The anteroposterior pericardial sac diameter measured by echocardiography correlates with the volume of pericardial effusion and with effort dyspnea

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Abstract Aims: To examine the value of the anteroposterior pericardial sac diameter (APD) for prediction of the volume of pericardial effusion. Methods and results: We measured the APD by echocardiography before 52 pericardiocentesis procedures and correlated it with the aspirate volume, etiology, symptoms, and clinical outcome. The volume of the aspirate ranged from 60 to 2300 ml (median 650 ml). The APD (range 8.0 cm–15.9 cm, median 12 cm) correlated well with the cubic root of the volume of the effusion [volume = (0.8APD – 0.6)\textsuperscript{3}, \( r^2 = 0.533, p < 0.01 \)]. An APD \( \geq 12 \) cm had a positive predictive value of 88% and a negative predictive value of 83% for effusion volume above the sample median (\( \geq 650 \) ml) and a positive predictive value of 100% for effusion in the middle or upper aspirate volume tertiles. Effort dyspnea was more common among patients with APD \( \geq 12.0 \) cm \( (n = 13) \) than in those with APD < 12.0 cm \( (n = 11) \) \( (p = 0.007) \). One-year survival after pericardiocentesis was closely related to the severity of the underlying etiology and was not influenced by the volume of the effusion before aspiration. Conclusions: The APD is a simple, valuable method for non-invasive prediction of pericardial fluid volume. A greater APD is associated with, and may explain, effort dyspnea.

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Introduction

According to current literature, the volume of pericardial fluid is a strong predictor of outcome in hospitalized patients with pericardial effusion. Patients with a large chronic pericardial effusion are at risk of sudden development of cardiac tamponade. Owing to the very low complication rate of echo-guided pericardiocentesis, some authors have suggested recently that symptomatic and possibly even asymptomatic patients with a large pericardial effusion should undergo this procedure. That has made the need for non-invasive quantitation of pericardial effusion greater than ever. Echocardiography is the best non-invasive method for the detection of pericardial effusion. However, because the pericardial sac has a complex morphology, assessment of its volume by geometric parameters is difficult. Only a few attempts have been made to develop methods for quantitation of pericardial fluid volume by echocardiography, some of them using M-mode echocardiography and necessitating multiple measurements and complex calculations. We hypothesized that because the pericardial sac assumes a more globular shape in patients with a large pericardial effusion, the anteroposterior pericardial sac diameter (APD) alone, which can be easily measured by echocardiography, may correlate with the size of the effusion. The aims of the present study were to assess the possible value of the APD for prediction of the volume of pericardial effusion and to examine the impact of the size of the effusion on symptoms and clinical outcome.

Methods

We measured the APD by echocardiography and the volume of the aspirate in 54 consecutive patients with pericardial effusion who underwent echo-guided pericardiocentesis. All the patients were admitted with pericardial effusion or developed an effusion during hospitalization. Pericardiocentesis was performed for indications stipulated in common practice guidelines and not for the purpose of this study. Patients with incomplete evacuation of the effusion (defined as presence of more than minimal effusion on the echocardiogram at the end of the procedure) were excluded.

The diagnosis of cardiac tamponade was based on the finding of pulsus paradoxus ≥ 20 mmHg (for specificity) and/or echocardiographic and Doppler findings characteristic of pericardial effusion with cardiac compression. To assess the possible impact of the underlying pathology on outcome, we divided the patients into 3 groups as follows: group A, advanced malignancy or other life-threatening illness; group B, other known or suspected conditions which can affect prognosis (for example, pneumonia, renal failure, unexplained anemia); and group C, patients without these conditions (e.g. idiopathic, post-viral).

Evacuation of the effusion was defined as complete if the echocardiogram at the end of the procedure showed no or minimal (barely visible) effusion.

Echocardiography

All the patients underwent a standard echocardiographic study before pericardiocentesis. All echocardiographic measurements were obtained at bedside from two-dimensional echocardiograms, using standard equipment and software (HP Sonos 1000 and HP Sonos 5000, Hewlett-Packard, USA). The APD was defined as the maximal short axis dimension of the pericardial sac in the parasternal view, or in the subcostal view if the parasternal echocardiographic window was inadequate.

All the pericardiocentesis procedures were performed in the intensive care unit, using a similar technique to that described recently by Tsang et al.

Statistical analysis

Data are presented as mean ± SD or numbers and percentages, as appropriate. The APD was studied as a continuous variable and also categorized according to the median of the sample as small or large. The volume of the effusion was analyzed as a continuous variable and as a categorical variable as appropriate. It was categorized according to the tertiles of the sample as small (lower tertile), moderate (middle tertile) or large (upper tertile). The Pearson correlation was used to correlate the APD and the volume of pericardial effusion (as continuous variables). The Student’s t test was used for comparison between means, and Fisher’s exact test for comparison of frequencies between small groups. The Kaplan–Meier method was used for analysis of survival. The difference in survival between categories was estimated with the log-rank test. A p value of <0.05 was considered significant.

Results

Complete evacuation of the effusion was achieved in 52 procedures in 44 patients, 18 men and
26 women, aged 20–86 years (mean ± SD, 61 ± 15 years). The etiology of the effusion was malignancy (n = 23), idiopathic (n = 10), cardiac surgery (n = 5), iatrogenic (n = 3), respiratory tract infection (n = 3), heart failure (n = 2) and other causes (n = 6). The indication for pericardiocentesis was tamponade (n = 17), dyspnea (n = 10), large effusion (n = 7), investigation (n = 3) and recurrence or exacerbation of a chronic effusion (n = 5). There were no deaths. One patient had a short asymptomatic nonsustained ventricular tachycardia. There were no other major complications.

The volume of the aspirate ranged from 60 to 2300 ml (median 650 ml) and the APD ranged from 8.0 cm to 15.9 cm (median 12.0 cm) (Fig. 1). The APD correlated well with the cubic root of the volume of the effusion (R² = 0.533, p < 0.01). The equation of the regression line was: volume (ml) = [0.8APD (cm)–0.6]³.

To calculate the predictive value of the APD, we analyzed the data according to the tertiles of the effusion volume, as follows: lower tertile (small effusion), 60–500 ml, middle tertile (moderate effusion), 501–890 ml, and upper tertile (large effusion), ≥900 ml. The findings are listed in Table 1. Only one (4%) of the patients with an APD below median (<12.0 cm) had a large pericardial effusion, whereas none of the patients with APD above median had a small effusion. An APD above the median (≥12.0 cm) had a positive predictive value of 88% and a negative predictive value of 83% for effusion volume above the median (≥650 ml). The predictive value of an APD ≥12 cm for non-small (moderate or large) effusion was 100%.

Data of one-year outcome after pericardiocentesis were available for 39 patients, and of two-year outcome, for 36 patients. The overall 1-, 6-, 12-, and 24-month survival rates were 79%, 61%, 56% and 50%, respectively. There were no differences between the patients who died and those who survived, either in the APD (11.2 ± 1.4 cm vs. 11.6 ± 1.8 cm, p = 0.5) or in effusion volume (604 ± 298 ml vs. 756 ± 510 ml, p = 0.25). This held true even when the analysis was restricted to patients with non-traumatic (‘‘medical’’) pericardial effusion.

Analysis of the patients’ outcome by the severity of the underlying cause of the effusion showed that survival was closely related to the underlying condition. Fig. 2 shows the Kaplan–Meier survival curves for the three groups. The one-year survival of patients without a known severe underlying disease (group C) was excellent (14/14, 100%); the survival of patients with advanced malignancy or other life-threatening conditions (group C) was poor, and the survival of group B patients was intermediate. The difference between the 3 groups was highly significant (p < 0.0001).

To examine the clinical significance of the APD (Fig. 3), we compared the prevalence of effort dyspnea, between patients with an APD above and below the median. Excluded from this analysis were 21 patients in whom the effort dyspnea could have been due to causes other than the effusion itself (malignant lung involvement, n = 18; lung disease, n = 2; and congestive heart failure, n = 1) and patients with unknown clinical status (n = 5) or sudden tamponade (n = 2). Among the remaining 24 patients, we found a significantly higher prevalence of effort dyspnea in the group of patients with APD above median (n = 13) than in

<table>
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<tr>
<th>Table 1</th>
<th>The anteroposterior pericardial sac diameter (APD) and pericardial fluid volume</th>
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<tbody>
<tr>
<td>APD (cm)</td>
<td>Pericardial fluid volume (tertiles)</td>
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<tr>
<td></td>
<td>I (60–500 ml)</td>
</tr>
<tr>
<td>&lt;12.0 (n = 26)</td>
<td>17</td>
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<tr>
<td>≥12.0 (n = 26)</td>
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The data are the number of patients in each tertile of pericardial effusion volume according to the APD category (above and below the median).
those with APD below median \((n = 11, \ p = 0.007, \text{ Fisher’s exact test})\).

**Discussion**

The results of this study, in a relatively large series of patients with pericardial effusion, show that the APD can predict the volume of the effusion, and successfully distinguish between patients with larger and smaller effusions. Moreover, to the best of our knowledge, this is the first report of an association between the size of the effusion and effort dyspnea.

The APD was readily obtained in all our patients, in most of them from the parasternal view, in which the APD is probably closest to the true short axis of the pericardial sac. Thus, this method does not have the limitation of frequent absence of adequate echocardiographic window(s) reported for the M-mode based methods. As our findings show, the volume of pericardial effusion can be estimated based on the APD using a relatively simple calculation \([\text{volume} = (0.8 \text{APD} - 0.6)^3]\), which can be easily done at the bedside.

The correlation between the APD and the volume of the effusion was good. Our method was less accurate in predicting the exact volume of very small or very large effusions and at the ends of the APD range \((< 9 \text{ cm and } > 13 \text{ cm})\). Nevertheless, the results suggest that for clinical purposes, using the correlation equation for prediction of the volume of pericardial effusion can be useful in spite of its simplicity and that this method may help physicians to decide on the need for pericardiocentesis when the size of the effusion is the only criterion.

The mechanism whereby large pericardial effusions cause effort dyspnea has not been well understood.

*Figure 2*  Survival by the underlying etiology of pericardial effusion. A: patients with advanced malignancy or other life-threatening illness; B: patients with other known or suspected conditions which can affect prognosis; C: patients without these conditions.

*Figure 3*  Measurement of the APD. Parasternal long axis view. The APD is measured where the distance between the anterior and posterior inner surfaces of the pericardial sac along its anteroposterior axis is the greatest. APD: Anteroposterior pericardial sac diameter (11.2 cm); PE, pericardial effusion; LV, left ventricle.
established. Effort dyspnea is a common complaint among patients with pericardial effusion.\textsuperscript{9,10} However, in many patients with pericardial effusion, other causes for effort dyspnea (for example, pleural effusion, malignant lung disease, anemia and congestive heart failure) are also present. We found a significantly higher prevalence of this symptom in patients with APD $\geq 12$ cm than in those with APD $< 12$ cm. As the APD relates to the size of the pericardial sac, its association with effort dyspnea noted here suggests that the effort dyspnea due to large pericardial effusions is a product of restriction or limitation of ventilation by the large space-occupying pericardial sac in the thorax.

References


