Table of Contents

<table>
<thead>
<tr>
<th>Section</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Epidemiology of Dengue Fever in Malaysia</td>
<td>3-4</td>
</tr>
<tr>
<td>2. Virology and Laboratory Diagnosis</td>
<td>4-6</td>
</tr>
<tr>
<td>3. Pathophysiology of Dengue Haemorrhagic Fever</td>
<td>7</td>
</tr>
<tr>
<td>4. Definition of Dengue Fevers (WHO 1997)</td>
<td>8-10</td>
</tr>
<tr>
<td>5. Clinical Features of Dengue Infection</td>
<td>11-14</td>
</tr>
<tr>
<td>6. Clinical Course of Dengue Haemorrhagic Fever</td>
<td>15</td>
</tr>
<tr>
<td>7. Criteria for Hospitalisation</td>
<td>16-18</td>
</tr>
<tr>
<td>8. Management of Dengue Infection</td>
<td>19-23</td>
</tr>
<tr>
<td>8.1 Management of Dengue Haemorrhagic Fever</td>
<td>19-20</td>
</tr>
<tr>
<td>8.2 Management of Dengue Shock Syndrome</td>
<td>21</td>
</tr>
<tr>
<td>8.3 Management of Pleural Effusion</td>
<td>21</td>
</tr>
<tr>
<td>8.4 Indications for Blood Products in Dengue Infection</td>
<td>22</td>
</tr>
<tr>
<td>8.5 Management of Upper Gastrointestinal Bleeding in Dengue Patients</td>
<td>22</td>
</tr>
<tr>
<td>8.6 Intensive Care in Dengue Infection</td>
<td>23</td>
</tr>
<tr>
<td>9. Criteria for Discharging Patients Hospitalised with Dengue Infection</td>
<td>24</td>
</tr>
<tr>
<td>10. References</td>
<td>25</td>
</tr>
</tbody>
</table>
EPIDEMIOLOGY OF DENGUE FEVER IN MALAYSIA

Dengue is the most common and widespread arthropod-borne arboviral infection in the world today. The geographical spread, incidence and severity of dengue fever (DF) and dengue haemorrhagic fever (DHF) are increasing in the Americas, South-East Asia, the Eastern Mediterranean and the Western Pacific. Some 2,500 million to 3,000 million people live in areas where dengue viruses can be transmitted. It is estimated that each year 50 million infections occur, with 500,000 cases of DHF and at least 12,000 deaths.¹

Dengue fever was first reported in 1902 in Penang² and has become a major public health problem in Malaysia, especially since the appearance of the first DHF outbreak also in Penang in 1962.³ Rapid industrial and economic development over the last two decades have brought about massive infrastructure development, creating man-made environment for breeding of Aedes mosquito.

Notification of DF and DHF in Malaysia was implemented in 1971. Under the Prevention and Control of Infectious Disease Act 1988, it is compulsory for all Medical Officers to notify all cases of DF, DHF and deaths due to dengue infection. All suspected, probable or confirmed cases are to be notified by the attending Physicians/Medical Officers to the nearest district health office within 24 hours. Early notification is essential for control measures to be instituted immediately to prevent outbreaks.

The incidence rate of clinically diagnosed DF and DHF reported is showing an upward trend from 8.5 cases/100,000 population in 1988 to 123.4 cases/100,000 population in 1998. Out of 16,368 cases reported in the year 2001, 22% were among children 14 years and below. Similarly the case fatality rate (CFR) for DHF is high, ranging 5% to 6% per annum for both children and adults. As expected, there are more cases of DF than DHF, with a ratio of 16 – 25:1 over the last 5 years. In the year 2001, the DF:DHF ratio in children was 6.7:1 as compared to 27.3:1 in adults⁴

Most of the cases are reported among the urban population (70 – 80%) with the highest incidence in the working and school going age group which correlates with the relatively high Aedes Index in construction sites, factories and schools.⁴
Table: NUMBER OF CASES, DEATHS AND INCIDENCE RATES (per 100,000) FOR DENGUE FEVER (1988-2001)

<table>
<thead>
<tr>
<th>YEAR</th>
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<th>TOTAL</th>
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<td>68.79</td>
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VIROLOGY AND LABORATORY DIAGNOSIS

Dengue virus is an Arbovirus that belongs to the family *Flaviviridae*, under the genus *Flavivirus*. In the past, it was classified under the Group B Arboviruses. It is a small enveloped virus measuring 50 to 60 nm in size containing a single stranded positive sense RNA genome.

Dengue virus is transmitted via the bite of Aedes mosquitoes in particular *A. aegypti* & *A. albopictus*. In human disease the cycle of transmission involves man-vector-man.

The virus is present in blood in early acute phase only, generally for 1-5 days. The incubation period varies between 3 to 10 days with an average of 4-6 days.5

There are 2 patterns of host serological responses namely the Primary and Secondary immune response. The primary response occurs in non immune individuals undergoing their first dengue virus infection, while the secondary (anamnestic) response occurs in individuals having memory cells during a repeat dengue infection.

*Dengue Virus Serotype*

There are four serotypes of dengue virus (DEN-1, DEN-2, DEN-3 and DEN-4). They are antigenically very similar to each other but different enough to elicit only transient partial cross-protection after infection by each one of them. Antibodies to one type cross react in tests with other antigens therefore serological antibody tests, in general, do not differentiate between dengue serotypes.
The 4 serotypes of the Dengue virus may all circulate concurrently in the same season but in different geographical regions. One serotype may predominate over other serotypes depending on the susceptibility or immunity of the population. The trends in circulating dengue viral serotypes in Malaysia have been studied and found to predominate in a cyclical pattern. In the period from 1990-1995, DEN-3 was the predominant serotype and followed by DEN-2 between 1997-2000. In the year 2001 DEN-3 reappeared and predominated over DEN-2.

**Laboratory diagnosis of dengue**

1) Serology
2) Virus isolation
3) Molecular technique (Amplification and detection of dengue ribonucleic acids by RT-PCR)

**Serology**

This is the most practical method available for the laboratory diagnosis of dengue infection. The traditional haemagglutination inhibition (HI) test is still being used. However, it is labour-intensive, requiring 3 days to perform with both acute and convalescent serum samples and hence the diagnosis is retrospective.

The serological test of choice today is the *Enzyme Linked Immunosorbent Assay (ELISA)*, which is simpler, faster and is also the mainstay of most hospital laboratories in this country. Some points are worth considering with respect to ELISA IgM:

- Dengue-specific IgM appears in both Primary & Secondary infection
- Only about 60% of Dengue infection may be diagnosed on day 5 or 6 of illness with single serum by IgM test and 100% with paired sera (7 to 14 days apart)
- IgM is more specific for flavivirus infection than IgG
- IgM testing can be done in one day (turnaround time of 5 hours).

**Note:**

- The interpretation of serological results must be carefully considered with respect to clinical features of the illness and NOT interpreted in isolation.
- A positive dengue IgM result indicates acute or recent past infection (up to 90 days) however a single positive dengue IgM is not necessarily indicative of a present dengue infection.
- ELISA IgM results may be negative in early acute blood specimen and a repeat specimen should be tested before confirming or excluding dengue infection.

Simple rapid tests such as the strip assays are available for the rapid detection of specific IgM and IgG but are very costly.
**Virus isolation**

This is the *most definitive method* for the diagnosis of dengue infection. It is only performed in a few research laboratories because it is laborious, time consuming, costly and it is sensitive only if the blood is collected in the early acute phase of illness.

**Molecular technique**

Available as a research tool in very few laboratories.

**Autopsy Specimens**

Post mortem specimens include biopsy from lymph nodes, spleen, liver, brain. Collect in sterile bottle/container. (No preservatives required)
Transport in dry ice. It is important to communicate with the relevant virology laboratory prior to dispatch of specimen. If delay in transport is anticipated, store the sample in minus 70 °C or in dry ice.
PATHOPHYSIOLOGY OF DENGUE HAEMORRHAGIC FEVER

DHF

↑ VASCULAR PERMEABILITY

plasma leakage

THROMBOCYTOPENIA

< 100 x 10^9/L

COAGULOPATHY

↑ PT/APTT

HYPOVOLEMIA

(↑ Hct)

HYPOVOLEMIC SHOCK

With appropriate fluid therapy

SEVERE HAEMORRHAGE

(normal/↓ Hct)

with appropriate blood/component support

Tissue Acidosis

MULTIORGAN FAILURE

INTRACTABLE SHOCK

RECOVERY

RECOVERY

DIC

Notes:

- Plasma leakage is a hallmark of DHF.
- The haematocrit (Hct) is usually > 40% and may be as high as 55-65%.
- Platelet deficiency and dysfunction are constant features in dengue infection.
- Vasculopathy, consumption coagulopathy, overt DIC (disseminated intravascular coagulation) and shock can enhance bleeding which increase the morbidity and mortality.
- Increased immune complexes, complement activation and various cytokine activities have been implicated in dengue infection.
DEFINITION OF DENGUE FEVERS (WHO 1997)^1

Classic Dengue Fever

Given the variability in the clinical illness associated with dengue infection, it is not appropriate to adopt a detailed clinical definition of dengue fever. Rather, the need for laboratory confirmation is emphasised.

The following classifications are proposed:

- **probable** – an acute febrile illness with two or more of the following manifestations:
  - headache
  - retro-orbital pain
  - myalgia
  - arthralgia
  - rash
  - haemorrhagic manifestations
  - leukopenia;

* (any case fulfilling the above criteria should be notified to the health authorities, even before serology is known)

**and**

- supportive serology (refer to laboratory diagnosis)

**or**

- occurrence at the same location and time as other confirmed cases of dengue fever.

- **Confirmed** – a case confirmed by laboratory criteria (see below)
- **Reportable** – any probable or confirmed case should be reported.

**Laboratory criteria for confirmation of dengue fever are:**

- Isolation of the dengue virus from serum or autopsy samples; or
- Demonstration of a fourfold or greater change in reciprocal IgG or IgM antibody titres to one or more dengue virus antigens in paired serum samples; or
- Demonstration of dengue virus antigen in autopsy tissue, serum or cerebrospinal fluid samples by immunohistochemistry, immunofluorescence or ELISA; or
- Detection of dengue virus genomic sequences in autopsy tissue, serum or cerebrospinal fluid samples by polymerase chain reaction (PCR).
**Dengue Haemorrhagic Fever**

The critical stage is reached at the end of the febrile phase of illness; accompanying or shortly after a rapid drop in temperature varying degrees of circulatory disturbances occurs. This phase rarely lasts longer than 48 hours.

The following must all be present:

1. Fever, or history of acute fever, lasting 2-7 days, occasionally biphasic.
2. Haemorrhagic tendencies, evidenced by at least one of the following:
   a. a positive torniquet test
   b. petechiae, ecchymoses, or purpura
   c. bleeding from the mucosa, gastrointestinal tract, injection sites or other locations
3. Thrombocytopenia (100,000/mm$^3$ or less)
4. Evidence of plasma leakage due to increased vascular permeability, manifested by at least one of the following:
   a. haemoconcentration (equal to or greater than 20% above average for age, sex and population)
   b. a drop in haematocrit following volume replacement equal to or greater than 20% of haematocrit at presentation.
   c. signs of plasma leakage evidenced by pleural effusion, ascites and hypoproteinemia.

Other clinical manifestations suggestive of DHF are

   a. hepatomegaly which may be tender
   b. circulatory disturbance

**Dengue Fever versus Dengue Haemorrhagic Fever**

- Thrombocytopenia with concurrent haemoconcentration differentiates Grades I and II DHF from classic DF.
- Increased capillary permeability is the main pathophysiology that differentiates DHF/DSS from DF. Based on the severity of capillary permeability, DHF is graded into grade I to IV.
- Therefore your notification is to be either DF or DHF.
- Case fatality rate is calculated only for DHF cases.

*(Revision of the final diagnosis from DF to DHF should be done for all cases if it meets the criteria. This must be followed by re-notification to the health authorities.)*

**Dengue Shock Syndrome**

All the above 4 criteria for DHF must be present, plus evidence of circulatory failure manifested by:
- rapid and weak pulse
- narrow pulse pressure less than 20mmHg
or manifested by
- hypotension for age
- cold clammy skin and restlessness

**WHO grading of DHF/DSS**

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Grade I</td>
<td>In the presence of haemoconcentration, fever and non-specific constitutional symptoms, a positive torniquet test is the only haemorrhagic manifestation</td>
</tr>
<tr>
<td>Grade II</td>
<td>spontaneous bleeding in addition to the manifestation from Grade I</td>
</tr>
<tr>
<td>Grade III*</td>
<td>circulatory failure, pulse pressure less than 20 mmHg but systolic pressure is still normal</td>
</tr>
<tr>
<td>Grade IV*</td>
<td>profound shock, hypotension or unrecordable blood pressure.</td>
</tr>
</tbody>
</table>

* Grades III and IV are classified as DSS.

**CLINICAL FEATURES OF DENGUE INFECTION**

Dengue virus infection may present in four different clinical syndromes.

1. Undifferentiated fever
2. Classic dengue fever
3. Dengue Haemorrhagic Fever [DHF]
4. Dengue Shock Syndrome [DSS]

**Pointers to the clinical diagnosis of dengue infection**

1. high continuous fever of 3 days or more
2. headache, backache and retro-orbital pain
3. abdominal pain, vomiting, loose stools
4. petechial haemorrhage and/or spontaneous bleeding
5. rash – generalised flushing/maculopapular
6. hepatomegaly
7. fall in platelet count that precedes or occurs simultaneously with a rise in the haematocrit
8. normal WBC or leukopenia with relative lymphocytosis
9. normal ESR (<20mm first hour)
10. shock

**Note:** all criteria need not be present at the same time
Other manifestations and organ involvement

1. Haemostatic Changes in Dengue

The mechanisms of bleeding in DHF are still not clearly defined. They are multifactorial involving the 3 major haemostatic systems:
   1. vasculopathy
   2. platelet abnormalities
   3. coagulation defects

Vasculopathy

a. High Haematocrit

The haematocrit in DHF is usually > 40%, but may be as high as 55 to 60%.\(^\text{10}\) This haemoconcentration is due to plasma leakage which starts at the end of the febrile stage and continues up to 24-48 hours after defervescence of fever. Increased capillary permeability leading to plasma leakage is by far the most common cause of shock in DHF.

b. Positive Tourniquet Test

A positive tourniquet test is seen in dengue infection even before the platelet starts to fall and this indicates capillary fragility.

Platelet Abnormalities

a. Thrombocytopenia

The platelet count begins to fall in the febrile stage and is lowest in the shock stage. It can reach a nadir of less than \(10 \times 10^9/L\). It then starts to rise by the second afebrile day and normalizes by 7 days.\(^\text{11}\)

b. Platelet Dysfunction

Platelet functions, in particular, ADP-induced platelet aggregation and ADP-releasing ability are impaired.\(^\text{12}\)

Coagulation Defects

In most cases of DHF there is prolongation of APTT (in 54.6% of patients) and PT (in 33.3%), with a variable degree of reduction in coagulation factors II, V, VII, VIII, IX and X. Fibrinogen is constantly decreased and the degree of reduction is related to clinical severity.\(^\text{12}\)

There is usually a mild form of consumption coagulopathy which reverts with appropriate intravenous fluid therapy and may not need specific blood products. Overt disseminated
intravascular coagulation (DIC) with significant bleeding occurs in association with prolonged uncorrected hypovolemic shock due to plasma leakage.

2. **Respiratory System**

- respiratory distress in patients with DHF
  - may be due to pleural effusions, acute respiratory distress syndrome (ARDS), shock or metabolic acidosis.

- increased vascular permeability causes
  - transudate leak, which leads to pleural effusion, characteristically on the right side.
  - shock, bilateral pleural effusion is common.

- acute respiratory distress syndrome (ARDS) is acute respiratory failure characterised by hypoxemia and a characteristic radiographic picture.

3. **Heart**

- sinus tachycardia in febrile phase
- sinus bradycardia with varying degrees of nodal block
- raised cardiac enzymes
- myocarditis, rarely

4. **Liver**

- hepatomegaly, which may be tender
- raised liver enzymes, especially aspartate aminotransferase but jaundice is not a common feature.
- there is usually associated hypoalbuminemia.
- acute liver failure has been associated with DHF and when this occurs is usually accompanied by encephalopathy and acute renal failure.

5. **Gastrointestinal System**

- gastrointestinal haemorrhage is a common manifestation in dengue haemorrhagic fever.
- gallbladder: thickening of the wall
- pancreatitis (rarely)

6. **Neurological System**

   Three forms of neurological manifestation of dengue fever have been described:
   - headache, dizziness, delirium, restlessness, mental irritability
   - depressed conscious level, confusion, lethargy, meningism, coma
• delayed symptoms, paralysis of upper and lower extremities (post-infectious encephalomyelopathy) and depression may occur during convalescence.

These manifestations may be due to viral encephalitis or secondary to liver involvement, hypovolemia, electrolyte imbalance or intracranial haemorrhage.

• CT scan of the brain is indicated to rule out intracranial haemorrhage when there are focal localising neurological signs or altered conscious level.

7. Renal System

• severe hypovolemia, haemorrhage and prolonged shock may progress to acute renal failure.
• Haemolytic uraemic syndrome has been described in dengue infections.

8. Reproductive system

• menorrhagia and post-partum haemorrhage have been noted to occur.
• transplacental infection may cause dengue infection in the newborn.
• transplacental dengue infection have been reported with variable severity of clinical manifestation.
CLINICAL COURSE OF DENGUE HAEMORRHAGIC FEVER

Fever days

<table>
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<th>1</th>
<th>2</th>
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<th>5</th>
<th>6</th>
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</table>

Viraemia

Temperature

Shock

Haematocrit

Platelet count

Antibody (IgG)

Phases:

Febrile

Critical

Reabsorption/Recovery

Note:
- It is important to recognise the febrile phase, critical phase and recovery phase so that appropriate therapy can be instituted.
CRITERIA FOR HOSPITALISATION

The patient must be assessed in totality and NOT by the absence or presence of any feature/criteria in isolation.

Lower threshold should be used for the elderly, pregnant women, patients with liver disease, peptic ulcer disease and multiple co-morbid conditions [cardiovascular, pulmonary, metabolic, renal, immuno-suppressed conditions] and patients who are living alone.

General condition
- Continuous fever ≥ 3 days
- Lethargy
- Restlessness
- Generalised flushing
- Excessive tiredness

Dehydration
- Unable to tolerate orally/ vomiting
- Diarrhoea / frequent loose stools

Abdominal discomfort
- Right hypochondrium / epigastric pain; tender hepatomegaly

Haemorrhagic manifestations as evidenced by:
- Positive *tourniquet test* (especially helpful in places without laboratory support, for example in general practice)
- Petechiae, ecchymoses or purpura
- Spontaneous mucosal bleeding from the gums, epistaxis, haematuria, menorrhagia, injection sites or other sites
- Haematemesis, melaena, haematochezia [painless fresh per rectal bleeding]

Thrombocytopaenia
Platelet count does not correlate with the severity of bleeding.
• Patients with active bleeding should be admitted regardless of the platelet count
• Without bleeding tendency but platelet count on a rapid down trend as monitored daily in outpatient clinic / A&E department.
• Platelet count of less than 100,000/mm³

Plasma leakage manifested by:
• Rapid rising haematocrit
• **Haematocrit** equal to or greater than 20% of baseline
  
  [Caution: This haematocrit criterion is not applicable in patients with prior anaemia or significant blood loss at presentation].

• Pleural effusion, ascites (serositis)
• Suspect plasma leakage in:
  o males with haematocrit above 47%
  o females with haematocrit above 40%

Evidence of circulatory failure/shock as manifested by:
• Rapid and weak pulse
• Diminished peripheral pulses
• Narrowing of the **pulse pressure** [<20mmHg]
• Hypotension for age [late finding representing uncorrected shock]
• Cool, mottled or pale skin
• Changes in mental status, restlessness, lethargy,
• Oliguria
• Tachypnea (due to metabolic acidosis)

If patient does not meet admission criteria but needs further follow-up

• Daily follow-up at the OPD with platelet and haematocrit
• Encourage patient to seek admission anytime [OPD or A&E] the condition is felt to have deteriorated
• encourage patient to drink lots of fluids
* The tourniquet test is performed by inflating a blood pressure cuff on the upper arm to a point midway between the systolic and diastolic pressures for 5 minutes. A test is considered positive when 20 or more petechiae per 2.5cm² area square is observed. However the test may be negative in shock.

** Pulse pressure = systolic blood pressure minus diastolic blood pressure. The narrow pulse pressure is observed early in the course of shock, whereas hypotension is observed later, or in patients who experience severe bleeding. Pulse pressure < 20 mmHg is significant.
MANAGEMENT OF DENGUE INFECTION

Treatment of classical Dengue Fever is the same as for other Acute Uncomplicated Viral Infections:

- Plenty of oral fluids
- Paracetamol for relief of fever and bodyache
- Avoid aspirin & NSAIDs (antiplatelet effect)
- Avoid intra-muscular injections
- Seek further medical advice if there is deterioration.

Management of Dengue Haemorrhagic Fever
(see flow chart)

After 24-48 hours of onset of increase in vascular permeability, plasma leakage stops. It is very important that fluid therapy is managed carefully.
MANAGEMENT OF DHF/ DSS

FLOW CHART:

START iv Fluids

- 0.9% sodium chloride (normal saline) [30-50 ml/kg/day]
- KCL supplement as required
- Caution in elderly/cardiac disease
- Diabetics: use all normal saline

MONITOR haematocrit closely; Vital signs and urine output hourly

IMPROVEMENT
- Haematocrit decreases
- Stable blood pressure & pulse rate
- Urine output increases

Reduce iv fluids
Assess oral intake

FURTHER IMPROVEMENT
Reduce/stop iv infusion after 24-48 hrs
Discharge when indicated

NO IMPROVEMENT
INTENSIVE MONITORING REQUIRED
- Haematocrit increases
- Pulse rate increases
- Pulse pressure < 20 mmHg
- Urine output decreases

Increase iv fluids to 10-15 ml/kg/hr
Monitor haematocrit, FBC, BUSE & vital signs closely

Unstable vital signs
Urine output decreases
Signs of SHOCK

haematocrit ↑↑ Normal/haematocrit ↓↓

Blood transfusion ↓Hb/ suspicion of bleeding/ overt bleed

IMPROVEMENT

?FLUID OVERLOAD (pulmonary oedema, ↑JVP, crepitations, heart rate > 120/min

Consider Frusemide/ Intensive Care

Discharge when indicated

Colloid infusion (20 ml/kg in 30 minutes)
Management of Dengue Shock Syndrome

- HDU/ ICU care indicated whenever available.
- Rapid volume replacement (colloids or plasma expander) – 20 ml/kg body weight/hourly.
- Continuous monitoring of vital signs.
- Avoid subclavian / jugular CVP [recommend Peripherally Inserted Central Catheter-PICC].
- CBD insertion for accurate intake & output chart.
- Haematocrit monitoring every 4 to 6 hours or as clinically indicated.
- Renal & liver profile/ ABG / DIC screening STAT and repeat as required.
- Try to avoid femoral / brachial artery puncture.
- CXR & ECG.
- Correct electrolyte imbalance and acidosis.
- Blood transfusion (packed cells) if there is
  a) obvious bleeding or
  b) persistent shock with falling haematocrit and hemoglobin following volume replacement.
- Inotrope support may also be required.
- When pulse rate and volume and blood pressure are improving, make sure the fluid is reduced to maintenance.
- Reabsorption of extravasated fluid occurs when capillary leakage ceases.
- Therefore the duration of intravenous fluid replacement should not exceed the duration of dengue shock or capillary leakage.
- Prolongation of intravenous fluid therapy can result in fluid overload manifest as pulmonary oedema.
- Good compression after blood taking is important to prevent bleeding from venepuncture site.

Management of pleural effusion

- Pleural effusions occur during the phase of plasma leakage.
- Pleural effusions decrease thoracic compliance and functional residual capacity leading to hypoxemia and increased work of spontaneous breathing.
- Massive pleural effusions can be prevented by judicious replacement of intravascular volume.
- Diuretic therapy should only be considered during the reabsorption phase.
- Intercostal drainage of pleural effusions is generally not recommended.

Management of bleeding in DF/DHF/DSS

Most cases of bleeding in DHF/DSS occur as a result of prolonged shock secondary to inadequately corrected plasma leakage. There is a category of patients with pre-existing peptic ulcers who develop haemorrhage in the course of DF. However there is no consensus on how these patients should be treated.
Management of gastrointestinal bleeding in dengue patients

The haematocrit obtained for a patient with acute bleeding poorly reflects the degree of blood loss due to concurrent haemoconcentration. Suspect bleeding if the haematocrit is lower than expected for the degree of shock.

There is no consensus on the placement of a nasogastric tube. However, a nasogastric tube may help to decompress the stomach and monitor for active bleed. In the setting of coagulopathy, the nasogastric tube may lead to trauma.

Indications for blood products in dengue infection

1. Blood transfusion (packed red cell) is indicated in significant bleeding* (falling haematocrit / haemoglobin in an unstable patient).
2. Platelet transfusion is generally avoided unless:
   a. there is significant bleeding regardless of platelet count
   b. platelet count < 10,000/mm³ with impending or established CNS bleed or continuous bleeding from a pre-existing peptic ulcer which needs a procedure such as an upper gastroscopy.
3. In established disseminated intravascular coagulopathy (low serum fibrinogen, reduced platelets along with prolonged PT and APTT**) with significant bleeding, infusion of cryoprecipitate (1 unit per 10 kg body weight), fresh frozen plasma (15 ml/kg) and platelet concentrates (4-6 units random platelet concentrate) are required.

Note:
There are no prospective studies and consensus on platelet transfusion based on low platelet count with or without bleeding in dengue infection in the adult population.
There are no randomised prospective studies to show that the administration of fresh frozen plasma or platelet concentrates have improved the outcome in DHF/DSS in adults.

* Significant bleeding includes:
  o extensive mucosal bleeds
  o upper GIT bleeding
  o impending intracranial haemorrhage (headaches, fundal haemorrhages)

** Prolonged PT and APTT may be seen in dengue infection even in the absence of disseminated intravascular coagulation.

Intensive care in dengue infection

Preview
- Patients are at greatest risk of organ impairment or organ failure during the phase of dengue shock which coincides with capillary leakage.
- Capillary leakage normally commences around the 4th to 5th day of illness and lasts for 24-48 hours.
- Dengue shock may resolve without organ impairment.
• The onset of multiple organ failure secondary to dengue shock is associated with an increased risk of mortality.

➢ Admission to an intensive care unit/high dependency ward of a patient with dengue shock should be considered
  o if facilities are available locally
  o for titration of fluid therapy
  o for monitoring of organ perfusion and function

Respiratory distress

• Respiratory distress can be due to the following and may indicate a need for specific interventions including mechanical ventilation:
  o ARDS
  o Massive pleural effusions
  o Pulmonary oedema, during reabsorption and in the presence of continued intravenous fluid therapy
  o Ascites
  o Concomitant sepsis including nosocomial sepsis
  o Shock and acidosis.

Indications for mechanical ventilation:

• Clinical respiratory muscle fatigue
• Hypoxemia unresponsive to high flow oxygen therapy
• Respiratory acidosis with hypercarbia
• Encephalopathy

Criteria for discharging patients hospitalised with DHF/DF

The following criteria should be met before patients recovering from DHF/DF are discharged.

• absence of fever for at least 24 hours without the use of anti-fever therapy
• at least 2 days after recovery from shock
• rising platelet count of more than 50,000 per mm$^3$
• stable haematocrit
• visible clinical improvement
• return of appetite
• good urine output
• no respiratory distress

REFERENCES