

---

ORIGINAL ARTICLE

---

Nutritional Status of Dengue Haemorrhagic  
Fever in Children

by

SUGIYANTO; TELUK SEBODO; SUTARYO; SUMINTA and ISMANGOEN

(From the Department of Child Health, Medical School, Gajah Mada  
University, Yogyakarta)

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.*

### 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 (restlessness).

#### Laboratory examination:

- thrombocytopenia of 100.000 per mm<sup>3</sup> 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 Anthropometri in Jakarta (1975), as follows:

I. The nutritional condition (is classified into):

1. Good nutrition or "well nourished",

### 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).

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.

TABLE 1: Number of patients according to sex, severe/mild DHF

Sex	Shock		Without Shock		Total	
	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
0 : 0	1 : 1.6		1 : 1.06		1 : 1.3	

6 died (10.3%)

TABLE 2: Nutritional status and sex of DHF patients who died

Nutritional status	D i e d				T o t a l	
	♂	%	♀	%	Σ	%
Well nourished	1	0.8	1	0.8	2	1.6
Underweight PCM I	—	—	4	3.2	4	3.2
Severe PCM PCM II	—	—	—	—	—	—
T o t a l	1	0.8	5	4.0	6	4.8

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

A g e	S h o c k		Without shock		T o t a l	
	Number of cases	%	Number of cases	%	Number of cases	%
< 2 years	1	0.8	—	—	1	0.8
2 — < 5 years	12	9.5	7	5.6	19	15.1
5 — < 10 years	38	30.2	48	38.1	86	68.3
10 — 14 years	7	5.6	13	10.3	20	15.8
T o t a l	58	46.0	68	54.0	126	100

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

Nutritional status	S h o c k		Without shock		T o t a l	
	Number of cases	%	Number of cases	%	Number of cases	%
Well nourished	24	19.0	30	23.8	54	42.8
Underweight	34	27.0	38	30.2	72	57.2
T o t a l	58	46.0	68	54.0	126	100

p &gt; 0.05

FIG. 1: Correlation between nutritional status and shock in DHF

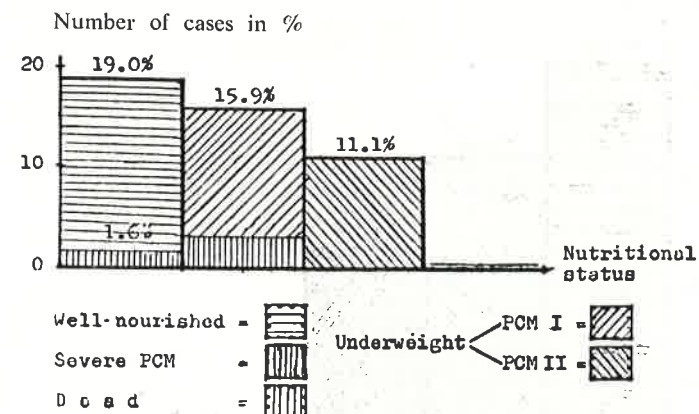


FIG. 2: Correlation between nutritional status and without shock in DHF

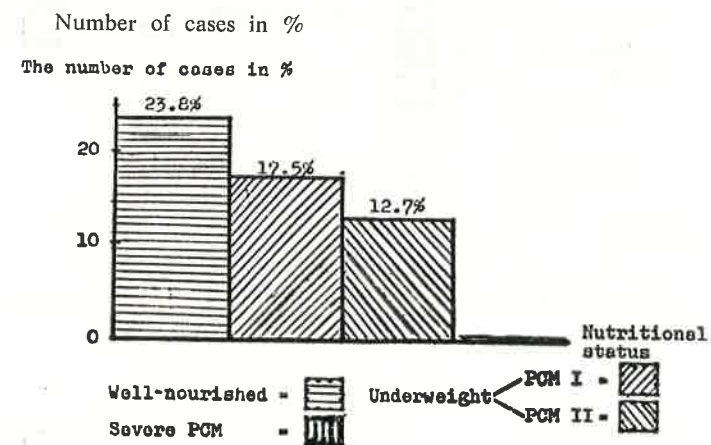
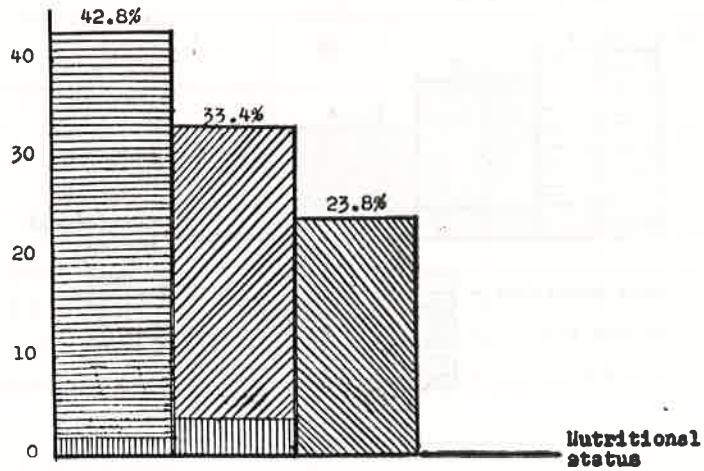


FIG. 3: Correlation between nutritional status and DHF

Number of cases in %






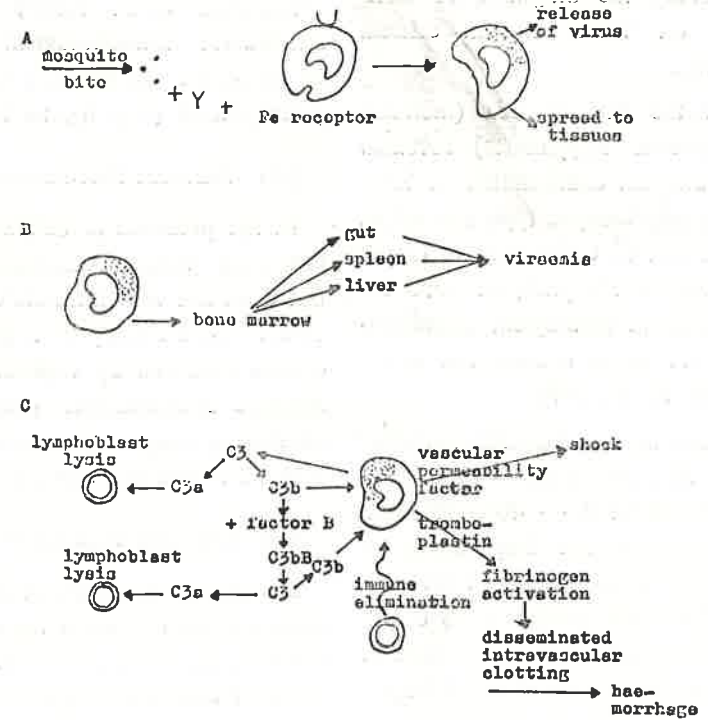
Well nourished =  Underweight  $\left\{ \begin{array}{l} \text{PCM I} = \text{diagonal lines pattern} \\ \text{PCM II} = \text{diagonal lines pattern} \end{array} \right.$   
 Severe PCM =   
 Died = 

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



( 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 out 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 Red-

dy, 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.

### Summary

This study may be summarized as follows:

- No significant difference was found in correlation with nutritional status and shock in DHF.
- No shock was found in severe PCM patients.
- More immunologic examinations on PCM patients and DHF were needed.

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.

### REFERENCES

1. BHASKARAM, C; REDDY, V.: Cell mediated immunity in protein calorie malnutrition. *Environ. Child Health* 20: 284 (1974).
2. CHANDRA, R.K.: Interaction of nutrition, infection and immune response. *Acta paediat. scand.* 68: 137 - 144 (1979a); T and B lymphocyte subpopulations and leucocyte terminal desoxynucleotidyl transferase in energy protein undernutrition. *Acta paediat. scand.* 68: 841 - 845 (1979 b).
3. GUBLER, D.J.; ERAM, S.; JUMALI; SETIOBUDI, Y.; SADONO, T.I.; SUTRISNO D.S.; SULIANTI SAROSO. J.: Dengue Haemorrhagic Fever in Bantul, Yogyakarta, Indonesia. *Bull. penelitian health studies in Indonesia.* Vol- II, No. I, 1979 (1976).
4. HALSTEAD, S.B.: Dengue Haemorrhagic Fever — a public health problem and a field for research. *Bull. WHO* 58 (I): 1 - 21 (1980).
5. HALSTEAD, S.B., MARCHETTE, D.N.; O'ROURHE, E.: Immunologically enhanced Dengue virus infection of mononuclear phagocyte. A mechanism which may regulate diseases severity. *Asian J. infect. Dis.* 2: 85 - 93 (1978).
6. ISMANGOEN; SAMIK WAHAB; RACHMAT SOETRISNO; ACHMAD SURYONO: Dengue Haemorrhagic fever in Yogyakarta, Central Java. *Pediatr. Indones.* 12: 49 - 54 (1972).
7. LOKAKARYA ANTROPOMETRI GIZI, Jakarta, 29 - 31 Mei 1975.
8. MORLEY, D.: Dengue Haemorrhagic Fever. *Paediatric priorities in the developing world*, 1st ed. (Butterworth, London/Boston 1976).
9. NELSON, W.E.: *Textbook of Paediatrics*, 11th Asia ed., pp. 942 - 944 (Igaku Shoin, Tokyo 1979).
10. NIMMANNITYA, S.: Dengue Haemorrhagic fever, *Problem and progress.* *Pediatr. Indones.* 15: 93 - 104 (1975).
11. PELITA III, KESEHATAN, Departemen Kesehatan R.I., 1978.
12. SUMARMO: Dengue Haemorrhagic fever, *Problems and progress.* Workshop of the national institute of health research and development, Ministry of Health, Jakarta, April 25 - 27, 1978.

13. TJOKRONEGORO, A. : Immunology in protein calorie malnutrition. Presented at the regional graduate applied nutrition course. CCBTM & PH — SEAMEO, Jakarta (1976).
14. W.H.O. : Technical guide No. 1. Clinical diagnosis and surveillance, preventive and control of Dengue Haemorrhagic Fever (1975).
15. YATI, S., SEBODO, T.; UTOMO; ISMANGOEN : Dengue Haemorrhagic Fever, Pertemuan Ilmiah Fakultas Kedokteran UGM. p. 56 - 69 (1977).