ORIGINAL ARTICLE

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 $PaCO_2$ 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 CO_2 is 20 times the diffusion coefficient of O_2, oxygen diffusion is more easily disturbed. Further more the patient will be hypoxemic which will be compensated by hyperventilation, resulting in a decreased $PaCO_2$. Hypoxemia leads to several grades of tissue hypoxia and will be followed by increased anaerob 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 O_2 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 O_2 therapy was given similar to the DSS patients, and to the other group O_2 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 ($PaCO_2$) 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 O_2) and 4 hours after the O_2 support.

Results

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 five 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 $PaCO_2$ in DHF II patients before and after oxygen support were 102.9 ± 39.5 mmHg and 119.6 ± 34.5 mmHg. In DSS, PaO_2 before and after O_2, 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 O_2 support, 26 patients fulfilled the criteria of having had hyperventilation state. From 15 DHF II patients without O_2 support, there were 9 patients with hyperventilation (table 4).

B.2. Improved respiration rate and $PaCO_2$ after O_2 support in DHF II patients with hyperventilation.

After O_2 support the respiration rate decreased significantly ($p < 0.01$), while $PaCO_2$ increased but not significantly (table 5).

B.3. Respiration rate and $PaCO_2$ changes in DHF II patients with Hyperventilation without O_2 therapy

Table 6 shows decreased respiration rate and $PaCO_2$ but the difference was not significant.

Table 1 : *Distribution of the hyperventilation state*

Stage	Hyperventilation state		Total
	(+)	(−)	
DHF II	35	10	45
DSS	39	1	40
Total	74	11	85

$X^2 = 5{,}685$ p = 0,0173 P<0,05

Table 2 : *Distribution of hypoxemia*

Stage	Hypoxemia		Total
	(+)	(−)	
DHF II	16	29	45
DSS	15	25	40
Total	31	54	85

$X^2 = 0{,}0015$ p = 0,9682 p > 0,05

Table 3 : *Distribution of metabolic acidosis state*

O_2 support	Metabolic acidosis		Total
	(+)	(−)	
DHF II	27	18	45
DSS	33	7	40
Total	60	25	85

$X^2 = 4{,}136$ p = 0,0419 p< 0,05

Table 4 : *Distribution of hyperventilation in DHF patients with O_2 support*

O_2 support	DHF II + Hyperventilation		Total
	(+)	(−)	
(+)	26	4	30
(−)	9	6	15
Total	35	10	45

Table 5 : *Improved respiration rate and $PaCO_2$ after O_2 therapy in DHF II patients with hyperventilation*

Improved	Oxygen therapy		d	SD	p
	Before	After			
Respiration-rate	31,97 ± 7,21	29,62 ± 4,73	−2,30	3,42	< 0,01 *
$PaCO_2$	21.65 ± 4,51	22,66 ± 3,89	1,003	4,86	> 0,05**

* t = 3,429 ** t = 1,052

Table 6 : *Respiration rate and $PaCO_2$ changes in DHF II with Hyperventilation not given O_2 therapy*

Improved	Oxygen therapy		d	SD	p
	Before	After			
Respiration-rate	35,78 ± 8,96	34,44 ± 6,33	−1,33	6,71	> 0,05 *
$PaCO_2$	22,87 ± 3,79	20,07 ± 3,72	−2,81	4,94	> 0,05**

* 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 O_2 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 PaO_2 after O_2 therapy. The FiO_2 in normal and healthy individual given O_2 therapy as in this study would be 40-50% therefore the FiO_2 should be around 200-300 mmHg. While in this study, the average of the maximal PaO_2 after O_2 therapy was only 154,1 mmHg. This condition showed the evidence of disturbed O_2 diffusion in DHF.

The persentage 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 Varavithya 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 O_2 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 O_2 diffusion that leads to hypoxemia which is later compensated by having hyperventilation. Metabolic acidosis happens because of lack of O_2 in the tissue which will further activate the anaerob metabolism, resulting in increased lactic acid (lactoacidosis).

After O_2 therapy, the respiration rate decreased significantly in the studied group, while in the control group the decrease of the respiration rate was not significant. O_2 therapy also increased $PaCO_2$ in both groups but not significantly (tables 5 and 6).

The occurrence of lung edema and pleural effusion in DHF disturbs O_2 diffusion in the alveoli and further disturbs the respiration such as hyperventilation (6). The first change is increased respiratory rate followed by decreased $PaCO_2$. 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 $PaCO_2$. In this study, oxygenisation decreased the respiration rate but was not followed by increased $PaCO_2$. Therefore, it can be said that O_2 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 hypoxemi 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 $PaCO_2$. 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.

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