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ORIGINAL ARTICLE

Nutritional Status of Dengue Haemorrhagic Fever in Children

by

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Abstracts

Shock in Dengue infection is especially caused by an immunologic process (secondary reaction).

This process may occur when the activity of Lymphocytes (C.M.I.), complement system and macrophages is good. This activity, however is disturbed in undernutrition.

In this study on 126 DHF/DSS patients, fulfilling the WHO criteria (1975), the cases of shock were mostly found in patients with a body weight of more than 80% Harvard standard.

There was no case of shock, in patients with a nutritional status under 60% of the Harvard standard.

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Introduction

Dengue Haemorrhagic Fever (DHF) is an endemic disease in Southeast Asia. In Indonesia it has been reported, that there are 5000 — 10,000 patients per year. The mortality was about 10% of the patients admitted to the hospital (Morley, 1979) and shock was found in 5 — 40% of all the patients (Nelson, 1979; Halstead, 1980).

The process of shock in DHF may be caused by the activity of lymphocytes (C.M.I.), complement system and macrophages. This activity in children with undernutrition is disturbed.

In Indonesia about 30% of children are suffering from mild and moderate PCM, and 3 — 5% from severe PCM (Pelita III Kesehatan, 1978).

The aim of this study is to know the correlation between the two factors above.

Material and Methods

One hundred and twenty six DHF patients admitted to the Department of Child Health, University of Gajah Mada Hospital from January 1979 until December 1980, were included in this study.

The diagnosis of DHF is primarily based on the WHO criteria (1975) such as:

Clinical criteria:
— acute and high fever for 2 — 7 days.
— haemorrhage:
— positive tourniquet test
— petechiae, purpura, ecchymoses — epistaxis, haemorrhage of the gum
— haematemesis, melena.
— hepatomegaly
— shock, with manifestation of rapid and small pulse, pressure difference of systolic and diastolic is short (20 mmHg or less) or signs of hypotension, cold skin, clammy and nervousness (restlesses).

Laboratory examination:
— thrombocytopenia of 100,000 per mm³ or less and
— haemoconcentration with increasing haematocrit of 20% or more.

The clinical diagnosis of DHF can be determined by two or three of the clinical criteria, followed by thrombocytopenia and haemoconcentration.

If shock with high haematocrit (except severe haemorrhage) followed by severe thrombocytopenia, there is a possibility of DHF/DSS.

Haemoconcentration is always concomitant with thrombocytopenia in DHF (Nimmannitya, 1975).

Severe and mild DHF are divided into 2 groups namely DHF with and without shock.

The nutritional status is evaluated according to the Workshop on Nutritional Anthropometry in Jakarta (1975), as follows:

I. The nutritional condition (is classified into):
1. Good nutrition or "well nourished",
2. Malnutrition or "underweight", included "mild" and "moderate PCM" (Protein Calorie Malnutrition).
3. Bad nutrition or "severe PCM" included marasmus, marasmic-kwashiorkor and kwashiorkor.

II. Limit line used is weight for age such as:
1. Limit line of percentile 50 Harvard is the same with 100%;
2. Under limit line of good nutrition is 80%;
3. Under limit line of malnutrition is 60%;
4. Under limit line of 60% is bad nutrition.

The body weight of the patient is measured on the first day of hospitalization.

Results

The Dengue Haemorrhagic Fever patients consisted of 55 (43.7%) males and 71 (56.3%) females.

Fifty eight (46%) out of 126 DHF patients were in the shock condition and 6 (10.3%) out of 58 shock patients died (Table 1).

Four (3.2%) out of the dead patients were in the state of undernutrition (mild PCM) (Table 2).

Shock was mostly found in patients aged between 5 to 10 years. (Table 3).

There was no significant difference (p > 0.05) between patients with good nutrition and undernutrition in correlation with shock (Table 4).

The relationship above is illustrated by histogram (Fig. 1, 2, 3).

| Table 1: Number of patients according to sex, severe/mild DHF |
| --- | --- | --- | --- |
| | Shock | Without Shock | Total |
| Sex | Number of cases | % | Number of cases | % | Number of cases | % |
| Male | 22 | 17.5 | 33 | 26.2 | 55 | 43.7 |
| Female | 36 | 28.6 | 35 | 27.8 | 71 | 56.3 |
| Total | 58 | 46.0 | 68 | 54.0 | 126 | 100 |

6 died (10.3%)
### TABLE 2: Nutritional status and sex of DHF patients who died

<table>
<thead>
<tr>
<th>Nutritional status</th>
<th>Died</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>8%</td>
<td>9%</td>
</tr>
<tr>
<td>Well nourished</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PCM I</td>
<td>1</td>
<td>0.8%</td>
</tr>
<tr>
<td>Underweight PCM II</td>
<td>4</td>
<td>3.2%</td>
</tr>
<tr>
<td>Severe PCM</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>1</td>
<td>8%</td>
</tr>
</tbody>
</table>

### TABLE 3: Number of patients according to age and severe/mild DHF

<table>
<thead>
<tr>
<th>Age</th>
<th>Shock</th>
<th>Without shock</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of cases</td>
<td>%</td>
<td>Number of cases</td>
</tr>
<tr>
<td>&lt; 2 years</td>
<td>1</td>
<td>0.8</td>
<td>-</td>
</tr>
<tr>
<td>2—&lt; 5 years</td>
<td>12</td>
<td>9.5</td>
<td>7</td>
</tr>
<tr>
<td>5—&lt; 10 years</td>
<td>38</td>
<td>30.2</td>
<td>48</td>
</tr>
<tr>
<td>10—14 years</td>
<td>7</td>
<td>5.6</td>
<td>13</td>
</tr>
<tr>
<td>Total</td>
<td>58</td>
<td>46.0</td>
<td>68</td>
</tr>
</tbody>
</table>

### TABLE 4: Correlation between nutritional status and severe/mild DHF

<table>
<thead>
<tr>
<th>Nutritional status</th>
<th>Shock</th>
<th>Without shock</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Number of cases</td>
<td>%</td>
<td>Number of cases</td>
</tr>
<tr>
<td>Well nourished</td>
<td>24</td>
<td>19.0</td>
<td>30</td>
</tr>
<tr>
<td>Underweight</td>
<td>34</td>
<td>27.0</td>
<td>38</td>
</tr>
<tr>
<td>Total</td>
<td>58</td>
<td>46.0</td>
<td>68</td>
</tr>
</tbody>
</table>

p > 0.05
FIG. 3: Correlation between nutritional status and DHF

Number of cases in %

40
30
20
10
0

Nutritional status

Well nourished - Underweight
Severe PCM - POM I - POM II
Died -

FIG. 4: Scheme for "Immunological Enhancement of Infection" Hypothesis of DHF/DSS Pathogenesis

(A) Mosquito bite → + Y → Fe receptor → released virus 
spread to tissues

(B) Gut → lymph node → liver → virus cascade

(C) Lymphoblast → C3a → C3b → Factor B → complement activation 
lymphoblast lysis 
immune elimination 
vasoconstriction 
shock

Disseminated intravascular clotting → hemorrage

(Halstead, 1980)
Discussion

In this study the diagnosis of DHF was based on WHO clinical criteria (WHO, 1975).

Study in the Bantul area (included Daerah Istimewa Yogyakarta) revealed positive virological examination in 73% and serologically Dengue type 3 in 65%. The Department of Child Health, Gajah Mada University Hospital, in 1970 found 80% positive serological examination of 48 cases (Ismangoen et al., 1972; Gubler et al., 1976).

In previous studies patients suffering from shock were about 10 — 40% (Halstead, 1980), about 5 — 40% (Nelson, 1979), whereas in the Department of Child Health, Gajah Mada University Hospital, DSS cases (average 10% of annual admission), had a mortality rate of about 10 — 20% (Yati et al., 1977).

In our study, there were 58 DSS patients (46%) and 6 (4.6%) out of 126 DHF cases died. This result is not much different from other studies.

Some investigators reported that patients suffering from DHF, generally had a well — nourished condition and DSS was rarely found in undernourished patients, especially in severe PCM (Gubler et al., 1976; Morley, 1979; Sumarmo, 1978; Halstead, 1980); Shock in DHF is an immunologic reaction, while in PCM patients the T lymphocyte function is disturbed, the number of lymphocytes decreases, and the macrophage function is also disturbed (Bhaskaram and Reddy, 1974; Tjokronegoro, 1976; Chandra, 1979).

Halstead et al. (1978) presented a scheme for "immunological enhancement of infection" hypothesis of DHF/DSS pathogenesis (Fig. 4), as follows:

(A) Afferent mechanism.

In the presence of enhancing antibody (antibody which complexes with virus but does not kill) immune complex attaches to an Fc receptor triggering phagocytosis followed by replication of virus. Because mononuclear phagocytes may be motile, they may spread infection as provide a site for replication.

(B) Efferent mechanism.

Dengue virus grows in bone marrow, liver, spleen, lymphoid tissue, and histiocytes in the skin. A central tenet of the immune enhancement hypothesis is that enhancing antibody regulates the number of cells infected: the more cells infected, the more severe is the disease.

(C) Effector mechanism.

It is believed that the major pathophysiological abnormalities are caused by factors released from activated mononuclear phagocytes infected with Dengue. The immune response, possibly T lymphocytes, may be important in activating mononuclear phagocytes.

Nutrition, sex, and genetic factors may be important in modulating individual infection through their regulatory effect on T lymphocytes function.

In our study the patients consisted of 54 (42.8%) who were well nourished and 72 (57.2%) who were underweight. Shock was found in 24 out of 54 well nourished cases and in 34 of 72 underweight patients.

There was no significant difference (p > 0.05) between patients with good nutritional status and undernutrition in correlation with shock.

No shock was found in severe PCM patients whose weight was under 60% Harvard standard.

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The Incidence of Congenital Malformation in the Gadjah Mada University Hospital Yogyakarta During 1974—1979

by

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Abstract

A study of the incidence of congenital malformation among 4625 newborns in the neonatal unit, Gadjah Mada University Hospital Yogyakarta, during a period of 5 years (1974 — 1979) is presented. The incidence of 1.64% out of 4625 newborns was found.

The three most frequent malformations were hydrocephalus (21%), cleft lip and cleft palate (9.2%) and Down’s Syndrome (9.2%).

A total of 40% were found in multiparous mothers between 20 — 34 years of age, and of 26% in grandmultiparous mothers older than 35 years.

According to Arey’s distribution of congenital anomalies, developmental arrest was most prevalent (59.2%).

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