Hyperventilation in Children with Dengue Hemorrhagic Fever (DHF)

by

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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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Introduction

Many studies and observations had been done by Sumarmo (1) concerning the clinical manifestations of DHF, but the respiratory disturbances were not much revealed. The result of this study on the profile of the blood gas analysis in DHF was similar to the study done by Pongpanich and Kumponpant (2) who mentioned the evidence of mild metabolic acidosis and compensated respiratory alkalosis. Miller et al., (3) presumed that respiratory alkalosis was found in relation to the increased ventilation due to stimulation by fever toward the respiratory center. Excitation and anxiety were also presumed as causative factors.

Bhamarapavati et al., (4) found evidence of pulmonary edema and pulmonary hemorrhage in the autopsy of 100 DHF cases. Radiologically, at early onset of the disease, Tamaela and Karjomanggolo (5) found evidence of lung edema and pleural effusion.

According to Kasim (6), lung edema found in DHF will disturb gas diffusion in the alveoli. Since diffusion coefficient of CO₂ is 20 times the diffusion coefficient of O₂, oxygen diffusion is more easily disturbed. Further more the patient will be hypoxicem which will be compensated by hyperventilation, resulting in a decreased PaCO₂. Hypoxemia leads to sever grades of tissue hypoxia and will be followed by increased anaerobic metabolism, lactic acid formation, and metabolic acidosis. Both will aggravate the hyperventilation state.

Materials and methods

Eighty five children with DHF who were hospitalized at the Department of Child Health, Medical School University of Indonesia since May 1988 to September 1988, were included in this study. The diagnosis was based on the criteria of the WHO (7) and was confirmed by serologic finding using hemagglutination inhibition test recommended by WHO (7).

The material consisted of 45 DHF II patients and 40 DSS patients. All of the DSS patients were given O₂ through a mask with a flow rate of 5-6 L/min for 4 hours. The patients with DHF II were again divided into 2 groups. To the first group O₂ therapy was given similar to the DSS patients, and to the other group O₂ therapy was not given. Both groups of DHF II were divided again into 2 other groups those with hyperventilation and those without. The condition of hyperventilation was recognized based on the result of blood gas analysis (PaCO₂) less-than 30 mmHg and respiration rate (more than normal rate for age).

Physical examination and blood gas analysis were done twice, at the time of admission (before giving O₂) and 4 hours after the O₂ support.

A. Descriptive

A.1. Characteristics of the samples

There were 85 patients of DHF who were hospitalized during this study (boys : girls = 1 : 1.5), and 47.06% patients were 5-9 years old. Radiologic findings of the lungs mostly revealed pleural effusions (49.41%) while 32.94% patients showed evidence of engorgement. The body temperature was 37°C - 38°C in 71.76% of cases and the serologic test were positive in 75.64% of patients.

A.2. Hyperventilation and the causing factors

A.2.1. Alveolar hyperventilation

Hyperventilation occurred in 77.78% DHF II patients and 97.5% DSS patients (table 1). The difference between these two groups was statistically significant (p < 0.05).

A.2.2. Hypoxemia

Thirty five point fifty percent (35.55%) of 45 DHF II cases and 37.5% of 40 DSS patients, were hypoxemic. The difference was not significant (p > 0.05) as shown in table 2.

The mean PaCO₂ in DHF II patients before and after oxygen support were 103.9 ± 34.5 mmHg and 119.6 ± 34.5 mmHg. In DSS, PaO₂ before and after O₂ support were 103.9 ± 34.5 mmHg and 11.4 ± 37.1 mmHg respectively.

A.2.3. Metabolic acidosis

Metabolic acidosis were noticed to happen in 60% DHF II patients and in 82.5% DSS patients. The difference was statistically significantly (table 3).

B. Clinical Trial

B.1. DHF with hyperventilation

From 30 DHF II patients with O₂ support, 26 patients fulfilled the criteria of having had hyperventilation state. From 15 DHF II patients without O₂ support, there were 9 patients with hyperventilation (table 4).

B.2. Improved respiration rate and PaCO₂ after O₂ support in DHF II patients with hyperventilation.

After O₂ support the respiration rate decreased significantly (p < 0.01), while PaCO₂ increased but not significantly (table 5).

B.3. Respiration rate and PaCO₂ changes in DHF II patients with Hyperventilation without O₂ therapy

Table 6 shows decreased respiration rate and PaCO₂ but the difference was not significant.
Table 1: Distribution of the hyperventilation state

<table>
<thead>
<tr>
<th>Stage</th>
<th>Hyperventilation state (+)</th>
<th>Hyperventilation state (-)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>DHF II</td>
<td>35</td>
<td>10</td>
<td>45</td>
</tr>
<tr>
<td>DSS</td>
<td>39</td>
<td>1</td>
<td>40</td>
</tr>
<tr>
<td>Total</td>
<td>74</td>
<td>11</td>
<td>85</td>
</tr>
</tbody>
</table>

\[X^2 = 5.685\] \[p = 0.0173\] \[P < 0.05\]

Table 2: Distribution of hypoxemia

<table>
<thead>
<tr>
<th>Stage</th>
<th>Hypoxemia (+)</th>
<th>Hypoxemia (-)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>DHF II</td>
<td>16</td>
<td>29</td>
<td>45</td>
</tr>
<tr>
<td>DSS</td>
<td>15</td>
<td>25</td>
<td>40</td>
</tr>
<tr>
<td>Total</td>
<td>31</td>
<td>54</td>
<td>85</td>
</tr>
</tbody>
</table>

\[X^2 = 0.0015\] \[p = 0.9682\] \[p > 0.05\]

Table 3: Distribution of metabolic acidosis state

<table>
<thead>
<tr>
<th>O₂ support</th>
<th>Metabolic acidosis (+)</th>
<th>Metabolic acidosis (-)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>DHF II</td>
<td>27</td>
<td>18</td>
<td>45</td>
</tr>
<tr>
<td>DSS</td>
<td>33</td>
<td>7</td>
<td>40</td>
</tr>
<tr>
<td>Total</td>
<td>60</td>
<td>25</td>
<td>85</td>
</tr>
</tbody>
</table>

\[X^2 = 4.136\] \[p = 0.0419\] \[p < 0.05\]

Table 4: Distribution of hyperventilation in DHF patients with O₂ support

<table>
<thead>
<tr>
<th>O₂ support</th>
<th>DHF II + Hyperventilation (+)</th>
<th>DHF II + Hyperventilation (-)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>(+)</td>
<td>26</td>
<td>4</td>
<td>30</td>
</tr>
<tr>
<td>(-)</td>
<td>9</td>
<td>6</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>35</td>
<td>10</td>
<td>45</td>
</tr>
</tbody>
</table>

Table 5: Improved respiration rate and PaCO₂ after O₂ therapy in DHF II patients with hyperventilation

<table>
<thead>
<tr>
<th>Improved</th>
<th>Oxygen therapy</th>
<th>Before</th>
<th>After</th>
<th>d</th>
<th>SD</th>
<th>p</th>
<th>Respiration-rate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>31.97 ± 7.21</td>
<td>29.62 ± 4.73</td>
<td>-2.30</td>
<td>3.42</td>
<td>&lt; 0.01 *</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>21.65 ± 4.51</td>
<td>22.66 ± 3.89</td>
<td>1.003</td>
<td>4.86</td>
<td>&gt; 0.05**</td>
<td></td>
</tr>
</tbody>
</table>

* t = 3.429 \[** t = 1.052\]

Table 6: Respiration rate and PaCO₂ changes in DHF II with Hyperventilation not given O₂ therapy

<table>
<thead>
<tr>
<th>Improved</th>
<th>Oxygen therapy</th>
<th>Before</th>
<th>After</th>
<th>d</th>
<th>SD</th>
<th>p</th>
<th>Respiration-rate</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>35.78 ± 8.96</td>
<td>34.44 ± 6.33</td>
<td>-1.33</td>
<td>6.71</td>
<td>&gt; 0.05 *</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>22.87 ± 3.79</td>
<td>20.07 ± 3.72</td>
<td>-2.81</td>
<td>4.94</td>
<td>&gt; 0.05**</td>
<td></td>
</tr>
</tbody>
</table>

* t = 0.594 \[** t = 1.706\]
Discussion

The highest incidence of the DHF was among the age group of 5-9 years and the ratio between girls and boys was 1.5 : 1. In 71.76% cases, the body temperature ranged from 37°C - 38°C and lung abnormalities were mostly pleural effusion (49.41%) and engorgement (32.94%). Serologic findings i.e. the hemagglutination inhibition test were found positive in 75.64% of cases. The conditions mentioned above were similar to many previous studies and therefore the cases in this study could be regarded as appropriate DHF patients.

Kasim (6) stated that in DHF, hypoxemia leads to hyperventilation. Hypoxia happens due to disturbance in oxygen diffusion because of lung edema and pleural effusion which are caused by plasma leakage.

Very probably the hyperventilation state was not due to fever since the body temperature was not too high, mostly ranging from 37°C - 38°C.

Hyperventilation occurs more frequently in DSS. It is presumed that hyperventilation in DSS is not only due to the disturbances in O₂ diffusion but also because of metabolic acidosis and shock.

There was no significant difference between the incidence of hypoxemia in DHF II and DSS patients. The hyperventilation state prevents the occurrence of hypoxemia.

In DHF II and DSS, there was only slight increase of PaO₂ after O₂ therapy. The FiO₂ in normal and healthy individual given O₂ therapy as in this study would be 40-50% therefore the FiO₂ should be around 200-300 mmHg. While in this study, the average of the maximal PaO₂ after O₂ therapy was only 154.1 mmHg. This condition showed the evidence of disturbed O₂ diffusion in DHF.

The percentage of metabolic acidosis was even higher in DSS than in DHF II and the difference was statistically significant. This result was not different with the previous studies done by Varathiyana et al. (8) and by Sumarmo (1), even though their studies were only DSS cases.

Shapiro et al., (9,10) stated that metabolic acidosis could be also lead to the hyperventilation state. Therefore, the occurrence of hyperventilation in DHF could be caused by 2 factors: disturbed O₂ diffusion and metabolic acidosis.

In DHF with no evidence of shock, the occurrence of metabolic acidosis shows that this is not caused by shock but very probably due to disturbed O₂ diffusion that leads to hypoxemia which is later compensated by having hyperventilation. Metabolic acidosis happens because of lack of O₂ in the tissue which will further activate the anaerob metabolism, resulting in increased lactic acid (lactoacidosis).

After O₂ therapy, the respiration rate decreased significantly in the studied group, while in the control group the decrease of the respiration rate was not significant. O₂ therapy also increased PaCO₂ in both groups but not significantly (tables 5 and 6).

The occurrence of lung edema and pleural effusion in DHF disturbs O₂ diffusion in the alveoli and further disturbs the respiration such as hyperventilation (6). The first change is increased respiratory rate followed by decreased PaCO₂. In DHF, the tidal volume change therefore there is only change in the respiration rate (6).

Adequate oxygenisation improves the hyperventilation state noted by decreased respiration rate and increased PaCO₂. In this study, oxygenisation decreased the respiration rate but was not followed by increased PaCO₂. Therefore, it can be said that O₂ therapy could improve the hyperventilation state by reducing the respiration rate.

Summary

1. Alveolar hyperventilation occurred in most cases of DHF II and DSS. The main cause is disturbance in oxygen diffusion which will be followed by hypoxemia and tissue hypoxia that leads to metabolic acidosis due to anaerob metabolism and accumulation of lactic acid. Therefore, adequate oxygenisation should already be given in DHF grade II with hyperventilation to improve the disturbed respiration state because of prolonged hyperventilation that will worsen the general condition due to exhaustion.

2. Giving oxygen for 4 hours through a mask with a flow rate of 5-6 l/min in DHF grade II with hyperventilation will improve the respiration rate even though not yet followed by the improvement of the PaCO₂. Oxygenisation should be given through a mask with a greater flow rate (not more than 7 l/min) if necessary depending on the patient's condition.

3. The ventilation state of the hospitalized DHF patients should be monitored to consider the needs of oxygen therapy.
REFERENCES


ORIGINAL ARTICLE

A Study of Aplastic Anemia at the Department of Child Health, School of Medicine University of North Sumatera/Dr. Pirngadi Hospital, Medan

by

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(From the Department of Child Health, School of Medicine, University of North Sumatera/Dr. Pirngadi Hospital, Medan)

Abstract

A study of aplastic anemia in children in a period of June 1980 to June 1989 was done to evaluate the pattern of aplastic anemia in children. The cases consisted of 55 children, 30 males (54.55%) and 25 females (45.45%). Most of the patients (47.27%) were found in the age group of 10 to 15 years. The complaints were paleness (90.91%), fever (56.45%) and bleeding (52.72%). The hemoglobin concentration was 4.25 ± 1.17 g/dl (mean ± SD), and the thrombocytopenia was generally severe. Prednisone or combination of prednisone and oxymethalone was given in addition to blood transfusions, antibiotics and vitamins. The outcome was difficult to evaluate because of the irregularity of treatments. Of the 55 children, 9 (18.02%) contracted leukemia after 1 - 5 months.