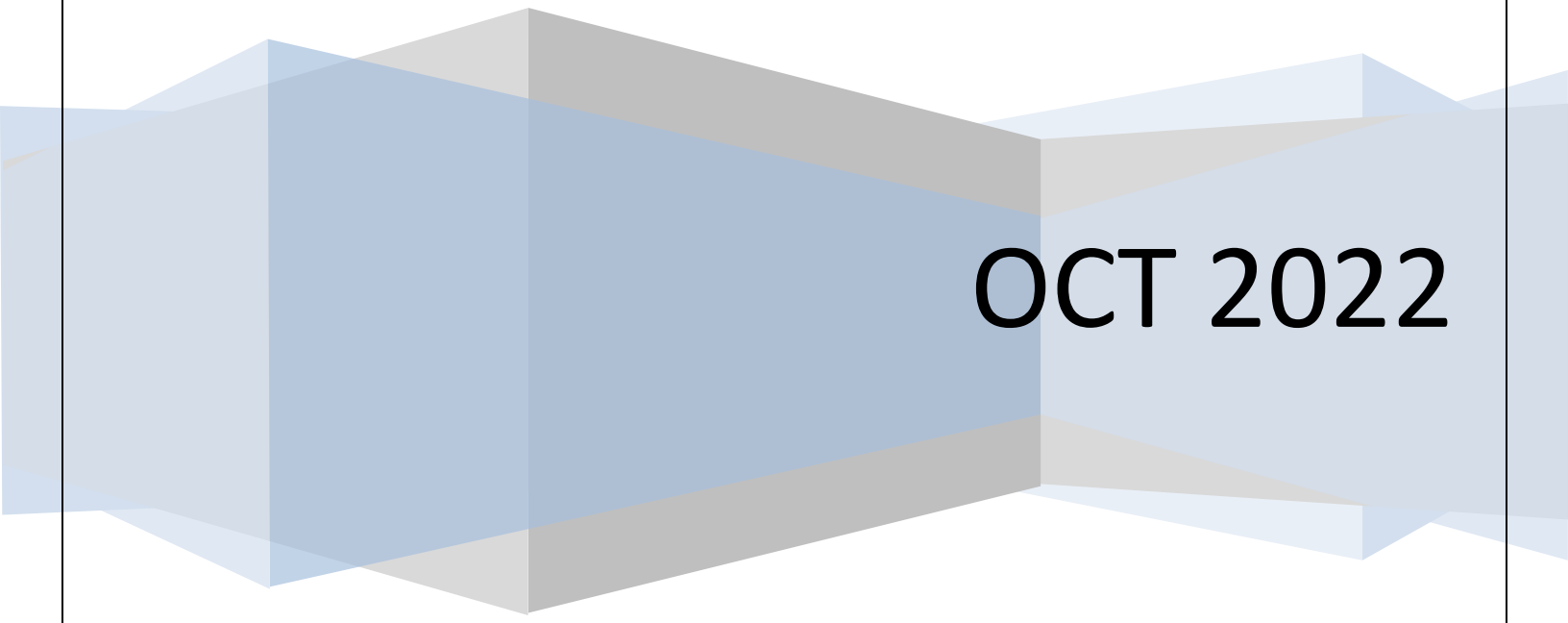
HARGEISA GROUP HOSPITAL

(HGH)

DENGUE VIRUS CLINICAL MANAGEMENT GUIDLINE



OCT 2022

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Overview and course of dengue illness

Dengue is a febrile illness caused by infection with one of four dengue viruses (DENV) transmitted by Aedes aegypti or Aedes albopictus mosquitoes during the taking of a blood meal.

Infection may be asymptomatic or present with a broad range of clinical manifestations including a mild febrile illness to a life-threatening shock syndrome.

There are four closely related but serologically distinct DENV types of the genus Flavivirus, called DENV-1, DENV-2, DENV-3, and DENV-4. There is transient cross-protection among the four types, which weakens and disappears over the months following infection; therefore, individuals living in a dengue-endemic area with all types co-circulating are at risk for infection with any and all DENV types.

Infection with one of the four serotypes of dengue virus (**primary infection**) provides life-long immunity to infection with a virus of the same (homologous) serotype

However, immunity to the other (heterologous) dengue serotypes is transient, and individuals can subsequently be infected with another dengue serotype (**secondary infection).**

After the incubation period, the illness begins abruptly and, in patients with moderate to severe disease, is followed by three phases - febrile, critical and recovery.

Due to its dynamic nature, the severity of the disease will usually only be apparent around defervescence i.e. during the transition of the febrile to the afebrile phase, which often coincides with the onset of the critical phase.

Activities (triage and management decisions) at the primary and secondary care levels (where patients are first seen and evaluated) are critical in determining the clinical outcome of dengue.

A well-managed front-line response not only reduces the number of unnecessary hospital admissions but also saves the lives of dengue patients. Early notification of dengue cases seen in primary and secondary care is crucial for identifying outbreaks and initiating an early response.

CLASSIFICATION SCHEMES

WHO 2009 classification

In 2009, the World Health Organization introduced a revised classification scheme consisting of the following categories:

1. Dengue without warning signs
2. Dengue with warning signs
3. Severe dengue

Dengue without warning signs — A presumptive diagnosis of dengue infection may be made in the setting of residence in or travel to an endemic area plus **fever** and two of the following:

- Nausea/vomiting
- Rash
- Headache, eye pain, muscle ache, or joint pain
- Leukopenia
- Positive tourniquet test

Dengue with warning signs — Dengue with warning signs of severe infection includes dengue infection as defined above in addition to any of the following:

- Abdominal pain or tenderness
- Persistent vomiting
- Clinical fluid accumulation (ascites, pleural effusion)
- Mucosal bleeding
- Lethargy or restlessness
- Hepatomegaly >2 cm
- Increase in hematocrit concurrent with rapid decrease in platelet count

Severe dengue — severe dengue infection includes dengue infection with at least one of the following:

- ❖ Severe plasma leakage leading to:
  - ✓ Shock
  - ✓ Fluid accumulation with respiratory distress
- ❖ Severe bleeding (as evaluated by clinician)
- ❖ Severe organ involvement:
  - ✓ Aspartate aminotransferase (AST) or alanine aminotransferase (ALT) ≥1000 units/L
  - ✓ Impaired consciousness
  - ✓ Organ failure

CLINICAL MANIFESTATIONS

Clinically apparent dengue infections are more common among adults among children; most dengue infections are asymptomatic or minimally symptomatic.

The incubation period of DENV infection ranges from 3 to 14 days; symptoms typically develop between 4 and 7 days after the bite of an infected mosquito.

Patients with suspected dengue should be assessed carefully and directed to the appropriate care setting. Early recognition of progression to severe disease and patients at increased risk for severe disease is essential, with prompt initiation of more aggressive therapy when necessary.

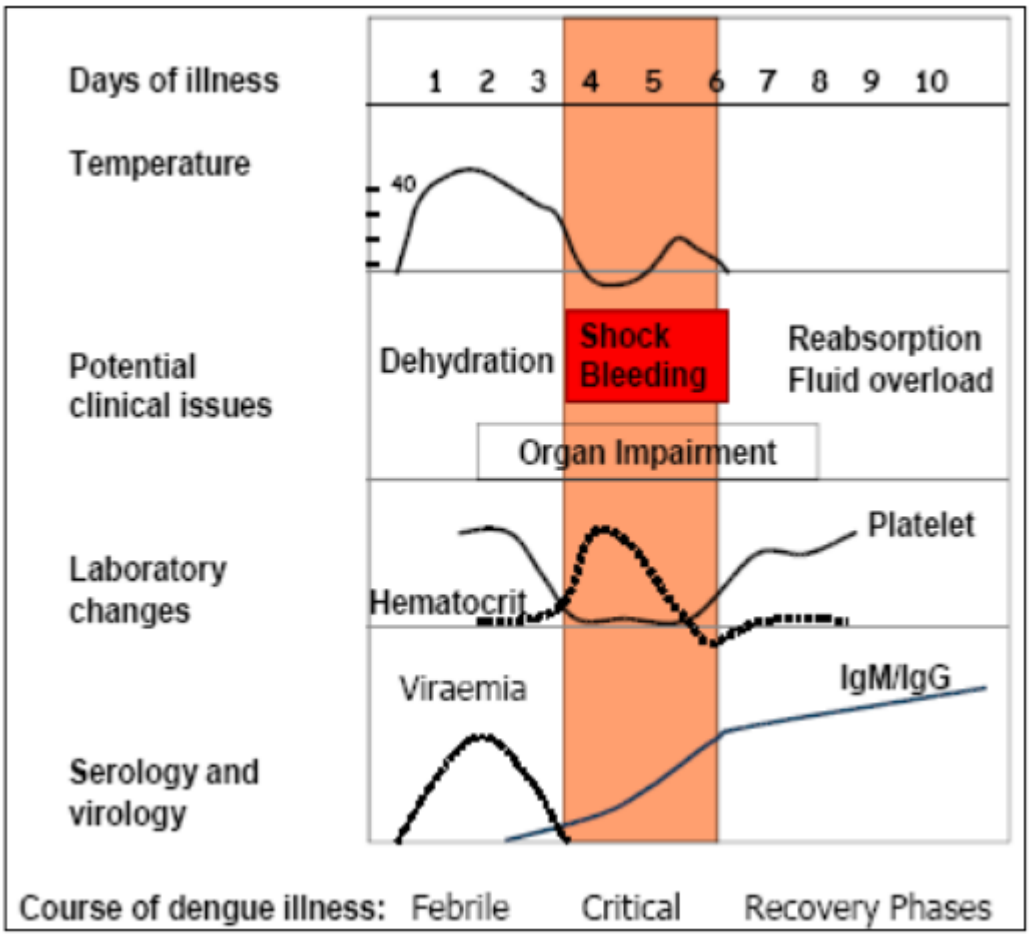
The course of dengue illness (Phases of infection)

There are three phases that can be seen in the setting of DENV infection

A) Febrile phase

B) Critical phase

C) Recovery phase



Febrile phase

Patients typically develop a high-grade fever (≥38.5°C) suddenly. This acute febrile phase usually lasts 2□7 days and is often accompanied by facial flushing, skin erythema, generalized body ache, myalgia, arthralgia, retro-orbital eye pain, photophobia, rubeliform exanthema and headache.

Some patients may have a sore throat, an injected pharynx, and conjunctival injection. Anorexia, nausea and vomiting are common.

It can be difficult to distinguish dengue clinically from non-dengue febrile diseases in the early febrile phase.

A positive tourniquet test in this phase indicates an increased probability of dengue.

However, these clinical features do not predict the severity of disease. Therefore it is crucial to monitor for warning signs and other clinical parameters in order to recognize progression to the critical phase.

Mild haemorrhagic manifestations such as petechiae and mucosal membrane bleeding (e.g. of the nose and gums) may be seen.

The liver may be enlarged and tender after a few days of fever.

The earliest abnormality in the full blood count is a progressive decrease in total white cell count, which should alert the physician to a high probability of dengue.

Thrombocytopenia (≤100,000 cells/mm) are common, Serum aspartate transaminase (AST) levels are frequently elevated, the elevations are usually modest (2 to 5 times the upper limit of normal values), but marked elevations (5 to 15 times the upper limit of normal) occasionally occur

In addition to these somatic symptoms, with the onset of fever patients may suffer an acute and progressive loss in their ability to perform their daily functions such as schooling, work and interpersonal relations.

Between days 3 and 7 of the illness, the clinician must watch for signs of vascular leakage. Significant vascular leakage reduces intravascular volume and decreases organ perfusion. Corresponding clinical manifestations may include persistent vomiting, increasingly severe abdominal pain, tender hepatomegaly, development of pleural effusions and/or ascites, mucosal bleeding, and lethargy or restlessness.

Laboratory findings may include a high or increasing hematocrit level (≥20 percent from baseline) concurrent with a rapid decrease in the platelet count.

Critical phase

During the transition from the febrile to afebrile phase, patients without an increase in capillary permeability will improve without going through the critical phase.

Instead of improving with the subsidence of high fever; patients with increased capillary permeability may manifest with the warning signs, mostly as a result of plasma leakage.

The warning signs mark the beginning of the critical phase. These patients become worse around the time of defervescence, when the temperature drops to 37.5-38°C or less and remains below this level, usually on days 3–8 of illness.

Progressive leukopenia followed by a rapid decrease in platelet count usually precedes plasma leakage.

An increasing haematocrit above the baseline may be one of the earliest additional signs and the period of clinically significant plasma leakage usually lasts24-48 hours.

The degree of haemoconcentration above the baseline haematocrit reflects the severity of plasma leakage; however, this may be reduced by early intravenous fluid therapy.

Hence,frequent haematocrit determinations are essential because they signal the need for possible adjustments to intravenous fluid therapy.

Moderate-to-severe thrombocytopenia is common during the critical phase,nadir platelet counts ≤20,000 cells/mm may be observed, followed by rapid improvement during the recovery phase.

A transient increase in the activated partial-thromboplastin time and decrease in fibrinogen levels are also common.

Pleural effusion and ascites are usually only clinically detectable after intravenous fluid therapy, unless plasma leakage is significant. A right lateral decubitus chest radiograph, ultrasound detection of free fluid in the chest or abdomen, or gallbladder wall edema may precede clinical detection.

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A right lateral decubitus chest radiograph, ultrasound detection of free fluid in the chest or abdomen, or gall bladder wall oedema may precede clinical detection.

Warning signs of dengue

Warning signs usually precede the manifestations of shock and appear towards the end of the febrile phase, usually between days 3–7 of illness. Persistent vomiting and severe abdominal pain are early indications of plasma leakage and become increasingly worse as the patient progresses to the shock state. The patient becomes increasingly lethargic but usually remains mentally alert.

These symptoms may persist into the shock stage.

Weakness, dizziness or postural hypotension occurs during the shock state. Spontaneous mucosal bleeding or bleeding at previous venepuncture sites are important haemorrhagic manifestations. Increasing liver size and a tender liver is frequently observed.

Recovery phase

As the patient survives the 24-48 hour critical phase, a gradual reabsorption of extravascular compartment fluid takes place in the following 48-72 hours.

Plasma leakage and hemorrhage resolve, vital signs stabilize, and accumulated fluids are resorbed.

General wellbeing improves, appetite returns, gastrointestinal symptoms abate, haemodynamic status stabilizes, and diuresis ensues.

Some patients have a confluent erythematous or petechial rash with small areas of normal skin, described as “isles of white in the sea of red”

The haematocrit stabilizes or may be lower due to the dilutional effect of reabsorbed fluid. The white blood cell count usually starts to rise soon after defervescence but the recovery of the platelet count is typically later than that of the white blood cell count.

Respiratory distress from massive pleural effusion and ascites, pulmonary oedema or congestive heart failure will occur during the critical and/or recovery phases if excessive intravenous fluids have been administered.

Medical complications seen in the febrile, critical and recovery phases of dengue

1	Febrile phase	Dehydration: high fever may cause neurological disturbances and febrile seizures in young children
2	Critical phase	Shock from plasma leakage: severe haemorrhage; organ impairment
3	Recovery phase	Hypervolaemia (only if intravenous fluid therapy has been excessive and/or has extended into this period) and acute pulmonary oedema

Severe dengue

A case of severe dengue is defined as a suspected dengue patient with one or more of the following:-

(i) Severe plasma leakage that leads to shock (dengue shock) and/or fluid accumulation with respiratory distress
(ii) Severe bleeding;
(iii) Severe organ impairment.

Severe plasma leakage and dengue shock

Dengue shock syndrome (DSS) is a form of hypovolaemic shock and results from continued vascular permeability and plasma leakage.

This usually takes place around defervescence, i.e. on days 4-5 of illness (range of days 3-8), and is often preceded by warning signs.

Dengue shock presents as a physiologic continuum, progressing from asymptomatic capillary leakage to compensated shock to hypotensive shock and ultimately to cardiac arrest

During **the initial stage of shock**, the compensatory mechanism that maintains a normal systolic BP produces tachycardia, quiet tachypnoea (tachypnoea without increased effort), and peripheral vasoconstriction with reduced skin perfusion (manifested as cold extremities and delayed capillary refill time of > 2 seconds and weak volume peripheral pulses).

As peripheral vascular resistance increases, the diastolic pressure rises towards the systolic pressure and the pulse pressure (the difference between the systolic and diastolic pressures) narrows.

In adults, a pulse pressure of ≤ 20 mmHg may indicate more severe shock. Compensated metabolic acidosis is observed when the pH is normal with low carbon dioxide tension and a low bicarbonate level.

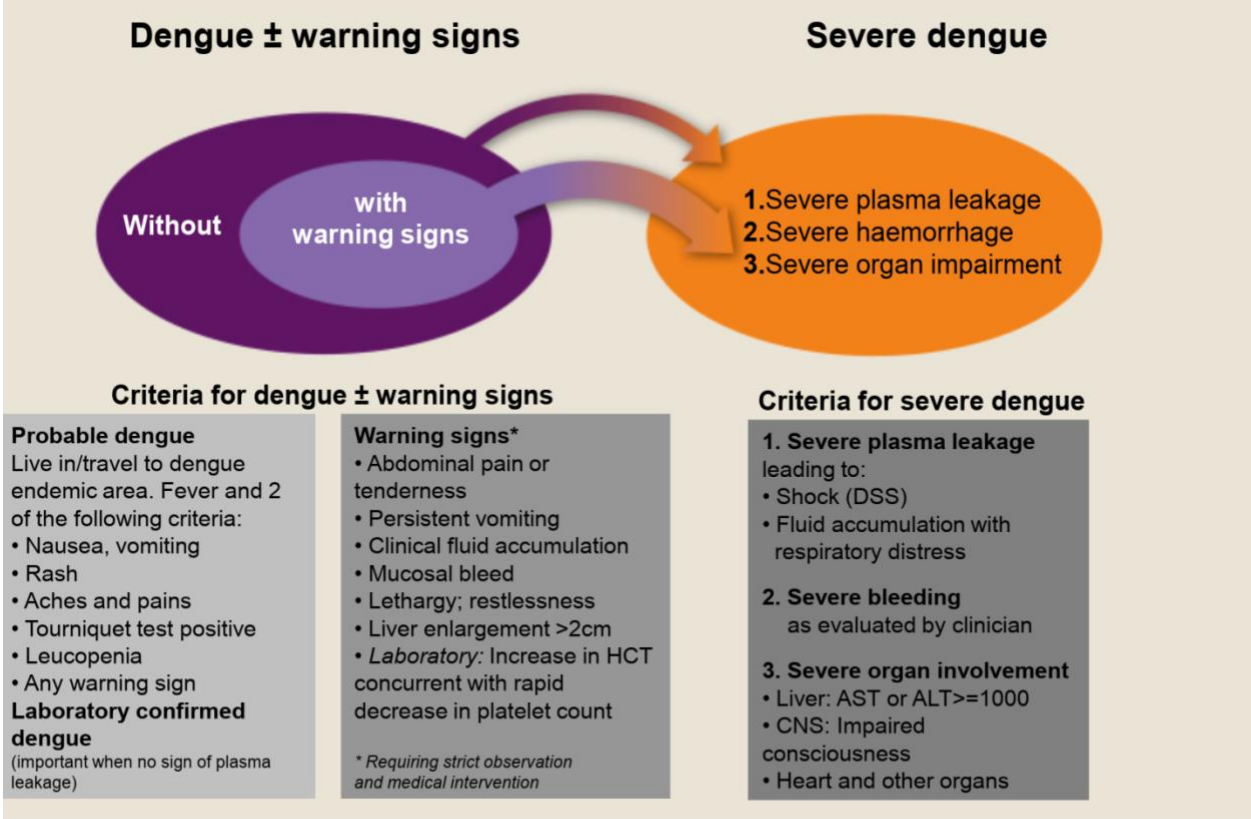
Patients who have dengue and are in compensated shock often remain conscious and lucid. The inexperienced physician may measure a normal systolic pressure and a normal pulse oximetry (SpO2 95–100%) in a conscious patient and underestimate the critical state of the patient.

Worsening hypovolaemic shock

Manifests as increasing tachycardia and peripheral vasoconstriction. Not only are the extremities cold and cyanosed but the limbs become mottled, cold and clammy.

By this stage the breathing becomes more rapid and increases in depth - a compensation for the metabolic acidosis (Kussmaul's breathing). Finally, there is decompensation, both systolic and diastolic BPs disappear suddenly and dramatically,and the patient is said to have hypotensive or decompensated shock.

Dengue case classification by severity



= alanine aminotransferase; AST = aspartate aminotransferase; CNS = central nervous system; DSS = ue shock syndrome; HCT = haematocrit

DIAGNOSIS

Laboratory diagnosis of dengue is made by detecting the virus and/or any of its components (infective virus, virus genome, dengue antigen) or by investigating the serological responses present after infection (specifically IgM and IgG levels).

The diagnostic method to confirm an acute infection depends on the time of clinical illness:
the febrile phase is coincident with the presence of viraemia, some viral components and replication products in blood,

The critical and convalescent phases coincide with the development of antibodies.

Febrile phase (day 1 to day's 4–5 of fever): Virus genome detection using reverse transcriptase polymerase chain reaction (RT-PCR) and real-time RT-PCR confirms an acute dengue infection.

NS1 Ag is a marker of acute dengue infection. Both enzyme-linked immunosorbent assay (ELISA) and rapid commercial tests are available for NS1 Ag detection.

Critical and convalescent phases (after days 4–5 of illness):

Specific IgM is the best marker of a recent dengue infection. MAC-ELISA and rapid tests are the most frequent methods for IgM detection;

however In addition to IgM, high levels of specific IgG in sera collected early after fever onset as detected by ELISA and haemagglutination inhibition assay (HIA) also suggest a recent dengue infection.
Primary infections are characterized by high levels of IgM and low levels of IgG, while low levels of IgM with high levels of IgG characterize secondary infections.

Confirmed and probable dengue diagnosis, interpretation of results and sample characteristics

	Method	Interpretation	Sample characteristics
Confirmed dengue infection	Viral isolation	Virus isolated	Serum (collected at 1–5 days of fever) Necropsy tissues
	Genome detection	Positive RT-PCR or positive real-time RT-PCR	
	Antigen detection	Positive NS1 Ag	
		Positive immunohistochemical	Necropsy tissues
	IgM seroconversion	From negative IgM to positive IgM in paired sera	Acute serum (days 1–5) and convalescent serum (15–21 days after first serum)
	IgG seroconversion	From negative IgG to positive IgG in paired sera or 4-fold increase IgG levels among paired sera	
Probable dengue infection	Positive IgM	Positive IgM	Single serum collected after day 5
	High IgG levels	High IgG levels by ELISA or HI (≥ 1280)	

ELISA = enzyme-linked immunosorbent assay; IgG = immunoglobulin G; IgM = immunoglobulin M; NS1 Ag = non-structural protein 1 antigen; RT-PCR = reverse transcriptase polymerase chain reaction

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of DENV infection includes:

- **Other viral hemorrhagic fevers** – Other viruses capable of causing hemorrhagic fever include Ebola virus, Marburg virus, Lassa virus, yellow fever virus, Crimean-Congo hemorrhagic fever, hantavirus (hemorrhagic fever with renal syndrome)
- Chikungunya and Zika virus infection
- Malaria, Typhoid fever, Acute HIV infection, Viral hepatitis
- Sepsis due to bacteremia
- Coronavirus disease 2019 (COVID-19)

Leukopenia and thrombocytopenia may be present in non-infectious diseases such as:-

- systemic lupus erythamatosis (SLE)
- acute leukaemia
- Henoch-Schönlein purpura
- thrombotic thrombocytopenic purpura
- Immunological thrombocytopenic purpura(ITP).

Dengue Fever Case Management

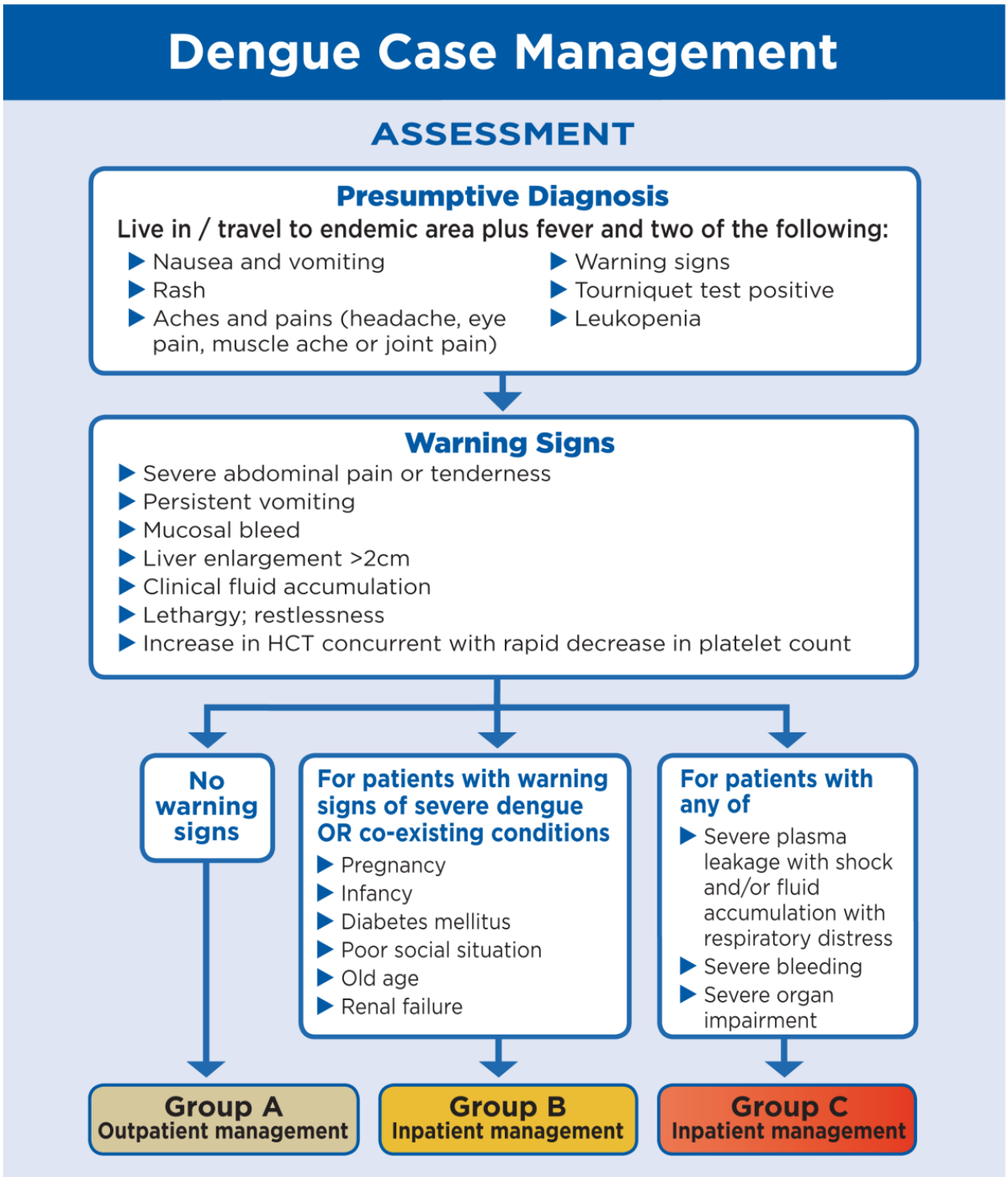
There is no direct antiviral therapy available against the DENVs. Management is supportive, which largely consists of maintaining adequate intravascular volume.

Outpatient Management Criteria:-

- ✓ Presumptive diagnosis of dengue infection in the absence of warning signs
- ✓ No coexisting conditions (pregnancy, infancy, old age, diabetes, renal failure, underlying hemolytic disease, obesity, or poor social situation)
- ✓ able to tolerate oral fluids
- ✓ urinate at least once every six hours
- ✓ Have near normal blood counts

Inpatient Management Criteria:-

- ✓ patients with dengue and warning signs of severe infection
- ✓ severe dengue infection
- ✓ dengue infection with coexisting conditions
- ✓ An elevated hematocrit(Hct>50%) is an indication for plasma leakage and shock
- ✓ Leukopenia (white blood cell count 5000 cells/mm or lower) and/or thrombocytopenia (platelet count 100,000 cells/mm or lower), especially in high-risk patients (infants, older adults, pregnancy, patients with comorbidities)
- ✓ Blood pressure <90/60 mmHg and Altered consciousness


Dengue Case Management
ASSESSMENT
Presumptive Diagnosis
Live in / travel to endemic area plus fever and two of the following:
Nausea and vomiting
Rash
Aches and pains (headache, eye pain, muscle ache or joint pain)
Warning signs
Tourniquet test positive
Leukopenia
Warning Signs
Severe abdominal pain or tenderness
Persistent vomiting
Mucosal bleed
Liver enlargement >2cm
Clinical fluid accumulation
Lethargy; restlessness
Increase in HCT concurrent with rapid decrease in platelet count
No warning signs
For patients with warning signs of severe dengue OR co-existing conditions
Pregnancy
Infancy
Diabetes mellitus
Poor social situation
Old age
Renal failure
For patients with any of
Severe plasma leakage with shock and/or fluid accumulation with respiratory distress
Severe bleeding
Severe organ impairment
Group A
Outpatient management
Group B
Inpatient management
Group C
Inpatient management

Treatment according to Groups A–C

Group A (Outpatient)

- These patients are able to tolerate adequate volumes of oral fluids, pass urine at least once every six hours and do not have any of the warning signs (particularly when fever subsides).
- ✓ Patients with dengue fever should be cautioned to maintain intake of oral fluid to avoid dehydration. Fever and myalgias can be managed with acetaminophen (maximum 60 mg/kg/day in children or **4 g/day in adults**).
- ✓ Aspirin or nonsteroidal antiinflammatory agents should generally be avoided because of the risk of bleeding complications.
- ✓ During the febrile phase (lasting two to seven days) and the subsequent critical phase (lasting one to two days), the patient should be evaluated daily from the third day of illness through the end of the critical phase for signs of dehydration and other warning signs of severe dengue.

Group A
Outpatient Management

During the febrile phase (may last 2–7 days) and subsequent critical phase (1–2 days), your clinic should

- Follow CBCs
- Watch for dehydration
- Watch for warning signs, including decreasing platelet count and increasing hematocrit
- Watch for defervescence (indicating beginning of critical phase)

Advise patient or their family to do the following

Control the fever

- Give acetaminophen every 6 hours (maximum 4 doses per day). Do not give ibuprofen, aspirin, or aspirin-containing drugs.
- Sponge patient's skin with tepid water when temperature is high.

Prevent dehydration which occurs when a person loses too much fluid (from high fever, vomiting, or poor oral intake). Give plenty of fluids (not only water) and watch for signs of dehydration. Bring patient to clinic or emergency room if any of the following signs develop:

- Decrease in urination (check number of wet diapers or trips to the bathroom)
- Few or no tears when child cries
- Dry mouth, tongue or lips
- Sunken eyes
- Listlessness, agitation, or confusion
- Fast heartbeat (>100/min)
- Cold or clammy fingers and toes
- Sunken fontanel in an infant

Prevent spread of dengue within your house

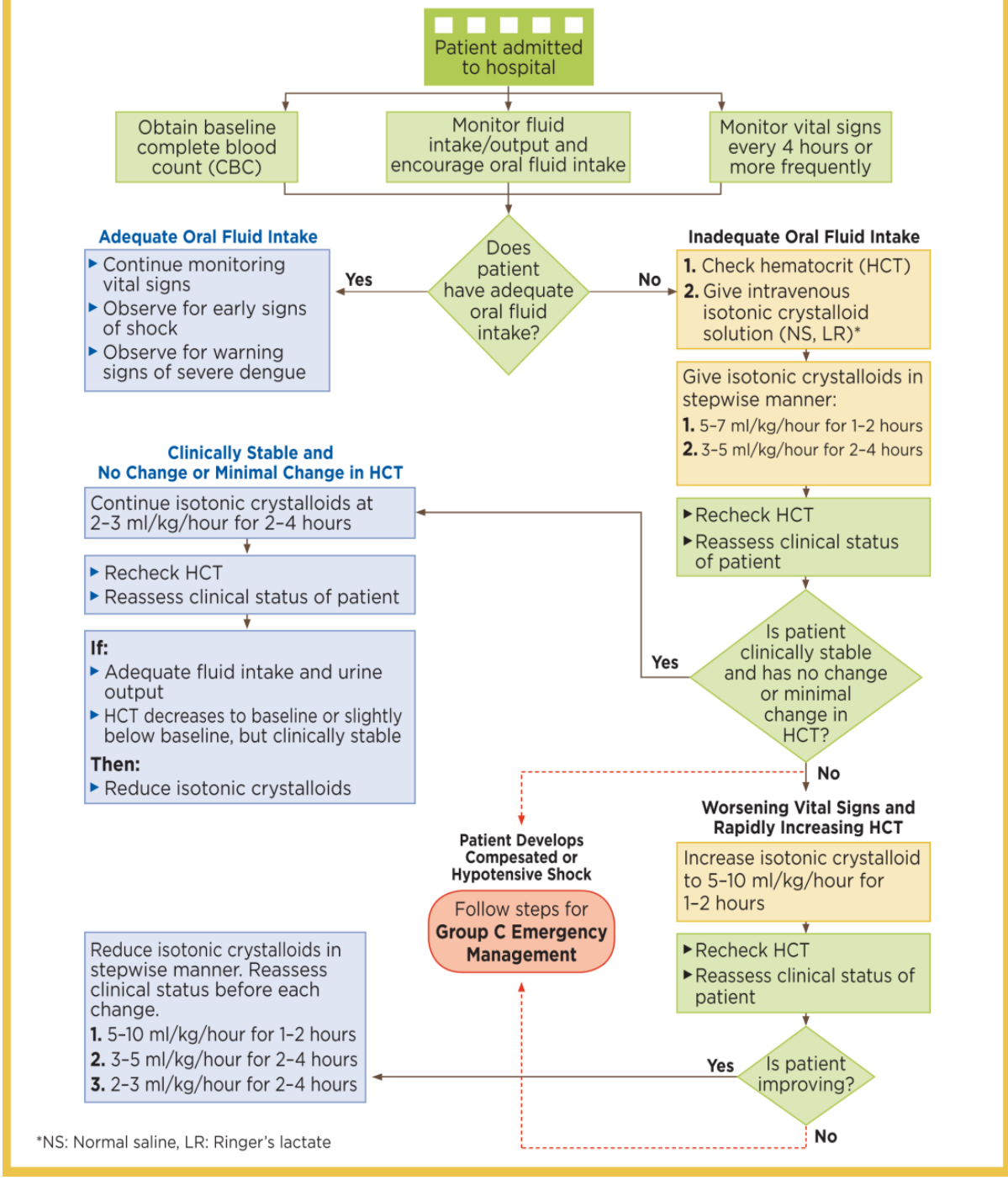
- Place patient under bed net or have patient use insect repellent while febrile to avoid infecting mosquitoes that can infect others within 2 weeks.
- KILL all mosquitoes in house.
- Empty containers that carry water on patio.
- Put screens on windows and doors to prevent mosquitoes from coming into house.

Watch for warning signs as temperature declines 3 to 8 days after symptoms began. Return IMMEDIATELY to clinic or emergency department if any of the following warning signs appear:

- Severe abdominal pain or persistent vomiting
- Red spots/patches on skin
- Bleeding from nose or gums
- Vomiting blood
- Black, tarry stools
- Drowsiness or irritability
- Pale, cold, or clammy skin
- Difficulty breathing

Group B — Inpatient Management for Dengue Patients with

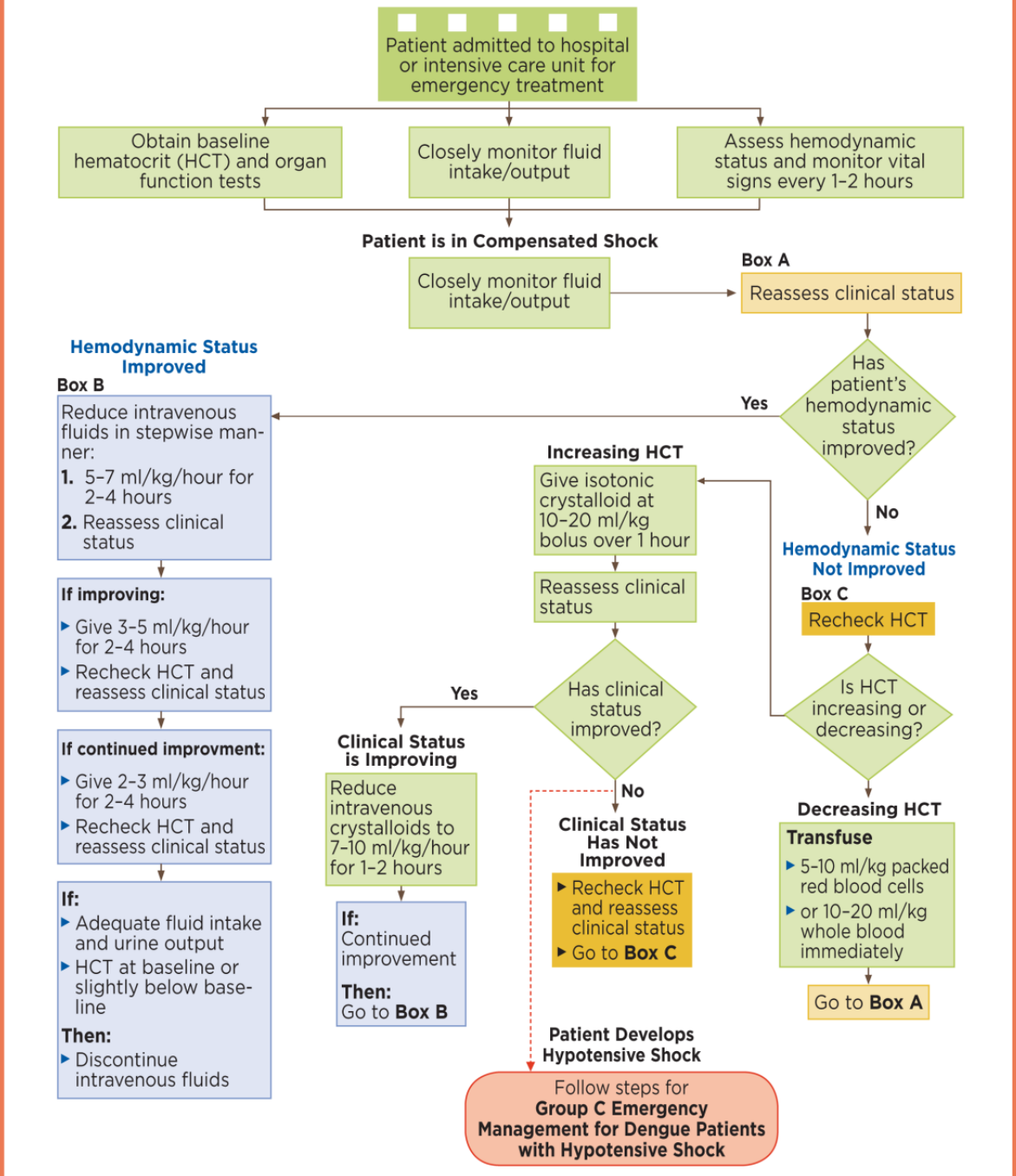
Warning Signs



*NS: Normal saline, LR: Ringer's lactate

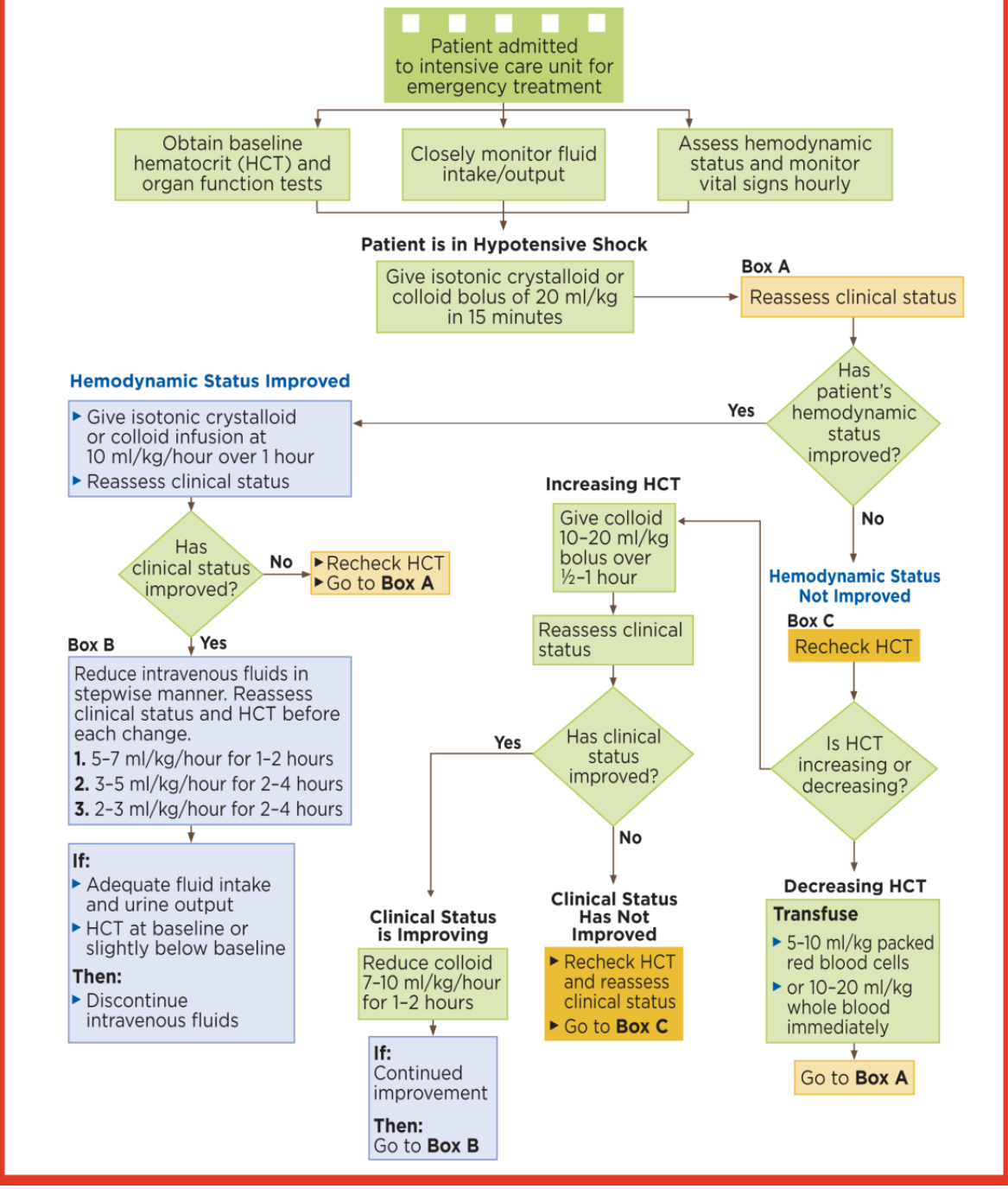
Group C — Emergency Management for Dengue Patients with

Compensated Shock



Group C — Emergency Management for Dengue Patients with

Hypotensive Shock



Hemodynamic Assessment

Hemodynamic Parameters	Stable Circulation	Compensated Shock	Hypotensive Shock
Conscious level	Clear and lucid	Clear and lucid	Restless, combative
Capillary refill	Brisk (≤2 sec)	Prolonged (>2 sec)	Very prolonged, mottled skin
Extremities	Warm and pink	Cool peripheries	Cold, clammy
Peripheral pulse volume	Good volume	Weak and thready	Feeble or absent
Heart rate	Normal heart rate for age	Tachycardia for age	Severe tachycardia or bradycardia in late shock
Blood pressure	▶ Normal blood pressure for age ▶ Normal pulse pressure for age	▶ Normal systolic pressure, but rising diastolic pressure ▶ Narrowing pulse pressure ▶ Postural hypotension	▶ Narrow pulse pressure (≤ 20 mmHg) ▶ Hypotension ▶ Unrecordable blood pressure
Respiratory rate	Normal respiratory rate for age	Tachypnea	Hyperpnea or Kussmaul's breathing (metabolic acidosis)
Urine output	Normal	Reducing trend	Oliguria or anuria

When to stop intravenous fluid therapy

Recognizing when to decrease or stop intravenous fluids as part of the treatment of severe dengue is crucial to prevent fluid overload**.** When any of the following signs are present, intravenous fluids should be reduced or discontinued:

- Signs of cessation of plasma leakage
- Stable BP, pulse and peripheral perfusion
- Haematocrit decreases in the presence of a good pulse volume
- Apyrexia (without the use of antipyretics) for more than 24–48 hours
- Resolving bowel/abdominal symptoms
- Improving urine output.

Continuing intravenous fluid therapy beyond the 48 hours of the critical phase will put the patient at risk of pulmonary oedema and other complications such as thrombophlebitis.

Treatment of haemorrhagic complications

Mucosal bleeding may occur in any patient with dengue but if the patient remains stable with fluid resuscitation/replacement, this should be considered as a minor issue.

The bleeding usually improves rapidly during the recovery phase.

In patients with profound thrombocytopenia, ensure strict bed rest and protection from trauma.

Do not give intramuscular injections.

No evidence exist that prophylactic platelet transfusions are beneficial in haemodynamically stable patients.

Patients at risk of severe bleeding are those who:

- ✓ Have profound/prolonged/refractory shock
- ✓ Have hypotensive shock and multi-organ failure or severe and persistent metabolic acidosis
- ✓ Take non-steroidal anti-inflammatory agents
- ✓ Have pre-existing peptic ulcer disease
- ✓ Use of Anticoagulant therapy
- ✓ Have any form of trauma, including intramuscular injection

Severe bleeding should be recognized in the following situations:

- Persistent and/or severe overt bleeding in the presence of unstable haemodynamic
  status, regardless of the haematocrit level
- A decrease in haematocrit after boluses of fluid resuscitation together with unstable
  haemodynamic status
- Refractory shock that fails to respond to consecutive fluid resuscitation of 40–60 ml/kg
- Hypotensive shock with inappropriately low/normal haematocrit

- Persistent or worsening metabolic acidosis in patients with a well-maintained systolic BP, especially in those with severe abdominal tenderness and distension.

Blood transfusion is life-saving and should be given as soon as severe bleeding is suspected / recognized. However, blood transfusion must be given with care because of the risk of fluid overload. Do not wait for the haematocrit to drop too low before deciding on blood transfusion.

Management of bleeding

Gastrointestinal bleeding, epistaxis, or heavy menstrual bleeding may be severe enough to warrant blood transfusion.
Significant internal bleeding should be suspected in patients with signs of intravascular hypovolemia without elevation of hematocrit.

Factors that contribute to bleeding include thrombocytopenia due to decreased platelet survival and, in severe cases, prolonged prothrombin time (international normalized ratio >1.3) and frank disseminated intravascular coagulation due to liver failure.

Platelet transfusion has not been shown to be effective at preventing or controlling hemorrhage but may be warranted in patients with severe thrombocytopenia (<10,000/mm) and active bleeding.

In general, the preponderance of data does not support a role for prophylactic platelet transfusion in patients with severe thrombocytopenia in the absence of active bleeding.

Administration of intravenous Vitamin K is warranted for patients with severe liver dysfunction or prolonged prothrombin time.

The action plan for the treatment of haemorrhagic complications is as follows:

- If possible, attempts should be made to stop bleeding if the source of bleeding is identified e.g. severe epistaxis may be controlled by nasal adrenaline packing.
- If blood loss can be quantified, this should be replaced. If not, give aliquots of 5–10 ml/kg of fresh -packed red cells or 10–20 ml/kg of fresh or fairly fresh whole blood (FWB) at an appropriate rate and observe the clinical response. It is important that fresh whole blood or fresh red cells are given.
- Oxygen delivery at tissue level is optimal with high levels of 2,3 diphosphoglycerate (2,3 DPG). Stored erythrocytes lose 2,3 DPG, low levels of which impede the oxygen-releasing capacity of haemoglobin, resulting in functional tissue hypoxia.
- A good clinical response includes improving haemodynamic status and acid-base balance.
- Consider repeating the blood transfusion if there is further overt blood loss or no appropriate rise in haematocrit after blood transfusion in an unstable patient.
- There is no evidence that supports the practice of transfusing platelet concentrates and/or fresh-frozen plasma for severe bleeding in dengue.
- Observational studies show that transfusions of platelet concentrates and fresh frozen plasma in dengue were not able to sustain the platelet counts and coagulation profile. However, in the case of massive bleeding, they often exacerbate the fluid overload.
- Nevertheless, in certain situations such as obstetrical deliveries or other surgeries, transfusions of platelet concentrates with or without fresh frozen plasma should be considered in anticipation of severe bleeding.

- In gastrointestinal bleeding, H-2 antagonist and proton pump inhibitors have been used, but their efficacy has not been studied.
- Great care should be taken when inserting a nasogastric tube or bladder catheters which may cause severe haemorrhage. A lubricated orogastric tube may minimize the trauma during insertion.
- Insertion of central venous catheters should be done with ultra-sound guidance or by an experienced person.
- It is essential to remember that blood transfusion is only indicated in dengue patients with severe bleeding. Unnecessary blood transfusions cause the haematocrit to rise sharply, thus giving a false impression of haemoconcentration and severe plasma leakage leading to unwarranted fluid therapy.

Complications and intensive care management

Many of the complications seen in dengue are preventable if clinical team members are alert to the physiological problems of the three different phases.

When hypovolaemic shock is adequately managed, patients appear to "sail out" of the critical phase with mere parenteral fluids. But that belies the effort that has been invested in the monitoring and careful titration of intravenous fluid therapy, guided by frequent clinical and haematocrit
evaluation.

Causes of complications in dengue include:

• missed diagnosis at the frontline;
• inadequate monitoring and misinterpretation of vital signs;
• inadequate monitoring of fluid intake and urine output;
• late recognition of shock leading to profound and/or prolonged shock;
• late recognition of severe bleeding;
• too much or too little intravenous fluids i.e. not following/understanding the treatment guidelines;
• careless attitude towards aseptic techniques.

Outcome: These lead to a life-threatening situation characterized by one or a combination of the following:

- prolonged and/or profound shock
- severe bleeding with severe disseminated intravascular coagulopathy
- fluid overload;
- respiratory distress and failure
- multi-organ dysfunction of liver, kidneys and neurological system;
- irreversible shock and death.

This group of patients should be referred to a hospital with intensive care facilities wherever possible.

Send the appropriate investigations such as (Chest X ray, Abdominal Ultrasound, ECG, Coagulation Profile …so on) with senior consultation.

PREVENTION

In endemic areas — Approaches for the prevention of DENV infection and disease in endemic areas include mosquito control, personal protective measures, and vaccination.

Personal protection from infection:-

- ✓ Mosquito repellants
- ✓ Insecticide spraying
- ✓ Vaccination (CYD-TDV (Dengvaxia)

Mosquito control:-

- ✓ Reducing breeding sites
- ✓ Larva control
- ✓ Endosymbiotic control (A novel dengue control strategy consists of releasing mosquitoes infected with Wolbachia, an obligate intracellular bacterium.
  A. aegypti mosquitoes infected with Wolbachia are less susceptible to DENV infection than wild-type A. aegypti.

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