Sensitivity and Specificity of Electrocardiographic Criteria for Left Ventricular Hypertrophy in Children with Rheumatic Heart Disease

by

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Abstract

Electrocardiographic criteria for left ventricular hypertrophy (LVH) were examined in 84 unselected pediatric patients with rheumatic heart disease. There were 47 male and 37 female patients, ranging in age from 6 to 19 years. Electrocardiographic LVH was detected in 41 patients (48.8%), i.e. in 35.3% (26/74) of boys and in 63.6% (15/41) of girls. Echocardiographically determined LVH was present in 42 cases (50%) if left ventricular mass (LVM) was indexed for height, or 47 cases (56%) if LVM was indexed for body surface area (BSA). The overall sensitivity of height-indexed electrocardiographic diagnosis of LVH was 71.4% (95% confidence interval = 57.7% to 85.1%), while its sensitivity was 73.8% (95% confidence interval = 60.0% to 87.0%). For BSA-indexed echocardiographic LVH, the sensitivity was 68.1% (95% confidence interval = 54.8 to 81.4%) and the specificity was 75.7% (95% confidence interval = 61.9% to 89.5%). When sex-adjustment was examined, there was no increase of sensitivity of electrocardiographic LVH. Sensitivity of the electrocardiogram for LVH increased when age-adjustment was examined with 13 years of age as a cut-off point, both for height-indexed and BSA-indexed echocardiographic LVH. Reasons for the difference between these findings and the findings in adult patients (remarkably low sensitivity and very high specificity of ECG LVH) were discussed. Electrocardiogram was a moderate diagnostic modality in the detection of LVH in our pediatric patients with rheumatic heart disease. Sex did not influence the sensitivity of ECG LVH, but older age group tended to increase its sensitivity.

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Introduction

Rheumatic heart disease is still prevalent in developing countries, including Indonesia. It is the most important and the only permanent sequel of rheumatic fever, and not infrequently causes disability of the affected children, leading to significant impairment of their growth and development and may even result in death (1,2). Typically, acute rheumatic fever affects young children; the peak incidence of the first attack of rheumatic fever is between 8-12 years of age.

In Dr. Cipto Mangunkusumo Hospital, Jakarta, more than one half of patients with acute rheumatic fever developed permanent valvular heart disease (3). Following hospitalization for acute management of rheumatic fever, the survivors are managed at the Out-patient Clinic for the prevention of streptococcal infections with benzathine penicillin injection or its substitutes once a month. Apart from clinical assessment, echocardiographic and chest X-ray examinations are performed every 3 to 12 months to detect any change of the cardiac status, including ventricular hypertrophy. In the last several years echocardiographic evaluation has been available in our hospital, enabling more accurate diagnosis of valvular lesions and detection of ventricular dilatation and/or hypertrophy. We have been impressed that many patients with valvular rheumatic heart disease who did not have left ventricular hypertrophy (LVH) on electrocardiogram (ECG) showed the evidence of increased left ventricular mass (LVM) measured echocardiographically.

There were several reports on the sensitivity and specificity of electrocardiographic LVH in adults (4-7), but we were unaware of those values in children. The only report on the determination on echocardiographic left ventricular mass in children was that of Daniels et al., (8). Even more, no report on the sensitivity and specificity of electrocardiographic criteria for LVH in children with rheumatic heart disease was published. The values may of benefit in interpreting patients with rheumatic heart disease, since ECG can be recorded at any time with low cost, while echocardiography is either not available in many places in Indonesia or, if available, the high cost is beyond the reach of most patients.

We report our cross sectional study specially designed to examine the sensitivity and specificity of electrocardiographic criteria for LVH in a group of pediatric patients with rheumatic heart disease.

Materials and Methods

The study population consisted of unselected pediatric patients with rheumatic heart disease visiting the Out-patient Clinic, Division of Cardiology, Department of Child Health, Dr. Cipto Mangunkusumo Hospital, Jakarta between July and September, 1990. They had been managed on a monthly basis for receiving benzathine penicillin or its substitutes to prevent streptococcal infection. No attempt was made to select any particular valvular lesions.

Clinical data

All patients underwent complete physical examination, including measurements of body weight, height, and clinical cardiovascular diagnosis. Body surface area was estimated using a nomogram based on the following formula (9):

$$BSA = 0.07184 \times \frac{W^{0.425}}{H^{0.725}}$$

Where S = body surface area in $m^2$, W = body weight in kg, and H = height in cm. Results of previous echocardiographic examination were reviewed to confirm the type of valvular lesions. The nutritional status was graded using modified classification of Wellcome Trust Party, i.e. overweight (>120%), normal (80-120%), mild to moderate undernutrition (60-80%), or severe undernutrition (<60%), where 100% represented the 50th percentile of Harvard Standard.

Echocardiographic methods

In nearly all patients, complete echocardiographic examination (i.e. 2-dimensional, M-mode, and echo-Doppler with colorflow mapping) had been performed, so that the specific valvular lesions was known. For the purpose of the study, standard M-mode echocardiography was performed using Toshiba SSH-650A with 2.5 or 3.75 MHz transducers. With the patient lying on a left lateral decubitus position, left ventricular measurement were performed from either the long axis or short axis parasternal view. Tracings were recorded on Toshiba Sonoprinter black and white paper at a paper speed of 25 or 50 mm/second. The tracings were coded and read blindly. LV measurements were performed by one of the authors (SS). Particular attention was paid to identifying the correct level for LV measurements. LV measurements were made just below or at the tips of the mitral leaflets. The left ventricular internal diameter, interventricular septum, and left ventricular posterior wall thickness were measured at end-diastole, as indicated by the beginning of the q wave. All measurements were made according to American Society of Echocardiography (ASE), i.e. leading-edge-to-leading-edge convention (11) (see Figure 1). Studies were excluded if any uncertainty of precision of measurements was found. Echocardiographic evidence of pericardial effusion was also a criterion for exclusion, since it might decrease the precordial ECG voltages.
Figure 1: Method of left ventricular measurements according to American Society of Echocardiography used in this study. All measurements were performed using leading-edge-to-leading-edge convention. LVID = left ventricular internal diameter; PWT = posterior wall thickness; IVST = interventricular septum thickness.

Left ventricular mass (LVM) was calculated in all patients using the modified ASE-cube criteria which was known to be close to anatomic LV mass (12):

\[
LVM = 0.8 \times 1.04 \times ([LVID + PWT + IVST]^3 - LVID^3) + 0.6 \text{ gm}
\]

where

- LVM = left ventricular mass
- LVID = left ventricular internal dimension in diastole
- PWT = ventricular posterior wall thickness in diastole
- IVST = interventricular septal thickness in diastole.

The LVM was then indexed for sex, height, and body surface area. In boys, LVM of more than 99.8 gm/m² or 103 gm/m² indicated LVH; whereas in girls the comparable values were 81.0 gm/m², or 84.2 gm/m² (8). Evaluation was then made for age-adjusted groups (both sexes combined, less than 13 years and 13 years or more), and for grading of the severity of LV mass in quartiles.

Method of analysis and presentation

Sensitivity, specificity, accuracy, and predictive values were presented in per cents; 95% confidence interval of each value was also supplied in per cent. The confidence interval was calculated using the following formula:

\[
CI = P \pm 1.96 \frac{PQ}{N}
\]

where CI = 95% confidence interval of proportion (in decimal)
- \(P\) = proportion of interest
- \(Q\) = 1 - \(P\)
- \(N\) = number of observed patients

The \(X^2\) test was used to test the differences between sensitivity and specificity of ECG LVH in sex-adjusted and in age-adjusted group. The Kolmogorov-Smirnov test was applied to detect changes of electrocardiographic LVH. All statistical results were considered significant at p level of 0.05 or less.

Results

Clinical data

Of the initial 90 patients enrolled in this study, 6 were excluded because of the evidence of left bundle branch block in 1, inadequate ECG recording in 1, and inadequate M-mode tracings in 3 patients. The other one was excluded because of the evidence of moderate pericardial effusion detected echocardiographically. Thus, there were 84 patients suitable for analysis, consisting of 47 boys and 37 girls, ranging in age from 6 years to 19 years (see Table 1). All of them had native (unoperated) rheumatic valvular heart disease. The specific valvular lesions were depicted in Table 2, while the nutritional status was depicted in Table 3.

Table 1: Age and sex distribution of 84 patients with rheumatic heart disease

<table>
<thead>
<tr>
<th>Age (yrs)</th>
<th>Sex</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>6 - 12</td>
<td></td>
<td>26</td>
<td>15</td>
<td>41</td>
</tr>
<tr>
<td>13 - 19</td>
<td></td>
<td>21</td>
<td>22</td>
<td>43</td>
</tr>
<tr>
<td></td>
<td></td>
<td>47</td>
<td>37</td>
<td>84</td>
</tr>
</tbody>
</table>

Table 2: Specific valvular lesions in 84 patients based on clinical findings and previously performed echo-Doppler examination

<table>
<thead>
<tr>
<th>Valvular lesion(s)</th>
<th>No of cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Isolated MR</td>
<td>34</td>
</tr>
<tr>
<td>Isolated MS</td>
<td>2</td>
</tr>
<tr>
<td>Isolated AR</td>
<td>2</td>
</tr>
<tr>
<td>MR + AR</td>
<td>26</td>
</tr>
<tr>
<td>MR + MS + AR</td>
<td>8</td>
</tr>
<tr>
<td>MR + TR</td>
<td>6</td>
</tr>
<tr>
<td>MR + MS</td>
<td>6</td>
</tr>
<tr>
<td>Total</td>
<td>84</td>
</tr>
</tbody>
</table>

MR = mitral regurgitation; MS = mitral stenosis; AR = aortic regurgitation; TR = tricuspid regurgitation.
Table 3: The nutritional status of 84 rheumatic heart disease patients

<table>
<thead>
<tr>
<th>Nutritional status</th>
<th>No of Patients</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Over-weight</td>
<td>0</td>
<td>0%</td>
</tr>
<tr>
<td>Normal</td>
<td>32</td>
<td>38.1</td>
</tr>
<tr>
<td>Mild to moderate malnutrition</td>
<td>44</td>
<td>52.4</td>
</tr>
<tr>
<td>Severe malnutrition</td>
<td>8</td>
<td>9.5</td>
</tr>
<tr>
<td>Total</td>
<td>84</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Overall Sensitivity and Specificity of Electrocardiographic LVH
Electrocardiographic LVH was found in 41 patients (48.8%), while echocardiographic LVH was detected in 42 patients (50.0%) if LV mass was indexed for height, or in 47 patients (56%) if it was indexed for body surface area (BSA). The overall sensitivity and specificity for electrocardiographic LVH were 71.4% and 73.8% for height-indexed echocardiographic LVH, or 68.1% and 75.7% for BSA indexed-echocardiographic LVH (Tables 4 and 5). The accuracy and predictive values of the diagnostic test were also shown.

Table 4: Sensitivity, specificity, accuracy, and predictive values of ECG LVH in 84 patients with rheumatic heart disease using height-indexed echocardiographic LVH as ‘gold standard’

<table>
<thead>
<tr>
<th>ECG LVH</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>30</td>
<td>11</td>
<td>41</td>
</tr>
<tr>
<td>No</td>
<td>12</td>
<td>31</td>
<td>43</td>
</tr>
<tr>
<td>Total</td>
<td>42</td>
<td>42</td>
<td>84</td>
</tr>
</tbody>
</table>

Sensitivity = 30/42 = 71.4% (95% CI = 57.7% to 85.1%)
Specificity = 31/42 = 73.8% (95% CI = 60.5% to 87.1%)
Accuracy = 61/84 = 72.6% (95% CI = 63.1% to 82.1%)
Positive predictive value = 30/41 = 73.2% (95% CI = 59.6% to 86.8%)
Negative predictive value = 31/43 = 72.1% (95% CI = 58.7% to 86.6%)

Table 5: Sensitivity, specificity, accuracy, and predictive values of ECG LVH in 84 patients with rheumatic heart disease using BSA-indexed echocardiographic LVH as ‘gold standard’

<table>
<thead>
<tr>
<th>Echocardiographic LVH</th>
<th>Yes</th>
<th>No</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yes</td>
<td>32</td>
<td>9</td>
<td>41</td>
</tr>
<tr>
<td>No</td>
<td>15</td>
<td>28</td>
<td>43</td>
</tr>
<tr>
<td>Total</td>
<td>47</td>
<td>37</td>
<td>84</td>
</tr>
</tbody>
</table>

Sensitivity = 30/42 = 68.1% (95% CI = 54.8% to 81.4%)
Specificity = 28/37 = 75.7% (95% CI = 61.9% to 89.5%)
Accuracy = 60/84 = 71.4% (95% CI = 61.7% to 81.1%)
Positive predictive value = 32/41 = 78.0% (95% CI = 65.3% to 80.7%)
Negative predictive value = 28/43 = 65.1% (95% CI = 50.9% to 79.3%)

Sex-adjusted Sensitivity and Specificity
When sex-specific index was performed, the sensitivity of electrocardiographic LVH were not significantly changed, both for height (x² test, df = 1, x² = 1.16, p > 0.10) and BSA-indexed (x² test, df = 1, x² = 0.04, p = 0.10) echocardiographic LVH (Table 6).

Influence of Age
When age-specific index with 13 years of age as cut-off point was worked out, the sensitivity of electrocardiographic LVH increased significantly in older patients (x² test, df = 1, x² = 8.3, p = 0.02 p < 0.01 for height-indexed LVH, x² = 10.82, p = 0.001 for BSA-indexed LVH). The improved sensitivity was not accompanied by a significant change of specificity (x², df = 1, p > 0.05). The positive predictive value of the electrocardiographic LVH improved accordingly (see Table 7). In this calculation, both sexes were combined, since the number of patients was too small to allow proper analysis.

Table 6: Sensitivity, specificity, accuracy, and predictive values of sex-specific electrocardiographic criteria for LVH

<table>
<thead>
<tr>
<th>LVH/HR : Boy</th>
<th>Ech+</th>
<th>Ech-</th>
<th>Ech+</th>
<th>Ech-</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Accuracy (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LHV/HR</td>
<td>11</td>
<td>1</td>
<td>9</td>
<td>12</td>
<td>16/23 (73.1)</td>
<td>16/24 (66.7)</td>
<td>34/47 (72.3)</td>
<td>18/26 (69.2)</td>
<td>16/31 (52.9)</td>
</tr>
<tr>
<td>Girl</td>
<td>12</td>
<td>1</td>
<td>7</td>
<td>1</td>
<td>15/19 (80.0)</td>
<td>15/18 (83.3)</td>
<td>37/37 (100)</td>
<td>12/12 (100)</td>
<td>15/12 (62.5)</td>
</tr>
<tr>
<td>LVH/BSA : Boy</td>
<td>19</td>
<td>7</td>
<td>9</td>
<td>12</td>
<td>12/19 (63.2)</td>
<td>12/19 (63.2)</td>
<td>34/47 (72.3)</td>
<td>12/26 (46.2)</td>
<td>12/26 (46.2)</td>
</tr>
<tr>
<td>Girl</td>
<td>13</td>
<td>2</td>
<td>6</td>
<td>16</td>
<td>13/19 (68.4)</td>
<td>16/18 (88.9)</td>
<td>29/37 (78.4)</td>
<td>15/37 (40.5)</td>
<td>16/32 (50.0)</td>
</tr>
</tbody>
</table>

LVH/HR = Left ventricular hypertrophy, indexed for height; LVH/BSA = Left ventricular hypertrophy, indexed for body surface area; Echo = echocardiographic; ECG = electrocardiographic; PPV = positive predictive value; NPV = negative predictive value.
Table 7: Sensitivity, specificity, accuracy, and predictive values of age-specific electrocardiographic criteria for LVH*

<table>
<thead>
<tr>
<th></th>
<th>Echo+</th>
<th>Echo-</th>
<th>Echo+</th>
<th>Echo-</th>
<th>Sensitivity (%)</th>
<th>Specificity (%)</th>
<th>Accuracy (%)</th>
<th>PPV (%)</th>
<th>NPV (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVH/H :</td>
<td>13 yrs</td>
<td></td>
<td>17</td>
<td>17</td>
<td>10/17 (58.8)</td>
<td>14/17 (82.4)</td>
<td>14/17 (82.4)</td>
<td>14/17 (82.4)</td>
<td>14/17 (82.4)</td>
</tr>
<tr>
<td></td>
<td>17 yrs</td>
<td>22</td>
<td>4</td>
<td>3</td>
<td>12/16 (75.0)</td>
<td>14/16 (87.5)</td>
<td>14/16 (87.5)</td>
<td>14/16 (87.5)</td>
<td>14/16 (87.5)</td>
</tr>
<tr>
<td>LVH/BSA :</td>
<td>13 yrs</td>
<td>10</td>
<td>3</td>
<td>15</td>
<td>10/14 (71.4)</td>
<td>15/14 (107.1)</td>
<td>15/14 (107.1)</td>
<td>15/14 (107.1)</td>
<td>15/14 (107.1)</td>
</tr>
<tr>
<td></td>
<td>15 yrs</td>
<td>22</td>
<td>4</td>
<td>3</td>
<td>12/16 (75.0)</td>
<td>14/16 (87.5)</td>
<td>14/16 (87.5)</td>
<td>14/16 (87.5)</td>
<td>14/16 (87.5)</td>
</tr>
</tbody>
</table>

* See legend of Table 6 for abbreviations.

Figure 2: Sensitivity of ECG LVH according to quartiles of severity of electrocardiographically determined LVH (BSA-indexed). Trend for increasing sensitivity with increasing left ventricular mass was seen, although statistically not significant (p > 0.05).

Discussion

Despite the wide practice of using electrocardiogram in the detection of LVH in patients with proven or suspected heart disease, its ability to do so has been questioned recently (14). Indeed, mostly in adult patients, the sensitivity of electrocardiographic LVH is astonishingly low, both when compared with autopsy findings (12,13) or with echocardiographically determined LVH (4-7).

Our series consisted of pediatric patients with rheumatic heart disease. We are unaware of any study examining the sensitivity and specificity of electrocardiographic LVH in such patients. Our results indicated that the sensitivity and specificity of electrocardiographic LVH were of moderate degree, i.e. around 70% for sensitivity and similar value for specificity (Table 4). These were different with numerous reports on adult patients, which usually have coronary heart disease or hypertension as the main cause of LVH. The sensitivity of ECG LVH was as low as 6.9% in Levy and his co-workers series (7) involving adult patients in general population. In clinical setting, the sensitivity of ECG LVH varied from 6 to 53% (14). The specificity of ECG LVH in both population and clinic-based studies (4-7), however, was much higher than ours (95% vs 70%). It means that in adult population with common heart problems in western countries, electrocardiogram is a poor tool in the detection of LVH. If no LVH is detected by ECG, there is still a great possibility that the left ventricular mass increased abnormally, but if LVH is determined electrocardiographically, the LV mass increased almost always abnormally.

In our opinion, the moderately high sensitivity found in our subjects was probably caused by several reasons, i.e.: (1) the severity of anatomic LVH in most of our study population; most of them have moderate to severe mitral regurgitation with or without other valvar lesions; (2) most of our patients were mildly to moderately undernourished (Table 3) as a result of the heart disease, the low socioeconomic level of the family, or both; thin patients tended to have higher R waves, which was the main criteria for ECG LVH; (3) we used the corrected ASE-cube formula for LV mass calculation, which certainly gave much lower values than the uncorrected formula, as proposed by Devereux et al., (12). Had we used the uncorrected formula, the LV mass would have been higher, and more LVH would have been diagnosed echocardiographically, so that the ECG LVH would have had lower sensitivity and higher specificity (see discussion below).

Sex-adjusted sensitivity of ECG LVH was not significantly different with the overall sensitivity. This also disagreed with most studies, which showed that sensitivity of ECG LVH in males was consistently higher than in females (4-7). The main explanation of the difference in adult patients is that there is attenuation of QRS voltage by greater spatial separation of the myocardium from precordial leads because of breast tissue in women (7). Certainly breast tissue would not be a problem in pediatric population, except in adolescents.

In contrast, age-adjusted sensitivity of ECG LVH showed statistically significant difference; i.e. in patients age 13 years or more, the sensitivity of ECG LVH was higher than in patients less than 13 years of age. In spite of previously described attenuation of QRS voltage with increasing
age in normal subjects, our data, like many others did, showed a trend toward increasing sensitivity of ECG for LVH with advancing age. We are of the opinion similar to that of Levy et al. (7) that this apparently paradoxical phenomena might be a consequence of increasing prevalence of echocardiographic LVH in older patients with rheumatic heart disease, and a shift toward more severe extremes of LVH in adolescents with rheumatic heart disease. This suggestion was supported by the evidence of trend of increasing sensitivity of ECG for LVH with the increasing severity of echocardiographic LVH (Figure 3). With more study population, Levy et al. (7) found a statistically significant result concerning this matter.

Regarding the use of echocardiography in the estimation of LV mass, there are several methods with good correlation coefficient with LV mass measured at necropsy. One of the most popular is the simplified ASE-cube method with the following formula:

\[ \text{LV mass (ASE-cube)} = 1.04 \times (\text{PWT} + \text{LVID} + \text{IVS})^3 - \text{LVID}^3 \]

However this method tends to overestimate the anatomic LV mass. Devereux et al. (12) suggested to correct the formula that approximated to anatomic LV mass:

\[ \text{LV mass} = 0.80 \times \text{LV mass (ACE-cube)} + 0.6 \text{gm} \]

which was used in this study.

According to Devereux et al., (12) the best method of LV mass measurement was that LV mass cube formula with Penn convention. In this method, endocardial thickness was excluded from ventricular wall and ventricular septal thickness, and included in ventricular dimension measurement. We did not use this method since we found significant difficulty in determining the exact thickness of endocardial tissue, especially in young children. In our experience, leading-edge-to-leading-edge measurements gave more reproducibility, even in a very young baby.

In contrast to in adult population, there is no standardized values for echocardiographically determined LV mass index for children because of the lack of age-appropriate upper normal limits for LVH. The study of Daniels and others (8) on the normal values of echocardiographically determined LV mass index was very important although it must be critically examined before the proposed criteria for LVH are accepted as definitive (15). We used Daniels upper limits for echocardiographically determined LVH both for height-indexed and BSA-indexed LV mass, as suggested by Snider and Serwer (16).

Of particular interest is our finding that echocardiographically determined LVH was more commonly found if LV mass was indexed for height. Many other authorities have reported that the reverse was true: more LVH is determined echocardiographically in height-indexed LV mass than in BSA-indexed LV mass (4-8). Again, we highly suspected that this conflicting observation was caused by the fact that most of our study population showed some degree of undernutrition. Even in children with normal nutritional status, the BSA/height is usually lower than in adults. With the presence of undernutrition, the body mass was considerably reduced, so that tall patients would have further decreased of BSA. In consequence, the denominator for indexing the LV mass was almost always lower in BSA-indexed LV mass than of height-indexed LV mass. Consequently, LV mass/height was mostly lower than LV mass/BSA. Our final note is that we included the 95% confidence interval in each values of sensitivity, specificity, accuracy, and predictive values in accordance with current recommendation, since it would better indicate the spread of the values of interest in relation with the number of patients studied (17,18).

In conclusion, we found that ECG was a moderate tool in detecting LVH in unselected pediatric patients with rheumatic heart disease. Its sensitivity, specificity, accuracy, and predictive values were around 70%, and they did not change significantly if sex-adjustment was performed. The sensitivity of ECG LVH was significantly higher in adolescents with rheumatic heart disease than in preadolescent patients. When applying these results, however, it should be remembered that our series consisted mainly of thin children with moderate to severe valvular lesions.

Acknowledgement
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REFERENCES


Hyperventilation in Children with Dengue Hemorrhagic Fever (DHF)

by KASIM, Y.A., ANKY TRI RINI K.E. and SUMARMO S.P.S.

(From the Department of Child Health, Medical School University of Indonesia, Jakarta)

Abstract

Many studies of Dengue Hemorrhagic Fever (DHF) have been done but only a few revealed the respiratory status. Respiratory problems arise because of plasma leakage through the damaged capillaries, causing lung edema and in turn result in hypoxemia. This later on will be compensated by a hyperventilation state.

During a 6-month period (May to September 1988), two aspects were studied in 85 patients hospitalized with DHF. First, the ventilatory pattern and second, the result of giving oxygen support in improving the respiratory disturbance, in this case alveolar hyperventilation.

The incidence of alveolar hyperventilation in DHF grade II (DHF II) and Dengue Shock Syndrome (DSS) differed significantly. Hypoxemia occurred in DHF II and DSS with no significant differences. The difference of the incidence of metabolic acidosis in DHF II and DSS were significant.

In DHF II patients having had hyperventilation state, oxygen therapy decreased respiration rate significantly and increased the PaCO₂ though not significantly.

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