A DISSERTATION ON

CLINICAL PROFILE OF DENGUE FEVER IN PATIENTS OF > 13 YRS – A STUDY OF 122 CASES

Dissertation submitted to
TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY
Chennai

In partial fulfillment of the regulations

for the award of the degree of

MD BRANCH – I

GENERAL MEDICINE

MARCH 2010

GOVT. STANLEY MEDICAL COLLEGE

CHENNAI

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

CHENNAI – TAMILNADU

CERTIFICATE

This is to certify that this dissertation entitled "CLINICAL PROFILE OF DENGUE FEVER IN PATIENTS OF > 13 YRS – A STUDY OF 122 CASES" submitted by DR.RAJASEKAR.D to The Tamil Nadu Dr. M.G.R. Medical University Chennai is in partial fulfillment of the requirement of the award of M.D DEGREE BRANCH I(General medicine) and is a bonafide research work carried out by him under direct supervision and guidance.

Signature of the Unit Chief

Signature of Professor and HOD

Signature of the Dean

DECLARATION

I solemnly declare that the dissertation titled "CLINICAL PROFILE OF DENGUE FEVER IN PATIENTS OF > 13 YRS – A STUDY OF 122 CASES" was done by me at Stanley Medical College and Hospital during 2007-2009 under the guidance and supervision of **PROF.S.TITO. M.D.** The dissertation is submitted to the Tamil Nadu Dr. MGR Medical University towards the partial fulfillment of requirements for the award of M.D. DEGREE (BRANCH-I) in General Medicine.

Place: Chennai.

Date: Dr.Rajasekar.D

ACKNOWLEDGEMENT

I owe my thanks to the dean, Stanley medical college and hospital, **Prof. Dr. A.PRIYA. M.S.,D.O.,** for allowing me to avail the facilities needed for my dissertation work.

I am grateful to **Prof. S.RAMASAMY. M.D.,** Professor and Head of the Department of Medicine, Stanley medical college for permitting me to do the study and for his encouragement.

I have great pleasure in expressing my deep sense of gratitude and respect for **PROF. S.SHIVAKUMAR. M.D.,** Former Professor & Head of Dept. of Medicine, Stanley Medical College and Hospital and **Prof. S.TITO. M.D.,** Additional Professor, Department of medicine and chief of medical unit V, Stanley medical college and hospital, Chennai, for approving this study and giving suggestions and guidance in preparing this dissertation.

I am extremely thankful to my unit assistant professors, **DR. THILAGAVATHY. M.D., DR. T.ARUN. M.D.,** and **DR.R. ARUN. M.D.,** and **DR. G.VASUMATHY. M.D.,** Registrar, Department of medicine for their valuable guidance and constant encouragement.

Last but not least, I sincerely thank all my colleagues who shared their knowledge and my patients without whose cooperation this study would have been impossible.

CONTENTS

INTRODUCTION

INTRODUCTION

Dengue infection is one of the commonest mosquito borne acute febrile viral haemorrhagic illness. Dengue epidemics were reported throughout the world, but most frequently from the region of South Asia. Most of the studies regarding Dengue infection/virus, epidemiological, Clinical and management pattern were studied in the region of South Asia.

Dengue infection presents with varied clinical manifestation ranging from asymptomatic or simple viral illness to circulatory shock (DSS). Dengue infection has the potential to cause severe bleeding, shock and death. So, early diagnosis and recognition of complication is cornerstone in management. Even though, Dengue infection admissions are common in pediatric age group, adult patients admissions has also increased in recent years. However, the datas of Dengue infection among adults are limited. This study is to get additional datas on Dengue infection among adults from Chennai, which is from the region of South Asia.

This study is done in Stanley Medical College, North Chennai which is highly endemic for communicable infectious diseases. This study deals with Clinical and Laboratory profile of Dengue infection among adults from North Chennai.

AIM OF THE STUDY

AIM OF THE STUDY

To study the Clinical profile of Dengue Infection among age of >13yrs in the North Chennai, attending Medical Department, Govt. Stanley Hospital.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Dengue fever (DF) is a mosquito borne acute febrile viral disease frequently presenting with headaches, bone or joint and muscular pains, rash and leukopenia. Dengue hemorrhagic fever (DHF) is characterized by four major clinical manifestations: Fever, Thrombocytopenia, Hemorrhagic phenomena and Plasma leakage manifestations. If DHF associated with circulatory shock is called Dengue Shock Syndrome (DSS).

EPIDEMIOLOGY

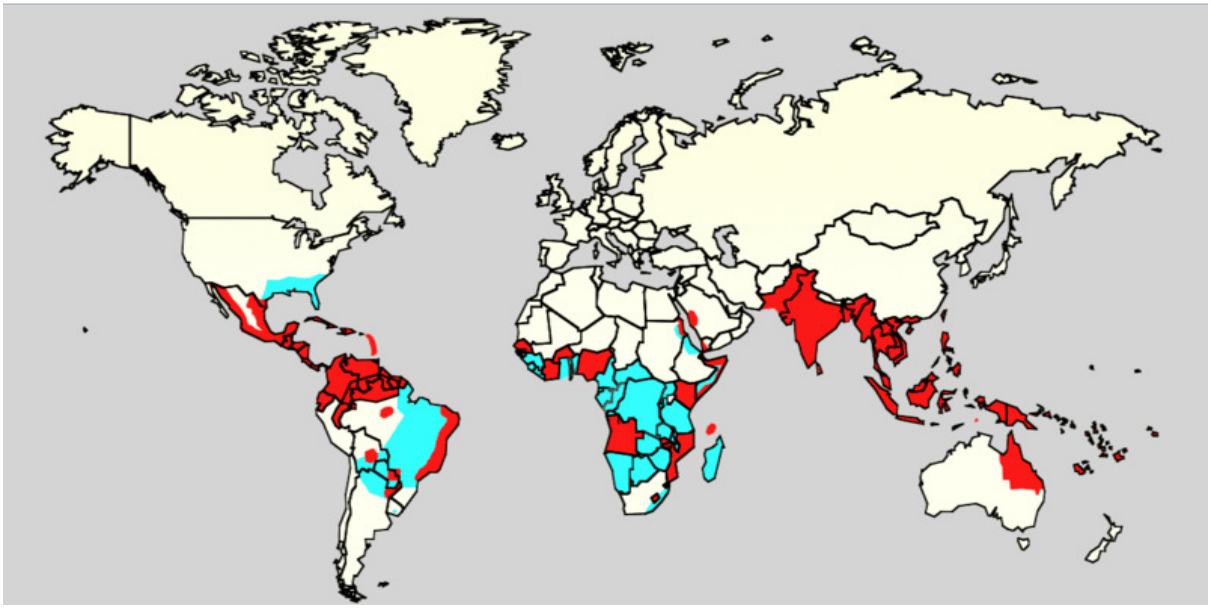
During the 19th century, dengue was considered a sporadic disease, causing epidemics at long intervals. However, dramatic changes in this pattern have occurred and currently, dengue ranks as the most important mosquito borne viral disease in the world. In the past 50 years, its incidence has increased 30-fold with significant outbreaks occurring in five of six World Health Organisation (WHO) regions. At present, dengue is endemic in 112 countries in the world[2].

Around 2.5 to 3 billion people, living mainly in urban areas of tropical and subtropical regions, are estimated to be at risk of acquiring dengue viral infections. Estimates suggest that annually 100 million cases of dengue fever and half a million cases of dengue haemorrhagic fever(DHF) occur in the world with a case fatality in Asian countries of 0.5%–3.5%.Of those with DHF, 90% are children less than 15 years of age[5].

The disease was first recognized in the Philippines in 1953. This gradually spread to other countries in the region. Major epidemics occurred in other regions of the world in the 1980s and 1990s and were caused by all four dengue viral serotypes. While the predominant

serotype in the 1980s and the early 1990s was DEN-2, in recent years it has changed to the DEN-3 serotype. In 1998, a pandemic of dengue viral infection occurred, when 1.2 million cases of dengue fever and DHF were reported from 56 countries worldwide. The world population was exposed to a new subtype of the DEN-3 virus (subtype III), which originated in the Indian subcontinent and later spread to involve other continents. Exposure of a non-immune population to this new subtype of DEN-3 may have been the cause of this pandemic[5].

Worldwide dengue distribution – 2006[2]

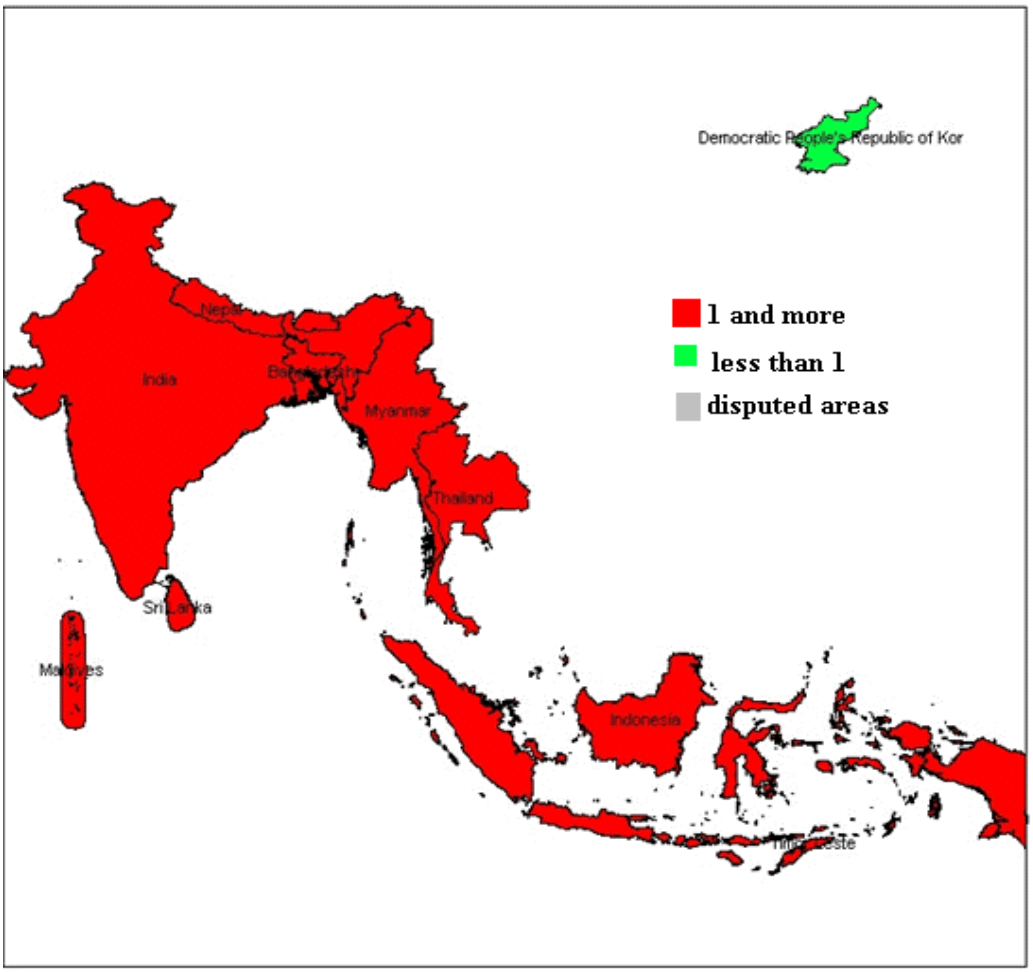


(Red: Epidemic dengue. Blue:Aedes aegypti.)

EPIDEMIOLOGICAL TRENDS IN SOUTH EAST ASIA[3,6]

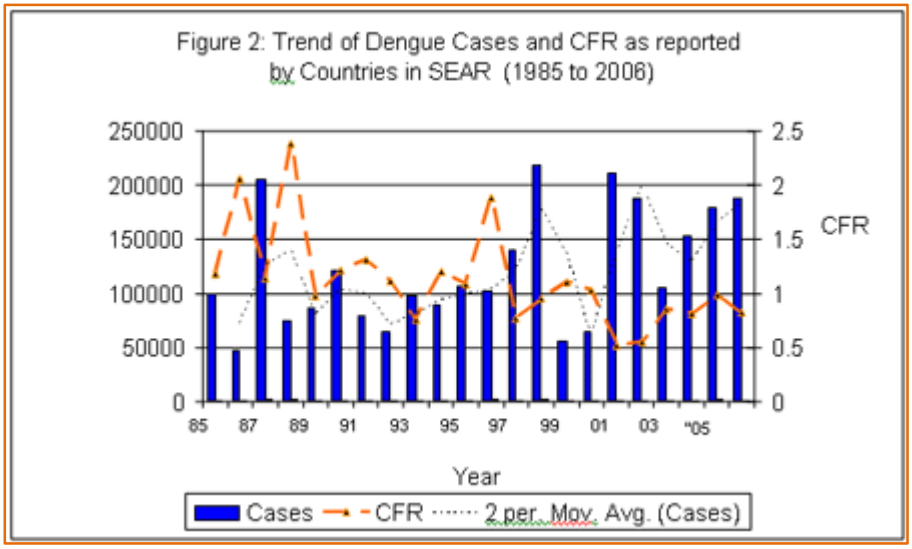
The first epidemic of DHF in South East Asia occurred in 1954 in Manila, Philippines.

Following this, epidemics have occurred in nearly all countries in this region. Although serological surveys conducted in Indonesia showed that DEN-1 and DEN-2 were the prevalent serotypes until the late 1980s, the DEN-3 serotype has been the predominant serotype in the



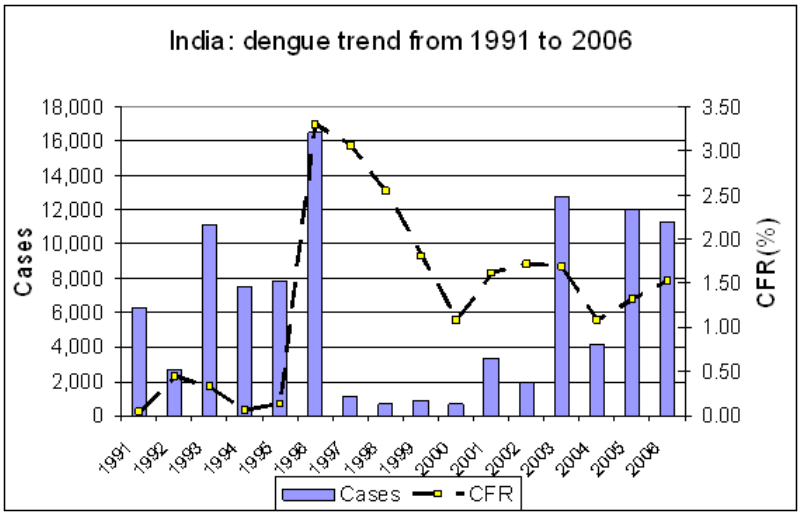
recent outbreaks. DEN-3 has been associated with severe dengue epidemics[10]. Although DEN–4 has been isolated in almost all epidemics, it is primarily detected in secondary dengue infections.

In 2003 only 8 countries in South East Asia Region reported dengue cases. As of 2006, ten out of the eleven countries in the Region[6] (Bangladesh, Bhutan, India, Indonesia, Maldives, Myanmar, Nepal, SriLanka, Thailand and Timor-Leste) reported dengue cases. Bhutan reported the first dengue outbreak in 2004. An outbreak, with a high case fatality rate (3.55%) was first reported in Timor-Leste in 2005[7]. Nepal reported dengue cases for the first time in November 2006. The Democratic Peoples'Republic of Korea is the only country in this Region of WHO that has no report of indigenous transmission of DF/DHF[5].



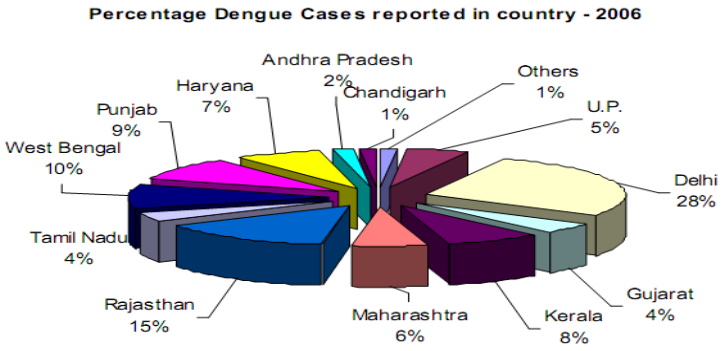
EPIDEMIOLOGICAL TRENDS IN INDIA

Dengue fever was first reported in 1963 from Calcutta city. Since then several outbreaks of dengue fever was reported from India with a major epidemic of dengue haemorrhagic fever that occurred in Delhi in 1996 when 10,252cases and 423 deaths were reported. Cases have been reported from the neighboring states of Haryana, Punjab, Rajasthan, Utter Pradesh and two southern and western states. DEN-2 was isolated during this epidemic and the proportion of DHF to DF was very high. The number of DF/DHF cases and deaths reported since the epidemic has been low till 2002 but again has risen in 2003[39]. In 2005, both the reported dengue cases and deaths show threefold increase as compared to 2004. The case fatality has been above 1% for the last 10 years[7]. However, the number of reported dengue cases and deaths are mainly from the capital city Delhi and the other states that have small outbreaks go unreported. Therefore the case surveillance needs further strengthening.



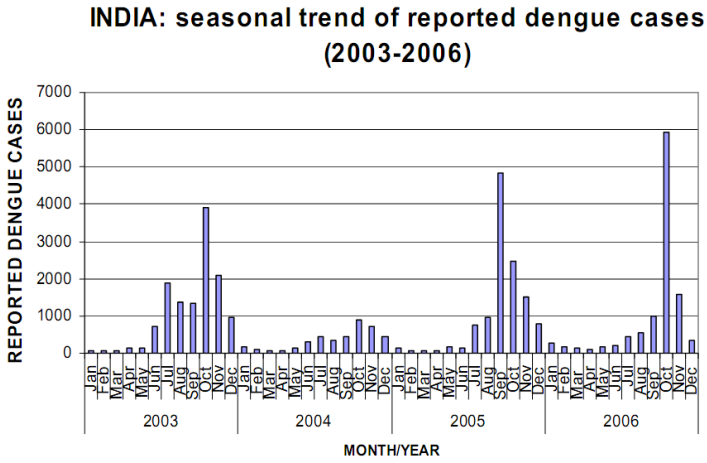
Aedes aegypti was reported from all the affected areas with house indices exceeding 20%. Surveillance activities are carried out on a limited scale by the National Institute of Virology, Pune and few other institutions in the country. Since 1996[16], dengue control activities are coordinated and carried out by the National Anti-Malaria Programme.

In 2006 the number of cases reported as compared to 2005 shows some reduction whereas the Case fatality rate has remained above 1%.



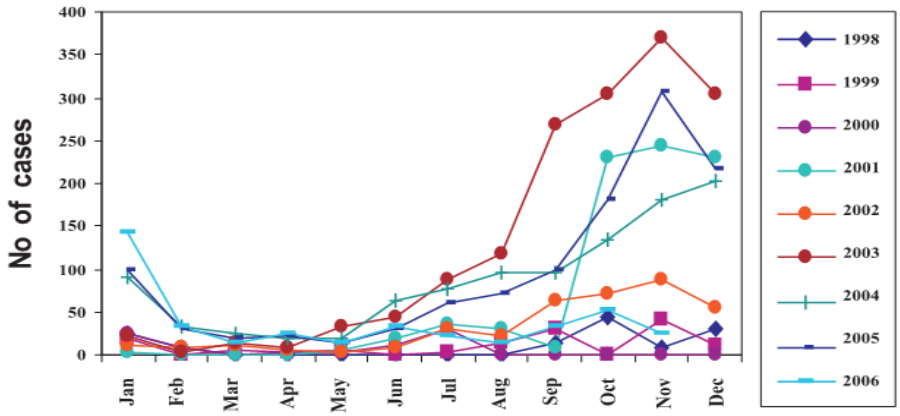
During 2007(upto 19 June), 383 cases and 6 deaths have been reported from Kerala (188 cases); Gujarat (93); Maharastra(15); Tamil Nadu(41); Karnataka(27); Haryana(5); Delhi (4); Rajasthan (4), Orissa (4), Chandigarh (1) and Uttar Pradesh (1).

The trend data from India shows that cases generally start to increase from august onwards, which is post monsoon season. More importantly, it is clear from data that breeding of Aedes mosquitoes however begins in June itself. Such data may be taken into consideration



,

while planning in advance for dengue prevention and control. Thus vector surveillance and control measures supported by community mobilization for behavioural change activites need to be taken before June and sustained throughout the rainy season.



Month-wise incidence of dengue cases in Tamil Nadu.

In Tamil Nadu, there has been an increase in the number of dengue cases reporting units during the last nine years. In 1998, dengue cases were reported from only 4 units which

increased to 33 units in 2006 due to the availability of serodiagnostic facilities at different centres in the State. Of the 30 districts in Tamil Nadu, dengue cases have been reported from 29 districts

between 1998 and 2005 which include DF/DHF outbreaks in Chennai in 2001[15], Nagercoil and Trichirapalli (2003) (unpublished data) and DF outbreaks in Krishnagiri and Dharmapuri districts8 in 2001. It is not clearly known why dengue cases have not been reported so far in Nilgiris district although the fever surveillance system is well in existence through Primary Health Care network. The probable reason could be that Nilgiris is a high altitude area where the abundance of vector population and vector competence for transmission of the disease needs to be studied. A total of 128 cases and 5 deaths were reported in 1998 which increased to 1600 cases and 12 deaths in 2003 and 1150 cases and 8 deaths in 2005.

Recently, between October 2001 and January 2002, an epidemic of dengue emerged in Chennai, affecting adults and children; majority affected were children less than 15 yrs of age.

DENGUE FEVER IN ADULTS

Children were predominantly affected, but in recent years clinicians have seen increasing numbers of adult dengue patients[17], with both significant morbidity and mortality. This rise in incidence among adults adversely affects developing countries economy. It also affects health planning, and is further compounded by the general lack of systematically collected information on the natural history of dengue in such patients. This often leads health planners and clinicians to base their decisions regarding resource allocation and clinical management on personal experiences[8], rather than on tangible evidence.

Trends of increasing numbers of adult dengue patients can also be seen in other South Asian, South-East Asian and Latin American countries. If we are to take effective steps to reduce this trend and treat this group optimally, pooling information from different countries is important. At present, information on adult dengue infections in South Asia is quite limited.

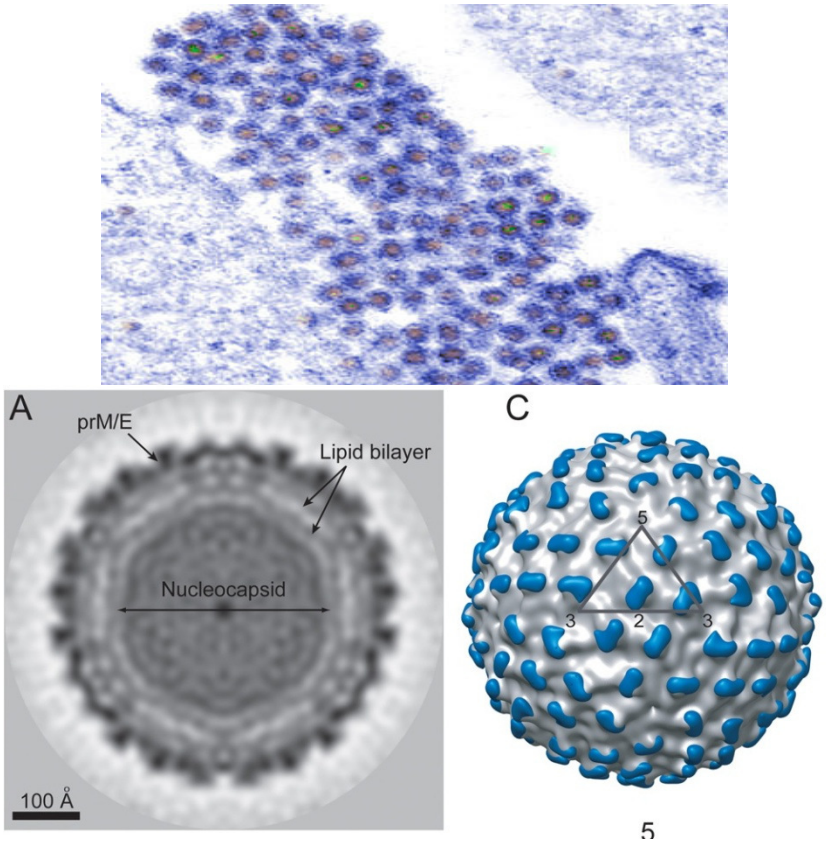
Adults[9] differed in the clinical manifestations of dengue infection from children. It is necessary for health personnel to take these differences into consideration when identifying probable cases of dengue infection. Such data should be put to use in the recognition and management of cases. DHF, DSS and deaths has been reported among adults in various studies done in SEA region.

G.N. Malavige *et al*[8] has done one study in adult patients with confirmed dengue infections (n=108) treated in a general medical ward in Sri Lanka from 24 April to 31 July 2004. In this study, there were 68 male and 40 female patients, mean age was 26.6 years. Dengue fever (DF) was seen in 33 (30.6%) and dengue haemorrhagic fever (DHF) in 75 (69.4%). Of the 37 (34.3%) with primary dengue infections, 19 (51.4%) developed DF and 18 (48.6%) developed DHF. Overall, 42 patients (38.9%) had bleeding manifestations. These adults showed differences in clinical and laboratory findings, disease severity and mortality, compared to children seen during the same epidemic. Secondary dengue infections were significantly associated with development of severe disease (OR 5.0, 95%CI 1.9–13.5, $p < 0.001$) Mortality was 3.7%. This study clearly demonstrates the Dengue infection pattern and complications among adults.

Dengue studies were also done by Adriana O *et al*, NP Singh *et al* and Janak Kishore *et al*. Tthese studies gives the clinical and epidemiological pattern of Dengue infection among adults.

DENGUE VIRUS[3]

The dengue virus[12] is a single stranded RNA virus belonging to the flaviviridae family. There are four serotypes (DEN 1–4), classified according to biological and immunological criteria. The viral genome is approximately 11 kb in length. The mature virion consists of three structural (core, membrane associated, and envelope) and seven non-structural (NS1, NS2a, NS2b, NS3, NS4a, NS4b, and NS5) proteins. The envelope protein is involved in the main biological functions of the virus. It binds to receptors on host cells, allowing the virus to be transported through it. In addition, the envelope protein is associated with haemagglutination of erythrocytes, induction of neutralising antibodies and protective immune responses.



5

Non-structural proteins (NS1–NS5) expressed as both membrane associated and secretory forms have also been implicated in the pathogenesis of severe disease.NS1 gets expressed on the surface of infected cells. Preliminary evidence suggests its involvement in viral RNA replication. Plasma levels of secreted NS1 (sNS1) correlate with viral titres, being higher in patients with DHF compared with dengue fever. Moreover, elevated free sNS1 levels within 72 hours of onset of illness identify patients at risk of developing DHF. Very high levels of NS1 protein are detected in acute phase samples from patients with secondary dengue infections but not primary infections. This suggests that NS1 may contribute to formation of circulating immune complexes, which are thought to have an important role in the pathogenesis of severe dengue infections.

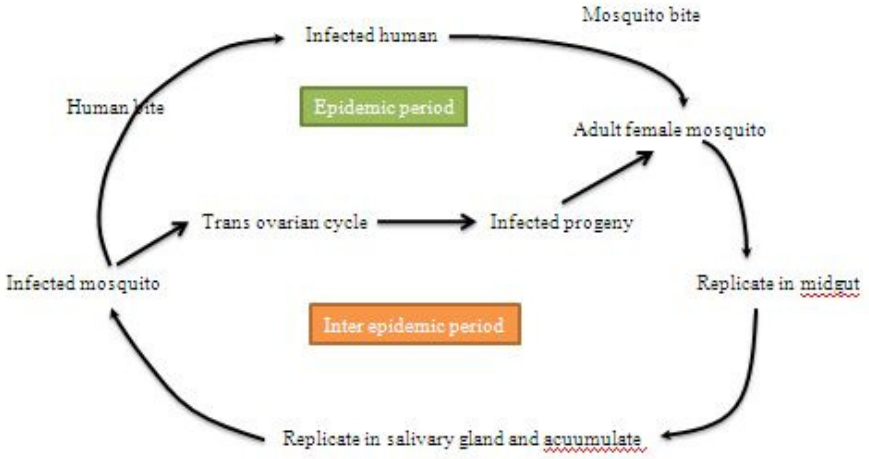
The dengue virus shares antigenic epitopes with other flaviviruses such as Japanese encephalitis virus. These shared epitopes may lead to production of cross reactive antibodies and hence interfere with serological diagnosis. However, antibodies directed to the prM protein of dengue viruses are species specific (not cross reactive with those of other flaviviruses) and may be useful for seroepidemiological[36] studies in dengue (especially in countries where other flaviviruses are endemic).

MOSQUITO VECTOR

Aedes[14] is the vector for Dengue transmission. The genus includes Aedes aegypti, Aedes albopictus, and Aedes polynesiensis.The primary and most important vector is A aegypti, but A albopictus and A polynesiensis may also be involved. Aedes aegypti, a container breeding, day biting mosquito is found in tropical and subtropical areas.They rest indoors, mainly in living

rooms and bedrooms. This maximises man vector contact and minimises contact with insecticides sprayed outdoors, hence contributing to difficulty in controlling this vector.

Aedes aegypti can breed in polluted water or small collections water such as flower vases or coconut shells.Eggs can survive for long periods, as they are capable of withstanding desiccation. Improper disposal of garbage or inadequate wastewater drainage facilitates, both consequences of unplanned urbanisation, may be responsible for high mosquito densities in endemic areas.



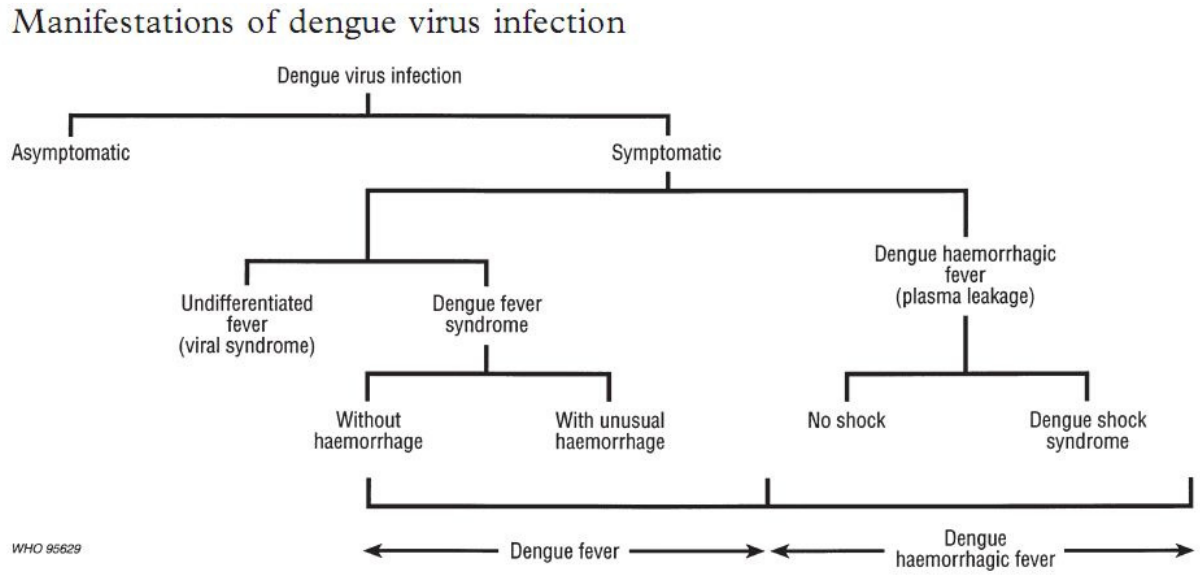
Significant increases in the mosquito larval populations are seen during the rainy season. This may be a reason why epidemics of dengue tend to coincide with the rainy season. Furthermore, ambient temperature and relative humidity affect viral propagation in mosquitoes; rates being highest in climates resembling the rainy season. Environmental temperatures also affect the time for acute viraemia in female mosquitoes, being shorter with rises in temperature.

After biting an infected human, dengue viruses enter an adult female mosquito. The virus first replicates in the midgut, reaches the haemocoel and haemolymph, and then gains access to different tissues of the insect. After viral replication in the salivary glands, the infected mosquito can transmit the virus to another human. Ultrastructural studies show viral particles within the nervous system, salivary glands, foregut, midgut, fat body, epidermal cells, ovary and internal body wall lining cells of the mosquito. In contrast, they are absent from muscle, the hindgut, and malphigian tubules. Compared with uninfected mosquitoes, infected ones take longer to complete a blood meal. This may contribute to the efficiency of A aegypti as a dengue viral vector.

The existence of transovarial dengue virus transmission in aedes infected female mosquitoes, allows propagation of virus to their progeny. Such a process would allow it to act as a reservoir for virus maintenance during interepidemic periods (without human or other vertebral host participation). Reports also suggest that dengue viruses may be transmitted sexually from the male to female mosquitoes, but not vice versa.

CLINICAL MANIFESTATIONS[1]

Dengue infection is a spectrum of disease ranging from asymptomatic to DSS.



UNDIFFERENTIATED FEVER

This usually follows a primary infection but may also occur during a secondary infection. Clinically it is indistinguishable from other viral infections.

DENGUE FEVER

Dengue fever may occur either as primary or secondary infection. The onset is sudden with high fever, severe headache (especially in the retro-orbital area), arthralgia, myalgia, anorexia, abdominal discomfort, and sometimes a macula-papular rash. The fever may be biphasic and tends to last for 2–7 days. Flushing, a characteristic feature is commonly observed on the face, neck, and chest. Coryza may also be a prominent symptom especially in infants. Younger children tend to present with coryza, diarrhoea, rash and seizure, and less commonly

with vomiting, headache, and abdominal pain. Although, haemorrhagic manifestations are uncommon in dengue fever, petechiae/pupura, gastrointestinal bleeding, epistaxis, and gingival bleeding have been observed in some individuals. A positive tourniquet test has been reported in many individuals with dengue fever possibly due to reduced capillary fragility. Recovery from dengue fever is usually uneventful, but may be prolonged especially in adults.

DENGUE HAEMORRHAGIC FEVER

DHF usually follows secondary dengue infections, but may sometimes follow primary infections, especially in infants. In such infants, maternally acquired dengue antibodies are presumed to enhance primary infections. Such a phenomenon has not been described in human infections other than dengue. DHF[11] is characterised by high fever, thrombocytopenia, haemorrhagic phenomena, and features of circulatory failure. For purposes of description DHF is divided into three phases—namely: febrile, leakage, and convalescent phases. Furthermore, according to severity DHF is divided into four grades.

The febrile phase begins with sudden onset fever accompanied by generalized constitutional symptoms and facial flush.The fever is high grade, intermittent, and associated with rigors. Epigastric discomfort, myalgia, vomiting, and abdominal pain are common and patients are usually quite miserable. Sore throats and febrile convulsions may be seen, especially among young children. Tender hepatomegaly is observed in almost all patients and splenomegaly may be seen in some. A macular papular rash similar to that seen in dengue fever is also seen in many patients. The fever lasts for 2–7 days and is followed by a fall in temperature to normal or subnormal levels. At this point, the patient may recover or progress to the phase of plasma leakage. Those who remain ill despite their temperature subsiding are more

likely to progress to DHF. Clinical deterioration usually occurs during defervescence (often between days 3 and 4)

PLASMA LEAKAGE MANIFESTATIONS[3]

Tachycardia and hypotension characterise the onset of plasma leakage. When plasma leakage is severe, patients may develop other signs of circulatory disturbance such as prolonged capillary refill time, narrow pulse pressures, and shock. Inadequate treatment of such patients often leads to profound shock. During the phase of plasma leakage (first 24–48 hours after onset of DHF), pleural effusions and ascites are common. Pleural effusion is usually seen on the right side; a right decubitus chest radiograph or ultrasound chest is best for detecting small effusions. Abdominal ultrasound scans may demonstrate ascites or a oedematous gall bladder wall. Pericardial effusions may also occur. This latter complication is uncommon, but is associated with high morbidity and mortality.

In DHF, bleeding may occur from any site and does not correlate with the platelet counts. Haemorrhagic manifestations usually occur once the fever has settled. Minor degrees of bleeding may manifest as gum bleeding and petechiae. The commonest site of haemorrhage is the gastrointestinal tract, which manifests as haematemesis or melaena, followed by epistaxis. Vaginal bleeding is commonly reported in females.

Convalescence in DHF is usually short and uneventful. The return of appetite is a good indicator of recovery from shock. Bradycardia is also seen in this period. If present, a confluent petechial rash with erythema and islands of pallor (usually known as a recovery rash) is

characteristic of dengue infections. During the convalescent stage, many patients also complain of severe itching especially on the palms and soles.

DENGUE SHOCK SYNDROME[3]

Severe plasma leakage leads to decreased intravascular volume followed by hypotension & shock. Usually occurs 3-7 days after the onset of fever. DSS manifests as cold clammy skin,circumoral cyanosis, severe abdominal pain, tachycardia, hypotension & shock. Prolonged shock may leads to metabolic acidosis which aggravates coagulopathy, leads to DIC and massive haemorrhage. Sometimes may be associated with encephalopathy. If shock is not corrected patient may die within 12-24hrs.patients may recover from shock in 2-3days with effective supportive management.

DIFFERENTIAL DIAGNOSIS OF DENGUE FEVER AND DHF[1]

DENGUE FEVER

Infectious mononucleosis, Chickengunya viral infections, Coxsackie and other enteroviral infections, Rickettsial infections, Leptospirosis and Influenza

DHF

Leptospirosis, Chikengunya viral infections, Kawasaki disease, Yellow fever, Hanta viral infections, Other viral haemorrhagic fevers and Meningococcal septicemia.

LABORATORY FINDINGS

In most cases of dengue fever, platelet counts and serum biochemistry are normal. However, leucopenia, thrombocytopenia, and raised liver enzymes may be seen. In contrast, DHF is always accompanied by a platelet count ,<$100x10^9$/l, haemoconcentration (a rise in the packed cell volume .20% of basal levels), leucopenia, and raised liver enzymes Elevation of both alanine and aspartate aminotransferase levels occur with plasma aspartate aminotransferase levels being higher in children who develop DHF than in those with dengue fever.

A leucopenia of $5x10^9$/l has been suggested to predict the onset of DHF. Initial leucopenia is followed by a relative lymphocytosis (with more than 15% atypical lymphocytes) towards the end of the febrile phase. Abnormal coagulation profiles (prolonged partial thromboplastin time and prothrombin time, raised fibrinogen degradation products), hypoalbuminaemia, and reduced serum complement levels are also seen. These coagulation abnormalities suggest that there is activation of both coagulation and fibrinolysis during acute infection and the degree of activation being greater in severe DHF and dengue shock syndrome.

COMPLICATIONS[3]

Severe dengue infections may give rise to many complications such as liver failure, disseminated intravascular coagulation, encephalopathy, myocarditis, acute renal failure, and haemolytic uraemic syndrome.

LIVER FAILURE

Since hepatocytes and Kupffer cells support viral replication, liver involvement is common in all forms of dengue infection. Levels of aspartate transaminase and alanine transaminase are significantly higher, and globulins significantly lower among patients with the more severe grades of DHF. Fulminant liver failure can occur due to hepatitis or focal necrosis of the liver causing hepatic encephalopathy and even death. Jaundice may be present. Neurological examination may show hyper-reflexia or an extensor plantar response. Electrolyte abnormalities and hypoglycaemia may accompany liver enzyme abnormalities.

ENCEPHALOPATHY[18]

Encephalopathy has been reported in 0.5% of patients with DHF, and has a mortality rate of 22%. Many factors contribute towards development of encephalopathy including: hepatic dysfunction, electrolyte imbalances, cerebral oedema (caused by vascular changes leading to fluid extravasation), hypoperfusion (due to circulatory disturbances), and dengue encephalitis. The dengue virus has been isolated from the cerebrospinal fluid of some patients having features of encephalitis. Other neurological manifestations such as altered consciousness, seizures, spasticity of limbs, hemiplegia, and a positive Kernig's sign have also been reported in 5.4% of patients with dengue.

MYOCARDITIS

Acute reversible myocarditis[19] has been reported in patients with dengue infections. ST segment and T wave changes in the electrocardiogram together with low ejection fractions and global hypokinesia on radionuclide ventriculography have been found. No myocardial necrosis

was detected in any of the patients. In another study, 16.7% of children had left ventricular dysfunction when assessed by two dimensional and colour Doppler echocardiography. The left ventricular failure may contribute to hypotension seen in DHF/dengue shock syndrome and may have implications in fluid management as fluid overload may worsen the condition.

DIAGNOSIS

CLINICAL DIAGNOSIS

WHO CASE DEFINITIONS FOR DENGUE INFECTION[1]

CASE DEFINITION FOR DENGUE FEVER

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

And

- Supportive serology (a reciprocal haemagglutination-inhibition antibody titre ≥ 1280, a comparable IgG enzyme-linked immunosorbent assay titre or a

positive IgM antibody test on a late acute or convalescent-phase serum specimen)

Or

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

Confirmed: case confirrmed by laboratory criteria

Reportable: any probable or confirrmed case should be reported.

LABORATORY CRITERIA

- 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 cerebro spinal fluid samples by immunohistochemistry, immunoflouorescence or ELISA; or
- Detection of dengue virus genomic sequences in autopsy tissue serum or cerebrospinal fluid samples by polymerase chain reaction (PCR).

CASE DEFINITION FOR DENGUE HAEMORRHAGIC FEVER

The following must all be present:

- Fever

- Haemorrhagic tendencies, evidenced by at least one of the following:
  - a positive tourniquet test
  - petechiae, ecchymoses or purpura
  - bleeding from the mucosa, gastrointestinal tract, injection sites or other locations.
  - haematemesis or melaena.
- Thrombocytopenia ($\leq$ 100000 cells per mm^3)
- Evidence of plasma leakage due to increased vascular permeability, manifested by at least one of the following:
  - rise in the haematocrit equal to or greater than 20% above average for age, sex and population;
  - drop in the haematocrit following volume-replacement treatment equal to or greater than 20% of baseline;
  - signs of plasma leakage such as pleural effusion, ascites and hypoproteinaemia.

CASE DEFINITION FOR DENGUE SHOCK SYNDROME

All of the above four criteria for DHF must be present, plus evidence of circulatory failure manifested by:

- Rapid and weak pulse.
- Narrow pulse pressure ($\leq$20mmHg).
- Hypotension for age and cold clammy skin and restlessness .

GRADING SEVERITY OF DENGUE HAEMORRHAGIC FEVER

Grade-I : Fever + Thrombocytopenia +Plasma leak + Tourniquet Test positive

Grade -II: Grade I + Spontaneous Bleeding

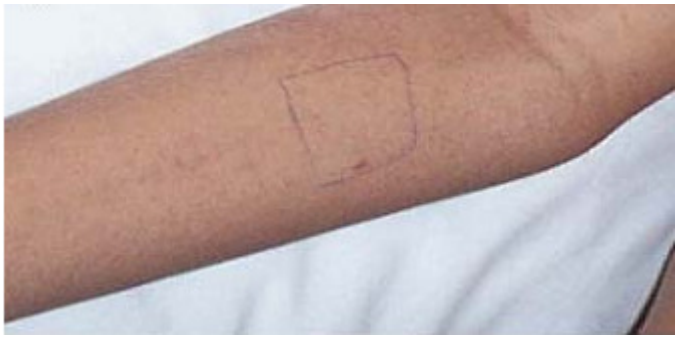
Grade III: Grade II + narrow pulse Pressure or hypotension

Grade IV: Grade II + Profound Shock

Grade III and IV are considered as Dengue Shock Syndrome (DSS)

TOURNIQUET TEST (HESS TEST)[1]

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 2.5cm (1 inch) square are observed. The test may be negative or mildly positive during the phase of profound shock. It usually becomes positive, sometimes strongly positive, if the test is conducted after recovery from shock.

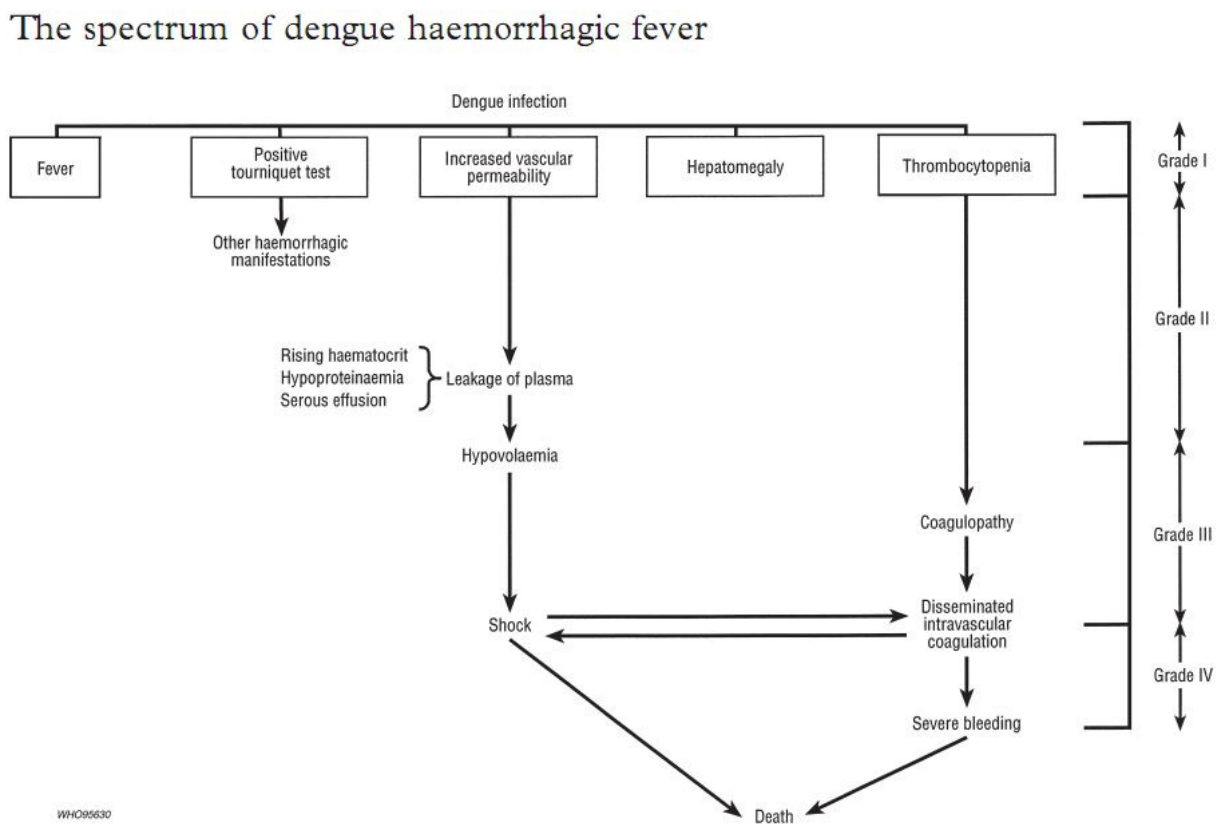


Tourniquet test positive

Overall, a positive standard tourniquet test is reasonably specific for dengue infection, if performed on children suspected to have dengue in an endemic area where the probability of

dengue is high. It should be remembered that a negative test does not exclude dengue infection. A careful inspection of the skin for petechiae or other bleeding can contribute importantly to the correct diagnosis.

The tourniquet test should be regarded as suggestive of dengue infection but not used as an absolute criterion for making the diagnosis. Nor is the test helpful in defining the severity of illness. It can be difficult to interpret in dark-skinned individuals. The fact that petechiae may be difficult to observe on dark skin may contribute to the differences seen in the results of the tourniquet test in different populations. The time taken to perform a tourniquet test may be better applied to an overall assessment of the patient with suspected dengue infection[1].

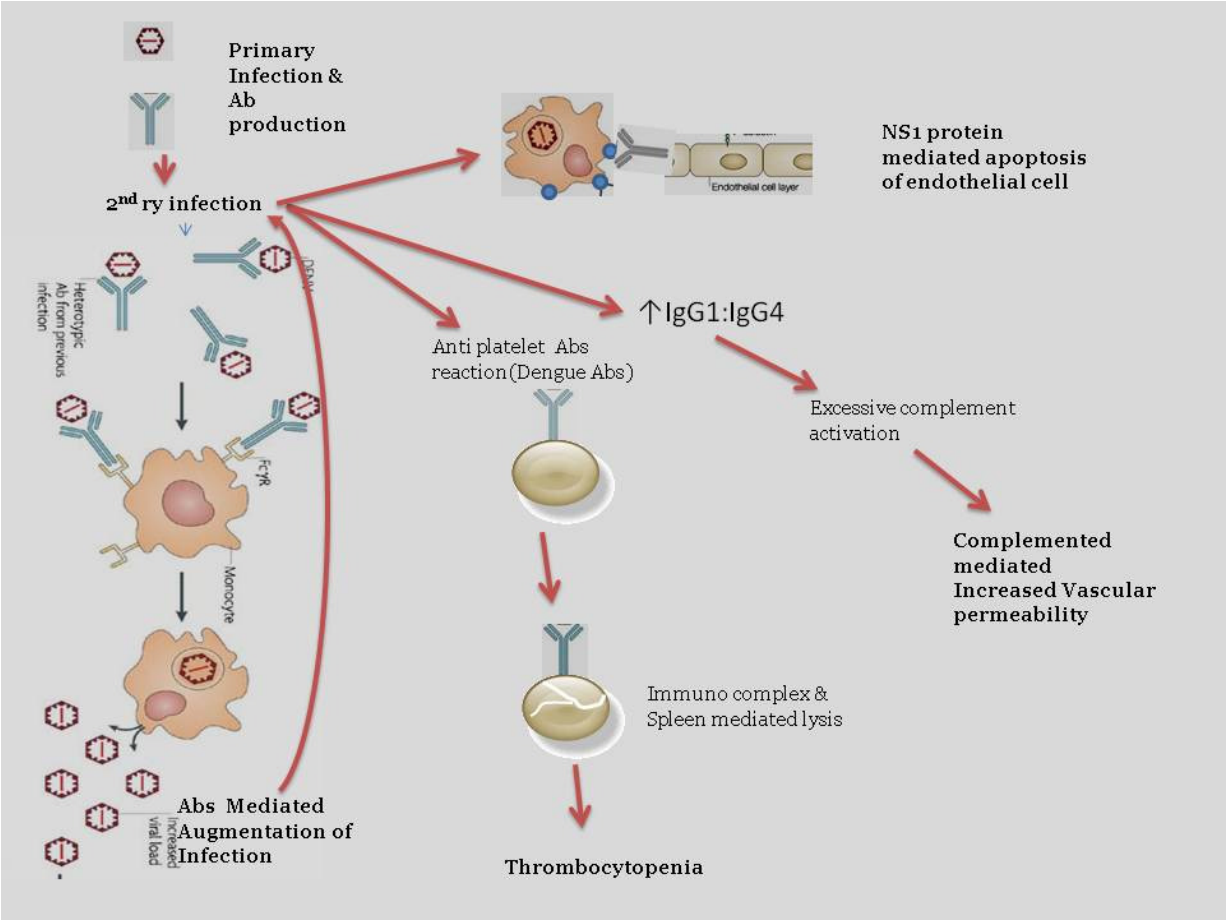


PATHOGENESIS OF DENGUE FEVER/DHF

Dengue may be caused by any of the dengue viral serotypes.Generally, infection with one serotype confers future protective immunity against that particular serotype but not against other serotypes. Furthermore, when infected for a second time with a different serotype, a more severe infection may occur. This is due to a phenomenon referred to as antibody dependent enhancement, where antibodies against the first serotype enhance infection with the second serotype. However, as only 2%–4% of individuals with a secondary dengue infection develop severe disease, antibody dependent enhancement alone cannot wholly explain this process. At present, reasons as to why only some individuals develop symptomatic infection are not known, but active research is being pursued by several groups to clarify such mechanisms.

After the bite of an infected mosquito, the dengue virus enters the body and replicates within cells of the mononuclear phagocyte lineage (macrophages, monocytes, and Bcells). Additionally, infection of mast cells, dendritic cells, and endothelial cells are known to occur. The incubation period of dengue infections is 7–10 days. A viraemic phase follows where the patient becomes febrile and infective. Thereafter, the patient may either recover or progress to the leakage phase, leading to DHF and/or dengue shock syndrome. Peak plasma viraemia correlates with the severity of dengue infections. Differences in **antibody, cytokine, and T-cell responses** are seen among patients with uncomplicated dengue fever or DHF/dengue shock syndrome.

1. ANTIBODY RESPONSES TO THE DENGUE VIRUS [3,20.22]



Antibody dependent enhancement is thought to play a key part in the pathogenesis of severe dengue infections. During secondary dengue infections, antibodies already present in the patient form complexes with the dengue virus. The Fc portion of these antibodies can then bind to FccRI and FccRII bearing cells and result in an increased number of cells being infected by the dengue virus. Antibody dependent enhancement is found to occur only in the presence of subneutralising concentrations of dengue antibodies.

2. CYTOKINE RESPONSES IN DENGUE INFECTIONS[3,21,23]

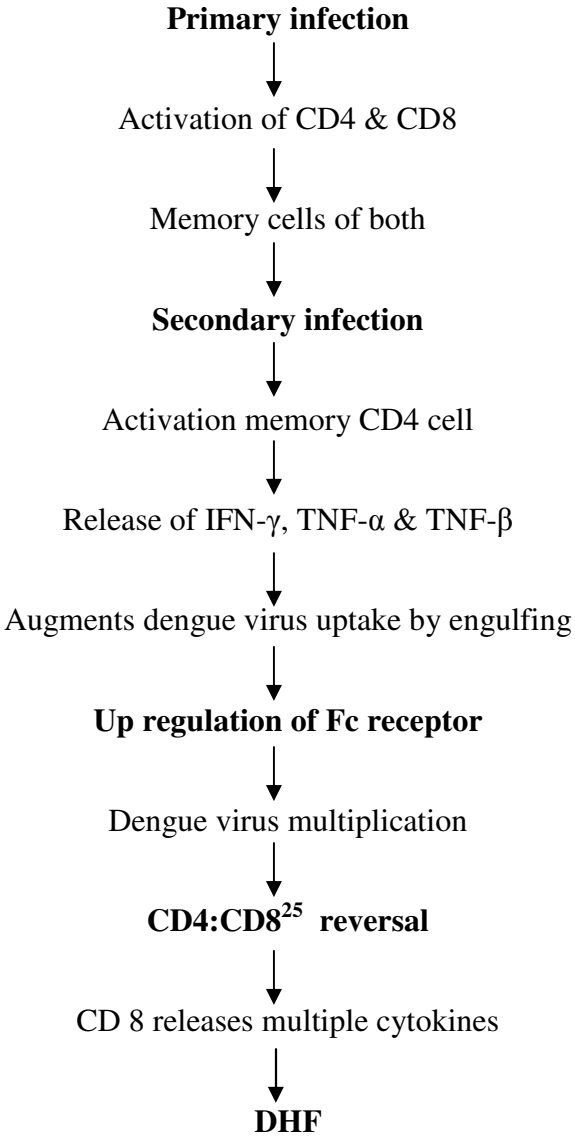
TNF-α, IL-2, IL-6 and IFN-γ increased in initial 3 days.IL-13 and IL-18 is very high in severe infection. IL-2 and IFN-γ are Th1 type, IL-5 and IL-4 Th2 type cytokines. Thus, it has been suggested that Th1 responses are seen during the first 3 days and Th2 responses occur later. Increased levels of IL-13 and IL-18 have also been reported during severe dengue infections, with highest levels seen in patients with grade IV DHF. Serum IL-12 levels are highest in patients with dengue fever, but undetectable in patients with grade III and IV DHF.

DHF patients have higher levels of TNF-α, IL-6, IL-13, IL-18, and cytotoxic factor compared with DF patients. These cytokines have been implicated in causing increased vascular permeability and shock during dengue infections[24].

Increased levels of TNF-α and IL-10 correlate with haemorrhagic manifestations and platelet decay respectively. IL-10 may also down-regulate platelet function and thus contribute t platelet defects associated with dengue infections.

3. CELLULAR IMMUNE RESPONSES IN DENGUE INFECTIONS[3,20]

Suppression of T-cell responses[25] can occur in dengue fever and DHF. This could persist for at least two weeks after the onset of fever. In one study, respiratory tract infections or diarrhoea were seen in 6% of patients after dengue infections. This suppression has been suggested to be due to a primary defect within antigen presenting cells. IL-10, whose levels are increased in DHF, is known to down-regulate antigen presenting cell responses and induce unresponsiveness in T-cells.



SPECIAL ISSUES

THROMBOCYTOPENIA[24]

- Early bone marrow suppression, prolonged megakaryocyte arrest
- Increased peripheral destruction – immune mediated, spleen mediated

Platelet dysfunction also occurs. Impairment of ADP releasing ability of platelets, thereby causing impairment of platelet aggregation.

COAGULOPATHY[24,41]

Coagulopathy is most important complication in dengue viral infection

Factors causing coagulopathy[37] are;

- Thrombocytopenia
- PT,aPTT prolongation; decreased fibrinogen level.
- Decreased II,V,VII,VIII,IX & X factors
- Mechanisms causes spontaneous activation of fibrinolysis.

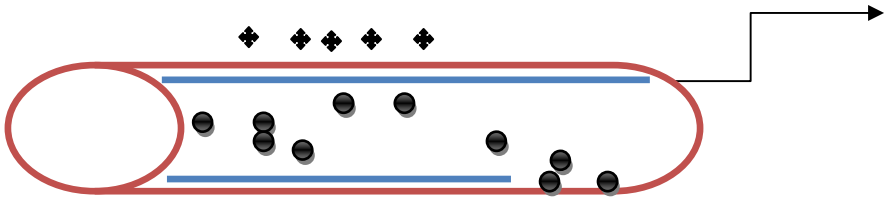
GI bleeding is most common bleeding manifestation followed by epistaxis.Due to plasma leakage the drop in hematocrit is not evident due to hemcoancentration[38]. In later stage thee will be a vicious cycle of unresponsiveness shock, massive fluid replacement, fluid overload, worsening of metabolic state and further worsens bleeding. So recognization of hypotension & shock and treatment of them is essential.

PLASMA LEAKAGE[41]

There is increase in microvascular permeability that leads to plasma leakage followed by hypotension & shock. The exact mechanism remains unknown. Vascular permeability present in all type of Dengue illness.

There will be sudden & marked increase in microvascular permeability which allows plasma water to flood out of the intravascular compartment and leads to sudden hypovolemic shock, as the compensatory mechanisms (increased lymphatic drainage, reabsorption capacity)

fail to cope. Capillary permeability of growing children is twice that of healthy adults. This higher microvascular permeability in childhood result from the greater density and surface area of microvessels in children than in adults, and explains why children are more prone to develop Dengue shock syndrome than adults.



Alterations occur in endothelial glycocalyx layer in vessel wall, which is act as a electrostatic barrier in vascular wall and repelling negatively charged plasma proteins from endovascular surface. Dengue NS protein or immune response directly damages glycocalyx and alters fiber matrix of the layer. The fast recovery from endothelial permeability evidences the possibilities of reversible factors disturbances rather than structural damage to vascular wall. Cytokines also play role in causing increased vascular permeability (IL-1,IL-2,IL-6,TNF-α,IFN-γ, VEGF)

HOST GENETIC FACTORS

Severe dengue infections are seen in only a minority (2%–4%) of patients with secondary dengue infections. A few studies have looked at the effect of polymorphisms at the major histocompatibility complex locus on susceptibility to DHF. Loke et al carried out molecular HLA typing of patients with DHF in Vietnam. They found that polymorphism at the HLA class I loci was significantly associated with DHF disease susceptibility, but polymorphism in the HLA-DRB1 or TNF genes were not. Furthermore, this association was confined to the HLA-A region

and not the HLA-B gene. Children with HLA-A*33 were less likely and those with HLA-A*24 more likely to develop DHF.

LABORATORY DIAGNOSIS

Methods used for diagnosis of dengue infections are:

1. Virus isolation,
2. Serology
3. Molecular techniques

1. VIRUS ISOLATION:

Detection of dengue virus by culture is the defininitive diagnostic test, but practical considerations limit its use. During the febrile phase, dengue viruses can be isolated from serum, plasma, or leucocytes. It can also be isolated from postmortem specimens such as liver, lung,\ spleen, lymph nodes, thymus, cerebrospinal fluid, or pleural/ascitic fluid. Ideally, blood should be collected during the febrile period, preferably before the fifth day of illness (that is, before formation of neutralising antibodies). Formation of immune complexes due to the presence of large quantities of neutralising antibodies in secondary dengue patients may interfere with virus isolation. For short periods of time (less than 24 hours) serum can be kept at 4–8˚ C, but for longer periods should be stored at 270˚ C.

Virus isolation done by

- Mosquito cell lines.
- Mosquito inoculation technique.

- Vertebral cell culture.

Currently, inoculation of C636 mosquito cell lines (obtained from A albopictus) is the method of choice. Virus isolation is done for research purposes only as it needs expertise, takes two weeks to read the results, and is expensive.

SEROLOGICAL DIAGNOSIS

Methods are:

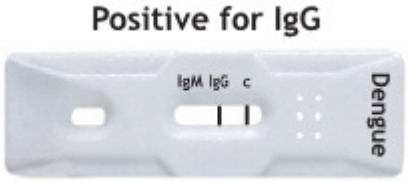
- Haemagglutination inhibition tests
- Enzyme linked immunosorbent assay (ELISA)
- Complement fixation test
- Neutralisation tests

2. HAEMAGGLUTINATION INHIBITION TESTS

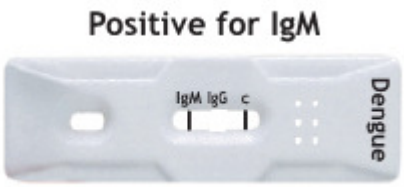
The HI test is simple, sensitive and reproducible and has the advantage of using reagents that may be prepared locally. HI test[26] requires paired sera. Paired sera are most easily obtained upon hospital admission (acute) and discharge (convalescent). The HI test is based on the ability of dengue virus antibodies[27] to inhibit this agglutination. A fourfold or greater rise in antibody titres is suggestive of a flavivirus infection (and not diagnostic of dengue infections). However, a single antibody titre >1/2560 is accepted as indicating secondary dengue infection if supported by a clinical history suggestive of dengue.Based on this principal Dengue Card Test (Panbio duocasste) formed.

DENGUE CARD TEST[40]

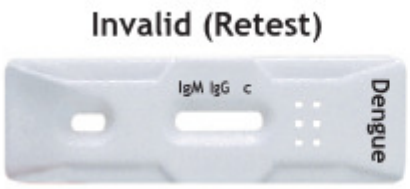
Sample addition & Running buffer addition

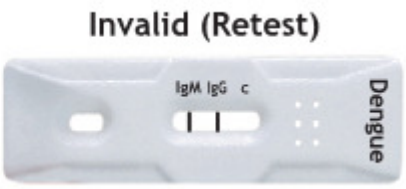


Secondary Dengue Infection



Primary Dengue Infection





Invalid test

MAC-ELISA

In primary or secondary dengue infections, MAC-ELISA[28] can measure a rise in dengue specific IgM, even in sera samples collected at 1-day to 2-day intervals in the acute phase. Specimens collected over an interval of 2–3 days spanning the day of defervescence are also usually diagnostic in MAC-ELISA. In cases where only a single specimen is available, detection of anti-dengue IgM permits the diagnosis of recent dengue infection.

Interpretation of MAC-ELISA results[a]

IgM antibody response	S1–S2 interval[b]	IgM to IgG ratio	Interpretation
Increase in molar fraction	2–14 days	High Low	Acute flavivirus infection, primary Acute flavivirus infection, secondary
Elevated, no change or decrease in molar fraction	2–14 days	High Low	Recent flavivirus infection, primary Recent flavivirus infection, secondary
Elevated	(Single specimen)	High Low	Recent flavivirus infection, primary Recent flavivirus infection, probably secondary

Detection of IgM in cerebrospinal fluid is a significant diagnostic finding, implying flavivirus replication within the CNS.

3. MOLECULAR DETECTION

RT-PCR is useful for the detection of dengue infection early in the disease when antibodies are not detected. RT-PCR is more sensitive than virus isolation, allows for rapid detection of dengue infections (results are usually available in 24 hours) and easier identification of the circulating serotype. It is useful for epidemiological studies as dengue serotypes could be identified without cross reactivity with other flaviviruses. The downside of molecular techniques is its relatively high cost and the expertise needed.

MANAGEMENT[1,4]

Febrile Phase:

In the initial phase the treatment of DF & DHF is the same and is as that of any other viral fever, i.e. symptomatic and supportive.

- Rest.
- **Paracetamo**l
- Do not give Aspirin or Brufen. Aspirin can cause gastritis and/or bleeding. In children, Reye's syndrome (Encephalopathy) may be a serious complication.
- Do not give antibiotics as these do not help.
- **Oral Rehydration Therapy** is recommended as there may be mild to moderate Dehydration due to vomiting & high temperature..

DENGUE HEMORRHAGIC FEVER

Patients with known or suspected DF[30] should have their platelet count and Hematocrit measured daily from the third day of illness until 1-2 days after defervescence. Those patients with a rising Hematocrit or falling platelet count should have intravascular volume deficits replaced. Those patients who improve can continue to be monitored in an outpatient setting. Those patients who do not improve should be admitted to the hospital for continued hydration.

INDICATIONS OF HOSPITALIZATION

- Patients who develop signs of Tachycardia
- ↑ Capillary Refilling Time (> 2 sec)
- Cool and clammy extremities
- Diminished peripheral pulses
- Changes in Mental status
- Oliguria
- Sudden rise in Hematocrit
- Narrowing of pulse pressure (< 20 mm Hg)
- Hypotension (Late finding-Uncorrected shock)

The fluid used to correct dehydration is chosen according to the nature of the fluid loss. In cases of isotonic dehydration, 5% glucose (50g/l) diluted 1:2 or 1:1 in physiological (normal)

saline should be used. Bicarbonate-containing solutions should not be used for the initial intravenous management of dehydration in DHF, and should be reserved for cases where there are persistent fluid losses from diarrhoea.

The necessary volume of replacement fluid is equivalent to the amount of fluid and electrolyte lost: thus, 10ml/kg should be administered for each 1% of normal body weight lost. Maintenance fluid requirements, calculated according to the Halliday & Segar formula, should be added to the replacement fluid volume. Since the rate of plasma leakage is not constant (it is more rapid when body temperature drops) the volume and rate of intravenous fluid therapy should be adjusted according to the volume and rate of plasma loss. Plasma loss can be monitored by changes in the haematocrit, vital signs or volume of urine output.

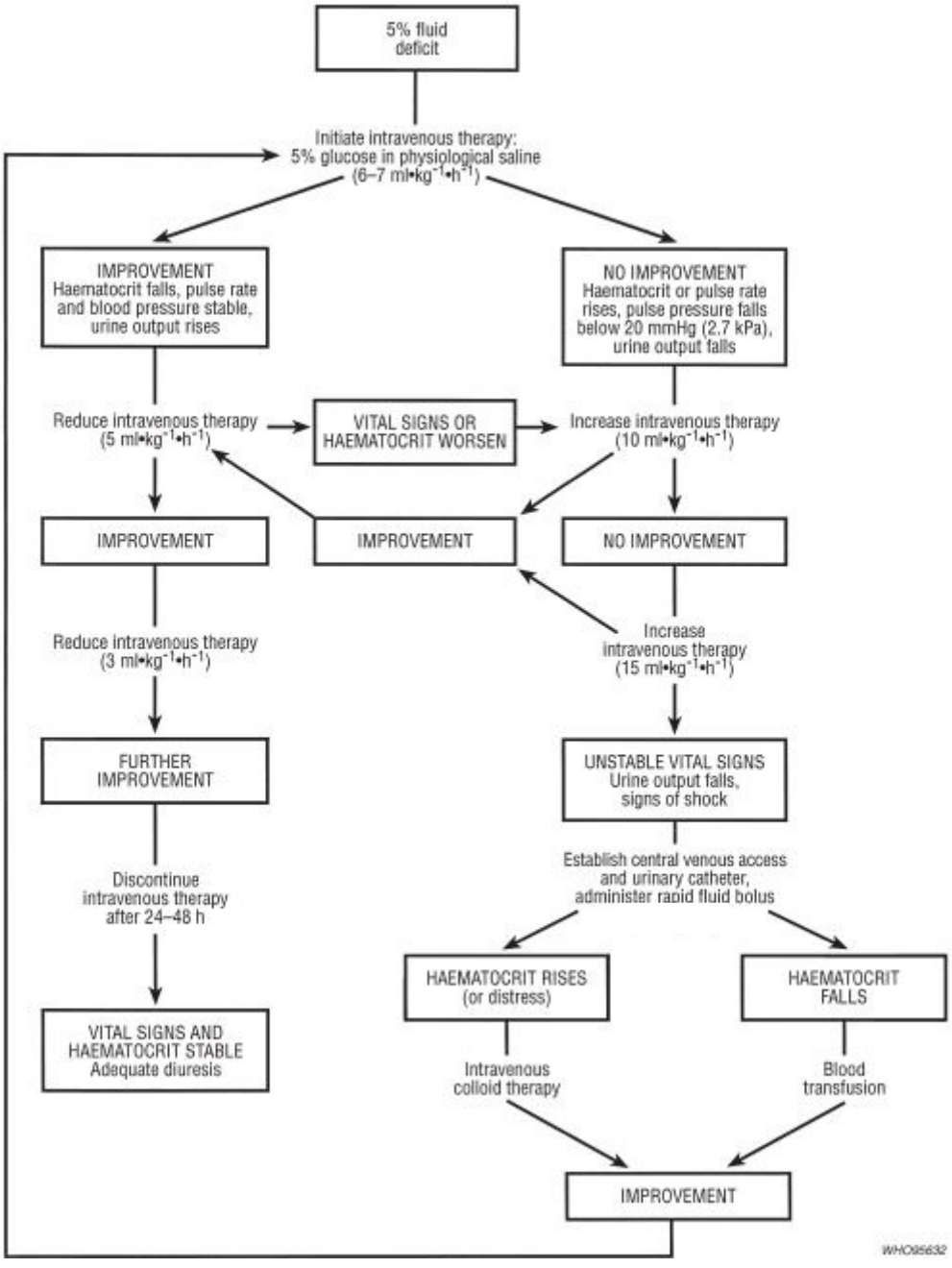
HALLIDAY & SEGAR FORMULA

Body weight (kg)	Maintenance volume (ml) administered over 24 hours
10	100/kg
10-20	1000 + 50 per each kg in excess of 10
>20	1500 + 20 per each kg in excess of 20

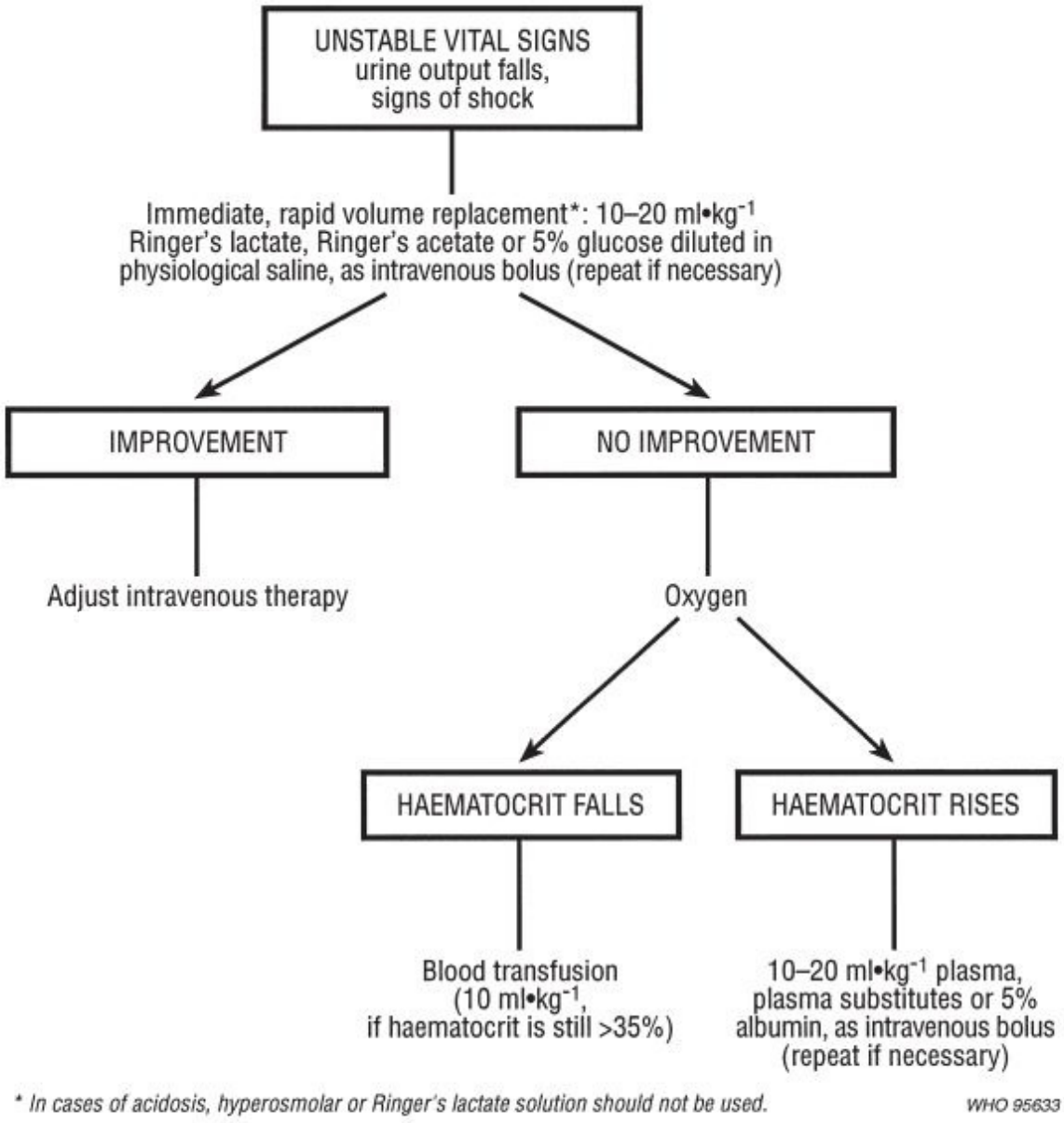
BLOOD TRANSFUSION

Blood transfusion is only indicated in cases with significant clinical bleeding. Internal bleeding may be difficult to recognize in the presence of haemoconcentration. A drop in haematocrit, e.g. from 50% to 40%, with no clinical improvement despite adequate fluid administration, indicates a significant internal haemorrhage. Transfusion with fresh whole blood is preferable. Fresh frozen plasma or concentrated platelets (if <20,000 or <50,000 with bleeding) may be indicated in cases where coagulopathy causes massive bleeding.

VOLUME REPLACEMENT FLOW CHART FOR DHF[32]



VOLUME REPLACEMENT FLOW CHART FOR DS[31,32]



FLUIDS RECOMMENDED:

Crystalloids[33] :

- 5% Dextrose in Isotonic NS
- 5% Dextrose in ½ NS

- 5% Dextrose in RL
- Shock Correction NS or RL

Colloids :

- Dextran 40
- Hemaccel
- Plasma

Monitoring of patients in DSS

- Check vitals every 15-30 minutes until shock is overcome.
- Check HCT / Platelets for every 2 hours for the first 6 hours and every 4 hours until stable.
- Fluid balance sheet to be maintained. Frequency & volume of urine output to be recorded. In refractory shock catheter may be needed.

CRITERIA FOR DISCHARGE

Patients who are resuscitated from shock recover rapidly. Patients with DHF or dengue shock syndrome (DSS) may be discharged from the hospital when they meet the following criteria:

- Afebrile for 24 hours without antipyretics
- Good appetite, clinically improved condition
- Adequate urine output
- Stable Hematocrit
- At least 48 hours have passed since recovery from shock
- Absence of respiratory distress
- Platelet count greater than 50,000.

PREVENTION AND CONTROL OF DHF

Since there is no effective vaccine against dengue, the prevention and control of dengue infections depends largely on preventing man-vector contact. Numerous strategies have been adopted and include: environmental control, biological control, chemical control, and active

case surveillance. While each of these methods have some effect, successful control programmes should incorporate all appropriate methods and also foster a strong partnership between the different dengue control agencies and the community. The dengue control programmes in the South East Asian and South Asian regions have been generally unsuccessful, largely because they have relied solely on insecticide spraying.

ENVIRONMENTAL CONTROL METHODS

These include: reducing vector breeding sites, solid waste management, modification of manmade breeding sites, and improvements in house design. Public education programmes play a vital part if they are to be effective. Personal protection is important in preventing man-vector contact. Sufficiently thick and loose fitting clothes reduce contact with the mosquitoes, but may not be the most practical clothes to wear in hot tropical climates. Other measures such as using household insecticidal products (mosquito mats and liquid vaporisers) or mosquito repellents may also be effective. Naturally occurring repellents (citronella oil, lemon grass) or chemical repellents (DEET) are available. However, unlike in the control of malaria,insecticide treated mosquito nets have limited utility in dengue as the vector is chiefly a day biting mosquito.

BIOLOGICAL CONTROL OF VECTOR:

Biological control methods are targeted against the larval stages of the dengue vector. They include the use of larvivorous fish such as Gambusia affinis and Poecilia reticulate, endotoxin producing bacteria (Bacillus thuringiensis serotype H-14 and Bacillus sphaericus are currently used), and copepod crustaceans. Bacillus thuringiensis serotype H-14 is more effective against A aegypti with very low levels of mammalian toxicity, and has therefore been accepted

for use in household containers storing water.2 The use of mesocyclops (a copepod crustacean) in the Northern Province of Vietnam led to the eradication of the vector in a many areas.

CHEMICAL CONTROL

This includes the application of larvicidal insecticides or space spraying. Space spraying is more widely used as larvicidal insecticides cost more. Insecticides used for treating containers that hold water includes Temephos 1% sand granules andinsect growth regulators. Regular monitoring of resistance patterns is essential as resistance to Temephos has been reported among some aedes mosquito species in the South East Asian Region. Insect growth regulators interfere with the development of the immature forms of the mosquito and have extremely low mammalian toxicity. Space spraying may be applied as thermal fogs or as ultralow volume sprays. Although both methods are equally effective in killing adult mosquitoes, thermal fogging tends tobe used more widely.

CURRENT STATUS OF THE DENGUE VACCINE[34]

Much research has been carried out to develop a dengue vaccine that is safe and immunogenic against all four serotypes. Although many of the vaccines developed so far(live attenuated, chimeric, DNA, and subunit vaccines) show promising results, none are sufficiently immunogenic for routine use.

MATERIALS AND METHODS

MATERIALS AND METHODS

This study was done in >13yrs patients admitted to adult General Medical ward of Stanley Medical College Hospital, Chennai from August 2007 – January 2009.

All patients with clinical features of Dengue infection and positive for Dengue Card Test (PANBIO card Test) were taken up for the study. Their Clinical profile, Laboratory data and Outcome were recorded.

1. All the patients were evaluated for

a). Clinical Features

- Fever
- Headache
- Retro orbital pain (ROP)
- Myalgia and Arthralgia
- Abdominal pain
- Nausea/ Vomiting
- Diarrhea/ Constipation
- Sleeplessness/ Lethargy
- Facial flush
- Conjunctival injection
- Lymphadenopathy
- Hepatospleenomegaly

- ❖ Bleeding manifestations
- ❖ Plasma leakage manifestations

b). Laboratory parameters

- ❖ Haemogram – TC, DC, Platelets
- ❖ Renal function – Urea, Creatinine
- ❖ Blood sugar
- ❖ Liver Function Test
- ❖ Chest X-ray
- ❖ Ultra-Sound Abdomen
- ❖ Dengue Card Test

2. Statistical Analysis

The relationship between the frequencies of clinical parameter of Dengue Fever (DF) and Dengue Haemorrhagic Fever (DHF) were analyzed after construction of 2x2 table and applying the Statistical Test of significance Chi-Squared Test of significance or Fisher`s eχ^2 test.

3. Exclusion criteria

Patients with malaria, Enteric fever, Leptospirosis and Pneumonia were excluded by doing appropriate investigations.

4. Following WHO criteria were adopted to define DF/DHF/DSS

CASE DEFINITION FOR DENGUE FEVER

The following classifications are proposed:

An acute febrile illness with two or more of the following manifestations:

- Headache
- Retro-orbital pain
- Myalgia
- Arthralgia
- Rash
- Haemorrhagic manifestations

CASE DEFINITION FOR DENGUE HAEMORRHAGIC FEVER

The following must all be present:

- Fever
- Haemorrhagic tendencies, evidenced by at least one of the following:
  - a positive tourniquet test
  - petechiae, ecchymoses or purpura
  - bleeding from the mucosa, gastrointestinal tract, injection sites or other locations.
  - haematemesis or melaena.
- Thrombocytopenia ($\leq$ 100000 cells per mm^3)

- Evidence of plasma leakage due to increased vascular permeability, manifested by at least one of the following:
    - rise in the haematocrit equal to or greater than 20% above average for age, sex and population;
    - drop in the haematocrit following volume-replacement treatment equal to or greater than 20% of baseline;
    - signs of plasma leakage such as pleural effusion, ascites and hypoproteinaemia.

CASE DEFINITION FOR DENGUE SHOCK SYNDROME

All of the above four criteria for DHF must be present, plus evidence of circulatory failure manifested by:

- Rapid and weak pulse.
- Narrow pulse pressure (≤20mmHg).
- Hypotension for age and cold clammy skin and restlessness .

GRADING SEVERITY OF DENGUE HAEMORRHAGIC FEVER

Grade-I : Fever + Thrombocytopenia +Plasma leak + Tourniquet Test positive

Grade -II: Grade I + Spontaneous Bleeding

Grade III: Grade II + narrow pulse Pressure or hypotension

Grade IV: Grade II + Profound Shock

Grade III and IV are considered as Dengue Shock Syndrome (DSS)

5. Management

All patients who suffered from fever were treated with paracetamol and bed rest. Those patients, who went for complications like DHF, were treated with WHO protocol. 5% DNS was used for all cases of Dengue Hemorrhagic Fever as IV fluid therapy. All DHF –II patients were started with IV crystalloids with rate of 6-7 ml/kg/hr and their improvement noted with Haematocrit value. If Patient deteriorated further aggressive management for Dengue Shock Syndrome was started with 10ml/kg/hr. If there is no improvement with this management, then 15ml /kg/hr rate of IV crystalloid was given. If Patient developed intractable Shock, further management was based on Haematocrit value. If there is fall in Haematocrit, Blood transfusion was given. If Haematocrit showed a rise, IV colloid therapy was indicated. Platelet transfusion was done in patients with bleeding manifestations with platelet <50,000 or platelet count <20,000 even without Bleeding manifestation.

RESULTS

RESULTS

Total no of patients : 122

No of Dengue Fever (DF) : 71 (58%)

No of Dengue Haemorrhagic Fever : 51 (42%)

DHF – I : 4 (3%)

DHF – II : 35 (30%)

DHF – III : 9 (7%)

DHF – IV : 3 (2%)

AGE – SEX DISTRIBUTION FOR DENGUE INFECTION

Mean Age : 29 ± 13.5 yrs

About 65% of patients were in the age group of 13 to 40 yrs.

66% were Male and 34% were Females

Sex ratio - M:F = 1.9:1

SPECTRUM OF DENGUE INFECTION

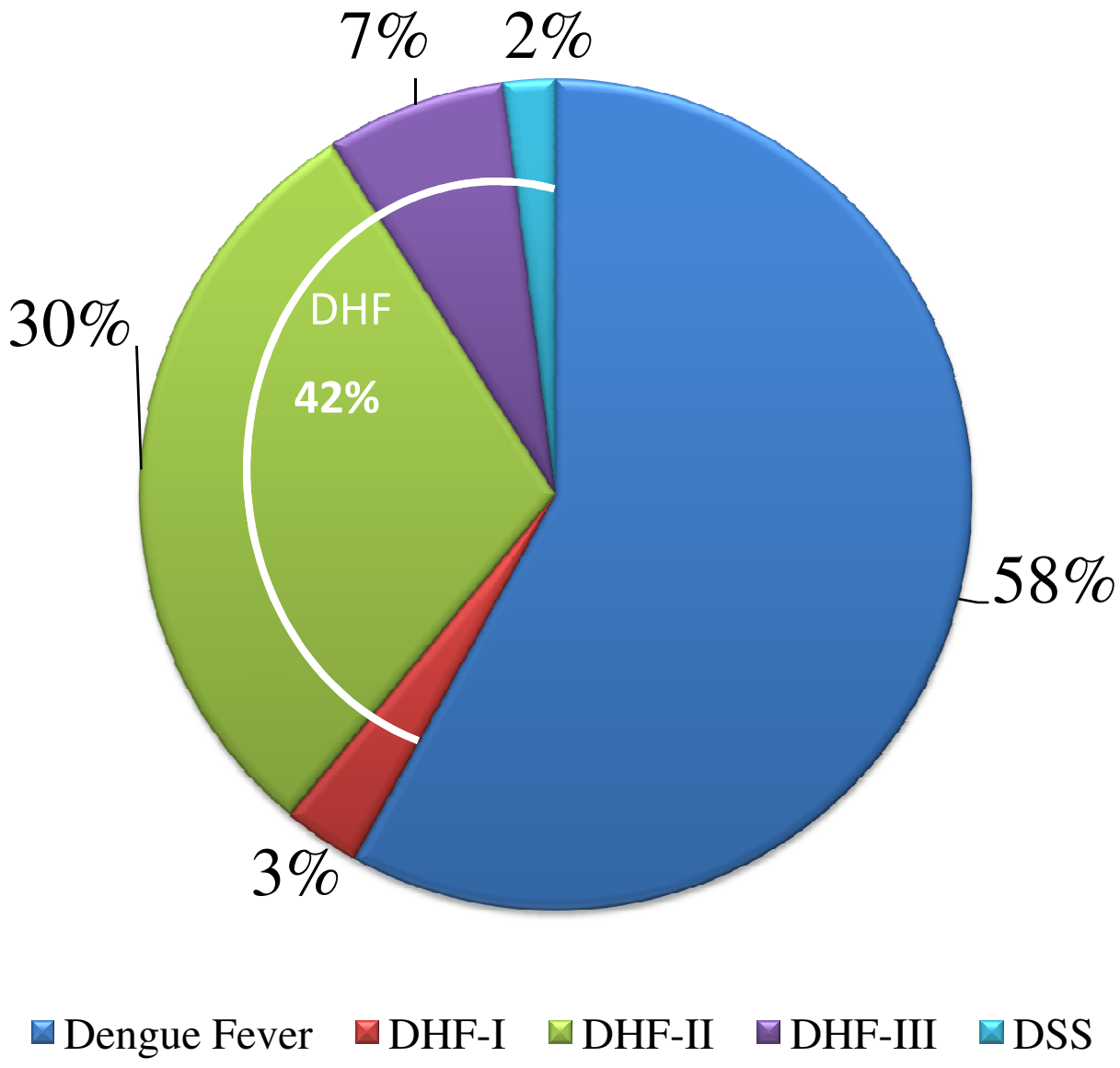


Table1. Age – Sex distribution for dengue infection

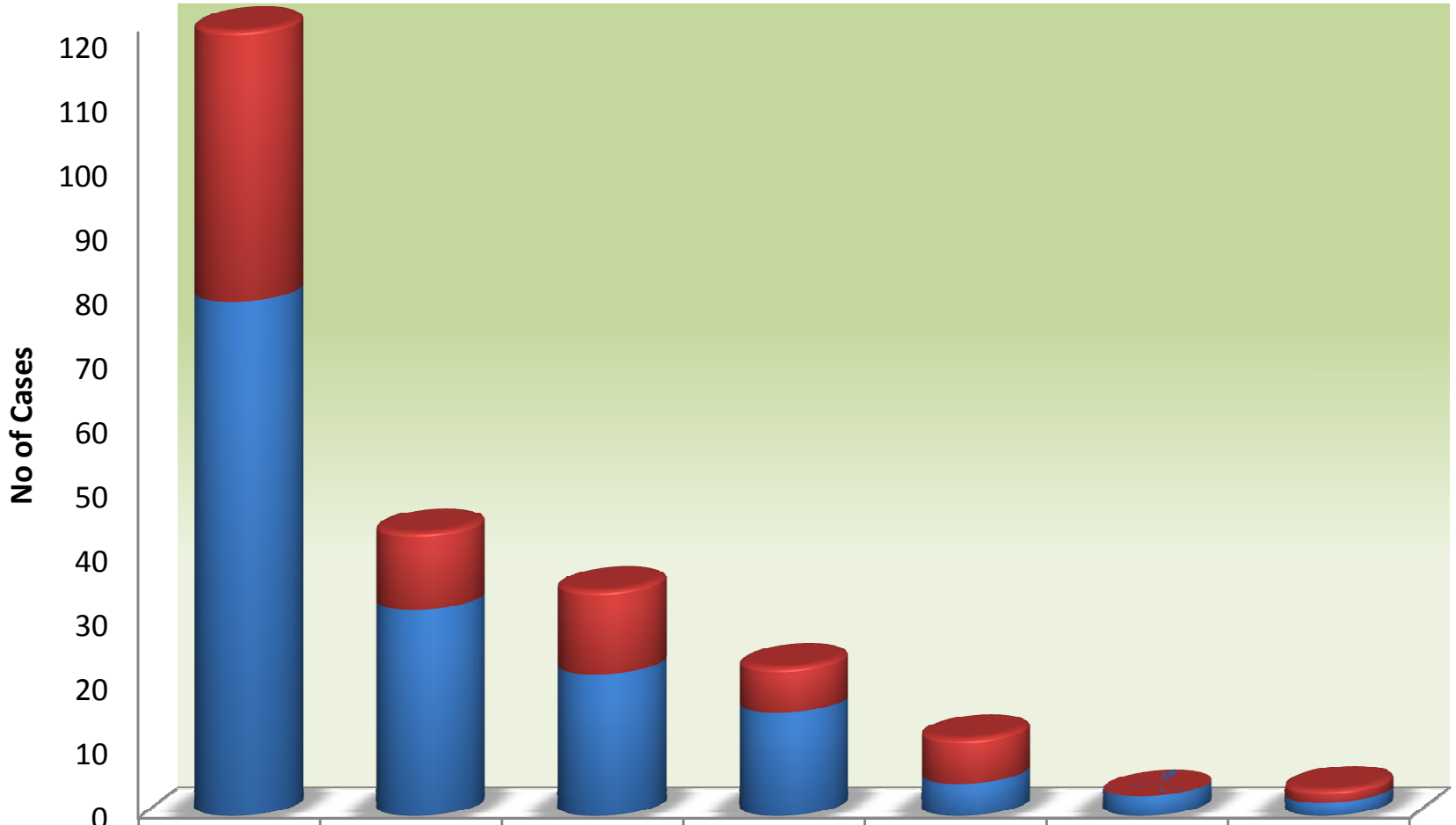
AGE GROUP	MALE	FEMALE	TOTAL
13 – 20 yrs	32	12	44
21 – 30 yrs	22	13	35
31 – 40 yrs	16	7	23
41 – 50 yrs	5	7	12
51 – 60 yrs	3	0	3
>60 yrs	2	3	5
TOTAL	80 (66%)	42 (34%)	122

AGE – SEX DISTRIBUTION FOR DHF

Table 2. Age – Sex distribution for DHF

n = 51	**DHF** n=51	**DHF – I** n=4	**DHF – II** n=35	**DHF – III** n=9	**DHF – IV** n=3
MALE	32 (26%)	3	22	6	1
FEMALE	19 (16%)	1	13	3	2
13 – 30 yrs	32	3	19	8	2
31 – 50 yrs	18	1	15	1	1
51 – 65 yrs	1	0	1	0	0
> 65 yrs	0	0	0	0	0

AGE – SEX DISTRIBUTION



	Total yrs	13-20	21 -30	31 -40	41-50	51-60	>60
Female	42	12	13	7	7	0	2
Male	80	32	22	16	5	3	2

Dengue Haemorrhagic Fever (DHF) commonly in the Age group of 13 – 40 yrs. Elderly patients were not affected with DHF.

Mean Age : 29 ± 11 yrs

63% were males and 37% were females

Sex ratio - : M:F = 1.7:1

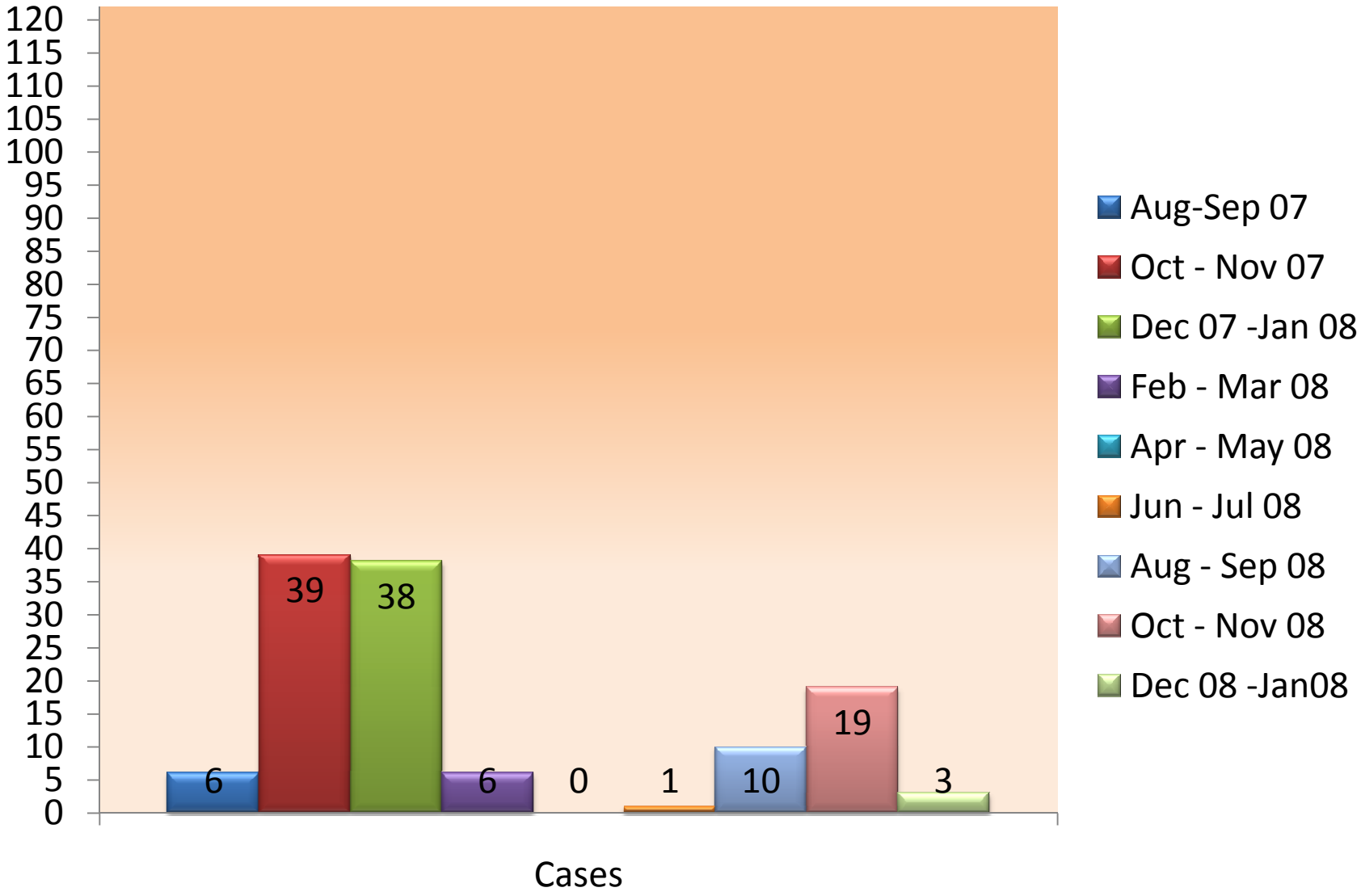
SEASONAL DISTRIBUTION

Most cases occurred in the month between September to January. Around 78 (64%) cases were reported in the year of 2007 and the number reduced to 44 (36%) in 2008.

Table 3. Seasonal Case Incidence

MONTH	**NO OF CASES** (n=122)
Aug – Sep 07	6
Oct – Nov 07	39
Dec 07 – Jan 08	38
Feb – Mar 08	6
Apr – May 08	0
Jun – Jul 08	1
Aug – Sep 08	10
Oct – Nov 08	19
Dec 08 – Jan 09	3

SEASONAL DISTRIBUTION



Primary Infection : 29 (24%)

Secondary Infection : 93 (76%)

Secondary infection causes DHF in 48 cases; only 3 cases were primary infection.

CLINICAL FEATURES

FEVER

Fever occurs in all (100%) patients.

Mean duration of fever : 4.9 ± 1 days

Mean duration of fever before admission : 2.5 days

High grade fever : 69% patients

Continuous type of fever : 41% patients

OTHER CLINICAL SYMPTOMS

Myalgia (82%), Arthralgia (65%) and Headache (77%) were common symptoms after fever.

Arthralgia, Abdominal pain, Nausea/Vomiting, altered bowel movements, Sleeplessness and lethargy were statistically significant symptoms that occurred in DHF as compared to Dengue fever.

Table 4. Clinical Symptoms

SYMPTOMS	Total n=122	DF n=71	DHF n=51	P value
HEADACHE	77%(94)	73%(52)	82%(42)	0.2797
RETRO ORBITAL PAIN	12%(15)	13%(9)	12%(6)	1
MYALGIA	82%(100)	86%(61)	76%(39)	0.02336
ARTHRALGIA	65%(79)	63%(45)	67%(35)	0.0288
ABDOMINAL PAIN	57%(69)	46%(33)	71%(36)	0.0097
NAUSEA/VOMITING	59%(72)	51%(36)	71%(36)	0.0397
ALTERED BOWEL MOVEMENTS	30%(37)	18%(13)	47%(24)	0.0012
CORYZA	52%(64)	54%(38)	51%(26)	0.8549
SLEEPLESSNESS/ LETHARGY	57%(70)	44%(31)	75%(39)	0.0004

Past History of viral fever within 2yrs was present in around 27(22%) pts. Among which, 17(33%) had DHF, Which is statistically significant (p=0.0553).

General and systemic examination findings were noted except bleeding and plasma leakage manifestations.

CLINICAL PROFILE - SYMPTOMS

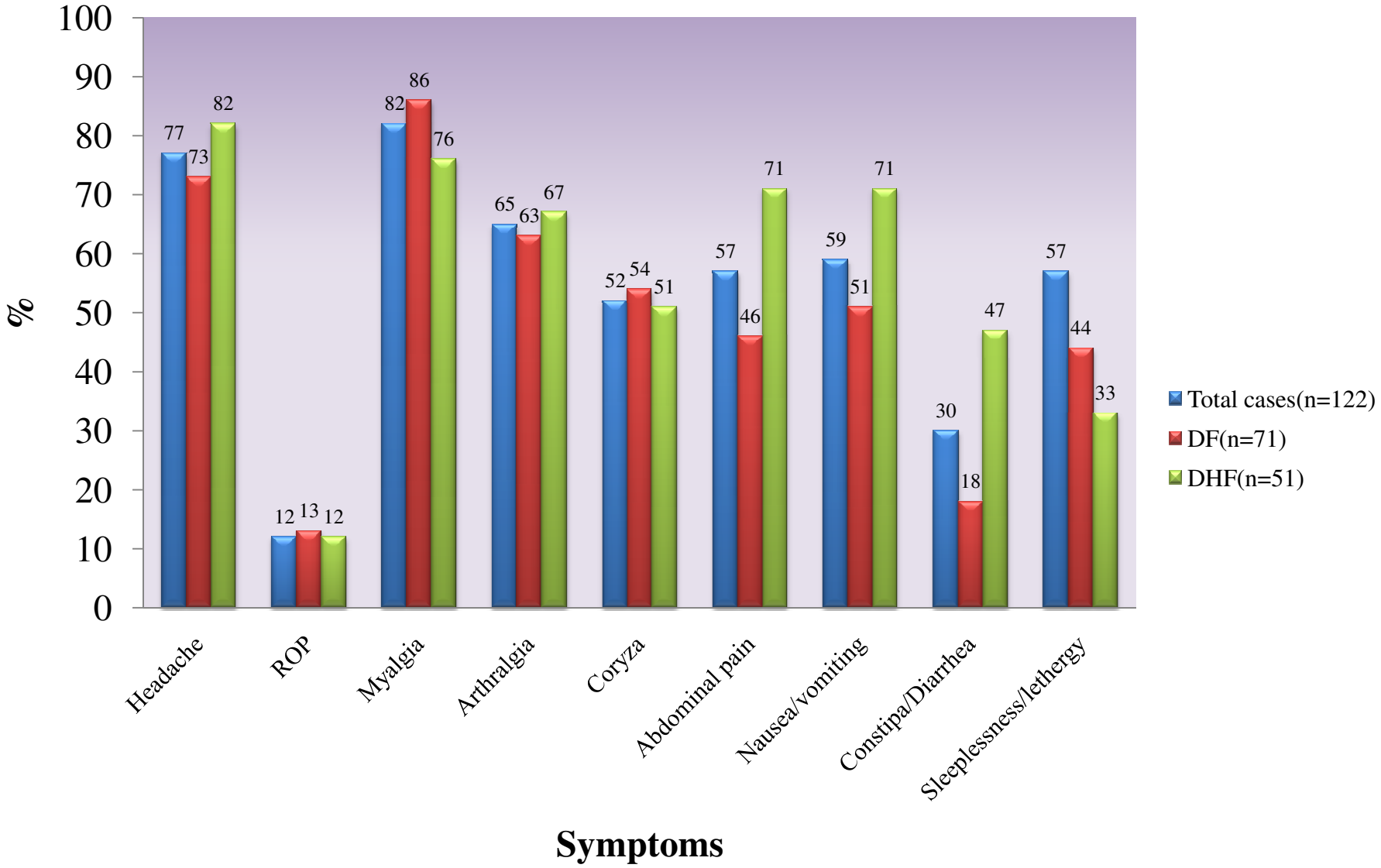


Table 5. Clinical signs

SIGNS	Total n=122	DF n=71	DHF n=51	P value
LYMPHADENOPATHY	31%(38)	23%(16)	44%(22)	0.0183
FACIAL FLUSH	39%(36)	10%(7)	57%(29)	0.0001
CONJUNCTIVAL INJECTION	47%(57)	35%(25)	63%(32)	0.0033
HEPATOMEGALY	11%(13)	8%(6)	14%(7)	0.3849
SPLENOMEGALY	15%(18)	13%(9)	18%(9)	0.4518
ENCEPHLOPATHY	2.5%(3)	-	-	-

Conjunctival injection, Facial flush and Lymphadenopathy occurred among statistically significant population with DHF.

Dengue Encephalopathy occurs in 3(2.5%) patients.

LAB PARAMETERS

Complete Haemogram, Liver function tests, Renal function test, electrolytes and Chest X-ray were done. Only Haemogram and Liver function test results were showed abnormal values. These investigations were analyzed. Other investigations were mostly within normal limits.

CLINICAL PROFILE - SIGNS

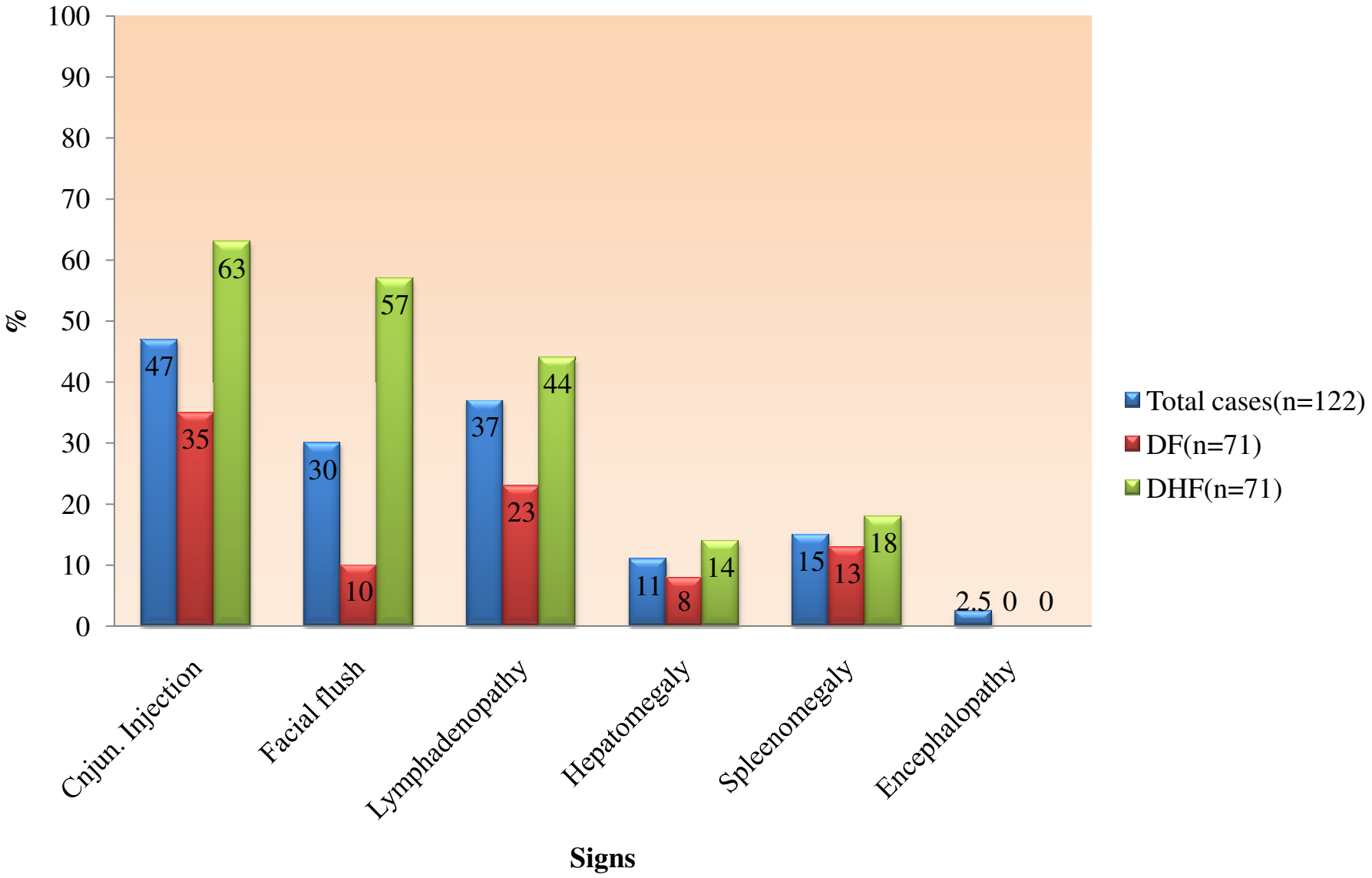


Table 6. Complete Blood Count

CBC	Total n=122	DF n=71	DHF n=51	P value
TC <3,500	17%(21)	15%(11)	19%(10)	0.6296
TC > 11,000	13%(16)	11%(8)	16%(8)	0.5886
EOSINOPHIL >8%	21%(26)	11%(8)	35%(18)	0.0018
HAEMATOCRIT > 45%	20%(24)	14%(10)	27%(14)	0.1047

Liver enzymes

Mean SGOT in DHF : 126 IU

Mean SGPT in DHF : 108 IU

Elevation of Liver enzymes in DHF is statistically significant.

Table 7. Liver Enzyme levels

LFT	Total n=122	DF n=71	DHF n=51	P value
SGOT >40IU	52%(64)	31%(22)	78%(40)	0.0001
SGOT >100IU	26%(32)	6%(4)	55%(28)	0.001
SGPT >40IU	39%(47)	13%(9)	75%(38)	0.0001
SGPT >100IU	22%(27)	4%(3)	47%(24)	0.0001

THROMBOCYTOPENIA

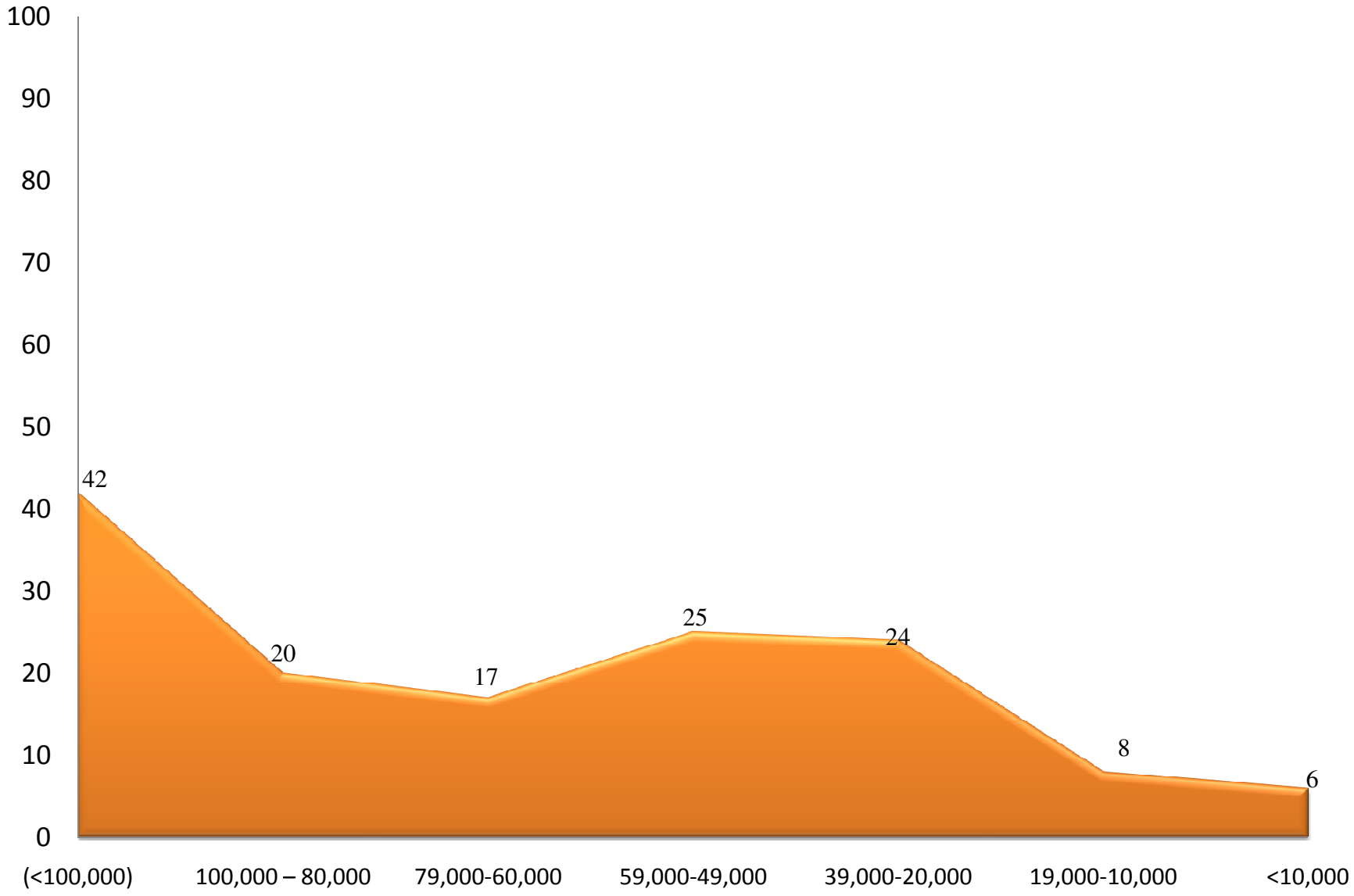
Around 51 (42%) patients had platelet count less than 1,00,000.

Table 8. Thrombocytopenia and Platelet Count Distribution

PLATELET COUNT	**PERCENTAGE** (n=122)
<1,00,000	42% (51)
PLATELET COUNT DISTRIBUTION	**PERCENTAGE**(n=51)
1,00,000 – 80,000	20% (10)
79,000 – 60,000	17% (9)
59,000 – 40,000	25% (13)
39,000 – 20,000	24% (12)
19,000 – 10,000	8% (4)
< 10,000	6% (3)

Mean platelet count in thrombocytopenic patient was 47,176 platelets/mm^3.

THROMBOCYTOPENIA



HAEMORRHAGIC MANIFESTATIONS

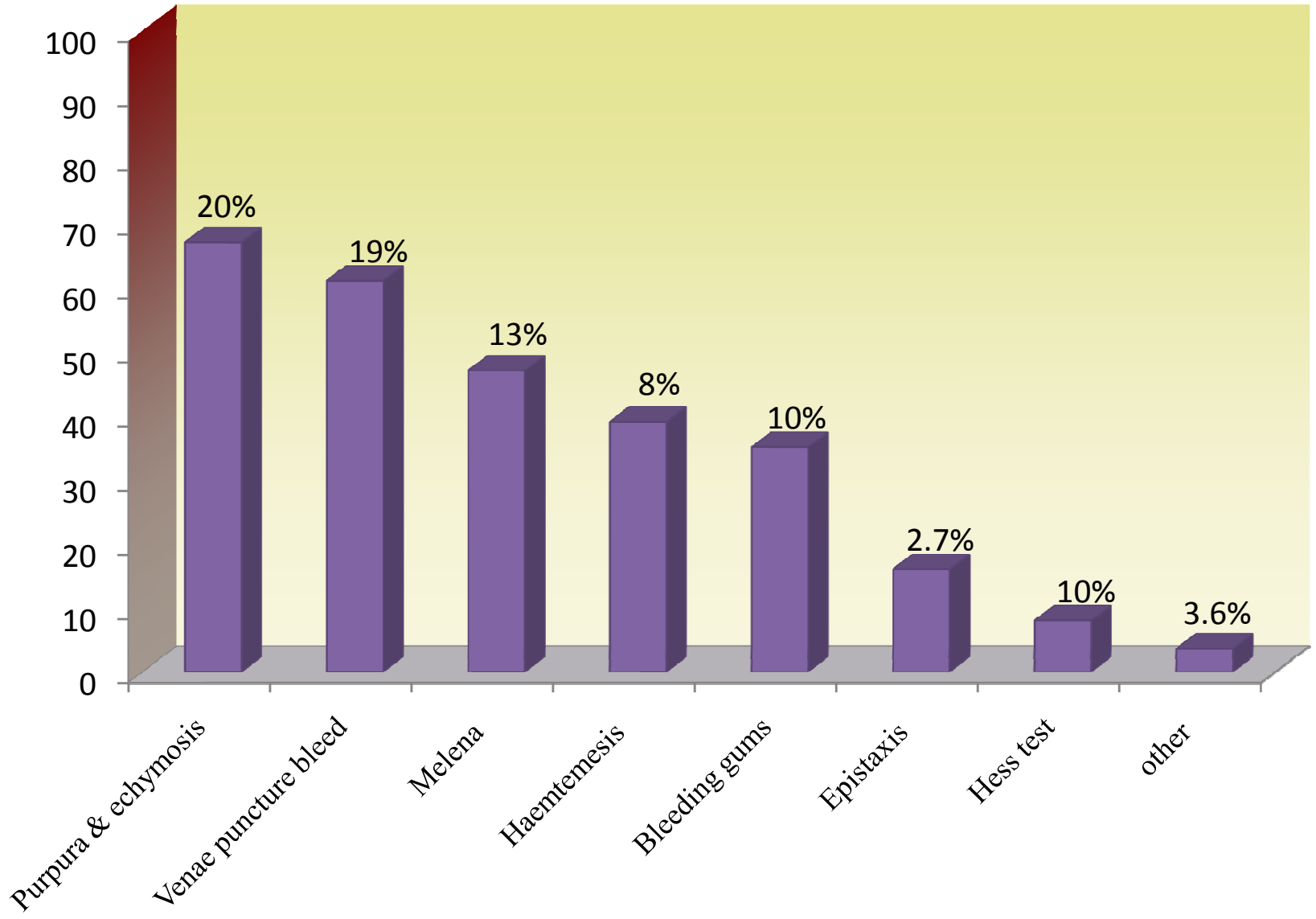
Total No of patients with Bleeding manifestations : 42% (51)

Table 9. Haemorrhagic manifestations

BLEEDING MANIFESTATION	OCCURRENCE (n=51)
EPISTAXIS	16% (8)
MALENA	47% (24)
HAEMATEMESIS	39% (20)
BLEEDING GUMS	35% (18)
RASHES	67% (34)
VENAE PUNCTURE BLEED	61% (31)
HESS TEST	8% (4)
OTHERS	3.6% (7)

Rashes and Venae puncture Bleeding were common bleeding manifestations. But overall GI bleeding was most common.

HAEMORRHAGIC MANIFESTATIONS (N=51;42%)



PLASMA LEAKAGE MANIFESTATIONS

Total No of patients with Plasma Leakage manifestations : 42% (51)

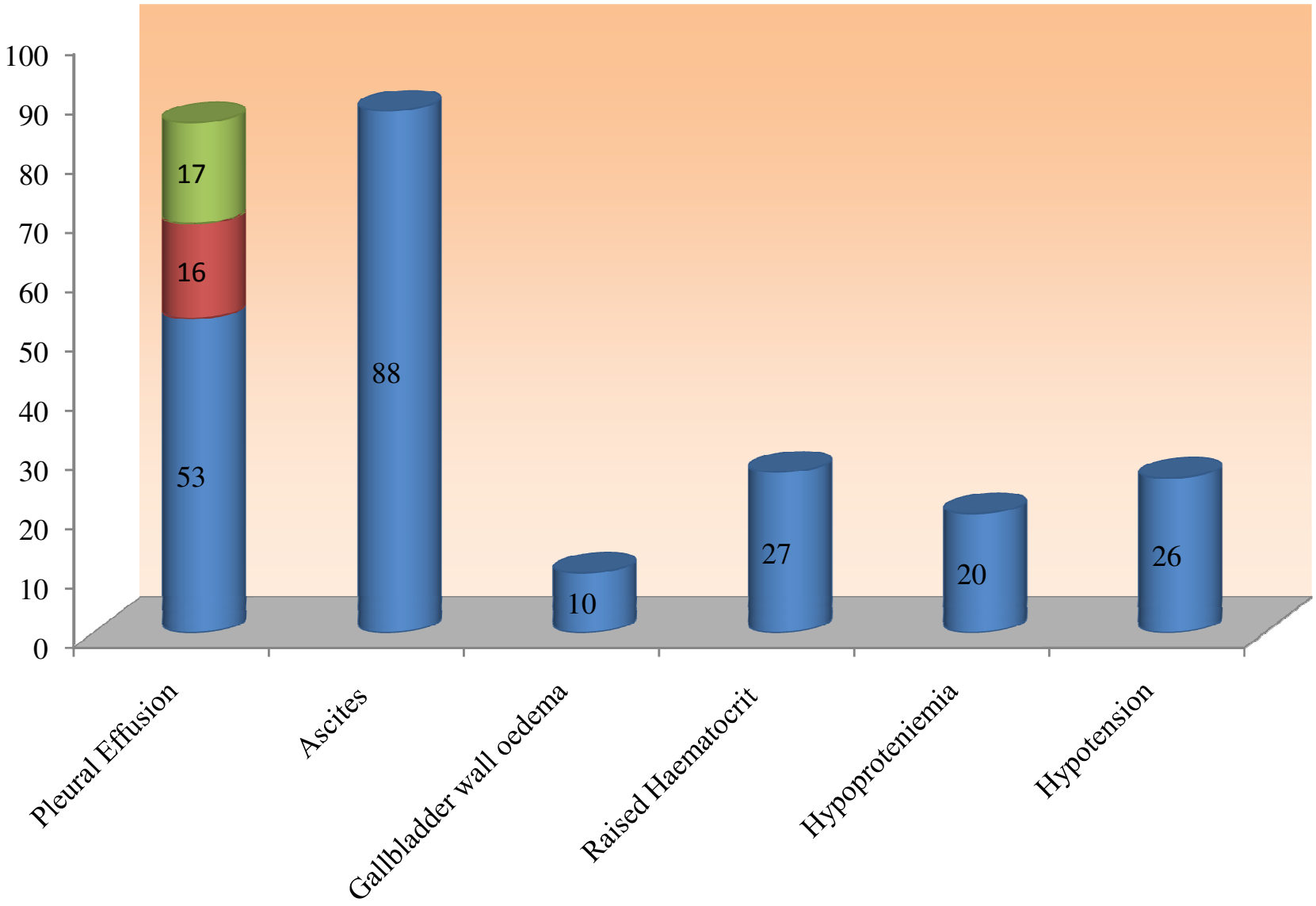
Table 10. Plasma leakage manifestations

PLASMA LEAKGE MANIFESTATION	**OCCURANCE** (n=51)
PLEURAL EFFUSION (PE)	84% (43)
	RIGHT PE – 62% (27)
	LEFT PE – 18% (8)
	BILATERAL PE – 20% (9)
ASCITES	88% (45)
GALL BLADDER WALL OEDEMA	10% (5)
INCREASED HAEMATOCRIT	27% (14)
HYPOPROTEINEMIA	20% (10)
HYPOTENSION	26% (11)

Ascites & Pleural effusion were common Plasma leakage manifestations. All cases of pleural effusion and Ascites were detected by USG examination.

All patients with Gall Bladder wall edema were affected with severe Dengue Infection. So Gall bladder wall edema is a significant finding to detect DHF.

PLASMA LEAKAGE (N=51;42%)



OUTCOME

DF patients were treated with paracetamol and close observation for complications for at least two days to detect complications.

All DHF – II (35) patients were started with IV fluid at the rate of 6-7 ml/kg/hr with 5%DNS. If patient maintained a stable BP, the fluid infusion rate was reduced to 3-4 ml/kg/hr. Vitals, Platelet count and Haematocrit were scrutinized regularly and managed according to the state of BP. No patients developed hypotension or mortality.2 patients had platelet count < 20,000 with bleeding, they were treated with 4unit Platelet concentrate.

Of the 12 patients with DSS 10 were treated in IMCU of Stanley Medical Hospital. 2 patients were managed in medical ward itself. Those 2 patients were managed only with IV crystalloids.

Patients with DHF – III (9) were carefully monitored for changes in Blood pressure. Patients were treated with 5%DNS in the rate of 6-7ml /kg/hr. 4 patients did not improve with this treatment. Then the infusion rate was increased to 10 ml/kg/hr and then to 15 ml/kg/hr. With this treatment 2 patients improved. But 2 patients remained hypotension. Their PCV was investigated and after confirmation of its elevated value, the patients were infused with 4packs of FFP (fresh Frozen Plasma) each. Both of them improved after 1 day. Among 9 patients, 2 pts had platelet <20,000. They were treated with 4 unit of platelet concentrate. Mortality of the 4 patients was prevented by timely measures against hypotension and thrombocytopenia. One patient had low haematocrit, in who was transfused with whole blood.

DHF – IV (3) patients were treated with IV crystalloids at 20ml/kg/hr. 2 patients had profound bleeding. Both patients were treated with platelet transfusion. Two patients were

recovered with IV fluid therapy and platelet transfusion. But one patient developed severe thrombocytopenia and low haematocrit. Around 6 units of platelets was transfused. 3unit of whole Blood were transfused. Even with this effective management in IMCU, bleeding didn't get arrested. Shock could not be corrected. Patient expired with intractable shock.

Fresh frozen Plasma given for : 2patient

Platelet concentrate given for : 7patients

Blood transfusion given for : 2patients

Case Fatality Rate (CFR) : 0.008%

Around 12 patients (DSS) were in a critical condition, but improved with effective treatment. Only one patient expired, due to intractable shock.

DISCUSSION

DISCUSSION

In India, 5,534 cases of Dengue were reported in 2007 with 80deaths. In 2008, 12,419 cases of Dengue were reported with 69deaths. Case Fatality Rate was 0.6%. In 2006, Tamilnadu contributed to 4% cases, most of which were from Chennai. Researchers are considering these datas as only tip of the iceberg of the actual situation. Most Dengue clinical studies were done in paediatric age group. Adult studies are quite limited, especially in India.

This present study was done in Stanley Medical College Hospital, North Chennai, which is highly endemic for most infectious diseases. As Dengue fever is one of the common infectious diseases, the incidence is relatively high in North Chennai. Most Dengue cases were managed as OP which is commonly presenting as simple viral illness in adults. In this study, we have taken into consideration only those who were admitted cases in Medical ward.

Here, we want to analyse present study by comparing with other Dengue adult studies. Indian and other South Asian region studies are taken up for the discussion.

Total of 122 cases of Dengue infection admitted to Stanley Medical college Hospital were analysed. Dengue Fever (DF) occurred in 71(58%) patients. Dengue Haemorrhagic Fever (DHF) occurred in 51 (42%) patients. Among patients with DHF around 3(2%) patients developed suffered with Dengue Shock Syndrome (DSS).

Dengue infection commonly occurred in Males (66%). M: F ratio is 1.9:1. Mean age was 29 ± 13.5 yrs. Majority of patients around 65%, were in the Age group of 13 – 30 yrs.

Among Dengue infections, DHF occurred in 42% of cases. DHF commonly presented in the Age group of 13 – 30 yrs, same as Dengue Infection. Elderly Patients were not affected with DHF. Mean Age was 29 ± 11yrs. 63% were males. M: F ratio was 1.7:1.

Parameters	**Present study n=122**	**Malavige *et al* Srilanka[42] n=108**	**NPSingh *et al* Delhi[44] n=185**	**Janak Kishore *et al;* Lucknow[45] n=100**	**Adriana O *et al;* Brazil[43] n=185**
DF	58%	30.6%	-	46%	-
DHF	42%	69.4%	-	54%	-
Mean Age	29±13.5yrs	26.6yrs	26±10yrs	30±14yrs	32±12yrs
Age Distribution	13-40yrs	13-56yrs	12-29yrs	15-30yrs	-
M:F	1.9:1	1.4:1	3:1	2:1	1.7:1

Most of the other Dengue studies were comparable with present study. In other studies the incidence of DHF is more than 50%, where most of the patients were admitted for complications by referrals. Age – Sex Distribution, Mean age and Male: Female ratio of the other studies was equally comparable with our studies.

Malavige et al & Adriana et al studies show the primary infection occurs in 34% patients, whereas present study shows it to be 24%.

In present study, the incidence of cases was mostly during the period of September 2007 to January 2008. The incidence reduced drastically in 2008 compared to 2007. This decline in incidence may be explained by effective step of the Government via mosquito control

programme in 2008, after the high incidence of Dengue fever in 2007. Seasonal distribution of case incidence corresponds mostly to the period of August to December in both years. This same Seasonal pattern was classically reported by WHO for Indian Seasonal Pattern of Dengue Infection for 2003 – 2006.

	Present study n=122	**Malavige *et al* Srilanka[42] n=108**	**Janak Kishore *et al;* Lucknow[45] n=100**	**Adriana O *et al;* Brazil[44] n=185**
DHF	42% (51)			23%
DHF I & II	76%	81%	76%	74.4%
DHF III & IV	24%	19%	24%	25.6%

The incidence of Dengue Haemorrhagic Fever (I & II) & DSS (III & III) are similar with other adult studies. In all studies including presnt study, DHF -II was common type.

CLINICAL FEATURES:

Fever has been documented 100% in all adult Dengue studies including present study. The average duration

	Present study n=122	**Malavige *et al* Srilanka[42] n=108**	**Janak Kishore *et al;* Lucknow[45] n=100**	**NP Singh *et al* Delhi[44] n=185**
Average Fever Duration	4.9 ± 1 days	4.7 days	5.9 days	4.5 ± 1 days

In all studies, including Present study high grade & continuous fever were commonly recorded. The average duration of fever is also nearly same.

Parameters	**Present study n=122**	**Malavige *et al* Srilanka[42] n=108**	**NP Singh *et al* Delhi[44] n=185**	**Janak Kishore *et al;* Lucknow[45] n=100**
Headache	77%	66%	61.6%	17%
Myalgia	82%	76%	57.8%	18%
Arthralgia	65%	57%	-	21%
Vomiting/ diarrhea	59%	63%	50%	16%
Abdominal pain	57%	16%	21%	-

These common symptoms of Present study were comparable with other adult Dengue studies. Abdominal pain was more common in Present study when compared with other studies. But abdominal pain was common in DHF than DF in all studies including Present study and the p value of Present study is significant for DHF.

Past History of viral illness was commonly recorded in DHF than DF. This past history was not analysed in other studies. In Present study, around 22% patients had viral illness in the recent past.

Parameters	Present study n=122	Malavige *et al* Srilanka[42] n=108	pradeep *et al*	Janak Kishore *et al;* Lucknow[45] n=100
Lymphadenopathy	37%	-	26%	-
Facial flush	30%	42%	42%	53%
Hepatomegaly	11%	45%	45%	-
Spleenomegaly	15%	2.7%	13%	-

Lymphadenopathy was not studied in most studies. In the Present study lymphadenopathy occurred significantly in DHF. Other clinical parameters occurred in the lower percentage in Present study when compared to other studies.

LAB PARAMETERS

Elevated liver enzymes were commonly studied in paediatric studies. In Present study SGOT & SGPT were elevated to statistically significant levels in DHF as compared to DF.

Parameters	Present study n=122	NP Singh *et al* Delhi[44] n=185	Janak Kishore *et al;* Lucknow[45] n=100
SGOT >40 IU	52%	16%	48%
SGPT >40 IU	39%	17%	49%

Janak Kishore et al study shows the elevation of liver enzymes, similar to Present study.

THROMBOCYTOPENIA

Thrombocytopenia	**Present study n=122**	**Malavige *et al* Srilanka[42] n=108**	**NP Singh *et al* Delhi[44] n=185**	**Janak Kishore *et al*; Lucknow[45] n=100**	**Adriana O *et al;* Brazil[43] n=185**
Total	42%	74%	61%	70%	70%
Mean platelet count in DHF	47,176	-	66,000	43,210	-

Thrombocytopenia was the most commonly studied parameter in previous Dengue studies. Other clinical features need to be monitored when the patient suffered from DHF, with plasma leakage manifestations. 42% patients had thrombocytopenia in Present study. Mean platelet count was 47,176 cells/mm^3. Around 49% patients had platelet count between 20,000 – 60,000 cell/mm^3. Only 6% patients had platelet count less than 10,000cells/mm^3.

HAEMORRHAGIC MANIFESTATION

Bleeding manifestation	**Present study n=122**	**Malavige *et al* Srilanka[42] n=108**	**NP Singh *et al* Delhi[44] n=185**
Total	42%	39%	40%
Epistaxis	16%	24%	14%
Malena	47%	34%	16%
Bleeding gums	35%	17%	-
Hematemesis	37%	40%	22.2%
Rashes	67%	54%	31.9%
Venae puncture bleed	61%	-	-
Hess test	8%	-	21%

In Present study, rashes, Venae puncture Bleed, Malena and Hematemesis were the common bleeding manifestations. Malavige *et al* study describes rashes and hematemesis as common bleeding manifestations. In all other studies rashes were predominant bleeding manifestations.

PLASMA LEAKAGE MANIFESTATIONS

Plasma Leakage manifestation	**Present study n=122**	**Malavige *et al* Srilanka[42] n=108**	**NP Singh *et al* Delhi[44] n=185**	**Adriana O *et al;* Brazil[43] n=185**
Total	42%	74%	61%	70%
Pleural Effusion	84%	16%	2%	56%
Ascites	88%	17%	2%	56%
Increased Haematocrit	27%	26%	5.6%	-
Hypotension	26%	18%	-	26%

In Present study, Ascites & Pleural Effusion played a major role in diagnosis of DHF. Hypotension occurred in 26% patients. In other studies the plasma leakage manifestations were limited. Plasma leakage, manifestations very difficult to diagnose clinically except Hypotension. So in most studies hypotension was the common plasma leakage manifestation. Recognition and early IV fluid therapy for hypotension is necessary.

Past history of previous similar viral illness present in 22% in total of 122 patients. Among patients with DHF past history of fever was present in 33% cases.

Gall bladder wall edema is a marker for DHF, was present in 5 patients of the present study.

Encephalopathy occurred in 3 patients. All the patients had fever for 2-3 days, followed by altered level of consciousness and acute confusional state. The confusional state recovered over 2-3 days. No focal neurological deficit had been demonstrated. Metabolic state, CSF analysis and Imaging were normal in all 3 patients. They were all are positive for dengue card test along with clinical features of Dengue fever.

One 14yr old boy expired in Present study due to intractable Dengue Shock Syndrome. Case fatality rate was 0.0008% in present study. But in other studies it was high.

STUDY	**CASE FATALITY RATE**
Present study (n=122)	0.008%
Malavige *et al;* Srilanka[42] **(n=108)**	3.7%
NP Singh *et al*: Delhi[44] **(n=185)**	2.7%
Harris et al[46]	0.001%
Kulatate et al[47]	0.02%

The mortality varies in many studies. Most of the studies were hospital based studies in which the hospital admissions were individual based and the mortality is largely prevented my effective management in hospital. So recognition of early haemorrahgic fever and special medications like FFP, Platelet concentrate and whole Blood act as cornerstone of management of complications.

In present study platelet concentrate were given for 7 patients, FFP for 2 patients and Whole Blood transfusion for 2 patients. The transfusion rate was low in present study compared to other studies. In Malavige et al study it was 12%, which is done in Srilanka. So effective early recognition and management of complications markedly decreases mortality.

SUMMARY

SUMMARY

1. Dengue fever occurred in 122 patients in present study. 42% patients had DHF. DHF-II was common type of DHF.
2. Dengue Shock syndrome occurred in 9% patients.
3. DF as well as DHF commonly occurred in the age group of 13 – 30 yrs
4. Mean Age – 29 ± 13.5 yrs; males (66%) were commonly affected than female; M : F is 1.9 : 1.
5. Most of the Dengue infection occurred during the period of October to January of the year.
6. Secondary infection (76%) was common compared to primary infection (24%) especially among DHF.
7. Other than Fever (100%) Myalgia (82%), arthralgia (65%) and Headache (77%) were common symptoms described in present study.
8. Abdominal symptoms, Sleeplessness/ lethargy, facial flushing and conjunctival injection occurred significantly in DHF than DF.
9. Elevated Eosinophil count and Transaminases occurred in DHF than DF.
10. Thrombocytopenia, Haemorrhagic manifestations and Plasma leakage manifestations occurred in 42% patients.
11. Rashes (67%), Venaepuncture bleed(61%) and Malena (47%) were common bleeding manifestations.
12. Ascites (88%) and Pleural Effusion (84%) were common plasma leakage manifestations.
13. Hypotension occurred in 26% patients. All patients were treated with IV fluid according to WHO protocol.

14. IV crystalloids were the primary modality of treatment in patient with DHF. According to the guidelines Whole blood transfusion (2patients), FFP (3patients) and Platelet concentrate (4patients) were also given for DSS.
15. All DHFs were diagnosed early with appropriate investigations and managed effectively with transfusions and IV crystalloids. These effective measures saved 11 DSS patients except one.
16. One patient expired. Case Fatality Rate was 0.008%.

CONCLUSION

CONCLUSION

A total 0f 122 cases with Dengue were analysed. Results show that Dengue fever and Dengue haemorrhagic fever were noted in adults particularly during epidemics. Fluid Therapy is the corner stone of management in Dengue Haemorrhagic fever. Early recognition and aggressive management of complications is essential to prevent mortality in adults.

ANNEXURE

BIBILIOGRAPHY

1. World Health Organisation; Prevention and control of dengue and dengue haemorrhagic fever: comprehensive guidelines. WHO Regional publication, SEARO, No 29, 1999; 86-88,46-48..

2. Yizhao Lang; Dengue fever; from Wikipedia, the free encyclopedia; August 24,2009; 1-3. http://en.wikipedia.org/wiki/Dengue_fever

3. N Malavige, S Fernando, D J Fernando, S L Seneviratne; Dengue viral infections; Postgrad Med J 2004;80:588–601.

4. Dengue haemorrhagic fever: diagnosis, treatment, prevention and control. 2nd edition. Geneva : World Health Organization. 1997.

5. Pinheiro FP, Corber SJ. Global situation of dengue and dengue haemorrhagic fever and its emergence in the Americas. World Health Stat Q 1997; 50:161–8.

6. Dengue – situation in south east region; WHO released on 06 July 2007. http://www.searo.who.int/en/Section10/Section332_1098.htm

7. Dengue – situation in selected countries of the south east region; WHO released on 06 July 2007. http://www.searo.who.int/en/Section10/Section332_9482.htm

8. G.n. malavige, v.g.n.s. velathanthiri et al; Patterns of disease among adults hospitalized with dengue infections; Q J Med 2006; 99:299–305

9. Halstead SB. Is there an inapparent dengue explosion? Lancet 1999; 353:1100–1.

10. King CC, Wu YC, Chao DY, et al. Major epidemics of dengue in Taiwan in 1981–2000: related to intensive virus activities in Asia. Dengue Bulletin 2000;24:1–10.

11. Sukri NC, Laras K, Wandra T, et al. Transmission of epidemic dengue hemorrhagic fever in easternmost Indonesia. Am J Trop Med Hyg 2003;68:529–35.

12. Nisalak A, Endy TP, Nimmanitya S, et al. Serotype-specific dengue virus circulation and dengue disease in Bangkok, Thailand from 1973 to 1999. Am J Trop Med Hyg 2003;68:191–20

13. Ha DQ, Tien NT, Huong VT, et al. Dengue epidemic in Southern Vietnam,1998. Emerg Infect Dis 2000;6:422–5.

14. latt KB, Linthicum KJ, Myint KS, et al. Impact of dengue virus infection on feeding behavior of Aedes aegypti. Am J Trop Med Hyg 1997;57:119–25

15. Narayanan M, Aravind MA, Thilothammal N, et al. Dengue fever epidemic in Chennai—a study of clinical profile and outcome. Indian Pediatr 2002;39:1027–33.

16. Anuradha S, Singh NP, Rizvi SN, et al. The 1996 outbreak of dengue hemorrhagic fever in Delhi, India. Southeast Asian J Trop Med Public Health 1998;29:503–6.

17. Wali JP, Biswas A, Handa R, et al. Dengue haemorrhagic fever in adults: a prospective study of 110 cases. Trop Doct 1999;29:27–30.

18. Lum LC, Lam SK, Choy YS, et al. Dengue encephalitis: a true entity? Am J Trop Med Hyg 1996;54:256–9.

19. Kabra SK, Juneja R, Madhulika, et al. Myocardial dysfunction in children with dengue haemorrhagic fever. Natl Med J India 1998;11:59–61.

20. Littaua R, Kurane I, Ennis FA. Human IgG Fc receptor II mediates antibody- dependent enhancement of dengue virus infection. J Immunol 1990;144:3183–6.

21. Yang KD, Yeh WT, Yang MY, et al. Antibody-dependent enhancement of heterotypic dengue infections involved in suppression of IFNgamma production. J Med Virol 2001;63:150–7.

22. Hathirat P, Isarangkura P, Srichaikul T, et al. Abnormal hemostasis in dengue hemorrhagic fever. Southeast Asian J Trop Med Public Health 1993;24(suppl 1):80–5.

23. Wang S, He R, Patarapotikul J, et al. Antibody-enhanced binding of dengue- 2 virus to human platelets. Virology 1995;213:254–7

24. Chaturvedi UC, Elbishbishi EA, Agarwal R, et al. Cytotoxic factor autoantibodies: possible role in the pathogenesis of dengue haemorrhagic fever. FEMS Immunol Med Microbiol 2001;30:181–6.

25. Liu CC, Huang KJ, Lin YS, et al. Transient CD4/CD8 ratio inversion and aberrant immune activation during dengue virus infection. J Med Virol 2002;68:241–52.

26. Henchal EA, McCown JM, Seguin MC, et al. Rapid identification of dengue virus isolates by using monoclonal antibodies in an indirect immunofluorescence assay. Am J Trop Med Hyg 1983;32:164–9.

27. Branch SL, Levett PN. Evaluation of four methods for detection of immunoglobulin M antibodies to dengue virus. Clin Diagn Lab Immunol 1999;6:555–7.

28. Nawa M, Ichikawa Y, Inouye S. Serotyping of dengue viruses by an enzyme- linked immunosorbent assay. Jpn J Med Sci Biol 1985;38:217–21.

29. Tripathi BK, Gupta B, Sinha RS, et al. Experience in adult population in dengue outbreak in Delhi. J Assoc Physicians India 1998;46:273–6.

30. Workshop on case management of dengue hemorrhagic fever, June 2002, Bangkok, Thailand.

31. Wills B. Volume replacement in dengue shock syndrome. Dengue Bulletin 2001;25:50–5.

32. Soni A, Chugh K, Sachdev A, et al. Management of dengue fever in ICU. Indian J Pediatr 2001;68:1051–5.

33. Dung NM, Day NP, Tam DT, et al. Fluid replacement in dengue shock syndrome: a randomized, double-blind comparison of four intravenous-fluid regimens. Clin Infect Dis 1999; 29:787–94.

34. Perikov Y. Development of dengue vaccine. Dengue Bulletin 2000;24:71–76.

35. Guha-Sapir D, Schimmer B. Dengue fever: new paradigms for a changing epidemiology. Emerg Themes Epidemiol 2005; 2:1.

36. Harris E, Videa E, Perez L, et al. Clinical, epidemiological and virological features of dengue in the 1998 epidemic in Nicaragua. Am J Trop Med Hyg 2000; 63:5–1

37. Krishnamurti C, Kalayanarooj S, Cutting MA, Peat RA, Rothwell SW, Reid TJ, et al. Mechanisms of hemorrhage in dengue without circulatory collapse. Am J Trop Med Hyg 2001; 65:840–7.

38. Shivbalan S, Anandnathan K, Balasubramanian S, Datta M, Amalraj E. Predictors of spontaneous bleeding in Dengue. Indian J Pediatr 2004; 71:33–6.

39. NP Singh, Rajat J *et al;* The 2003 outbreak of Dengue Fever in Delhi, India; Vol 36 No. 5 September 2005

40. Manual for using Dengue card test ; released by Bhat – Bio tech limited; Bangalore.

41. Scot B Halstead; Dengue; Pathophysiology; 307-310.

42. G.N. Malavige, V.G.N.S. Velathanthiri *et al*; Patterns of disease among adults hospitalized with dengue infections; Srilanka; Q J Med 2006; 99:299–305.
43. Adriana O. Guilarde, Marilia D. Turchi *et al;* Dengue and Dengue Hemorrhagic Fever among Adults: Clinical Outcomes Related to Viremia, Serotypes, and Antibody Response; JID 2008:197; 817 – 823
44. NP Singh, Rajat J *et al;* The 2003 outbreak of Dengue Fever in Delhi, India; Southeast Asian j trop med public health; Vol 36 No. 5 sep 2005.1174-1178.
45. Janak Kishore, Jagdeep Singh, T.N. Dhole and A. Ayyagari; Clinical and Serological Study of First Large Epidemic of Dengue in and around Lucknow, India, in 2003; Dengue Bulletin – Volume 30, 2006; 72-79.
46. Harris E, Videa E, Perez L, et al. Clinical, epidemiological and virological features of dengue in the 1998 epidemic in Nicaragua. Am J Trop Med Hyg 2000; 63:5–11.
47. Kularatne SA, Gawarammana IB, Kumarasiri PR. Epidemiology, clinical features, laborotory investigations and early diagnosis of dengue fever in adults: a descriptive study in Sri Lanka. Southeast Asian J Trop Med Public Health ;2005; 36:686–92.

PROFORMA

Name:

Age: Sex: I.P.No: DOA:

Symptoms:

Fever: Days: Days prior to admission: High/Low

Continuous/Intermittent Chills:

Headache: Days: Area:

Retro orbital Pain: Myalgia: Arthralgia:

Hemorrhagic manifestations:

Epistaxis/ malena/ Gum bleed/ Hematemesis/ Vena.Punc.Bleed/ Hess Test/ Rashes(area:)/ other- menorrhagia, prolonged bleed

Abdominal Pain: Nause/Vomiting: Constip/Diarrhea:

Coryza: Sleep/Lethargy: Past h/o fever

BP

Pulse rate:

Temp; <39/>39^0 : Palor: Face\flush:

Conj.Injection: Lyphadenopathy: Systemicexamination

Laboratory Investigations:

TC	DC-N	DC-L	DC - E	PCV	HB	Platelet
BT	CT	Sugar	Urea	Crea	S.Bil	SGOT
SGPT	SAP	T.Protein	S.Alb			

Dengue Test IgM: IgG:

CXR

USG Abdomen:

Blood transfusion:

FFP transfusion

Platelet transfusion:

Dengue :Infection Type: Primary?secondary Dengue Type: DF/DHF/DSS

Grade of DHF:I/II/III/IV

Outcome:

MASTER CHART

S. No	Name	Age	Sex	IP No	DOA	Occupation	Address	Fever	Days	Gap to admission	H/L	Chills	Day/Ni	Headache	Days	Area	ROP	Myalgia
1	Aravind	14	1	421342	28/8/07	student	vanarpaettai	1	4	2	h	1	i	1	2	f	1	1
2	saritha	24	f	39079	28/8/07	hw	chennai	1	10	6	1	0	1	0	0	0	0	1
3	ravi	40	1	39124	20/11/07	driver	achara	1	7	4	h	0	i	1	3	f,p	0	0
4	thomas	20	1	29475	2/9/2007	shopper	chennai	1	5	2	h	1	b	1	2	f		1
5	Radha	43	1	41307	11/12/2007	farmer	padi	1	3	2	h	0	i	1	2	f	0	0
6	Baskaran	23	1	32123	22/9/08	student	chennai	1	5	3	h	1	b	1	3	p,t	0	1
7	Sathish	17	1	31995	21/9/08	colley	chennai	1	7	2	1	1	b	1	4	p,t	0	1
8	Velmurugan	18	1	32667	25/9/07	student	chennai	1	3	1	h		i	0	0	0	0	1
9	Premalatha	19	f	32286	23/9/07	student	chennai	1	3	1	h	0	i	1	1	f,t	0	1
10	soniya	19	f	32189	22/9/07	student	chennai	1	4	1	h	1	b	0	0	0	0	1
11	Vignesh	15	1	33305	1/10/2007	student	chennai	1	8	3	h	1	b	1	5	p,t	0	1
12	Asokan	25	1	34618	11/10/2007	driver	thiruthani	1	4	2	h	0	1	1	2	f,t	0	1
13	Mohan	19	1	35140	16/10/2007	mechanic	pudhukuppam	1	7	5	h	0	b	0	0	0	0	1
14	Raghu	50	1	35141	16/10/07	farmer	thiruthani	1	6	4	h	1	1	1	2	f,t	0	1
15	Shanthy	35	f	35236	16/10/07	hw	chennai	1	7	5	1	0	i	1	5	f,t	0	1
16	Mallika	42	f	35222	16/10/07	hw	chennai	1	8	4	h	1	b	1	3	f	0	1
17	Selvi	19	f	34997	15/10/07	hw	chennai	1	6	3	h	1	b	1	3	f	0	1
18	Yasodha	70	f	35003	15/10/07	cooly	thiruvani	1	4	3	1	1	1	0	0	0	0	1
19	Parvathy	40	f	34970	23/10/07	hw	chennai	1	5	3	h	0	b	0	0	0	0	0
20	Bakiyalakshmi	42	f	35093	15/10/07	hw	chennai	1	3	2	1	0	i	0	0	0	0	0
21	Maran	22	1	35607	19/10/07	farmer	vellore	1	5	2	1	1	b	1	3	all	0	1
22	Sankar	30	1	36055	23/10/07	road work	ponndamalee	1	8	4	h	1	b	1	3	all	0	1
23	Babu	30	1	36079	23/10/07	cooly	chennai	1	5	3	1	0	i	1	4	occ	1	1
24	Shanker	23	1	36020	24/10/07	cooly	chennai	1	5	2	h	1	1	1	3	f	1	0
25	Rajesh kumar	16	1	36026	23/10/07	student	chennai	1	6	3	h	1	i	1	3	f	0	1
26	Prasanth	14	1	36121	24/10/07	student	chennai	1	6	3	1	1	1	1	3	f,o	0	1
27	Jothi	28	1		1/11/2007	driver	tiruvan	1	6	4	h	1	i	0	0	0	0	1
28	Ramesh	32	1	38969	19/11/07	shopper	padi	1	4	2	h	0	1	1	2	f,t,o	0	1
29	Mani	60	1	39692	26/11/07	painter	chennai	1	4	2	h	0	1	1	2	f	0	1
30	Gnanaprakasam	73	1	39733	26/11/07	cooly	chennai	1	3	1	h	1	1	1	1	f,t	0	1
31	Arunkumar	15	1	39839	27/11/07	student	chennai	1	6	3	1	1	i	0	0	0	0	0
32	Anandhan	37	1	41842	27/11/07	mason	chennai	1	5	3	h	1	b	1	3	f	0	1

33	Aseemdoss	20	1	40	1/1/2008	colley	chennai	1	6	4	1	0	b	1	3	occ	1	1
34	Krishnakumar	19	1	88	1/1/2008	shopper	chennai	1	6	3	h	1	i	1	3	f	1	1
35	Indirani	45	f	347	2/1/2008	hw	chennai	1	5	2	1	0	1	0	0	0	0	0
36	Mohamad Haniff	45	1	56431	4/12/2007	worker	chennai	1	3	2	h	1	1	1	2	occ	0	1
37	Vijayan	18	1	32944	29/12.07	rikshaw	chennai	1	5	4	h	1	1	1	4	f	0	1
38	Praveen kumar	13	1	33144	29/9/07	student	kasimedu	1	6	4	h	1	1	0	0	0	0	1
39	Vijayababu	22	1	33036	28/12/07	farmer	thiruthani	1	5	3	h	0	i	1	2	occ	1	1
40	Kumar	22	1	33728	21/10/07	shopper	chennai	1	3	1	h	0	1	1	2	f	0	1
41	Vinoth	21	1	33614	4/10/2007	student	chennai	1	3	1	h	0	1	1	2	f,t	1	1
42	Vimalraj	19	1	33532	3/10/2007	farmer	tiruporur	1	4	3	1	1	i	1	2	f,t	0	0
43	Dessapan	19	1	33779	6/10/2007	rikshaw	chennai	1	6	4	h	1	i	0	0	0	0	1
44	Meyal Joy	33	1	34052	10/12/2007	student	chennai	1	3	1	h	1	1	1	3	0,f,t	1	1
45	Venkataesen	23	1	42423	12/12/2007	cooly	chennai	1	6	3	1	1	i	1	2	o,f	0	1
46	Sindhu	27	f	42563	16/12/07	hw	chennai	1	7	4	1	0	i	1	3	f,t	0	1
47	Anbu	40	1	43423	28/12/07	driver	chennai	1	7	4	h	1	i	0	0	0	0	1
48	Usha	24	f	36737	30/11/07	hw	chennai	1	6	3	h	1	i	0	0	0	0	0
49	Ramanammal	70	f	36816	30/11/07	hw	chennai	1	4	2	h	0	1	1	2	all	0	1
50	Vasanthi	14	f	37063	1/11/2007	hw	chennai	1	4	2	h	0	1	1	2	f,t	0	1
51	jaya	26	f	36395	11-Mar	hw	chennai	1	6	3	h	0	i	1	5	f	0	1
52	Kanniammal	37	f	37063	11-Mar	hw	chennai	1	5	2	h	1	i	0	0	0	0	0
53	Vasudevan	25	1	36862	31/11/07	shopper	chennai	1	4	3	1	0	i	1	3	f	1	1
54	Subhashini	35	f	37538	5/12/2007	hw	chennai	1	6	2	h	0	1	1	4	all	1	1
55	Rajaesh	19	1	38121	12/12/2007	student	chennai	1	6	2	h	0	1	1	4	all	0	1
56	Sundar	32	1	2179/08	22/1	shopper	chennai	1	6	4	1	1	i	0	0	0	0	1
57	Syed umar	15	1	1213	11/1/2008	student	chennai	1	5	3	h	1	i	1	2	all	0	0
58	Guna	19	f	33368	2/11/2007	student	chennai	1	4	2	h	1	i	1	3	all	0	1
59	Yamuna	34	f	33348	21/12/07	hw	chennai	1	4	2	h	0	1	1	3	f	0	1
60	Tamilarasi	20	f	24118	24/2/08	hw	chennai	1	6	4	1	1	i	0	0	0	0	0
61	Mareeswari	18	f	33334	5/3/2008	student	chennai	1	3	1	h	1	1	1	2	f	0	0
62	Sharmila	25	f	37393	5/3/2008	hw	chennai	1	4	2	h	0	1	0	0	0	0	0
63	Maheswari	19	f	37950	10/3/2008	student	chennai	1	3	1	h	0	1	1	2	f	0	1
64	Pugazathi	30	f	38584	12/3/2008	hw	chennai	1	4	2	h	1	i	1	2	f	0	1
65	Kalaivanan	20	1	11172	7/9/2008	student	chennai	1	3	1	h	0	1	0	0	0	1	1

66	Raziyabee	42	f	37435	8/10/2008	hw	chennai	1	3	1	h	0	1	1	2	0	0	0
67	Munusamy	40	f	37435	16/11/08	farmer	tirutani	1	6	4	h	1	i	1	2	occ	1	0
68	Anadhanarayanan	21	1	38386	15/9/08	mason	chennai	1	4	2	h	0	1	1	2	f	1	1
69	sundar	20	1	32453	9-Jul	cooly	chennai	1	6	4	1	0	i	1	2	f	0	1
70	Vanitha	25	f	34733	12/9/2008	hw	chennai	1	3	1	h	0	1	0	0	0	0	0
71	Kannan	28	1	34784	15/9/08	cooly	chennai	1	4	2	h	0	1	0	0	0	0	0
72	Saranraj	19	1	34822	17/9/08	student	chennai	1	5	2	1	0	i	1	1	all	0	0
73	Soniya	21	f	35207	20/9/08	student	chennai	1	3	2	h	1	1	1	2	all	0	1
74	satheeshvaran	15	1	36162	25/9/08	student	chennai	1	5	3	1	1	i	1	2	all	0	1
75	Lakshmi	45	f	33336	6/9/2008	hw	chennai	1	5	3	h	0	i	1	4	all	0	1
76	Ranjitham	48	f		1/10/2008	hw	chennai	1	3	1	h	0	1	1	2	f	0	1
77	Kasthuri	45	f	33306	3/10/2008	hw	chennai	1	6	3	h	0	i	1	2	f	0	1
78	Udhaya reagen	24	1	33408	1/10/2007	electrician	chennai	1	7	3	1	1	i	1	2	o	0	1
79	Suresh	28	1	33455	3/10/2008	cooly	chennai	1	6	4	1	1	i	1	3	all	0	1
80	Srinivasan	40	1	43224	6/1o/08	cooly	chennai	1	4	1	h	1	1	1	3	all	0	1
81	Saravanan	14	1	33970	8/10/2007	student	chennai	1	3	3	h	0	1	1	3	all	0	1
82	Lakshmi	26	f	326002	2/11/2007	hw	chennai	1	6	3	1	0	i	1	3	f	0	1
83	Ashnaf	23	1	35407	13/10/07	hw	chennai	1	3	2	h	0	1	1	2	f	0	1
84	Mohamed Hussain	16	1	34763	13/10/07	hw	chennai	1	4	2	h	1	1	1	2	f,o	0	1
85	Bakyalakshmi	23	f	33829	5/10/2007	hw	chennai	1	3	1	h	1	1	0	0	0	0	1
86	Rajesweri	13	f	34024	6/10/2007	student	chennai	1	4	1	h	0	1	1	2	a	0	1
87	Ramadevi	19	f	34192	7/10/2007	student	chennai	1	4	2	h	1	1	0	0	0	0	1
88	Deepa	19	f	35876	22/10/07	student	chennai	1	3	2	h	1	1	0	0	0	0	1
89	Sivagami	40	f	35287	17/10/07	hw	chennai	1	3	2	h	0	1	1	2	a	0	1
90	Anand	20	1	40344	3/12/2007	shopper	chennai	1	4	2	h	0	1	1		2	a	1
91	Mohamed Iqbal	20	1	40611	5/12/2007	business	chennai	1	4	2	h	1	i	1	2	a	0	0
92	Annadurai	36	1	40637	5/12/2007	colley	chennai	1	4	3	h	0	i	1	2	a	0	1
93	Suresh	27	1	40841	6/12/2007	labour	chennai	1	4	2	1	1	i	1	2	a	0	1
94	Thangaraj	16	1	40976	9/12/2007	student	chennai	1	6	4	1	1	i	1	4	a	0	1
95	Durai	55	1	41190	11/12/2007	labour	chennai	1	6	3	1	0	i	1	3	f	0	1
96	Goutham shanker	20	1	41115	10/12/2007	student	chennai	1	6	3	1	0	i	1	2	a	0	1
97	Dhanasekaran	13	1	41166	10/12/2007	student	chennai	1	7	4	1	0	i	1	2	a	0	1
98	Lisban	49	1	41361	10/12/2007	shopper	chennai	1	2	2	h	0	1	0	0	0	0	1

99	Devamoorthy	29	1	41701	16/12/07	farmer	manali	1	6	4	1	0	i	1	2	o	0	0
100	Harikrishnana	15	1	41956	18/12/07	student	chennai	1	6	4	1	1	i	1	2	o	0	1
101	Allirajan	32	1	42013	17/12/07	driver	chennai	1	5	3	1	0	i	1	4	a	0	1
102	Munivel	25	1	42314	19/12/07	labour	chennai	1	7	4	1	1	i	1	5		0	1
103	Paneerselvam	52	1	42346	20/12/07	labour	chennai	1	5	3	h	1	1	1	3	a	0	1
104	Vijaya	30	f	42619	25/12/07	hw	chennai	1	6	3	h	0	1	1	3	a	1	1
105	Jothy	40	1	42655	27/12/07	shopper	chennai	1	7	4	1	1	i	1	4	o	0	1
106	Antony	65	1	42953	30/12/07	farmer	chennai	1	3	2	h	1	1	1	2	o	0	1
107	Harikrishnan	43	1	32145	31/11/07	labour	chennai	1	4	3	1	1	1	1	3	f	0	1
108	Thirumal	19	1	638	4/1/2008	student	chennai	1	4	2	h	1	1	1	2	a	0	1
109	sharmila	18	f	34506	11/10/2007	student	chennai	1	3	2	h	0	1	0	0	0	0	1
110	chithra	26	f	38395	11-Mar	hw	chennai	1	6	3	h	0	i	1	5	f	0	1
111	kuppan	15	1	32305	1/10/2008	student	chennai	1	8	3	h	1	b	1	5	p,t	0	1
112	Balu	37	1	41842	27/11/08	mason	chennai	1	5	3	h	1	b	1	3	f	0	1
113	Saravanan	23	1	35407	13/10/08	hw	chennai	1	3	2	h	0	1	1	2	f	0	1
114	seetha	30	f	38584	12/3/2008	hw	chennai	1	4	2	h	1	i	1	2	f	0	1
115	Tirupathi	40	1	42635	27/12/08	shopper	chennai	1	7	4	1	1	i	1	4	o	0	1
116	Sundar	32	1	32412	22/1/08	shopper	chennai	1	6	4	1	1	i	0	0	0	0	1
117	Moorthy	40	1	68435	16/11/08	farmer	tirutani	1	6	4	h	1	i	1	2	occ	1	0
118	Vedhan	18	1	32237	25/9/08	student	chennai	1	3	1	h		i	0	0	0	0	1
119	Sooranammal	70	f	36216	30/11/08	hw	chennai	1	4	2	h	0	1	1	2	all	0	1
120	prbakaran	40	1	46224	6/1o/08	cooly	chennai	1	4	1	h	1	1	1	3	all	0	1
121	Mayilsamy	22	1	33607	19/10/08	farmer	vellore	1	5	2	1	1	b	1	3	all	0	1
122	Sandha kumar	37	1	41842	27/11/08	mason	chennai	1	5	3	h	1	b	1	3	f	0	1

Arthralgia	Hemorrhagic	Epistaxis	malena	Gum bleed	Hematemesis	Vena.Punc.Bleed	Hess Test	Rashes	Rashes Area	Abdominal Pain	Nause/Vomiting	Constip/Diarrhea
1	1	1		0	1	1	0	1		1	1	0
1	1			1		1		1	legs,arms,back		1	
0	0	0		0	0	0	0	0	0	0	0	0
1	1	0	1	0	0	1	0	0		1	1	1
1	0	0	0	0	0	0	0	0	0	1	1	1
1	0	0	0	0	0	0	0	0	0	0	0	1
0	0	0	0	0	0	0	0	0	0	1	0	0
1	1	0	0	0	0	1	0	1	0	1	0	0
0	0	0	0	0	0	0	0	0	0	1	1	0
0	0	0	0	0	0	0	0	0	0	1	1	0
1	0	0	0	0	0	0	0	0	0	1	1	0
1	1	0	1	1	1	1	0	1	trunk	1	1	1
1	0	0	0	0	0	0	0	0	0	0	1	0
1	1	0	1	1	0	0	0	0	0	1	0	0
1	1	0	0	0	1	1	0	1	trunk	0	1	0
1	1	1	1	0	0	0	0	0	0	1	1	0
1	0	0	0	0	0	0	0	0	0	1	1	1
1	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0
0	1	0	0	0	0	1	0	1	trunk	1	0	0
1	1	0	1	0	1	1	0	1	body	1	1	1
1	1	0	0	0	1	0	0	0	0	1	1	1
1	0	0	0	0	0	0	0	0	0	0	0	0
0	1	0	1	0	0	0	0	0	0	1	1	1
1	0	0	0	0	0	0	0	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	1	1	1
1	0	0	0	0	0	0	0	0	0	0	1	1
1	0	0	0	0	0	0	0	0	0	1		0
1	0	0	0	0	0	0	0	0	0	0	1	0
1	0	0	0	0	0	0	0	0	0	0	1	1
0	1	1	0	0	0	0	0	1	sub conj	0	1	1
1	0	0	0	0	0	0	0	0	0	0	0	0

1	1	0	0	0	1	1	1	1		1	0	1
1	1	0	1	0	1	0	1	1	chest,back	1	1	1
0	1	1	0	0	1	1	0	0	0	0	1	1
1	1	0	0	1	0	0	0	1	limbs,chest	1	1	1
1	1	0	0	1	1	0	0	1	0	0	1	0
1	0	0	0	0	0	0	0	0	0	0	0	0
1	1	0	1	1	1	0	0	0	0	1	1	1
1	0	0	0	0	0	0	0	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	1	0	0
0	1	0	0	0	0	0	0	1	back ,leg	0	0	0
1	0	0	0	0	0	0	0	0	0	0	1	1
0	0	0	0	0	0	0	0	0	0	0	0	1
1	1	0	1	0	1	0	0	0	0	1	1	0
1	1	1	1	1	0	0	0	1	forarm,leg	1	1	0
1	0	0	0	0	0	0	0	0	0	0	1	0
0	0	0	0	0	0	0	0	0	0	0	0	0
1	0	0	0	0	0	0	0	0	0	1	1	0
1	0	0	0	0	0	0	0	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0
1	0	0	0	0	0	0	0	0	0	0	1	0
0	0	0	0	0	0	0	0	0	0	0	1	0
1	1	0	1	0	1	1	1	1	sub conj,back	1	1	1
0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	0	1	1	0	0	0	1	trunk	1	0	0
1	0	0	0	0	0	0	0	0	0	0	0	0
1	1	0	0	1	0	1	0	1	hands	1	1	1
0	1	0	0	0	0	1	0	1	legs,arms,back	0	0	1
0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	1	1	0
1	1	0	1	1	0	1	0	1	legs,arms,back	1	1	1
0	0	0	0	0	0	0	0	0	0	0	0	0

0	1	0	0	0	0	0	0	0	0	0	0	0
0	1	0	1	0	0	1	0	1	arms,legs	1	0	0
1	1	0	1	1	1	1	0	0	0	1	1	1
1	0	0	0	0	0	0	0	0	0	1	1	1
0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	0	1	0
0	0	0	0	0	0	0	0	0	0	0	1	0
0	1	0	0	0	0	1	0	1	0	0	0	0
1	1	0	1	0	1	0	0	1	0	1	1	1
0	1	1	0	1	0	0	0	0	0	1	1	1
0	1	0	0	0	0	1	0	1	0	0	1	1
1	0	0	0	0	0	0	0	0	0	1	1	0
1	0	0	0	0	0	0	0	0	0	1	1	0
0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	1	1	1	1	1	1	1	all over	1	1	0
0	1	0	1	0	1	0	0	0	0	1	1	0
1	1	0	0	0	0	1	0	0	0	0	1	0
0	0	0	0	0	0	0	0	0	0	1	1	0
0	1	0	1	0	0	1	0	1	0	1	0	0
1	1	0	0	0	0	0	0	1	trunk	1	1	0
0	0	0	0	0	0	0	0	0	0	0	1	0
0	0	0	0	0	0	0	0	0	0	0	0	0
1	0	0	0	0	0	0	0	0	0	1	1	0
1	0	0	0	0	0	0	0	0	0	1	0	0
0	0	0	0	0	0	0	0	0	0	0	1	0
0	1	0	1	1	0	1	0	0	0	1	1	0
1	0	0	0	0	0	0	0	0	0	0	1	0
1	0	0	0	0	0	0	0	0	0	1	1	0
1	1	0	0	0	0	0	0	1	arms,legs	1	1	0
0	0	0	0	0	0	0	0	0	0	1	1	1
0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	0	0	0	0	1	0	1	back ,leg	1	1	1

0	1	0	0	1	1	0	0	1	arms,legs	1	1	1
1	0	0	0	0	0	0	0	0	0	1	1	1
1	1	0	0	1	0	0	0	0	0	0	0	0
0	0	0	0	0	0	0	0	0	0	1	1	1
1	0	0	0	0	0	0	0	0	0	1	1	0
1	0	0	0	0	0	0	0	0	0	0	1	0
1	1	0	1	0	1	1	0	0	0	1	0	1
1	0	0	0	0	0	0	0	0	0	1	1	0
1	1	1	0	1	0	1	0	0	0	1	1	0
1	0	0	0	0	0	0	0	0	0	0	1	0
1	0	0	0	0	0	0	0	0	0	0	0	0
1	0	0	0	0	0	0	0	0	0	1	0	0
1	0	0	0	0	0	0	0	0	0	1	1	0
1	0	0	0	0	0	1	0	0	0	0	0	0
1	1	0	0	0	0	1	0	1	0	0	1	0
1	1	0	1	1	0	1	0	1	legs,arms,back	1	1	1
1	0	0	0	0	0	0	0	0	0	1	0	1
0	0	0	0	0	0	0	0	0	0	0	0	0
0	1	0	1	0	0	1	0	1	arms,legs	1	0	0
1	1	0	0	0	1	0	0	1	0	1	0	0
1	0	0	0	0	0	0	0	0	0	1	1	0
0	0	0	0	0	0	0	0	0	0	0	0	0
1	1	0	1	0	1	1	0	1	body	1	1	1
1	0	0	0	0	0	0	0	0	0	0	0	0

Coryza	Sleep/Lethargy	Last Episode of fever	Temp <39	Temp >39	Paloor	Face flush	Conj.Injection	Lyphadenopathy	Systemicexamination	TC	DC-N	DC-L	DC - E	PCV
0	1	0		1	1	1	1	1		6500	72	25	3	44
1	1	1	1			1	1			4500	64	36		42
1	1	0		1	0	0	0	0	0	10000	84	16		36
1	1	0		1	0	0	1	1	0	2500	70	25	5	48
1	1	1	1		0	0	0	0	0	8000	74	26		38
0	0	0		1	0	0	1	0	0	2000	60	27	13	42
0	0	0		1	0	1	1	1	0	6500	56	44		36
0	0	0		1	0	0	1	0	0	9000	62	34	4	38
0	0	0		1	0	0	0	0	0	8000	60	30	10	34
0	1	0		1	0	0	0	0	0	2500	58	38	4	34
1	1	0	1	1	0	0	0	0	0	4000	50	49	1	42
1	1	1	1	1	1	1	1	1	0	9400	76	20	4	50
1	1	0		1	0	0	1	1	0	4000	42	56	2	37
1	1	0	1		0	1	1	0	0	11000	54	37	9	40
1	0	0		1	1	0	0	0	0	3600	83	42	4	56
1	1	1	1		1	0	0	0	0	3200	57	33	10	26
1	1	0	1		0	0	0	0	0	4400	50	50		38
1	0	0	1		0	0	0	0	0	7300	82	15	3	36
0	1	0		1	0	0	0	0	0	4000	40	50	10	43
0	1	0		1	0	0	0	1	0	4500	40	50	10	43
1	1			1		1	1	1	0	8000	53	37	10	43
0	1	0		1	0	1	1	1	0	3000	44	46	0	48
0	0			1	0	0	0	1	0	4800	57	40	3	44
1	1	0		1	0	0	1	1	0	5200	60	30	10	49
1	0			1	0	1	0	1	0	4000	30	70	0	36
1	1			1	0	0	0	0	0	3000	64	35	1	35
1	1	0	1		0	0	1	0	0	3000	60	30	10	46
1	1	0		1	0	0	0	0	spleen	4500	45	50	5	46
0	0	1		1	0	0	0	0	0	7000	77	25	3	38
1	1	0		1	0	0	0	0	0	6300	73	27	0	30
0	1	0	1		1	1	1	1	0	4100	56	44		36
0	0	0	0	1	0	0	1	0	0	12000	70	24	6	42

1	1	1	1		0	1	1	1	0	12800	60	30	5	40
1	1	1	1		1	0	1	0	0	9600	85	15	0	40
1	0	0	1		0	0	0	0	0	5400	60	35	5	38
1	1	1	1		0	0	0	0	0	5600	65	33	2	40
0	1	1	0	0	0	1	1	0	0	3900	50	40	10	44
0	1	0	1		0	0	1	0	0	8000	68	30	2	38
1	1	0		1	1	1	1	1	0	12000	67	25	8	52
1	1	0	0	1	0	0	0	0	0	4400	60	40	0	42
0	0	0		1	0	0	0	0	0	6300	68	31	1	40
0	0	1	1		0	1	1	0	0	2900	56	44	0	48
1	1	0		1	0	0	1	0	0	7100	60	40	0	40
1	1	0		1	0	0	1	1	0	5500	80	20	0	42
0	1	1	1		0	1	1	1	0	6500	64	36	0	48
1	1	1	1		1	1	1	1	0	4000	48	52	0	36
1	1	0	1		0	0	1	0	0	13000	84	11	0	28
1	1	0	1		1	0	0	0	0	8900	70	26	4	38
0	1	0	0		0	0	0	0	0	4000	54	42	4	30
1	1	0		1	0	0	1	0	0	6800	57	43	0	37
1	0	1	1	1	0	0	1	1	0	5700	80	20	0	36
0	0	0	1		1	1	1	1	0	8000	60	37	3	32
1	0	0	1		0	0	1	1	0	2900	76	24	0	44
1	0	1		1	0	0	0	0	0	4000	71	29	0	36
1	1	1		1	0	1	1	1	0	3400	25	45	20	50
1	0	0	1	1	0	0	0	0	0	14000	70	27	3	38
1	1	1		1	1	1	1	0	hepa	2800	38	42	20	38
1	0	0	1		0	0	0	0	0	5600	70	24	6	36
1	1	1		1	1	1	1	1	0	13000	60	26	14	44
1	1	0	1		1	1	1	0	hepa	4200	63	37	0	36
0	0	0	0	1	0	1	0	0	0	5700	52	40	8	36
0	0	0	0	1	0	0	0	0	0	3600	58	35	11	40
1	0	0		1	0	0	1	0	0	2600	54	40	6	38
0	1	0	1	0	0	1	1	0	0	3600	60	30	10	50
0	1	0	0	1	0	0	0	0	0	4200	74	25	1	48

0	0	0	0	1	0	0	0	0	0	3300	62	38	0	48
1	1	1	1		0	1	0	0	0	11000	62	38	0	48
1	1	0	1	0	0	1	0	0	spleen	5700	78	11	11	45
0	0	1	1		0	0	1	0	0	2800	60	38	2	35
0	0	0	0	1	0	0	0	1	0	3200	60	36	4	38
0	0	0	0	1	0	0	0	0	0	6400	64	30	6	40
0	0	0	1	0	0	0	0	1	0	4000	60	38	2	38
1	0	0	0	1	0	1	0	0	0	5100	66	30	4	36
0	0	0	1		0	1	0	1	0	3000	50	30	10	46
0	1	1	1		0	0	0	0	0	4300	56	44	0	32
0	1	1		1	0	0	1	1	spleen	4600	53	43	9	37
0	1	1	1	1	0	1	1	1	hepa	4800	55	40	5	32
1	1	0	1	0	0	0	0	0	0	4000	50	37	3	38
1	0	0	1		0	0	1	0	0	3200	74	20	6	41
0	1	0		1	0	0	0	0	psychiatric behaviour	3600	72	27	1	36
0	1	0		1	1	1	1	1	0	11000	50	45	4	55
0	0	0	1	0	0	0	0	0	0	6200	66	32	2	40
0	0	0	1	0	0	0	0	0	0	4000	70	24	6	36
0	0	0	1	0	0	0	0	0	0	4100	50	48	2	40
0	1	0	1		0	0	1	1	0	4500	45	50	5	38
0	1	0		1	0	1	1	0	hepa	5200	57	26	17	43
0	0	0	1		0	0	0	0	0	7200	70	30	0	40
0	0	0	0	1	0	0	0	1	0	10000	75	23	2	36
1	0	1		1	0	0	1	0	0	4800	65	30	5	36
0	0	0	0	1	0	0	0	0	0	3600	55	35	10	40
1	0	0		0	0	0	0	0	0	2800	39	41	20	40
1	1	0	1		0	0	1	0	0	3600	48	42	10	42
1	0	0	1		0	0	0	0	0	8000	60	30	10	37
1	0	0	0	1	0	0	1	1	0	9200	62	34	4	38
0	1	0	1		0	0	0	1	0	11000	54	37	9	40
1	0	0	0	1	0	0	0	1	0	4000	70	28	2	42
1	0	0	1	0	0	0	0	0	0	6300	64	35	1	42
1	1	0	0	1	0	1	0	0	0	7200	90	10	0	46

0	1	0	1	0	0	1	1	1	0	3200	70	23	7	51
0	1	0	0	1	0	0	0	0	0	4000	50	49	1	40
1	0	0	1	0	0	0	0	1	0	3200	72	21	7	43
1	1	1	1	0	0	0	1	1	0	2700	60	30	10	46
1	1	0	0	1	0	0	1	0	0	12000	84	11	5	48
1	1	1	0	1	0	1	0	0	0	4000	71	29	0	36
0	1	0	1	0	0	0	0	0	0	10000	80	20	0	49
0	0	0	0	1	0	1	0	0	0	6300	72	28	0	46
1	1	0	1		0	0	1	0	0	12300	54	37	9	40
0	0	0	0	1	0	0	0	0	0	5600	70	24	6	38
0	0	0	0	1	0	0	1	0	0	9000	83	15	2	41
1	0	1	1	1	0	0	1	1	0	6800	57	43	0	37
1	1	0	1	1	0	0	0	0	0	4000	50	49	1	42
0	0	0	0	1	0	0	1	0	0	12000	70	24	6	42
0	0	0	1	0	0	0	0	0	0	4000	70	24	6	36
0	1	0	1	0	0	1	1	0	0	3600	60	30	10	50
0	1	0	1	0	0	0	0	0	0	10000	80	20	0	49
1	0	0	1	1	0	0	0	0	0	14000	70	27	3	38
1	1	1	1		0	1	0	0	0	11000	62	38	0	48
o r	0	0		1	0	0	1	0	0	6500	56	44		36
0	1	0	0		0	0	0	0	0	4000	54	42	4	30
0	1	0		1	0	0	0	0	psychiatric behaviour	3600	72	27	1	36
1	1			1		1	1	1	0	8000	53	37	10	43
0	0	0	0	1	0	0	1	0	0	12000	70	24	6	42

HB	Platelet	BT	CT	Sugar	Urea	Crea	LFT	S.Bil	SGOT	SGPT	SAP	T.Protein	S.Alb	IgM	IgG	CXR	USG Abdomen	Hepato/Spleen	Ascites	Pleural Eff	Hypotension
14	12000	n	n	140	42	1	ab	0.7	232	62	125	5.5	2.8	p	n	n	gall bladder wall	hep;spleen	1	b pe	1
13	55000	n	n	68	35	0.9	ab	1.1	129	94	59	5.7	3.4	p	p	n	gall bladder wall	spleen	1	rt pe	0
11	300000	n	n	108	28	0.9	n	1	36	26	93	5.6	3.5	n	p	n	0	0	0	0	0
15	40000	n	n	108	26	0.8	ab	0.9	148	160	154	5.8	3.4	p	p	n	rt pe	0	0	rt pe	0
14	145000	n	n	138	18	0.8	n	1	76	39	47	5.9	4	p	p	copd	0	0	0	0	0
14	84000	n	n	100	26	0.6	ab	1.1	55	49	62	5.5	3.6	n	p	n	0	0	0	0	0
11	130000	n	n	72	21	1.1	n	1	36	40	67	6.1	3.4	p	n	n	0	0	0	0	0
13.3	60000	n	n	96	28	0.9	n	0.8	32	30	66	6	3.2	n	p	n	1	0	1	bl pe	1
11	280000	n	n	72	20	0.8	n	1	36	39	58	5.6	3.6	n	p	n	0	0	0	0	0
11	160000	n	n	76	16	0.9	n	0.8	40	38	88	5.8	3.6	p	p	n	0	0	0	0	0
13.2	210000	n	n	80	20	0.7	n	1.1	46	33	76	5.5	3.7	n	p	n	1	hep;spleen	0	0	0
15	15000	n	n	125	26	0.9	ab	1.4	156	98	110	5.9	3.4	p	p	rt pe	1	0	1	b pe	0
12	75000	n	n	114	32	0.82	ab	1	55	36	112	5.8	3.6	p	p	n	1	spleen	0	0	0
13	55000	n	n	124	42	1	n	1.1	36	32	68	5.8	4	p	p	rt pe	rt pe	0		rt pe	0
18	65000	n	n	128	31	1.2	ab	1	47	55	60	6.4	4	p	p	n	0	0	0	0	0
7	35000	n	n	112	32	1	ab	o.9	196	148	140	6.3	4.2	p	n	n	0	0	1	1	1
11	47000	n	n	98	34	1	n	0.8	36	34	102	6	3.8	p	n	n	0	n	0	0	0
12	180000	n	n	121	32	0.98	n	1	43	36	84	5.8	4	p	p	n	1	spleen	0	0	0
14	120000	n	n	118	34	1.1	n	0.9	38	43	87	6	3.9	p	p	n	0	0	0	0	0
13	50000	n	n	96	32	1.1	ab	1	195	160	78	5.6	3	p	p	n	1	0	1	0	0
14	18000	n	n	90	20	0.9	ab	1.2	240	153	115	6.1	4	p	p	n	1	0	1	rt pe	1
17.2	40000	n	n	96	26	0.8	ab	1.1	192	121	122	5	3	p	p	n	1	hep	1	rt pe	0
14	154000	n	n	56	21	0.8	n	1	46	25	128	6.7	3.8	p	p	n	0	0	0	0	0
16.4	25000	n	n	123	34	1	ab	1	183	128	71	6.2	3.6	p	p	rt pe	gall bladder wall	spleen	1	b pe	0
11	249000	n	n	132	41	1	n	1	48	32	121	6.3	3.5	n	p	n	0	0	0	0	0
10	156000	n	n	141	38	0.8	n	0.9	38	32	86	5.8	3.6	p	p	n	0	0	0	0	0
14	150000	n	n	121	35	1	n	0.8	42	36	78	6.2	4	p	0	n	0	0	0	0	0
14	110000	n	n	75	21	0.8	n	1	46	36	78	5.8	3.6	p	0	n	1	spleen	0	0	0
9.8	127000	n	n	146	32	0.9	n	1.1	42	37	96	6	3.8	p	p	n	0	0	0	0	0
12	35000	n	n	113	36	1	n	1	36	38	78	5.8	3.8	p	0	n	0	0	0	0	0
11	83000	n	n	72	36	0.8	ab	1	83	76	112	5.8	3.4	p	p	n	1	0	1	lt pe	0
13	280000	n	n	122	40	1	ab	1.1	88	45	104	6	4	p	0	n	1	hep	0	0	0

14	34000	n	n	100	36	1	ab	1	116	123	102	6.1	3.9	0	p	n	1	hep:spleen	1	rt pe	0
13	50000	n	n	84	36	0.8	n	1.1	38	41	83	5.7	3.9	p	p	n	1	0	0	rt pe	0
10	67000	n	n	100	34	0.7	ab	0.8	148	122	98	6	3.5	p	p	n	gall bladder wall	hep	1	0	0
13	54000	n	n	73	28	0.9	n	1	28	24	64	5.8	3.7	p	p	n	1	0	1	bl pe	0
14	80000	n	n	100	17	0.8	ab	0.8	270	127	122	5.6	3.6	p	0	n	1	0	1	lt pe	0
13	220000	n	n	87	21	1	n	1	34	36	112	6.7	3.6	p	p	n	0	0	0	0	0
16	29000	1	n	120	32	0.8	ab	o.8	112	94	110	6.4	3.4	p	p	n	1	spleen	1	rt pe	0
13	180000	n	n	113	28	0.9	ab	1	117	116	68	7	3.7	p	p	n	0	0	0	0	0
13	177000	n	n	98	23	0.9	n	0.8	35	32	69	5	3.2	0	p	n	0	0	0	0	0
13	80000	n	n	112	32	0.5	ab	0.9	92	42	88	5.6	3.4	p	p	n	1	0	1	lt pe	1
13	90000	n	n	103	28	0.9	n	o.8	43	35	110	6.1	3.6	p	0	n	0	0	0	0	0
12	220000	n	n	98	28	0.8	n	1	22	16	84	5.6	3	p	p	n	0	0	0	0	0
12	88000	1	n	100	26	1	ab	1.1	92	82	112	6	3.6	p	p	n	1	0	1	rt pe	0
12	44000	n	n	102	28	0.8	ab	1	52	48	105	5.4	3.2	p	p	n	1	0	1	b pe	1
10	310000	n	n	98	32	0.9	n	1.1	38	32	96	6	3.5	p	p	n	0	0	0	0	0
12	420000	n	n	103	31	1	n	1	24	19	101	5.8	3.2	p	p	n	0	0	0	0	0
13	110000	n	n	112	30	1	n	0.8	30	28	98	5.9	3.5	p	p	n	0	0	0	0	0
13	150000	n	n	102	32	1	n	1	30	26	112	5.6	3	p	0	n	0	0	0	0	0
12	120000	n	n	102	30	0.8	n	0.9	28	30	95	6	3	p	p	n	0	0	0	0	0
11	180000	n	n	101	28	0.7	n	1	32	28	88	5.6	3.2	p	p	n	0	0	0	0	0
13	147000	n	n	98	23	1	n	1	38	19	61	5.8	3.2	p	p	n	0	0	0	0	0
12	105000	n	n	100	21	0.9	n	0.9	32	28	87	5.4	3	p	0	n	0	0	0	0	0
14	27000	1	n	98	28	0.9	ab	0.9	294	130	123	5	3.4	p	p	n	1	spleen,GB	1	rt pe	0
13	220000	n	n	96	30	1	n	0.9	36	30	112	5.6	3.2	p	p	n	0	0	0	0	0
13	22000	n	n	100	32	0,9	ab	1	106	98	110	5.6	3.4	p	p	n	1	hep	1	lt pe	0
12.8	320000	n	n	99	21	0.8	n	0.8	34	25	78	6	3	p	p	n	0	0	0	0	0
14	30000	n	n	80	23	0.9	ab	0.9	128	118	106	5.4	3.5	p	p	n	1	gb	1	b pe	1
14	42000	n	n	99	21	1	ab	1.1	130	120	115	5.6	3.5	p	p	n	1	gb	1	rt pe	0
12	180000	n	n	98	25	0.7	n	1	32	28	98	5.6	3.4	0	p	n	0	0	0	0	0
12	140000	n	n	74	29	0.6	n	0.8	28	25	87	5.4	3.4	0	p	n	0	0	0	0	0
13	140000	n	n	78	6	1	n	0.8	32	28	98	5.8	3.5	0	p	n	0	0	0	0	0
15	62000	n	n	98	38	0.7	ab	0.9	182	170	110	5	3.4	p	p	n	1	0	1	rt pe	0
14	114000	n	n	102	34	1	n	0.8	34	32	98	5.8	3.5	p	p	n	1	spleen	0	0	0

15	60000	n	n	80	19	1	ab	o.8	74	41	78	5.5	3.3	p	0	n	1	0	1	lt pe	0
13	28000	n	n	92	18	0.7	ab	1	130	140	112	5.8	3.4	p	p	n	1	0	1	rt pe	0
14	45000	n	n	92	32	0.7	ab	1	110	106	130	6.2	3.5	p	p	n	0	0	0	0	1
10	71000	n	n	83	26	0.7	n	0.8	32	23	89	5.3	3.8	p	p	n	1	spleen	0	0	0
11	120000	n	n	59	23	0.8	n	1	34	32	98	5.4	3	0	p	n	0	0	0	0	0
14	101000	n	n	64	36	0.8	n	1	38	28	108	5.8	3.2	p	p	n	0	0	0	0	0
12	109000	n	n	87	23	0.9	n	1	32	23	56	5.4	3.2	p	0	n	0	0	0	0	0
11	180000	n	n	96	40	0.9	n	0.7	28	22	94	5.8	3.2	p	0	n	0	0	0	0	0
13	80000	n	n	98	31	1	n	1	178	150	110	5.8	3.6	p	p	n	1	0	1	rt pe	0
11	80000	n	n	85	40	1	n	0.8	28	34	112	5.4	3.2	0	p	n	gall bladder wall	0	1	rt pe	0
11	83000	n	n	97	23	0.7	n	o.8	48	40	84	5.9	3.2	p	p	n	1	spleen	1	lt pe	0
10	59000	n	n	66	23	0.5	ab	0.8	126	118	178	6.1	4	p	0	n	1	hep,spleen	1	rt pe	0
11	80000	n	n	87	22	0.7	ab	1	89	44	87	5.4	3	p	0	n	1	spleen	1	rt pe	1
14	80000	n	n	98	24	0.8	n	1	26	19	57	6.3	3.5	p	p	n	0	0	0	0	0
13	130000	n	n	96	28	0.9	n	0.9	42	34	89	5.6	3.5	p	0	n	0	0	0	0	0
15	10000	1	1	55	41	1	ab	1	112	100	78	5	2.2	0	p	n	1	0	1	rt pe	1,shock
14	66000	n	n	68	32	1	ab	0.9	72	36	100	5.6	3.4	p	p	n	1	0	1	0	0
11	10000	n	n	86	32	0.9	n	1	43	34	98	5.4	3.5	p	p	n	1	0	1	0	0
13	134000	n	n	84	34	1	n	0.8	43	45	100	5.5	3.4	p	p	n	0	0	0	0	0
12	26000	n	n	77	29	0.9	ab	1	256	143	112	6	3.2	p	p	n	1	0	1	b pe	0
13	12000	n	n	96	20	0.8	n	0.8	34	32	98	5.4	3.2	p	p	n	1	hep	0	bpe	1,shock
12	210000	n	n	87	23	0.9	n	0.5	32	26	89	5.9	3.5	0	p	n	0	0	0	0	0
11	189000	n	n	89	32	0.8	n	0.6	25	23	87	5.7	3.4	0	p	n	0	0	0	0	0
12	110000	n	n	69	43	1	n	0.9	23	22	98	5.3	3.3	p	p	n	0	0	0	0	0
14	112000	n	n	78	32	1	n	0.8	24	34	78	5.4	3.2	p	0	n	0	0	0	0	0
12	60000	n	n	98	23	0.8	n	1	28	24	86	5.3	3	p	0	n	0	0	0	0	0
13	40000	n	n	84	23	0.6	n	0.8	32	28	78	5.9	3.5	p	0	n	1	0	1	rt pe	0
11	280000	n	n	78	25	0.8	n	0.9	43	34	98	6	3.7	p	0	n	0	0	0	0	0
12	256000	n	n	78	34	1	n	0.5	32	24	96	5.7	3.4	p	p	n	0	0	0	0	0
13	56000	n	n	160	43	1	n	1	44	32	55	5.3	3.2	p	p	n	1	0	1	rt pe	0
12	249000	n	n	126	34	1	n	0.4	33	26	87	6	3.8	0	p	n	0	0	0	0	0
11	156000	n	n	129	19	0.8	n	0.6	48	42	89	5.6	3.4	0	p	n	0	0	0	0	0
12	90000	n	n	112	32	0.8	n	0.7	32	25	89	5.4	3.2	p	p	n	1	0	1	rt pe	0

17.2	40000	n	n	96	29	0.4	ab	1	192	121	112	5.3	3	p	p	n	1	hep;spleen	1	0	1
12	120000	n	n	98	32	0.6	n	0.7	25	22	78	5.4	3.4	p	p	n	1	hep;spleen	0	0	0
12	40000	n	n	96	32	0.8	n	0.8	142	106	68	6	3.6	p	p	n	1	0	1	rt pe	0
14	158000	n	n	139	34	1	n	0.6	28	23	87	5.4	3.9	p	p	n	0	0	0	0	0
14	340000	n	n	154	36	0.8	n	0.4	28	23	89	5.8	3.6	p	p	n	0	0	0	0	0
11	105000	n	n	112	32	0.9	n	0.8	34	25	88	5.8	3.8	p	0	n	0	0	0	0	0
13	28000	n	n	76	34	0.8	n	0.9	110	103	88	6	3.4	p	0	n	1	0	1	lt pe	0
12	35000	n	n	113	32	0.7	n	0.8	24	22	98	5.4	3.6	p	p	n	0	0	0	0	0
15	55000	n	n	80	23	0.8	n	0.8	36	26	88	5.9	3.5	p	p	n	1	0	1	rt pe	0
13	320000	n	n	85	34	0.5	n	0.5	29	24	79	5.8	3,2	p	0	n	0	0	0	0	0
14	118000	n	n	112	32	0.6	n	0.5	32	25	88	5.6	3.4	0	p	n	0	0	0	0	0
13	150000	n	n	102	32	1	n	1	30	26	112	5.6	3	p	0	n	0	0	0	0	0
13.2	210000	n	n	80	20	0.7	n	1.1	46	33	76	5.5	3.7	n	p	n	1	hep;spleen	0	0	0
13	280000	n	n	122	40	1	ab	1.1	88	45	104	6	4	p	0	n	1	hep	0	0	0
11	10000	n	n	86	32	0.9	n	1	43	34	98	5.4	3.5	p	p	n	1	0	0	rt pe	0
15	62000	n	n	98	38	0.7	ab	0.9	182	170	110	5	3.4	p	p	n	1	0	1	rt pe	0
13	28000	n	n	76	34	0.8	n	0.9	110	103	88	6	3.4	p	0	n	0	0	0	0	0
13	220000	n	n	96	30	1	n	0.9	36	30	112	5.6	3.2	p	p	n	0	0	0	0	0
13	28000	n	n	92	18	0.7	ab	1	130	140	112	5.8	3.4	p	p	n	1	0	1	rt pe	0
11	130000	n	n	72	21	1.1	n	1	36	40	67	6.1	3.4	p	n	n	1	0	1	lt pe	0
13	110000	n	n	112	30	1	n	0.8	30	28	98	5.9	3.5	p	p	n	0	0	0	0	0
13	130000	n	n	96	28	0.9	n	0.9	42	34	89	5.6	3.5	p	0	n	0	0	0	0	0
14	18000	n	n	90	20	0.9	ab	1.2	240	153	115	6.1	4	p	p	n	1	0	1	rt pe	1
13	280000	n	n	122	40	1	ab	1.1	88	45	104	6	4	p	0	n	1	hep	0	0	0

tachycardia	↑HCT	Plasma leak	Fever	Thrombocytopenia	Plasma leak	Hges	Bp	Infection Type	Dengue	Blood trans	Platelet trans.	Comments
1	1	1	1	1	1	1	1	1	DHF-3	0	0	recoverd in 3days
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	antenatal 6months
0	1	1	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	1	0	0	0	1	DF,T	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
1	0	1	1	1	1	1	1	2	DHF-3	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
1	1	1	1	1	1	1	0	2	DHF-2	0	1	4uplatelet,3uffp transfused
0	0	0	1	1	0	0	0	2	DF,T	0	0	0
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0
0	1	1	1	1	1	1	0	2	DHF-2	0	0	0
1	0	1	1	1	1	1	1	1	DHF-3	1	1	
0	0	0	1	0	0	0	0	1	DF,T	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	1	1	1	1	1	0	2	DHF-1	0	0	0
1	0	1	1	1	1	1	1	2	DHF-3		5unit platelet	
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	
0	1	1	1	1	1	1	0	2	DHF-2	0	0	
0	0	0	1	0	0	0	0	2	DF	0	0	
0	0	0	1	0	0	0	0	2	DF	0	0	
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	0	0	1	1	1	1	0	2	DHF-2			
0	0	0	1	0	0	0	0	1	DF	0	0	0

0	0	1	1	1	1	1	0	2	DHF-2	0	0	
0	0	1	1	1	1	1	0	2	DHF-1	0	0	0
0	0	0	1	1	1	1	0	2	DHF-2	0	0	
0	0	0	1	1	1	1	0	2	DHF-2	0	0	0
0	0	1	1	1	1	1	0	1	DHF-2	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	1	1	1	1	1	1	0	2	DHF-2	0	1	4units platelets
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	Df	0	0	
1	1	1	1	1	1	1	1	2	DHF-3	0		in fluid mangaement
0	0	o	1	0	0	0	0	1	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0
1	0	1	1	1	1	1	1	2	DHF-3	4ffp, 2 bllod	2platelet	1u blood,2 uplatelet
0	0	0	1	0	0	0	0	2	DF,encheplalitis			
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	1	1	1	1	1	1	0	2	DHF-2	0	4plate	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
1	0	1	1	1	1	1	1	2	DSS	0	4platelet	ivfliud
1	0	1	1	1	1	1	1	2	DHF-3	0	0	iv fliud
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	Df	0	0	0
0	1	1	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0

0	0	1	1	1	1	1	0	2	DHF-2			
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0
1	0	1	1	1	1	1	1	2	DHF-2	0	0	0
0	0	0	1	1	0	0	0	2	DF,T	0	0	0
0	0	0	1	0	0	0	0	2	DF,encheplalitis	o	o	recovered 2days
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	1	1	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	1	1	1	0	2	DHF-2	0	0	0
0	0	1	1	1	1	1	0	1	DHF-2	0	0	0
1	1	1	1	1	0	0	0	1	DF,T	0	0	0
0	0	0	1	1	0	0	0	2	DF,T	0	0	0
0	0	0	1	0	0	0	0	1	DF,encheplalitis	0	0	encephalopathy
1	1	1	1	1	1	1	1	2	DSS,Death	4ffp, 2 bllod	8platelet	irrestable shoick
0	0	0	1	1	1	1	0	2	DHF-2	0	0	0
0	0	1	1	1	1	1	0	2	DHF-1	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0
1	0	1	1	1	1	1	1	2	DSS	0	4platelet	iv fluid reco in 1day
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	1	Df	0	0	0
0	0	0	1	1	0	0	0	1	DF,T	0	0	0
0	0	1	1	1	1	1	0	1	DHF-2	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0

1	1	1	1	1	1	1	1	2	DHF-3	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	0	0	1	1	1	1	0	1	DHF-2	0	0	0
0	0	0	1	1	0	0	0	2	DF,T	0	0	0
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
0	0	1	1	1	1	1	0	2	DHF-1	0	0	0
0	1	1	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	1	0	0	0	1	DF,T	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	1	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	1	1	1	0	2	DHF-2	0	0	0
0	0	0	1	0	0	0	0	2	DF	0	0	0
0	0	0	1	0	0	0	0	1	DF	0	0	0
1	0	1	1	1	1	1	1	2	DHF-3		5unit platelet	
0	0	0	1	0	0	0	0	1	DF	0	0	0