50, 0.55, 0.60 and 0.65 as cutoffs (Tables 4, 5).

A receiver operating characteristics (ROC) curve is also provided (Fig. 7), which demonstrated an area under the curve (AUC) of 0.67 (0.58–0.77, 95% confidence interval), p = 0.001. The "optimal" threshold was calculated at 0.545 with a sensitivity of 63.8% and specificity of 64.0%, similar to results displayed in Table 3 for the original 0.55 cutoff value (61.3% sensitivity, 66.0% specificity).

Disscussion

The size of the heart assessed by CTR on chest radiography is often used as a screening test for the presence of heart failure (HF) and for assessing its severity. A CTR ≤ 0.5 is commonly accepted as normal for an upright PA chest radiograph. A bedside AP chest radiograph is routinely substituted in deconditioned hospitalized patients who cannot stand upright for a PA film. Due to differences in technique and projection of the AP radiograph, assessment of the CTR on AP radiographs can often be confusing and misleading. Increased CTR on chest radiograph often prompts further imaging evaluation; this can be time-consuming and expensive, as well as concerning for radiation exposure, renal injury, and contrast allergy.

There are several factors which affect the CTR on AP chest radiography. During the performance of AP chest

Table 3 Analysis of various cardiothoracic ratio values when evaluating AP chest radiograph for cardiac chamber enlargement using echocardiography as gold standard

	0.50	0.55	0.60	0.65
Sensitivity	86.25% (CI: 76.73–92.93%)	61.25% (CI: 49.70–71.94%)	33.75% (CI: 23.55–45.19%)	5.00% (CI: 1.38–12.31%)
Specificity	32.00% (CI: 19.52–46.70%)	66.00% (CI: 51.23–78.79%)	92.00% (CI: 80.77–97.78%)	96.00% (CI: 86.29–99.51%)
Positive likelihood ratio	1.27 (CI: 1.03–1.56)	1.80 (CI: 1.18–2.75)	4.22 (CI: 1.57–11.34)	1.25 (CI: 0.24–6.58)
Negative likelihood ratio	0.43 (CI: 0.22–0.85)	0.59 (CI: 0.42–0.82)	0.72 (CI: 0.60–0.86)	0.99 (CI: 0.92–1.07)
Positive predictive value	69.99% (CI: 52.61–69.93%)	74.24% (CI: 65.36–81.49%)	87.10% (CI: 71.52–94.78%)	66.67% (CI: 27.55–91.32%)
Negative predictive value	59.26% (CI: 42.39–74.20%)	51.56% (CI: 43.11–59.93%)	46.46% (CI: 42.11–50.87%)	38.71% (CI: 36.93–40.52%)

CI 95% Confidence interval

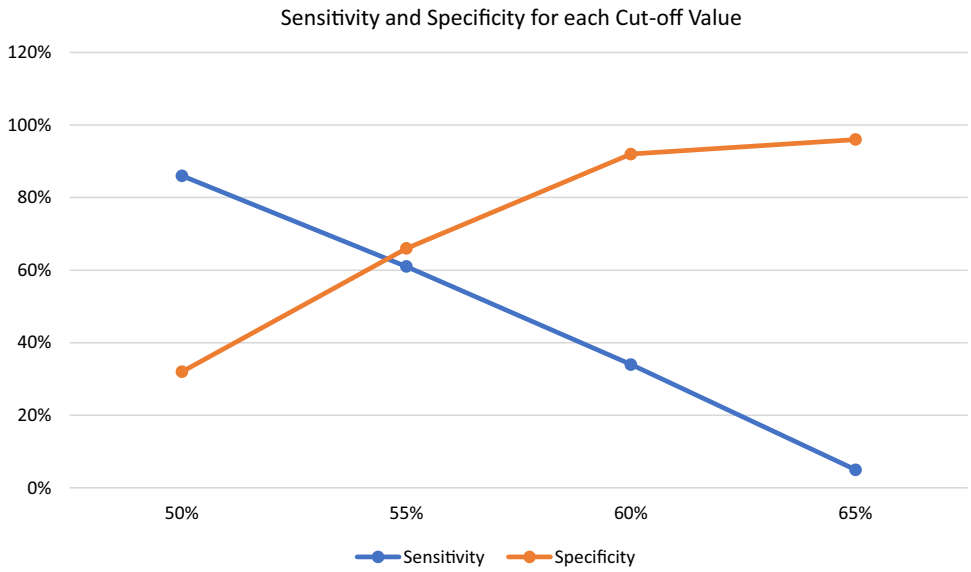

Fig. 5 Sensitivity and specificity of various cardiothoracic ratio values when evaluating AP radiograph for cardiac chamber enlargement using most recent echocardiography as gold standard

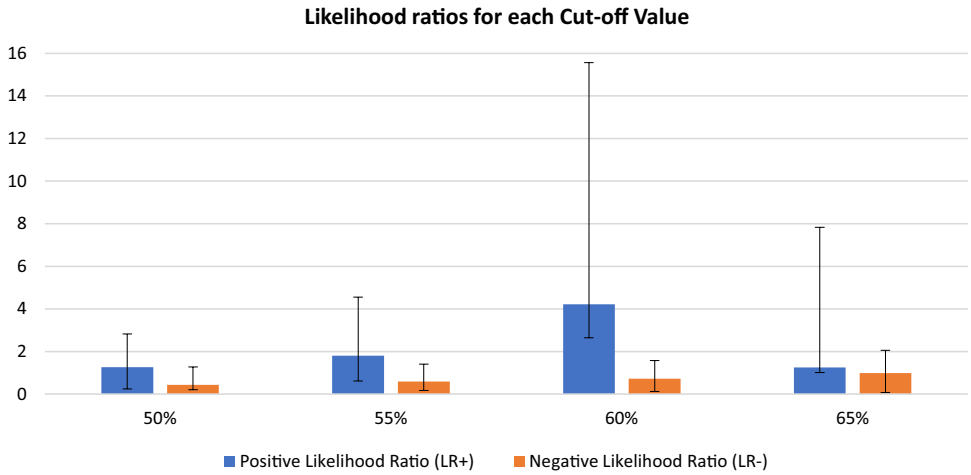

Fig. 6 Likelihood ratios of various cardiothoracic ratio values when evaluating ap radiograph for cardiac chamber enlargement using most recent echocardiography as gold standard

Table 4 Analysis of various cardiothoracic ratio values when evaluating chest CT scout image for cardiac chamber enlargement using echocardiography as gold standard

Cardiothoracic ratio: CT scout image	0.50	0.55	0.60	0.65
Sensitivity	41.25% (CI: 30.35–52.82%)	16.25% (CI: 8.95–26.18%)	1.25% (CI: 0.03–6.77%)	0.00% (CI: 0.00–4.51%)
Specificity	86.00% (CI: 73.26–94.18%)	96.00% (CI: 86.29–99.51%)	98.00% (CI: 89.35–99.95%)	100% (CI: 92.89–100%)
Positive likelihood ratio	2.95 (CI: 1.41–6.15)	4.06 (CI: 0.96–17.25)	0.62 (CI: 0.04–9.77)	n/a
Negative likelihood ratio	0.68 (CI: 0.55–0.85)	0.87 (CI: 0.78–0.98)	1.01 (CI: 0.96–1.06)	1.00 (CI: 1.00–1.00)
Positive predictive value	82.50% (CI: 69.33–90.77%)	86.67% (CI: 60.48–96.50%)	50.00% (CI: 6.01–93.99%)	n/a
Negative predictive value	47.78% (CI: 42.46–53.15%)	41.74% (CI: 39.05–44.48%)	38.28% (CI: 37.19–39.39%)	38.46% (CI: 38.46–38.46%)

CI 95% Confidence interval

Table 5 Analysis of various cardiothoracic ratio values when evaluating axial chest CT images for cardiac chamber enlargement using echocardiography as gold standard

Cardiothoracic ratio: axial CT image	0.50	0.55	0.60	0.65
Sensitivity	43.75% (CI: 32.68–55.30%)	20.00% (CI: 11.89–30.44%)	2.50% (CI: 0.30–8.74%)	1.25% (CI: 0.03–6.77%)
Specificity	82.00% (CI: 68.56–91.42%)	98.00% (CI: 89.35–99.95%)	98.00% (CI: 89.35–99.95%)	100% (CI: 92.89–100%)
Positive likelihood ratio	2.43 (CI: 1.28–4.62)	10.00 (CI: 1.37–73.10)	1.25 (CI: 0.12–13.43)	n/a
Negative likelihood ratio	0.69 (CI: 0.54–0.87)	0.82 (CI: 0.73–0.92)	0.99 (CI: 0.94–1.05)	0.99 (CI: 0.96–1.01)
Positive predictive value	79.55% (CI: 67.18–88.08%)	94.12% (CI: 68.64–99.15%)	66.67% (CI: 15.69–95.55%)	n/a
Negative predictive value	47.67% (CI: 41.92–53.49%)	43.36% (CI: 40.53–46.24%)	38.58% (CI: 37.34–39.84%)	38.76% (CI: 38.18–39.35%)

CI 95% Confidence interval

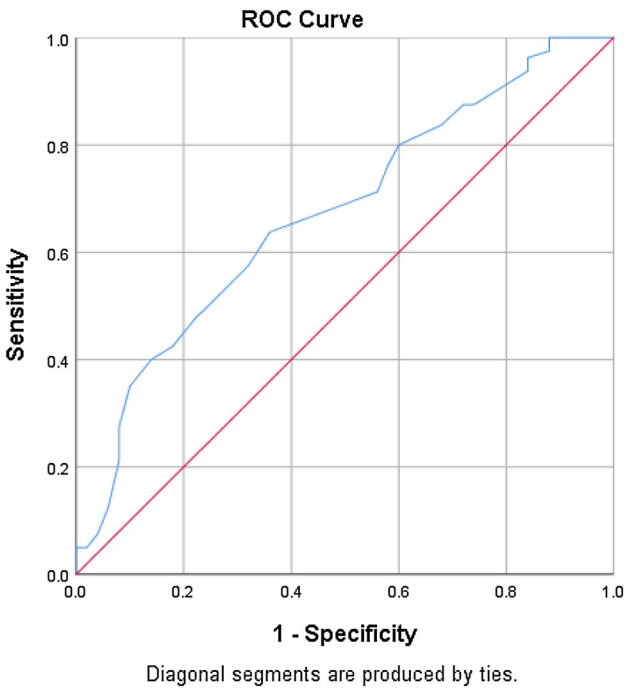

Fig. 7 A receiver operating characteristics (ROC) curve demonstrating the true positive rate (sensitivity) and true negative rate (specificity) when evaluating AP radiograph for cardiac chamber enlargement using most recent echocardiography as gold standard

radiography, the X-ray beams enter the chest of the patient from anterior wall of the chest and the projected image of the heart is projected on the detector (digital receiver or cassette) which is placed behind the patient after traveling through the entire chest. The heart is located anteriorly in the chest cavity just behind the anterior chest wall. The distance between the heart (anterior in the chest) and the digital receiver (placed behind the patient) causes a measurable, and very well known, magnification of apparent cardiac size on chest radiographs relative to the PA technique. Magnification of the chest does not occur to the same degree when using AP technique, which results in disproportionate magnification of the heart (centrally located in the radiograph and within the X-ray beam) relative to the overall thorax [1].It has even been shown that change in CTR due to AP radiography affected smaller hearts more than larger hearts [1]. Furthermore, portable AP chest radiography is typically performed from a shorter distance than standard PA chest radiography. In a study by Milne et al., it was also shown that even a change to the distance from chest wall to detector has a marked effect on CTR. At our institution portable AP chest radiographs may be acquired with an anode-to-film distance (AFD) of anywhere from 40 to 70 inches (or approximately 101–178 cm). The confined space in ICU departments often results in AFDs closer to 40 inches than to 70. By contrast, standard PA chest radiography is performed with a longer AFD 72 inches at our institution, or approximately 183 cm. Milne et al. suggested using approximate correction factors (− 12.5% of CTR for AP radiographs done with a 40 inch AFD and − 10% of CTR for those done with a 72 inch AFD) as a more useful determination of cardiac enlargement.

These principles, however, are not directly applicable to supine AP chest radiography because of altered gravitational effects. Portable AP chest radiography is usually performed while the patients are supine in their bed or semi upright if possible due to the patient's conditions (rarely performed in sitting upright position in the bed). Due to lack of the downward pulling effect of gravitational force, this supine position causes cephalad and peripheral redistribution of the circulating blood within the central cardiopulmonary vascular system which effectively results in further expansion and spread of the cardiovascular structures in the central thorax and loss of the natural gradual thinning of the mid and upper

lung vasculature seen during upright and standing position. Because of this redistribution physiologically happening in the supine position, the artefactual technical magnification of these central cardiovascular structures on radiographs due to AP technique is further accentuated.

Often the AP chest radiography technique is suboptimal in hospitalized patients due to the limitations of the environment or patient related factors and the technologists are not able to optimize the "angle of the X-ray beams" which also contributes to the magnification of the cardiovascular structures. A slight upward shift in the angle of the X-ray beam results in a lordotic view of the chest where the pulmonary vasculature appears augmented artefactual due to the technique instead of tapering naturally as seen on upright PA technique.

Additionally, the hospitalized patients are deconditioned due to variety of disease processes to hold their breath or they are not conscious enough to follow instructions of the technologists to suspend their respiration in fully expanded lung capacity. Because of that the relative size of the lungs and overall size of the chest appears substantially decreased compared to their actual size. Sub optimally expanded lungs can also result in increased pulmonary opacity which may simulate pulmonary edema, and may lead the interpreting radiologist towards an incorrect impression of congestive HF if there is a question of cardiomegaly.

It is also known that the cardiac silhouette on chest radiographs can appear different throughout the cardiac cycle [2–7]. Today the chest radiography is routinely done in a non-ECG gated fashion on nearly all patients regardless of their inpatient or outpatient status. Therefore, it is not possible to know the exact exposure timing and corresponding image capture point during the cardiac cycle on any chest radiography. Most of the cardiac cycle consists of diastolic phase (1/3 systole and 2/3 diastole) under resting conditions and it is safe to assume that non-gated radiographic image capture using conventional chest radiographic technique represents the diastolic appearance of the heart which results in larger transverse cardiac diameter (approximately 10% increase in transverse diameter from systole to diastole in healthy individuals). This change can be more than enough to cause apparently increased CTR (ratio of transverse diameter of the cardiac silhouette to transverse diameter of the thorax on PA radiograph) from normal (50–55%) to cardiomegaly (> 60%) which can imply left HF by itself without considering the magnification factor caused by the AP technique.

When all of these technical factors are combined together, the end product of the chest radiography examination results in artefactual enlarged cardio mediastinal silhouette and cephalized pulmonary vasculature in addition to an increased CTR which all implies a form of falsely diagnosed cardiomegaly associated with potential cardiac dysfunction or nonspecific pulmonary disease, virtually, in all patients who are examined with portable AP chest radiography.

This is a universal challenge for any institution which performs this type of imaging and results in poor service to the patients and referring clinical care team that radiology departments are trying to help.

Gollub et al. have examined the accuracy of the CTR using CT, Echocardiography and chest radiography, similar to our approach, to diagnose cardiomegaly and demonstrated that the CTR at routine CT scans was highly correlated with CTR at CXR [8]. Our results are in congruence with their observations regarding to the presence of cardiomegaly. They suggested that a CTR of lower than 0.49 indicate a low likelihood of LVH [8]. However our study focused on the AP radiography and quantified the artefactual magnification of the overall cardiac silhouette and association with enlargement of any of the cardiac chambers.

Chana et al. in their study of diagnostic accuracy of CTR on admission chest radiography to detect left or RV systolic dysfunction concluded that CTR measured on PA or AP films has limited value in detecting moderate left and/or RV systolic dysfunction and suggested unreliability of the previously established values compared to modern standards. In their study CTR of > 0.55 on PA films resulted in a sensitivity of 62.5% and a specificity of 76.5% for diagnosing any ventricular impairment (positive likelihood ratio of 2.56), with a PPV of 29.5%. However, in their study, the CTR on AP film was not predictive of ventricular impairment on echocardiography [9]. Our study has shown that using a CTR cutoff of 60% on AP chest radiography would help to reduce the over diagnosis of cardiomegaly/increased CTR thus reducing the incorrect implication of a cardiopulmonary pathology such as cardiac dysfunction and pulmonary edema.

In a systematic review of the literature conducted by Loomba et al. to identify studies comparing CTR by chest X-ray to LV dilation by echocardiography, the CTR had 83.3% sensitivity, 45.4% specificity, 43.5% PPV and 82.7% NPV. In their secondary analysis after exclusion of a pediatric study, the CTR had 86.2% sensitivity, 25.2% specificity, 42.5% PPV and 74.0% NPV. They concluded that, the CTR as determined by chest radiograph is sensitive but not specific for identifying LV dilation associated with a strong NPV for identifying LV dilation [10]. In our study, we have not conducted analysis for individual cardiac chambers.

Data provided regarding the ROC curve demonstrates an optimal threshold of 0.545, indicating that the 0.55 cutoff would provide the highest combined sensitivity and specificity given our data. However, this does not change our conclusion considering the artefactual magnification of the cardiac silhouette and the patient related factors usually limiting natural inspiratory thoracic expansion to the full capacity during the anteroposterior radiographic technique. High

sensitivity captures a larger portion of diseased patients, but also results in a higher number of false positives, which can result in an increased number of diagnostic tests for these patients. On the other hand, increased specificity captures a larger portion of the healthy population at the expense of misidentifying patients with mild or moderate disease. High sensitivity is generally preferred in cases where a disease is common or the consequences of misidentifying a diseased patient are high. Higher specificity is often favored in cases where a disease is rare.

Currently, there is no study which examined the effect of the phase of the cardiac cycle on appearance of the heart image on the supine AP portable chest radiography. Additionally, there is no scientific study which examined the clinical impact of the non-gated evaluation of the cardiac size on chest radiography in terms of additional extra imaging studies and laboratory work up of the patients. Future studies can be performed using ECG gated (or synchronized) radiography technology to accurately determine the effect of the phase of the cardiac cycle on global size of the heart and the appearance of the cardiac silhouette on supine imaging studies and compare with supine cardiac gated low dose CT imaging. The scientific evidence provided by such a study could also open the door for possible routine utilization of "ECG gated chest radiography", "temporal subtraction radiography" or "duel energy radiography". These could be used as a screening exam, or for follow up of patients who currently need recurrent imaging using cross sectional modalities (MRI, CT or echocardiography). Currently many cross sectional modality studies are time consuming, resource intensive and have associated risks including radiation exposure, contrast allergy, and reduced renal function, and may have limited availability based on patients' location or socioeconomic status.

One of the limitations of our study is that we did not evaluated the impact of mild to moderate enlargement of left atrium on CTR in supine or semi upright position. There is no scientific study which has examined impact of left atrial enlargement on appearance of cardiac silhouette on supine, semi upright or upright/standing chest radiography in terms of artefactual magnification. Exaggeration of the magnification effect of the AP technique can be expected in the presence of a moderate to severe enlargement of the left atrium due to repositioning of the normally diagonally oriented heart in thorax during upright position. This reorientation effect can be considered similar to the anterior displacement of the heart due to mass effect of a large hiatal hernia pushing the heart against the anterior thoracic wall and repositioning the heart more parallel to the chest wall. However mild left atrial enlargement may not affect the CTR neither on AP nor on PA technique regardless of the patient's position, although the clinical relevance depends on individual patients conditions. Another limitation of this study is that we have not recorded the number of the excluded cases due to the poor quality of the images and the cases with confounding disease processes on the radiographs which precludes optimal measurement of the CTR. All of the consecutive cases with adequate quality were included during the prospective registration process. We also did not record the number of the excluded cases due to lack of a chest CT or echocardiography.

Clinical relevance/application

There are several previous studies which suggests that increased CTR reflects enlargement of the right-sided heart. The patients with clinical signs of RV dysfunction (high jugular venous pressure and/or peripheral edema) shown to have a higher CTR than did those without these signs, despite having a similar EF [11]. In a HF trial, patients with atrial fibrillation (AF) had a higher CTR than did those with sinus rhythm, despite having a higher EF and a smaller LV dimension [12]. These observations indirectly suggest the greater contribution of a dilated right-sided rather than left-sided heart to roentgenographic cardiomegaly in patients with HF. More recently, Fukuta et al. have extended these observations and demonstrated that the right-sided cardiac chamber dimensions by echocardiography better correlate with the CTR than the leftsided cardiac chamber dimensions in patients with HF, specifically, the CTR correlated with major-axis dimensions of the right ventricle (RV) and right atrium [13]. Zakeri et al. showed that impairment in RV longitudinal contractility is more sensitive for the detection of RV disfunction than impairment in global contractility [14].

The left ventricle and the RV hypertrophies in response to chronic afterload elevation [15], resulting in maladaptive RV dilation and RV contractile dysfunction. Right ventricular hypertrophy (RVH) is present in 34–50% [16, 17] of HF with preserved ejection fraction (HFpEF) subjects, whereas RV dilation (RV basal diameter > 4.2 cm) is observed in about 30–35% [18, 19].

The increased left atrial pressure results in development of pulmonary hypertension (PH) [19]. It is estimated that up to 70–80% of HFpEF patients develop PH [20], which can cause RV dysfunction or failure [19]. Inclusion of the RVD in HFpEF diagnostic and risk stratification algorithms has been suggested [14] not only due to association with PH but also other disease processes such as AF, moderate to severe tricusbit regurgitation and RV pacing as well as poor prognosis, higher HF hospitalization rate, cardiovascular mortality and all-cause mortality, similar to the HF with reduced ejection fraction [18]. Left atrial enlargement is an important risk factor for atrial arrhythmias as shown in several groups of patients including the elderly [21–23]

patients with hypertrophic cardiomyopathy [24], valvular heart disease [25] and hypertension [26, 27].

The electrophysiologic studies have shown that in patients with AF most ectopic beats initiating paroxysmal AF originate from pulmonary veins (PV) but 4.7% originate from non-PV foci, localized in RA [28]. The role of non-PV foci is more significant in patients with right heart overload. In population with AF and chronic lung diseases with elevated mPAP non-PV arrhythmogenic foci were localized in the RA in as much as 26.7% of patients [29]. It has been shown that RA stretch due to elevated RA pressure leads to electrical remodeling characterized by increased AF inducibility upon its stimulation [30].

RA remodeling is also an important risk factor for atrial fibrillation (AFl). In one study RA remodeling was more advanced in patients with AFl than with AF [31]. In PAH patients RA enlargement is associated with increased prevalence of supraventricular arrhythmia. RA area index is an independent predictor of hospitalization due to supraventricular arrhythmia which is associated with increased morbidity and mortality [32].

Okute et al. have showed that CTR and NT-proBNP were positively correlated in the hemodialysis (HD) patients. A CTR of $>55\%$ was a significant predictor of CVD in HD patients, independent of the relevant clinical factors, and that it can be used in CVD risk stratification of HD patients when NT-proBNP is not available. CTR is increased with cardiac hypertrophy, but CTR is influenced by other factors such as extracellular volume [33].

Volume overload is common and associated with adverse outcomes in the HD population including systemic hypertension, PH, LVH, and mortality. Since the beginning of the era of maintenance dialysis, prescribing and maintaining a dry weight remains the standard of care for managing volume overload on HD. Maintaining an adequately low dry weight requires attention to sodium intake and adequate time on dialysis, as well as a high index of suspicion for volume overload. Reducing dry weight can provoke decreased cardiac chamber filling and is associated with risks including intradialytic hypotension [34], an independent factor predicting higher mortality in HD patients [35] and other safety concerns associated with reduction of extracellular volume including ischemia of heart, brain, and gut; loss of residual renal function; and vascular access thrombosis [36]. The ideal method to minimize intradialytic morbidity is unknown [34].

During HD, normotension without the use of antihypertensive medications and CTR below 48% on PA CXR have been considered evidence of achievement of dry weight ($\geq 50\%$ is proposed as a criterion for hypervolemia) [37]. In patients with high blood pressure despite a relatively low CTR (≤ 0.48), ACE therapy has been suggested. If blood pressure is normalized, continuation of the treatment proposed, whereas if hypertension persists, continuation of ultrafiltration (UF) has been suggested to normalize blood pressure. In patients with a CTR above 0.48 but normal blood pressure, echocardiography has been recommended to rule out cardiac structural/functional abnormalities [38]. Regression of cardiac remodeling (hypertrophy and dilatation) has been suggested with maintenance of dry-weight, using these criteria, in both peritoneal and HD patients [36, 39]. Even though alternative methods have been utilized, including natriuretic peptides, diameter of the inferior vena cava, continuous blood volume monitoring, and bioimpedance analysis, these methods have not been considered as completely reliable, practical or reproducible and they can be costly, time consuming, and require special equipment, which limit their usage [38].

Proper utilization of XCR to determine volume status requires special attention to the technique to prevent hypovolemia and associated unintended consequences of probing for "dry weight", especially in the elderly population who usually requires AP CXR. The thoracic diameter is lower in the elderly [40, 41] which increase the CTR and may suggests form of cardiac functional/structural abnormality in normotensive patients or volume overload, particularly in a patient with hypertension and may trigger unnecessary attempt for diuresis or vigorous UF. Additionally, a proper interpretation of AP CXR requires reconsideration of the currently practiced CTR criteria originating from PA CXR technique which may not be applicable to the portable CXR even though it is considered "usually appropriate" for evaluation of patients with cardiopulmonary signs and/or symptoms, with life-support devices, patients who are critically ill or medically unstable, patients who cannot be transported for standard PA CXR [42].

Currently, there is no study which examines the clinical impact of the AP CXR and increased CTR on a CXR often prompts further evaluation or intervention. Our study demonstrated exaggeration of the CTR on AP CXR and improved diagnostic accuracy and potentially the utility in predicting cardiac chamber enlargement when a 55 or 60% CTR threshold is used for noting cardiomegaly. Increasing the accuracy of chest radiographs in predicting cardiac chamber enlargement has the potential to improve patient triage in evaluation of cardiopulmonary status, communication with the referring clinicians as well as to decrease unnecessary imaging work up particularly in the elderly patient population.

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Compliance with ethical standards

Conflict of interests The authors declare that they have no conflict of interest.

Ethical approval All procedures performed in studies involving human participants were in accordance with 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.

References

1. Milne EN, Burnett K, Aufrichtig D, McMillan J, Imray TJ (1988) Assessment of cardiac size on portable chest films. J Thorac Imaging 3(2):64–72
2. Clark A, Coats A (2000) Unreliability of cardiothoracic ratio as a marker of left ventricular impairment: comparison with radionuclide ventriculography and echocardiography. Postgrad Med J 76(895):289–291
3. Gammill SL, Krebs C, Meyers P, Nice CM Jr, Becker HC (1970) Cardiac measurements in systole and diastole. Radiology 94(1):115–120
4. Dinsmore RE, Miller SW, Wernikoff RE, Potsaid MS (1978) Increasing the information content of chest radiographs: the use of an ECG gated interlaced radiography system for evaluation of the heart and great vessels. Presented at the annual meeting of the Radiological Society of North America
5. Dinsmore RE, Wernikoff RE, Miller SW, Pohost GM, Block PC, Potsaid MS (1979) Evaluation of left ventricular free wall asynergy due to coronary artery disease: use of an interlaced ECG-gated radiography system. Am J Roentgenol 132(6):909–914
6. Dinsmore RE, Phillips H, Boucher CA, Okada RD, Kushner F, Pohost GM (1983) A noninvasive radiographic technique for evaluation of exercise-induced changes in cardiac function. J Am Coll Cardiol 2(2):318–326
7. Dinsmore RE (1984) Improved use of interlaced ECG-gated radiography in cardiac evaluation. Am J Roentgenol 142(6):1295–1295
8. Gollub MJ, Panu N, Delaney H, Sohn M, Zheng J, Moskowitz CS, Rademaker J, Liu J (2012) Shall we report cardiomegaly at routine computed tomography of the chest? J Comput Assist Tomogr 36(1):67–71
9. Chana HS, Martin CA, Cakebread HE, Adjei FD, Gajendragadkar PR (2015) Diagnostic accuracy of cardiothoracic ratio on admission chest radiography to detect left or right ventricular systolic dysfunction: a retrospective study. J R Soc Med 108(8):317–324
10. Loomba RS, Shah PH, Nijhawan K, Aggarwal S, Arora R (2015) Cardiothoracic ratio for prediction of left ventricular dilation: a systematic review and pooled analysis. Future Cardiol 11(2):171–175
11. Philbin EF, Garg R, Danisa K, Denny M, Gosselin G, Hassapoyannes C, Horney A, Johnstone DE, Lang RM, Ramanathan K et al (1998) The relationship between cardiothoracic ratio and left ventricular ejection fraction in congestive heart failure. Arch Intern Med 158:501–506
12. Carson PE, Johnson GR, Dunkman WB, Fletcher RD, Farrell L, Cohn JN (1993) The influence of atrial fibrillation on prognosis in mild to moderate heart failure. The V-HeFT studies. The V-HeFT VA cooperative studies group. Circulation 87(suppl):VI102–VI110
13. Fukuta H, Ohte N, Brucks S et al (2007) Contribution of right-sided heart enlargement to cardiomegaly on chest roentgenogram in diastolic and systolic heart failure. Am J Cardiol 99:62–67
14. Rosita Z, Mohammed SF Epidemiology of right ventricular dysfunction in heart failure with preserved ejection fraction. Curr Heart Fail Rep (2015) 12:295–301
15. Poels EM, da Costa Martins PA, van Empel VP (2015) Adaptive capacity of the right ventricle: why does it fail? Am J Physiol Heart Circ Physiol 308:H803–H813
16. Burke MA, Katz DH, Beussink L, Selvaraj S et al (2014) Prognostic importance of pathophysiologic markers in patients with heart failure and preserved ejection fraction. Circ Heart Fail 7(2):288–299
17. Morris DA, Gailani M, Vaz Perez A et al (2011) Right ventricular myocardial systolic and diastolic dysfunction in heart failure with normal left ventricular ejection fraction. J Am Soc Echocardiogr 24:886–897
18. Mohammed SF, Hussain I, Abou Ezzeddine OF et al (2014) Right ventricular function in heart failure with preserved ejection fraction: a community-based study. Circulation 130:2310–2320
19. Guazzi M (2014) Pulmonary hypertension in heart failure preserved ejection fraction: prevalence, pathophysiology, and clinical perspectives. Circ Heart Fail 7:367–377
20. Lam CS, Roger VL, Rodeheffer RJ, Borlaug BA, Enders FT, Redfield MM (2009) Pulmonary hypertension in heart failure with preserved ejection fraction: a community-based study. J Am Coll Cardiol 53:1119–1126
21. Vaziri SM, Larson MG, Benjamin EJ, Levy D (1994) Echocardiographic predictors of nonrheumatic atrial fibrillation. Framingham Heart Study Circ 89:724–730
22. Psaty BM, Manolio TA, Kuller LH et al (1997) Incidence of and risk factors for atrial fibrillation in older adults. Circulation 96:2455–2461
23. Tsang TS, Barnes ME, Bailey KR et al (2001) Left atrial volume: important risk marker of incident atrial fibrillation in 1655 older men and women. Mayo Clin Proc 76:467–475
24. Tani T, Tanabe K, Ono M et al (2004) Left atrial volume and the risk of paroxysmal atrial fibrillation in patients with hypertrophic cardiomyopathy. J Am Soc Echocardiogr 17:644–648
25. Diker E, Aydogdu S, Ozdemir M et al (1996) Prevalence and predictors of atrial fibrillation in rheumatic valvular heart disease. Am J Cardiol 77:96–98
26. Dittrich HC, Pearce LA, Asinger RW et al (1999) Left atrial diameter in nonvalvular atrial fibrillation: an echocardiographic study. Stroke prevention in atrial fibrillation investigators. Am Heart J 137:494–499
27. Kanmanthareddy A, Reddy YM, Boolani H et al (2014) Incidence, predictors, and clinical course of atrial tachyarrhythmias in patients with pulmonary hypertension. J Interv Card Electrophysiol 41:9–14
28. Chen SA, Tai CT, Yu WC et al (1999) Right atrial focal atrial fibrillation: electrophysiologic characteristics and radiofrequency catheter ablation. J Cardiovasc Electrophysiol 10:328–335
29. Roh SY, Choi JI, Lee JY et al (2011) Catheter ablation of atrial fibrillation in patients with chronic lung disease. Circ Arrhythm Electrophysiol 4:815–822
30. Medi C, Kalman JM, Ling LH et al (2012) Atrial electrical and structural remodeling associated with longstanding pulmonary hypertension and right ventricular hypertrophy in humans. J Cardiovasc Electrophysiol 23:614–620
31. Medi C, Teh AW, Roberts-Thomson K, Morton JB, Kistler PM, Kalman JM (2012) Right atrial remodeling is more advanced in patients with atrial flutter than with atrial fibrillation. J Cardiovasc Electrophysiol 23:1067–1072
32. Waligóra M, Tyrka A, Miszalski-Jamka T et al (2018) Right atrium enlargement predicts clinically significant supraventricular arrhythmia in patients with pulmonary arterial hypertension. Heart Lung 47:237–242

33. Okute Y, Shoji T, Hayashi T (2017) Cardiothoracic ratio as a predictor of cardiovascular events in a cohort of hemodialysis patients. J. Atheroscler Thromb 24:412–421
34. Sinha AD, Agarwal R (2017) Setting the dry weight and its cardiovascular implications. Semin Dial 30(6):481–488
35. Shoji T, Tsubakihara Y, Fujii M, Imai E (2004) Hemodialysis associated hypotension as an independent risk factor for two-year mortality in hemodialysis patients. Kidney Int 66:1212–1220
36. Ok E, Levin NW, Asci G, Chazot C, Toz H, Ozkahya M (2017) Interplay of volume, blood pressure, organ ischemia, residual renal function, and diet: certainties and uncertainties with dialytic management. Semin Dial 30(5):420–429
37. Ozkahya M, Ok E, Toz H et al (2006) Long-term survival rates in hemodialysis patients treated with strict volume control. Nephrol Dial Transpl 21:3506–3513
38. Gunal AI (2013) How to determine 'dry weight'? Kidney Int Suppl 3:377–379
39. Gunal AI, Ilkay E, Kirciman E et al (2003) Blood pressure control and left ventricular hypertrophy in long-term CAPD and hemodialysis patients: a cross-sectional study. Perit Dial Int 23:563–567
40. Potter JF, Elahi D, Tobin JD, Andres R (1982) Effect of aging on the cardiothoracic ratio of men. J Am Geriatr Soc 30:404–409
41. Inoue K, Yoshii K, Ito H (1999) Effect of aging on cardiothoracic ratio in women: a longitudinal study. Gerontology 45:53–58
42. Robert D. Suh et al (2015) ACR appropriateness criterias intensive care unit patients. Expert panel on thoracic imaging. J Thorac Imaging 30:W63–W65