A STUDY ON OUTCOME OF CHILDREN WITH DENGUE INFECTION AND ITS CLINICAL CORELATION WITH HEPATIC DYSFUNCTION IN GMKMCH

Dissertation submitted to

THE TAMILNADU DR. M. G. R. MEDICAL UNIVERSITY

in partial fulfillment of the regulations
for the award of

M.D.DEGREE IN PAEDIATRIC MEDICINE

BRANCH VII

GOVERNMENT MOHAN KUMARAMANGALAM

MEDICAL COLLEGE, SALEM.

APRIL 2016

CERTIFICATE BY THE GUIDE

This is to certify that this the dissertation titled **"A STUDY ON OUTCOME OF CHILDREN WITH DENGUE INFECTION AND ITS CLINICAL CORELATION WITH HEPATIC DYSFUNCTION IN GMKMCH"** submitted by **Dr.J.Anita Chrisbina** to the faculty of Paediatric Medicine, The Tamil Nadu **Dr.M.G.R.Medical University**, Chennai in partial fulfillment of the requirement for the award of **MD Degree Branch VII (Paediatric Medicine)**, is a bonafide research work carried out by her under my direct supervision and guidance at Government Mohan Kumaramangalam Medical College, Salem during the academic year 2014-2016.

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ENDORSEMENT

BY THE HEAD OF THE DEPARTMENT

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DECLARATION

DECLARATION BY THE CANDIDATE

I here declare that this dissertation entitled **"A STUDY ON OUTCOME OF CHILDREN WITH DENGUE INFECTION AND ITS CLINICAL CORELATION WITH HEPATIC DYSFUNCTION IN GMKMCH"** is a bonafide and genuine research work carried out by me under the guidance of **Dr.T.SUNDARARAJAN M.D.,D.C.H., Professor & HOD** Department of Paediatric Medicine,, Government Mohan Kumaramangalam Medical College, Salem.

I have not submitted this previously to this university or any other university for the award of any degree or diploma

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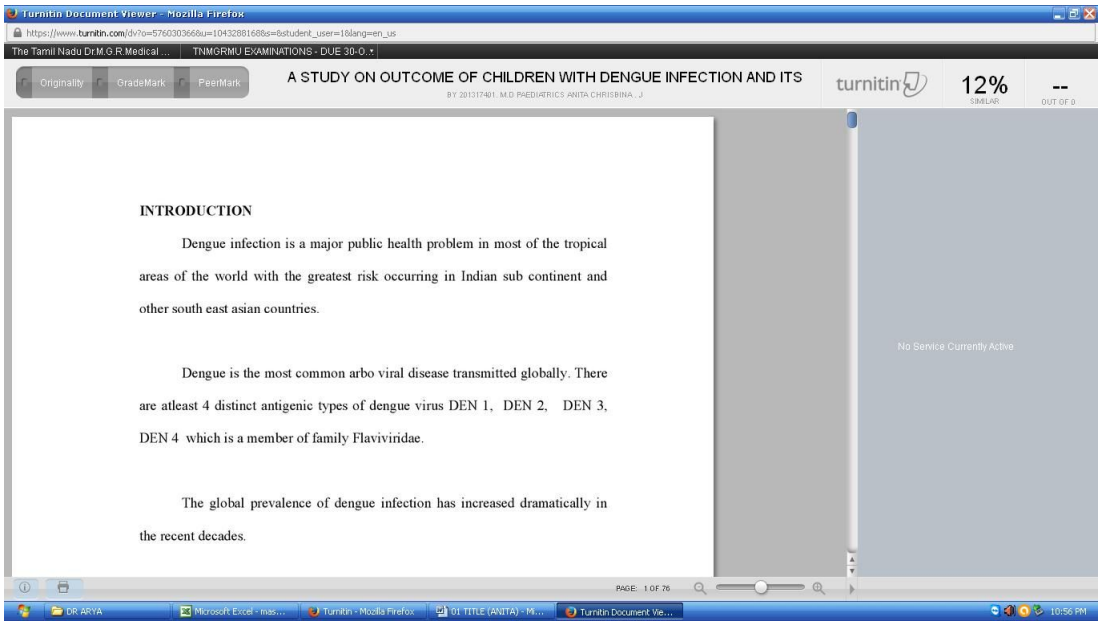
Grade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INTRODUCTION
Dengue infection is a major public health problem in most of the tropical areas of the world with the greatest risk occurring in Indian sub continent and other south east asian countries.
Dengue is the most common arbo viral disease transmitted globally. There are atleast 4 distinct antigenic types of dengue virus DEN 1, DEN 2, DEN 3, DEN 4 which is a member of family Flaviviridae.
The global prevalence of dengue infection has increased dramatically in the recent decades.
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Dated: 02.2015

Ethical committee meeting held on 08.01.2015 at 11.00 A.M in the seminar hall, IInd Floor, Medicine Block, Govt.Mohan Kumaramangalam Medical College Hospital, Salem 01

The following members were attended the meeting

MEMBERS:

1. Dr.N. Mohan, MS., FICS. FMMC. Dean, Govt.Mohan Kumaramangalam Medical College Hospital, Salem.
2. Dr.A.P.Ramasamy, MD., Chairman, ECRB, External Clinician.
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Sl.No	Name of the Presenter with Address	Title	Name of the Guide and Address	Whether it is approved or Not
1	Dr. J.Anita Chrisbina, III Year MD., P.G. Student, GMKMC., Salem-30.	A Study on outcome of Children with Dengue infection and its Clinical Corelation Hepatic Dysfunction in GMKMCH	Dr.T.Sundararajan MD., D.C.H. Professor & HOD Paediatrics Department GMKMC, Salem.	Approved

The ethical committee examined the studies in detail and is pleased to accord ethical committee approval for the above Post Graduate of this college to carry out the studies with the following conditions.

1. She should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to government.
2. She should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She should not deviate from the area of the work which applied for ethical clearance.
4. She should inform the IEC immediately, in case of any adverse events or serious adverse reactions.
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7. She should submit the summary of the work to the Ethical Committee on completion of the work.
8. She should not claim any funds from the institution while doing the work or on completion.
9. She should understand that the members of IEC have the right to monitor the worker with prior intimation.

Dr. R..RAVICHANDRAN M.S., MCH.,
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Salem.

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ABSTRACT

ABSTRACT :

A STUDY ON OUTCOME OF CHILDREN WITH DENGUE INFECTION AND ITS CLINICAL CORELATION WITH HEPATIC DYSFUNCTION IN GMKMCH

Dengue virus infection is a Major Public Health problem with upsurge in complicated and atypical manifestations in the recent years. Hepatic involvement in Dengue is known with protean of manifestations ranging from hepatomegaly elevated liver enzymes to fulminant hepatic failure.

OBJECTIVE :

To study hepatic dysfunction in childhood dengue infection.

To study clinical co-relation lilke severity, Clilnical Features, other lab parameters, morbidity and mortality.

STUDY DESIGN :

Prospective Cohort Study

SETTINGS :

GMKMCH , SALEM

SAMPLE SIZE :

100 Patients hospitalized with Dengue infection (Sero Positive for Dengue)

METHODOLOGY :

Dengue Sero positive patients are selected and examined for Hepatomegaly and Jaundice and subjected to complete blod count, Liver function tests, ultrasound abdomen, PT, APTT, HBsAg, HCV, Widal and analysed.

SIGNIFICANCE OF THE STUDY :

- The degree of Hepatic Dysfuction in dengue could be identified early and the severity of the disease could be assessed.
- Early interventions could prevent life threatening complications like massive Haemorrhage.
- The Role of Hepato-protective drugs in dengue could be tried for early recovery and there by decreasing morbidity and mortality in future studies.

ETHICAL CONSIDERATIONS :

Informed written consent is obtained from parents of patients.

CONCLUSION

In developing country like India, incidence of Dengue outbreaks ins increasing Hepatic Involvement of varying degrees have been reported. As hepatic dysfunction in Dengue is transient and reversible, early identification of the same should help to reduce life threatening complications. This can help to reduce the Morbidity and Morality due to Dengue infection. The Role of Hepato Protective Drugs in reducing morbidity and mortality should be analysed by further studies.

INTRODUCTION

INTRODUCTION

Dengue infection is a major public health problem in most of the tropical areas of the world with the greatest risk occurring in Indian sub continent and other south east asian countries.

Dengue is the most common arbo viral disease transmitted globally. There are atleast 4 distinct antigenic types of dengue virus DEN 1, DEN 2, DEN 3, DEN 4 which is a member of family Flaviviridae.

The global prevalence of dengue infection has increased dramatically in the recent decades.

Factors for dengue's spread include uncontrolled population growth, uncontrolled urbanization, overcrowding, inadequate health facilities, increased travel to epidemic areas, poor vector control, climate change and lack of awareness among people.

Dengue infections are known to present with a diverse clinical spectrum, ranging from asymptomatic illness to fatal outcome. Unusual manifestations have become more common .These include encephalitis, Guillian-Barre Syndrome, dengue hepatitis, myocarditis and acute respiratory distress syndrome.

Hepatic dysfunction varies from mild injury with elevation of transaminase activity, hepatomegaly to severe damage with jaundice and

fulminant hepatic failure. The severity of hepatic dysfunction varies with clinical presentation.

The cause for hepatic dysfunction may be due to inadequate perfusion, metabolic acidosis and disseminated intravascular coagulation.This in turn leads to ischaemia causing severe hepatic dysfunction.

Presence of fever, jaundice, hepatomegaly in endemic areas should arouse the suspicion of dengue hepatitis. Awareness of these manifestations of hepatic involvement in dengue may be helpful in arriving at early diagnosis and avoiding morbidity and mortality.

However only less number of studies have been reported regarding hepatic dysfunction in dengue. Hence it is with this objective this present study has been undertaken.

OBJECTIVE OF THE STUDY

1. To study the hepatic dysfunction in children with dengue infection

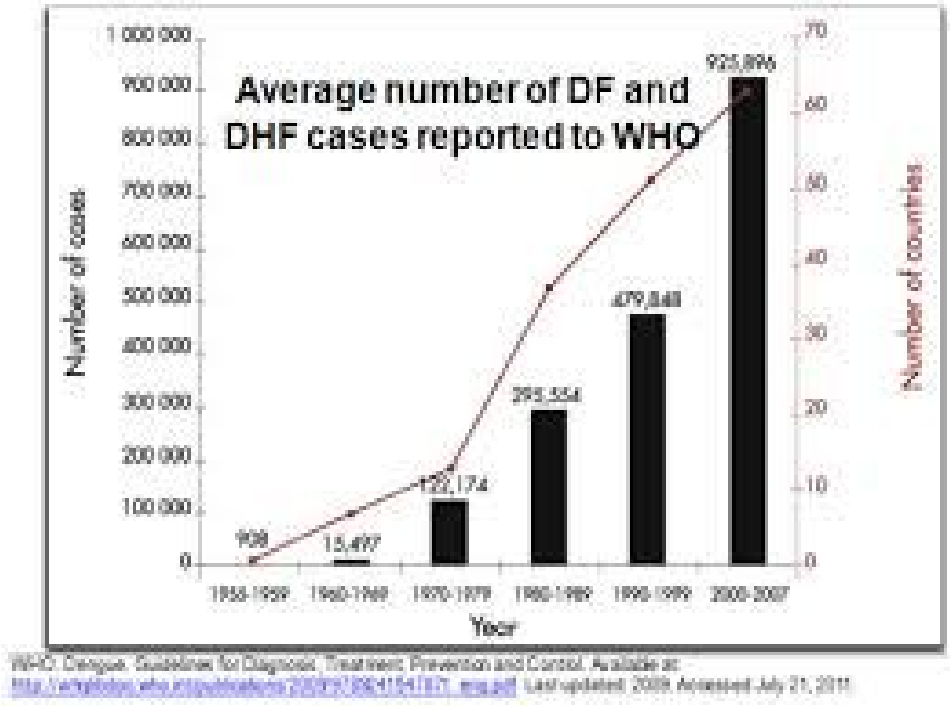
2. To study the clinical correlates like clinical features, laboratory parametres, morbidity and mortality.

BURDEN OF DISEASE:

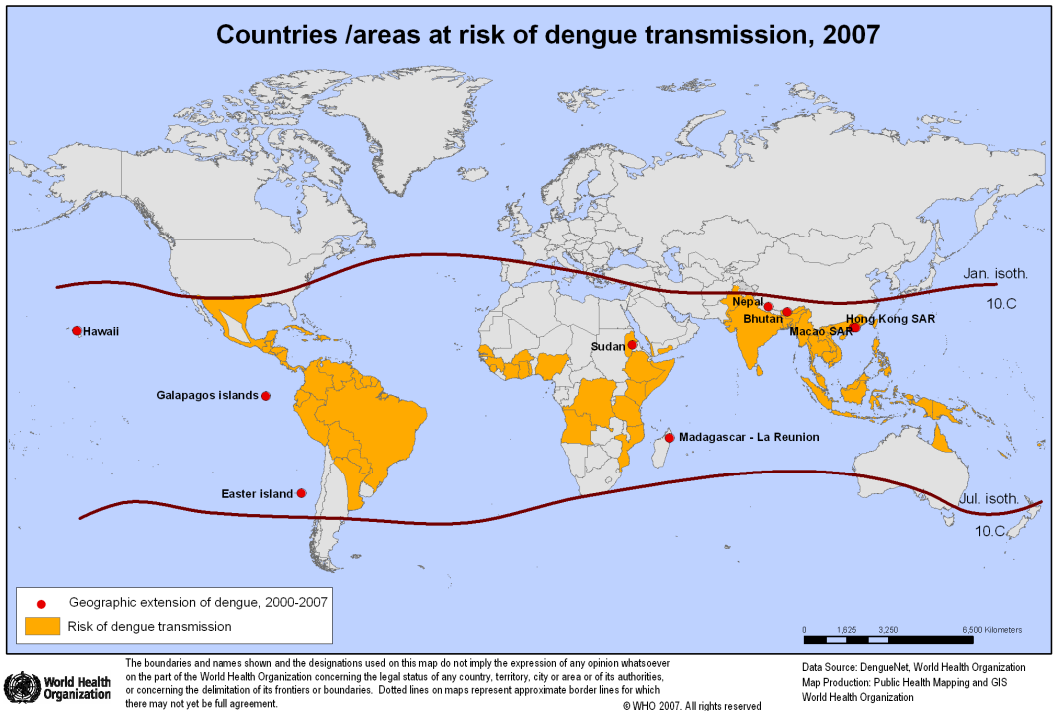
The first epidemic of dengue occurred in 1635 in the French westindies.

Prior to 1970, only 9 countries has experienced dengue hemorrhagic fever; since then the number has increased to more than 4 fold. The disease is now endemic in many countries. An estimated 2.5 billion people in about 100 countries are at risk of acquiring dengue viral infection with more than 50 million new infections, 20000 to 25000 deaths, mainly in children[4].

The number of cases reported annually to WHO ranged from 0.3 – 1.3millian in from 1996 – 2005. Misdiagnosis and under reporting are major obstracles in knowing the full burden.[3]



Average annual number of dengue cases reported to WHO and of countries reporting (1955-2007)



Since 2000, epidemic dengue has spread to new areas and has increased in already affected regions. In 2003 eight countries – India, Bangladesh, Maldives, Indonesia, Myanmar, Sri Lanka, Timor-Leste and Thailand reported Dengue cases.

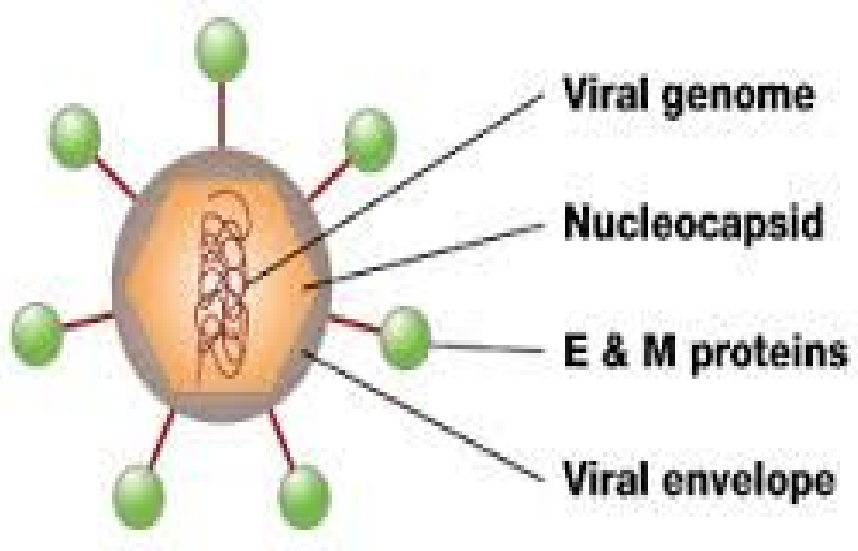
Reported case fatality rate in these regions is approximately is 1%. But due to focal outbreaks away from urban areas, India, Indonesia, and Myanmar have reported case fatality rates of 3-5%.

REVIEW OF LITERATURE:

- Study by Ole Wichmann et al[19] found that 43% of dengue fever & dengue hemorrhagic fever and 70% of dengue shock syndrome patients had hepatomegaly.
- Study by Nimmannitya et al[12] concluded that elevated aspartate transaminase levels are seenfrom day3 of illness, reachespeak by 1 week and returns to normal by 3-8 weeks.
- Study by Brijmohan et al [16]and Srivenuitha et al[21]found higher incidence of elevated liver enzymes are found in dendue shock syndrome than dengue fever & dengue hemorrhagic fever.
- Study by MMAFardi et al[22]found that elevatedalkaline phosphatase are found in 35.3% of dengue cases.
- Study by Luiz Jose de Souza [23]et al observed 90 % of cases with hepatomegaly.
- Study by DinhTheTrung et al[24] concluded that transaminase levels began to rise from day one and peaked by 2nd week.
- Most of the studies showed rise of aspartate transaminase was more than alanine transaminase.
- Study by M Narayanan et al[20] found rise in alanine transaminase in 76.1 % of cases.
- Manzhiwong et al[21] reported incidence of hypoalbuminemia in 16.53%

THE VIRUS:[5-7]

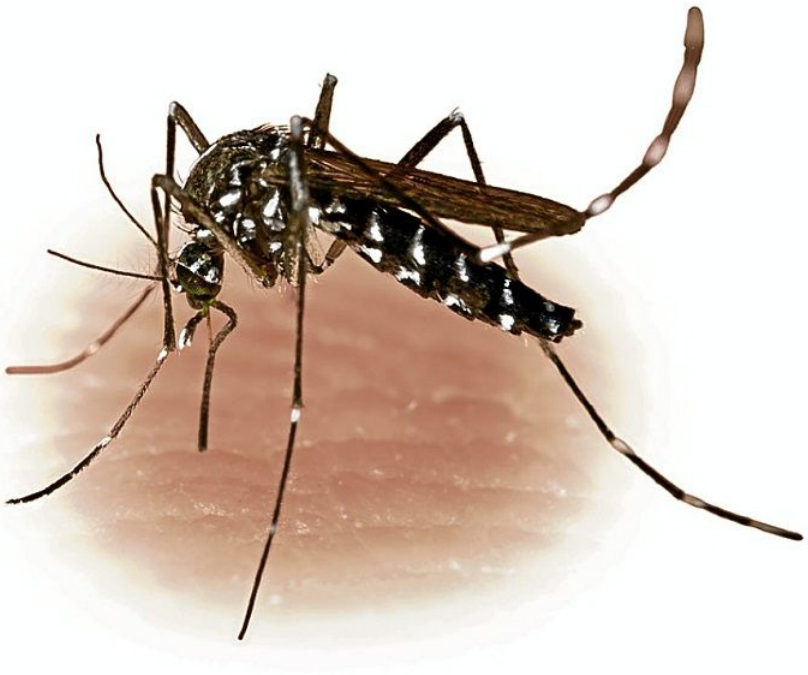
Denguevirus, a mosquito-borne flavivirus, is the causative agent, currently one of the most significant emerging disease challenges to global health. The dengue virus are single-stranded positive-sense RNA viruses with a genome of about 11000 bases that codes for three structural proteins, C(core protein), M (membrane protein), E (envelope protein); 7 nonstructural proteins, NS1, NS2a, NS2b, NS3, NS4a, NS4b, NS5; and short non-coding regions on the 5' and 3' ends. The 4 serotypes are DEN 1, DEN 2, DEN 3, DEN 4. All these 4 serotypes cause dengue epidemics. Asian serotypes DEN-2 and DEN-3 are associated with severe dengue



THE VECTOR:[4, 8, 9]

Aedes aegypti is a mosquito that can spread Dengue, Chikungunya yellow fever and other diseases. The mosquito is a small, dark of approximately 4 to 7 mm with typical white markings on the legs and thorax. Females are larger than males .

Aedes aegypti is a day biting mosquito. The mosquito is most active during daylight, for 2 to 3 hours after sunrise and several hours before sunset. The mosquito rests indoors, in dark places. Outside, they rest in cool shaded areas. The males do not bite humans or animals and they live on fruit. The females feed on fruit and also on blood. When seen under a microscope, male mouth parts are modified for nectar feeding, and female mouth parts are modified for blood feeding. Feeding on humans generally occurs at one to two hour intervals.



The Vector - Aedes aegypti

The mosquito attacks generally from underneath desks or chairs at the feet and ankles. Aedes aegypti lays its eggs in clean water. Aedes aegypti is highly domesticated, adapted well to human habitation. Spread increases with increasing humidity and temperature.

In dengue infected mosquito's, the virus is present in the salivary glands after 8 to 10 days of extrinsic incubation period .When a female bites a human for food, it injects saliva into the wound. It also injects the dengue virus into the host.

The mosquito is now present globally in tropical and sub-tropical regions. The mosquito has cosmo-tropical distribution annually, and spreads to temperate regions in summer. The insect is very fast in flight.

THE HOST:[4, 8, 9]

Several species of lower primates and humans are affected. Humans are the main reservoirs. Primary infection may provide life long immunity for that serotype. Young children may not be able to compensate for plasma leakage in adults, leading to frequent complications. The symptoms appear after an incubation period of 4 to 10 days.

Dengue affected child

PATHOPHYSIOLOGY: [9]

The main pathophysiological factor that decides the severity is plasma leakage and abnormal hemostasis. Exact cause for plasma leakage is not known. However complete recovery without any damage to blood vessels suggests that, the cause may be due to release of biological mediators.

It is observed that second infection with any of 4 serotypes causes severe dengue. The probable explanation is the serotype cross react with antibodies generated from primary infection. They bind with the virions without neutralizing them. These antiboby coated virionsare taken up rapidly by antigen presenting cells and further activation of t cells. The cytokines released by activated T cells causes most of the catastrophic events in dengue. These cytokines includes tumour necrosis factor, interferons and interleukins . Further endothelial dysfunction increases capillary permeability causing microvacular leakage and circulatory insufficiency.

Thrombocytopenia in dengue is due to molecular mimicry between viral proteins and platelet surface proteins. Blood fibrinolytic pathways are also activated. The associated liver injury and thrombocytopenia causes bleeding manifestations. Central nervous system may also be involved due to direct effect of the virus. Hepatic involvement includes diffuse hepatitis, steatosis and areas of focal necrosis.

Recently, the endothelial cell dysfunction, that occurs in Dengue is the main cause for plasma leakage. This is due to functional rather than structural damage.

CLINICAL MANIFESTATIONS:[10]

Incubation period of denue varies from 4 to 10 days. Most are sub clinical. Typical presentation is seen usually in older children. Presents in 3 phases:

(1) FEBRILE PHASE:

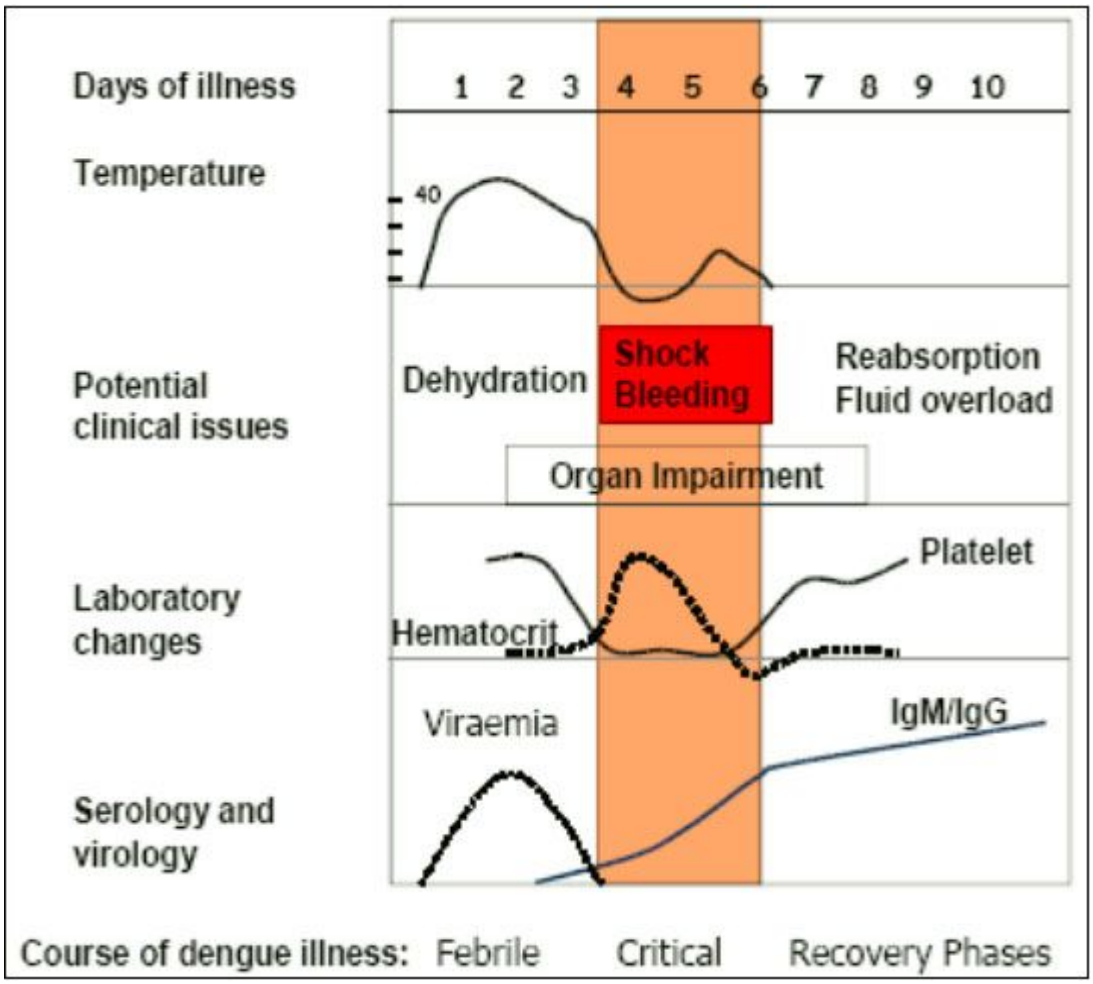
Characerised by

- high grade fever, usually lasts for 2-10 days.
- myalgia
- arthralgia
- generalized body ache
- skin erythema
- facial flusing
- anorexia
- nausea
- headache
- vomiting
- conjuctival congestion

- Positive tourniquet test and minor bleeding manifetations like mucosal bleed and petechiae may be seen.
- Hepatomegaly itself indicates severity of the disease. Platelet begins to fall.

(2) CRITICAL PHASE:

Occurs between 3 to 7 days of onset of fever, during the period of defervescence. Significant plasma leakage occurs. Chest X ray may show pleural effusion. Ultra-sound may show ascites, hepatomegaly ang gall bladder wall edema. Packed cell volume increases and platelet continues to fall. Organ dysfunction and circulatory failure occurs during this phase. Circulatory failure occurs when critical volume of plasma leaks out. But this is often preceded by warning signs.



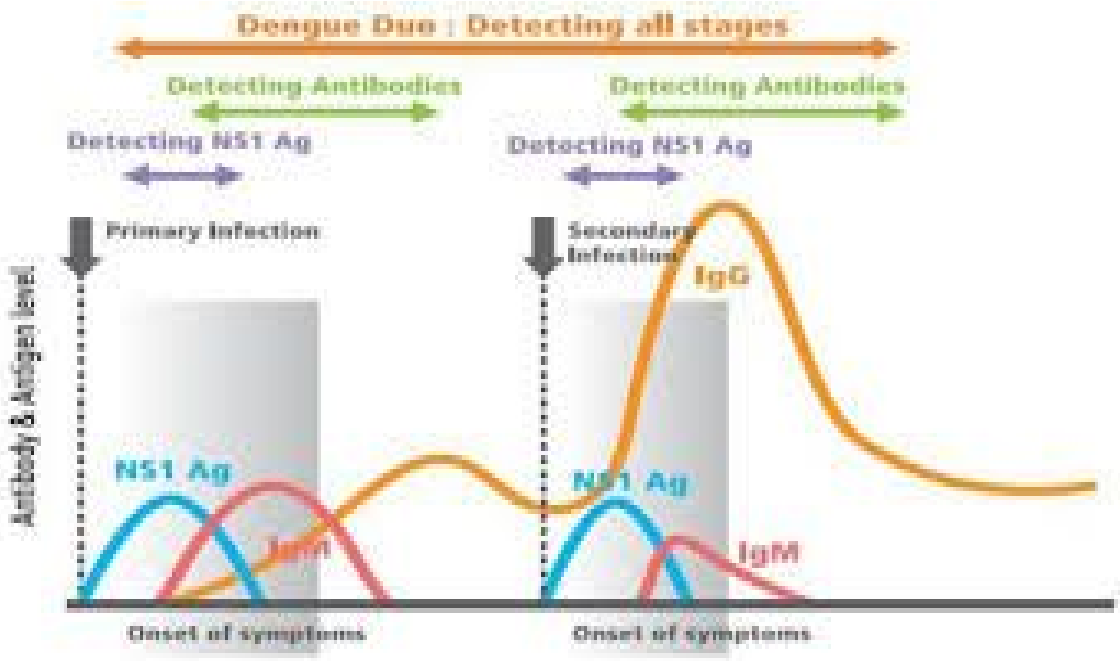
(3)RECOVERY PHASE:

After 24 to 48 hours of critical phase, recovery phase sets in. Gradual reabsorption of fluid from extravascular compartment to intravascular compartment occurs. Appetite improves. Respiratory distress may occur due to pulmonary edema. Leucocyte and platelet count begins to rise. Hemodynamic stability sets in and diuresis occurs. Some have rash of " islets of white in the sea of red " . Electrocardiac changes and bradycardia are common. Hematocrit may br normal or reduced due to fluid reabsorption.

LABORATORY DIAGNOSIS:[4]

Diagnosis may be established by

- Detection of virus
- Detection of antibodies against the virus



DETECTION OF VIRUS:

Isolation of virus by culture is a definitive diagnostic test, but is not practical. The period for dengue virus isolation is very short. After one to two days of defervescence, antibody titer begins to rise .This interferes with viral culture. Further the equipments are of high cost and difficult to maintain.

Viral RNA's are successfully detected using Reverse Transcriptase Polymerase Chain Reaction (RT-PCR) in several laboratories. It is faster and not much expensive. But the disadvantage is high false positivity due to viral

contamination.-dengue antigen may be also detected using in situ hybridization or immunocytochemistry. The latter is simple and used commonly .it has same specificity as viral isolation.

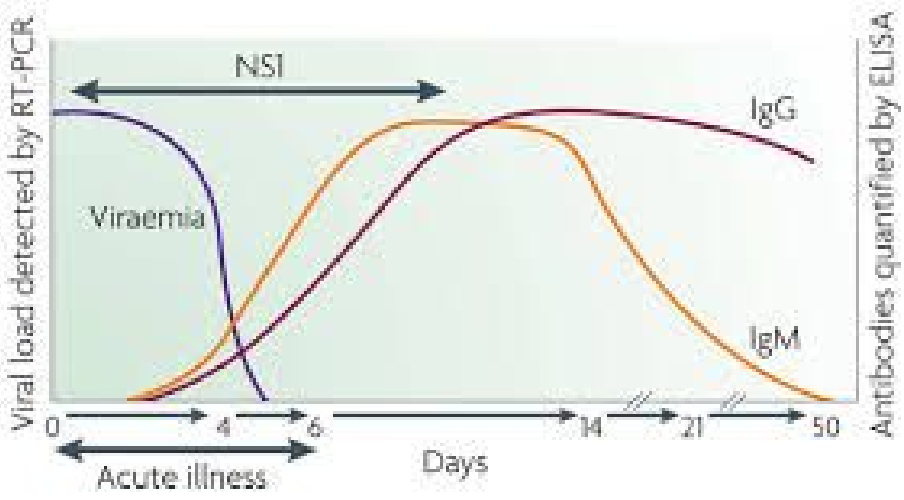
DETECTION OF ANTIBODY:

Serological diagnosis is simple. But there is high rate of false positive results.

MAC – ELISA:

It can measure the rising IgM dengue specific antibobytitre.

PRIMARY AND SECONDARY IMMUNOLOGIAL RESPONSE IN DENGUE INFECTION:



ATYPICAL MANIFESTATIONS OF DENGUE INFECTION[11]

NEUROLOGICAL:

- Encephalitis
- Encephalopathy
- Aseptic meningitis
- Guillianbarre syndrome
- Polyneuropathy
- Myelitis

GASTRO INTESTINAL:

- Hepatitis
- Acute pancreatitis
- Cholecstitis
- Diarrhea
- Parotisis
- Fulminant hepatic failure

RENAL:

- Acute renal failure

CARDIAC:

- Pericarditis
- Myocarditis
- Conduction abnormalities

RESPIRATORY:

- Pulmonary hemorrhage
- Acute respiratory distress syndrome

LYMPHO RETICULAR:

- Spontaneous spleen rupture

MUSCULO SKELETAL:

- Rhabdomyolysis
- Myositis

NEW WHO CLASSIFICATION OF DENGUE:

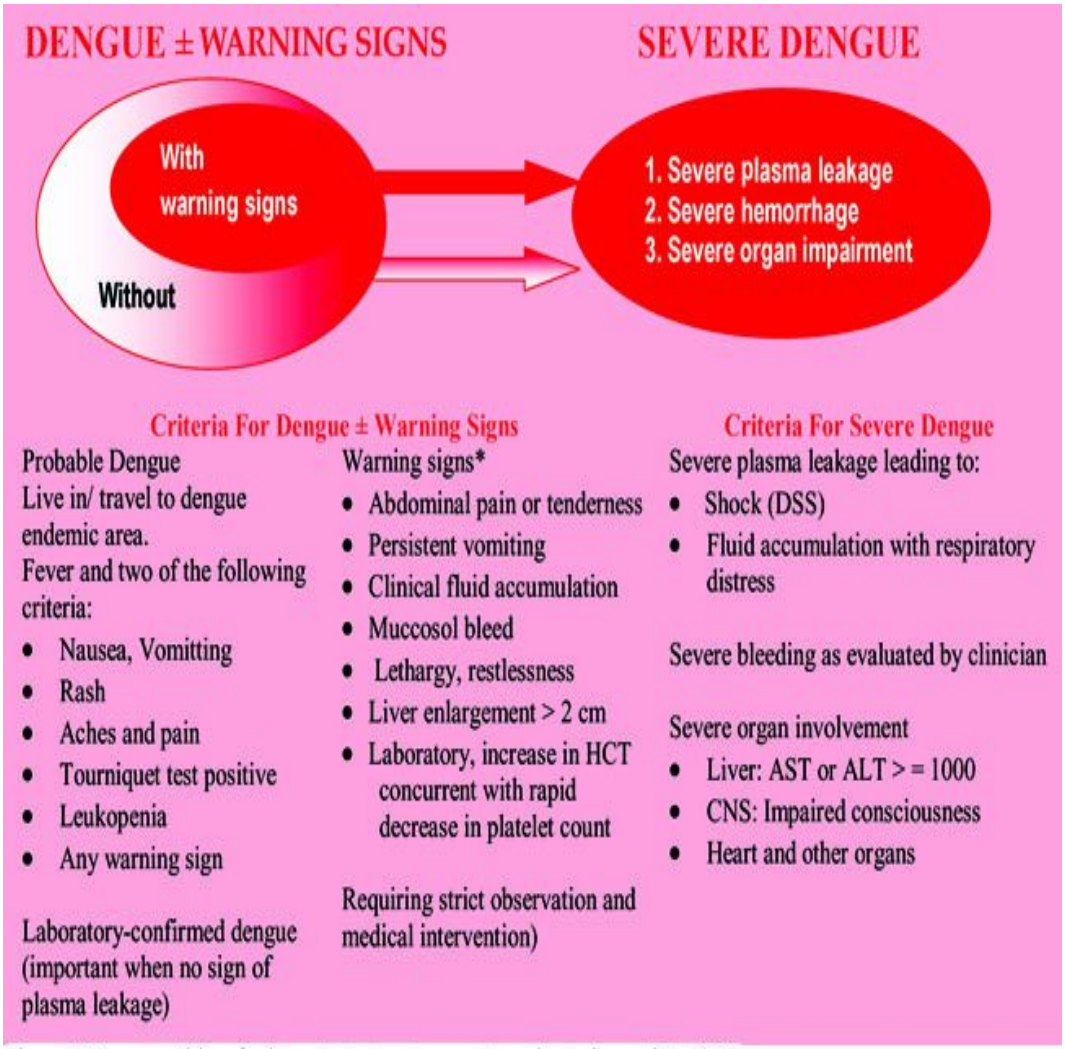


Figure-1: Dengue, guidelines for diagnosis, treatment, prevention and control new edition 2009.

WHO CLASSIFICATION OF DENGUE:[10]

The new WHO classification for dengue severity is divided into

- Probable dengue
- Dengue with Warning Signs
- Severe Dengue.

Dengue without Warning Signsor probable dengue:

Fever and two of the following after laboratory conformation

- Nausea
- Vomiting
- Rash
- Aches and pains
- Leukopenia
- Positive tourniquet test
- Any of warning signs

Dengue with Warning Signs**

Dengue as defined above with any of the following:

- Abdominal pain or tenderness
- Clinical fluid accumulation (ascites, pleural effusion)
- Mucosal bleeding
- Persistent vomiting
- Lethargy

- Restlessness
- Liver enlargement >2 cm
- Laboratory: increase in HCT concurrent with rapid decrease in platelet count

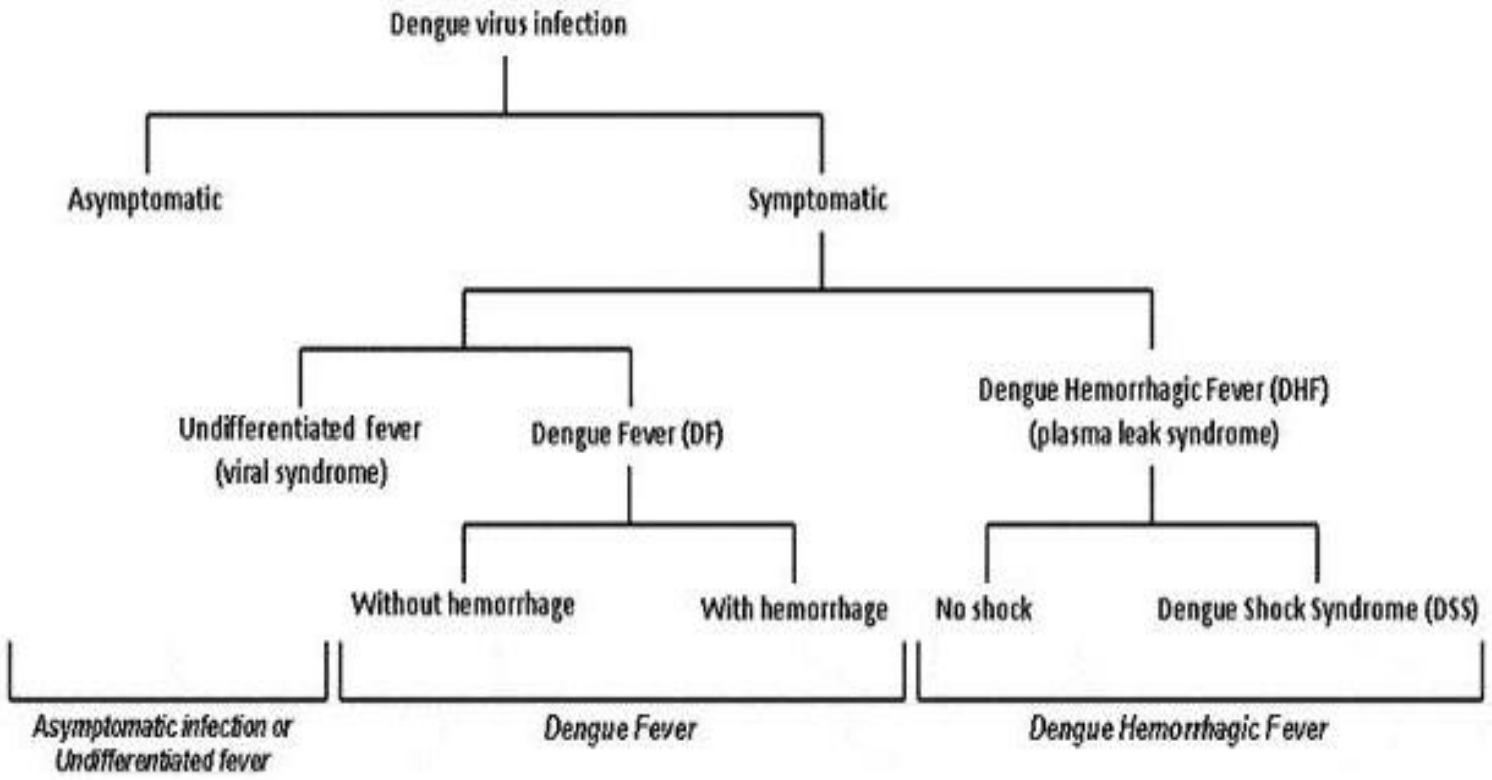
**requires strict observation and medical intervention

Severe Dengue:

Dengue with at least one of the following criteria:

- Severe Plasma Leakage leading to Shock
- Fluid accumulation causing respiratory distress
- Severe bleeding as evaluated by clinician
- Severe organ damage
  - Liver: Aspartate transaminase or Alanine transaminase ≥ 1000
  - Central nervous system: impaired consciousness
  - Failure of heart and other organs

OLDER CLASSIFICATION



*Adapted from *Dengue Haemorrhagic Fever: Diagnosis, Treatment, Prevention and Control. 2nd edition.* WHO, Geneva, 1997

HEPATIC INVOLVEMENT IN DENGUE:

- ✓ Hepatic involvement[11] in dengue is known with protean of manifestations ranging from
  - hepatomegaly
  - righthypochondrial tenderness
  - acute hepatitis
  - jaundice
  - elevation of liver enzymes
  - acute liver failure

- ✓ Histo pathological features [11]in dengue includes
  - central lobular necrosis
  - kupfer cell hyperplasia
  - Monocyte infiltration in portal tracts
  - Fatty changes

- ✓ Jaundice in dengue [11]may be due to
  - direct damage to liver by virus
  - hypoxic damage due to impaired perfusion

- ✓ Acute liver failure may lead to encephalopathy and life threatening hemmorhage. Elevation of liver enzymes may be an early marker to prevent these complications.

- ✓ Liver cells may be damaged by these mechanisms:[13]
  - effect of host immune reaction against virus infected cells
  - virus causing direct damage by cytopathic effect
  - impaired perfusion causing hypoxic damage

- ✓ Among the liver enzymes, aspartate transaminase was elevated more than alanine transaminase. This may be due to release of aspartate transaminase from damaged monocytes. The increase in liver enzymes has direct co-relation with the disease severity.

- ✓ Hepatic involvement usually prolongs the disease course of dengue.[12]

STEP WISE APPROACH TO DENGUE:[10]

HISTORY TAKING:

- Onset and duration of fever
- Assessment for warning signs
- Assessment of urine output
- History of travel to endemic areas
- Presence of co morbid conditions

EXAMINATION:

- Mental status
- Hydration status
- Hemodynamic status
- Look for breathlessness
- Look for abdominal tenderness, ascitis
- Look for rash and other bleeding manifestation
- tourniquet test

INVESTIGATIONS:

- IgM dengue positive
- Complete blood count
- Liver function test:

-SBR Total

- Direct

- Indirect

-SGOT

-SGPT

-ALP

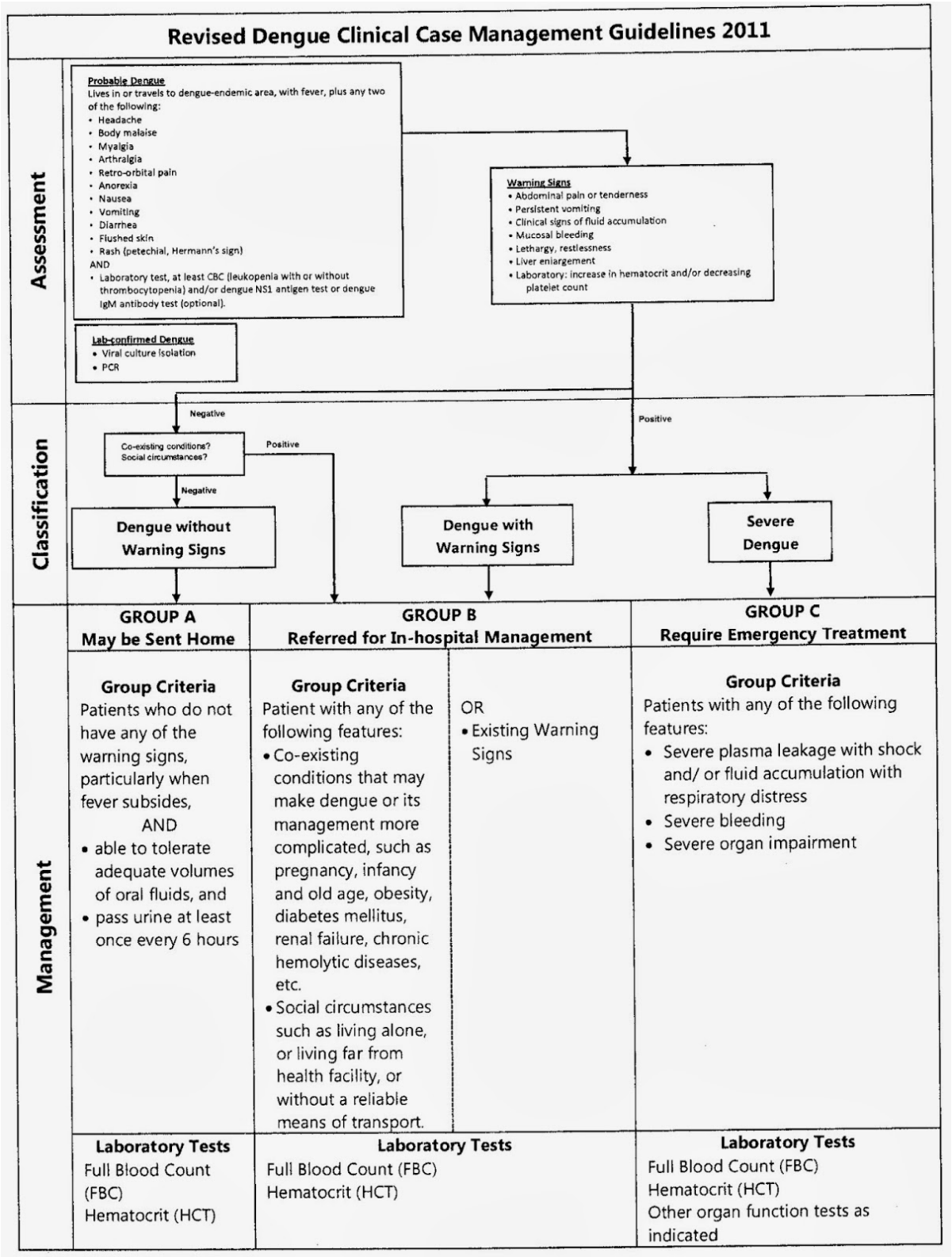
-S.protein

-Albumin

-Globulin

- PSS for Malarial parasite
- IgM anti hav
- HBsAg
- HCV
- Widal
- MAT for leptospirosis

MANAGEMENT PROTOCOL:[10]



MANAGEMENT PROTOCOL:[10]

Depending on the above findings, patients are classified into three groups A, B and C .

GROUP A: HOME MANAGEMENT

These patients tolerate adequate oral feeds, have adequate urine output, without any warning signs . These patients should be reviewed daily until they cross the critical phase. Warning signs should be explained to them and should be advised to report to the hospital immediately if they come across any of them.

TREATMENT:

- Advise adequate oral intake - ORS, fruit juice
- Paracetamol and tepid sponging to control fever

GROUP B: HOSPITAL MANAGEMENT:

These patients presents with warning signs and they require strict monitoring .

TREATMENT:

- Hematocrit should be obtained immediately .
- I.V Fluids at 5-10 ml/kg/hr to maintain adequate perfusion and urine output of 0.5ml/kg/hr. the inrta venous fluids are usually required for a period of 24 to 48 hours.

GROUP C: EMERGENCY MANAGEMENT:

This group includes patients with features of

- Severe plasma leakage and/or respiratory distress due to fluid accumulation
- Severe bleeding manifestations
- Severe organ impairment

TREATMENT:

These patients are classified as those with compensated shock and with decompensated shock

COMPENSATED SHOCK:

IV fluids with isotonic crystalloids at 5-10ml/kg/hr over one hour and then reassessed

-

PATIENT IMPROVES:

Reduce the fluids gradually to 5-7 ml/kg/hr for 1 to 2 hours and then to 3-5ml/kg/hr for 2 to 4 hours and then to2-3ml/kg/hr for 2 to 4 hours and reduced further depending upon the hemodynamic status.

Check hematocrit and other organ function tests periodically.

PATIENT DETERIORATES:

- monitor hematocrit after bolus
- if hct is still high, repeat 2nd bolus 10-20ml/kg/hr for one hour
- if improves, continue management as above.

- if hctreduces, indicates bleeding and consider transfusions.

DECOMPENSATED SHOCK:

- IV crystslloid or colloid at 20ml/kg over 15 minutes
- If hct is low, indicates bleeding and consider transfusions
- If hct is high, consider IV bolus with colloid at 20ml/kg/hr over 30 minutes to one hour
- If improves, treat as above
- If still unstable, give colloid 3rd bolus 10-20ml/kg/hr over 1 hour.
- Hemorrhagic complications are managed with fresh whole blood transfusions or packed cells.
- If still patient is unstable, consider supportive therpy with
    - inotropes and vasopressors
    - treatment of organ impairment
    - treatment of cardiac complications
    - renal replacement therapy.

DISCHARGE CRITERIA:[10]

- Afebrile for 48 hours
- Improvement of general condition with normal hemodynamic status, return of good appetite, adequate urine output, without any respiratory distress
- Stable hematocrit without any iv fluids and platelet in rising trend

MATERIALS AND METHODS

CASE SELECTION:

100 patients hospitalized with dengue infection (sero positive)

INCLUSION CRITERIA:

All serologically proven cases.

EXCLUSION CRITERIA:

Associated infections known to cause Hepatic involvement like Malaria, Enteric fever, Hepatitis, Leptospirosis

MODE OF EVALUATION:

Dengue sero positive patients are selected and examined clinically for hepatomegaly and jaundice and subjected to complete blood count, liver function tests, ultrasound abdomen, PT, APTT, Widal, HBsAg, HCV and analysed.

STUDY DESIGN:

- Prospective COHORT study.
- Study of patients selected as per criteria.

SAMPLE SIZE:

For the study to be statistically significant, a sample size of 100 was taken

METHODS:

COMPLETE BLOOD COUNT:

CBC was done using automated counter method.

LIVER FUNCTION TESTS:

LFT was done on the day of admission . 3ml venous blood was collected and the following were done

TOTAL AND DIRECT BILIRUBIN:

- Reagents: - Sodium nitrite 10mmol/L
- Sulphanilic acid 23mmol/L
- Sodium acetate 0.9 mol/L
- Sodium benzoate 0.5 mmol/L
- Caffine 0.25mol/L

Principle:

Bilirubin reacts with sulphanilic acid to produce an azo compound, the colour of which could be measured at 546nm and this gives an estimate of concentration of bilirubin.

PROCEDURE:

Serum sample is preferred.

Set the instrument using the settings below:

Method	Bichromatic
Wave length :	546 and 630 nm
Reaction slope:	Increasing
Reaction type :	end point
Flow cell temperature:	30 degree Celsius
Reagent volume:	1.1ml
Sample volume:	50 micro Litre
Incubation period:	Five minutes at room temperature
Factor:	26.312
Method:	monochromatic
Wavelength	546 nm

Other parameters are kept same as above.

Bichromaticmethod:

REAGENT	VOLUME
SAMPLE	50 micro L
Sodium Nitrite	100 micro L
Caffine	1 ml

Incubate for 5 minutes at room temperature . Read after 10 minutes.

Reference values:

Total bilirubin : 0.001 to 1.00 mg/dl

Direct bilirubin: 0.0 to 0.25 mg/dl

ALBUMIN:

Albumin binds with bromocresol green dye at ph 4.2 to form blue green coloured compound which is proportional to the concentration of albumin and when photometrically measured between 580 to 630 nm with maximum absorbance at 625 nm .

Sample: plasma or serum

REAGENT	BLANK	STANDARD	TEST
Albumin reagent 1	100 micro L	1000 micro L	1000 micro L
Distilled water	10 micro L	-	-
Standard 2	-	10 micro L	-
Sample	-	-	10 micro L

Mix well and read the results after one minute at 630 nm (580 nm–630 nm)

ALBUMIN (g/dl) = Absorbance of test / Absorbance of standard x concentration of standard (g/dl)

Reference values: 3.2 to 5 g/dl

SGOT / AST:

L-Aspartate + alpha ketoglutarate = oxaloacetate + L- glutamate

Oxaloacetate + NADH + H = L-Malate + NAD

AST – Aspartate aminotransferase

MDH – Malate dehydrogenase

There is decrease in absorption at 340nm since NADH is converted to NAD. The rate of decrease is proportional to AST activity in sample. The samples are reconstituted.

Reaction type	:	Kinetic
Reaction slope	:	Decreasing
Wavelength	:	340nm
Temperature	:	37 degree Celsius
Interval	:	60 seconds
Delay time	:	60 seconds
Sample volume	:	100 micro litre
Reagent volume	:	1 ml
Path length	:	1 centimetre
Factor	:	1746
No of readings	:	4

REAGENT	**VOLUME**
Recontituted reagent	1 ml
Sample	100 micro litre

Mix and read.

Reference value: upto 46 U / L

SGPT:

Principle:

L-Alanine + Alpha ketoglutarate = L- Glutamate + Pyruate

The rate of decrease is proportional to AST activity in sample.

The samples are reconstituted.

Reaction type : Kinetic

Reaction slope : Decreasing

Wavelength : 340nm

Temperature : 37 degree Celsius

Interval : 60 seconds

Delay time : 60 seconds

Sample volume : 100 micro litre

Reagent volume : 1 ml

Path length : 1 centimetre

Factor : 1746

No of readings : 4

REAGENT	VOLUME
Recontituted reagent	1 ml
Sample	100 micro litre

Mix and read .

Reference value: upto 49 U / L

ALKALINE PHOSPHATASE:

Principle:

P-Nitrophenyl phosphate + H2O = p-nitro phenol + phosphate

Add AQUA 4 supplied with kit to the reagent .

REAGENT	VOLUME
Working reagent	1000 micro litre
sample	20 micro litre

Mix and read

Reference value: 30-120 U/L

DENGUE IgM CAPTURE ELISA:

IgM estimation can be done using commercial kits available .

Sample: serum 0.5 ml is added to strip

Principle:

Serum antibodies combine with human IgM antibodies attached to polystyrene surface of microwell . An equal volume of HRP conjugated monoclonal antibodies are added to diluted antigen . This allows formation of antigen antibody complexes. Residual serm is washed . The substrate is hydrolysed by enzyme, which produces blue colour.

RESULTS:

PANBINO UNITS	RESULTS
< 9 UNITS	Negative
9-11 UNITS	Equivocal
>11 UNITS	Positive

PROTHROMBIN TIME:

Thromborel S Reagent is used to measure prothrombin time. It can determine the activity of factors II, V, VII, X.

Principle:

Incbation of plasma with optimal thromboplastin and calcium activates the coagulation process.

PROCEDURE:

Mix one part of sodium citrate (0.11 mol/L) with nine parts of venous blood. Centrifuge for 15 mins at 1500Xg at room temperature.

Take a pre warmed test tube to 37 degree Celsius	
Citrated plasma	100 micro Litre
Incubate for one minute at 37 degree Celsius	
Thromborel S Reagent	200 micro Litre
Note the timer on coagulation analyser after adding the reagent .	

The results can be reported in seconds or INR.

Reference Value: 1 – 1.5 INR

ACTIVATED PARTIAL THROMBOPLASTIN TIME:

It determines the coagulation abnormalities of intrinsic pathway

Principle:

Incubating plasma with optimal amount of phospholipids and calcium activates intrinsic pathway.

PROCEDURE:

Calcium chloride solution is pre warmed to 37 degree Celsius

Dade Actin Reagent is pre warmed to 37 degree Celsius for one minute

Pipet into tubes as follws:

	TEST SAMPLE	CONTROL SAMPLE
Dade Actin Reagent (pre warmed)	0.1 ml	0.1 ml
Plasma	0.1 ml	-
Control plasma	-	0.1 ml
Mix well and incubate for 3 minutes at 37 degree Celsius		
Calcium chloride solution (pre warmed)	0.1 ml	0.1 ml

Start timer after addition of calcium chloride. Observe for clot formation .

RESULTS:

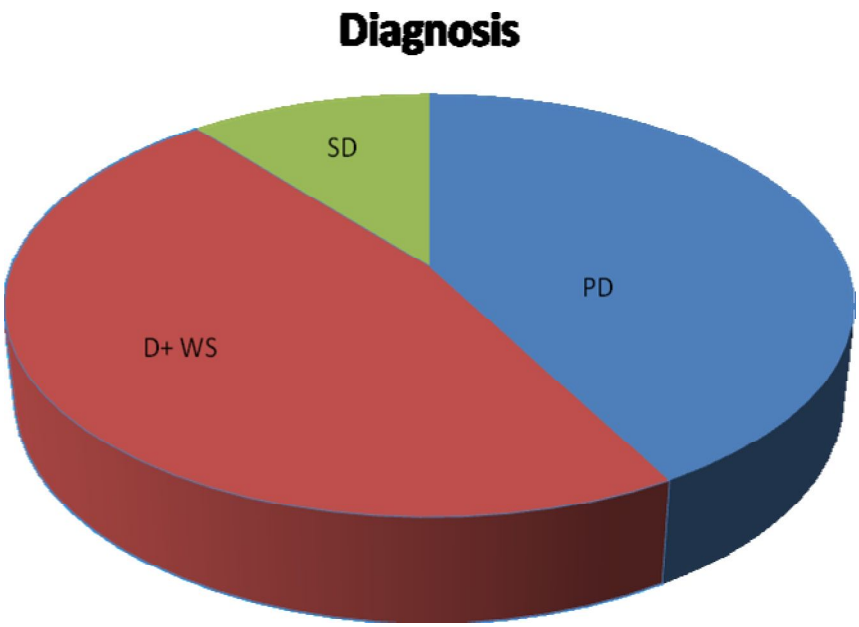
The results is expressed in seconds .

RESULTS

The study was conducted at the department of paediatrics , Govt. Mohan Kumaramangalam Medical College hospital , Salem from AUG 2014 TO JULY 2015.

This study was conducted on 100 serologically IgM dengue antibody positive cases between age group 2 months to 12 years of age fulfilling the WHO criteria for the diagnosis of dengue infection.

Diagnosis	Frequency	Percent
PROBABLE DENGUE (PD)	42	42
DENGUE WITH WAARNING SIGNS (D+WS)	47	47
SEVERE DENGUE (SD)	11	11
Total	100	100

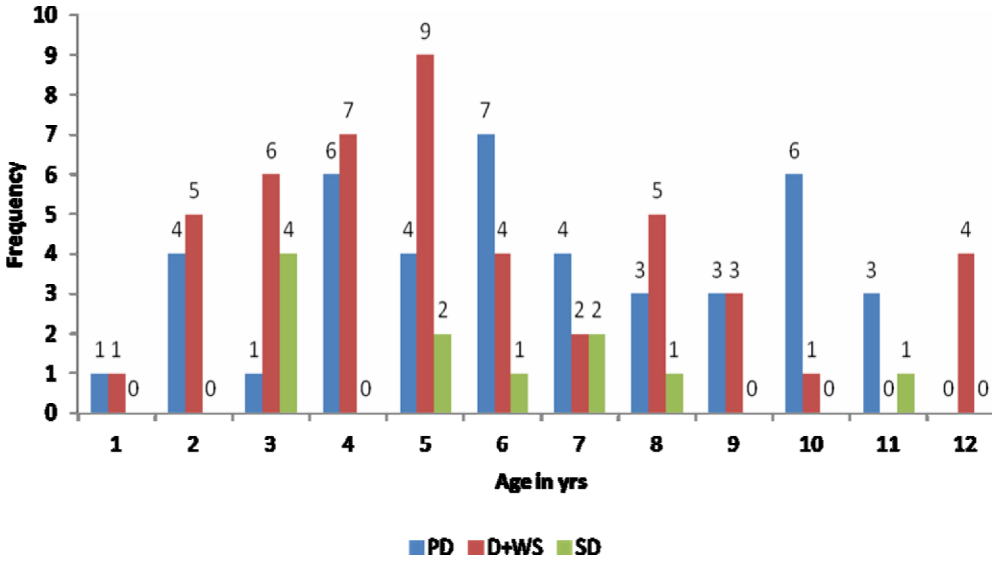


Of the 100 patients hospitalized with dengue infection, 42 were classified as having Probable Dengue, 47 were with Warning Signs and 11 were suffering from Severe Dengue.

AGE WISE DISRIBUTION OF CASES

	Diagnosis			Total
Age in yrs	**PD**	**D+SD**	**SD**	
1	1	1	-	2
2	4	5	-	9
3	1	6	4	11
4	6	7	-	13
5	4	9	2	15
6	7	4	1	12
7	4	2	2	8
8	3	5	1	9
9	3	3	-	6
10	6	1	-	7
11	3	-	1	4
12	-	4	-	4
Total	42	47	11	100

AGE WISE DISRIBUTION OF CASES

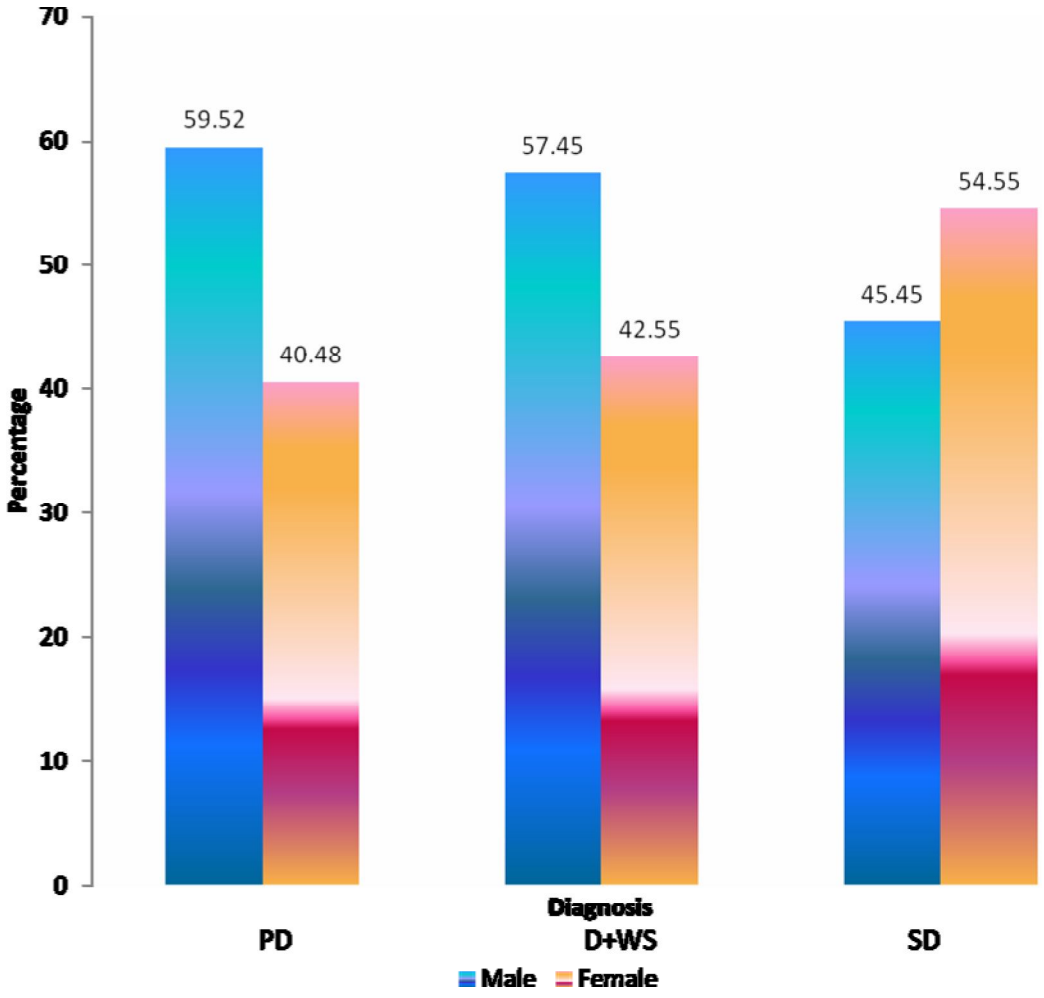


Dengue mainly affected children of age group 5 to 7 years.

GENDER DISTRIBUTION OF CASES

Diagnosis	Gender of the child				Total
	Male		Female		
	N	%	N	%	
PD	25	59.52	17	40.48	42
D+WS	27	57.45	20	42.55	47
SD	5	45.45	6	54.55	11
Total	57	57.00	43	43.00	100

GENDER DISTRIBUTION OF CASES

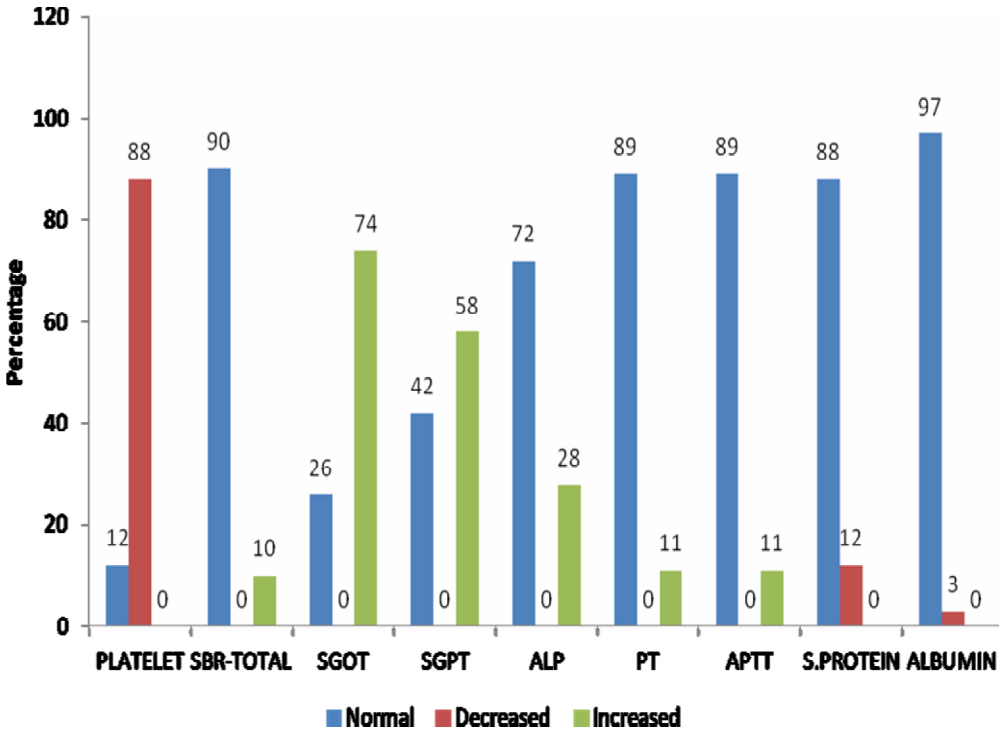


Dengue affected male and female children almost equally.

COMPARISION OF CHANGES IN LIVER FUUNCTION TESTS AND PLATELET COUNT:

	Normal		**Decreased**		**Increased**		**Total**
	N	**%**	**N**	**%**	**N**	**%**	
PLATELET	12	12	88	88	-	-	100
SBR-TOTAL	90	90	-	-	10	10	100
SGOT	26	26	-	-	74	74	100
SGPT	42	42	-	-	58	58	100
ALP	72	72	-	-	28	28	100
PT	89	89	-	-	11	11	100
APTT	89	89	-	-	11	11	100
S.PROTEIN	88	88	12	12	-	-	100
ALBUMIN	97	97	3	3	-	-	100

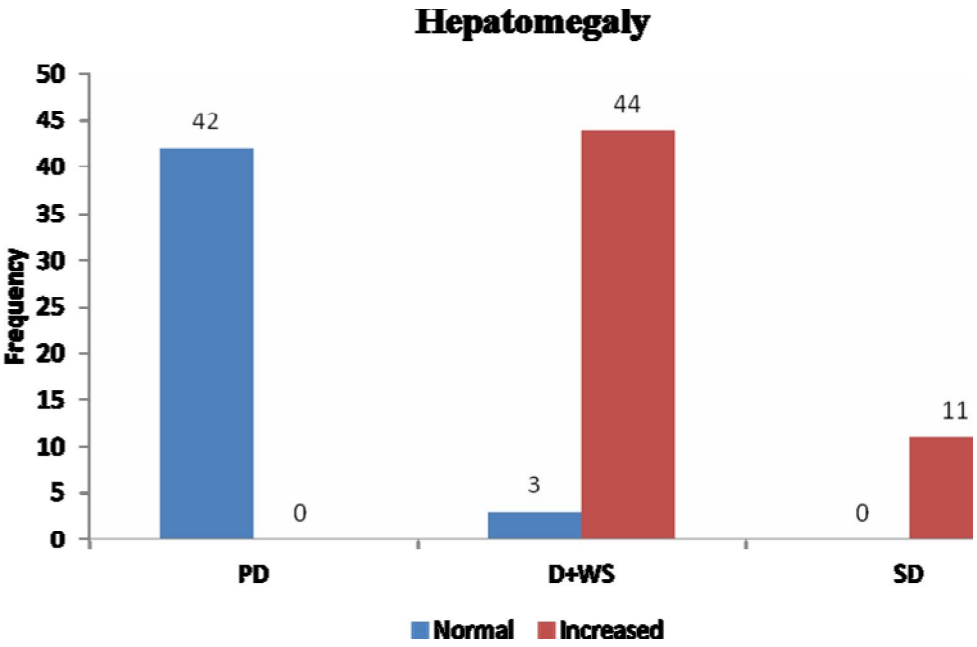
COMPARISION OF CHANGES IN LIVER FUUNCTION TESTS AND PLATELET COUNT



COMPARISION BETWEEN GROUPS WITH RESPECT TO HEPATOMEGALY

	Diagnosis			Chi square	p
Liver span	**PD**	**D+WS**	**SD**		
Normal	42	3		88.65	0.001**
Increased		44	11		
Total	42	45	11		

COMPARISION BETWEEN GROUPS WITH RESPECT TO HEPATOMEGALY



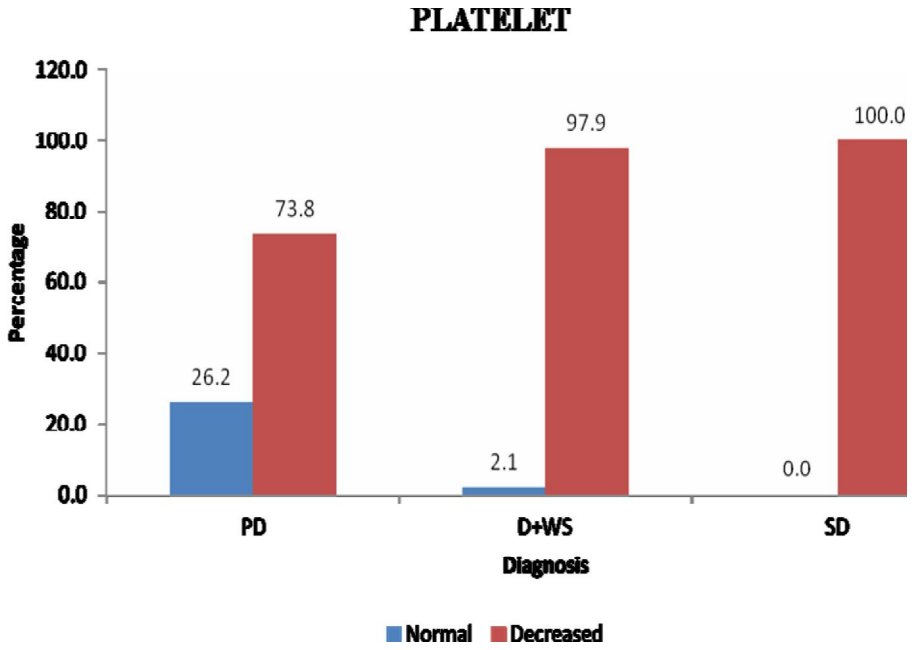
Hepatomegaly was seen in 55% of patients. When compared between the groups , 93.6% in patients with warning signs and 100% in seen severe dengue.

COMPARISION BETWEEN GROUPS WITH RESPECT TO PLATELET COUNT

PLATELET	Diagnosis						Total	Chi square	p
	PD		D+WS		SD				
	N	%	N	%	N	%			
Normal	11	26.2	1	2.1	-	-	12	13.85	0.001**
Decreased	31	73.8	46	97.9	11	100.0	88		
Total	42	100.0	47	100.0	11	100.0	100	-	-

** Significant at 1 % (Highly significant)

COMPARISION BETWEEN GROUPS WITH RESPECT TO PLATELET COUNT



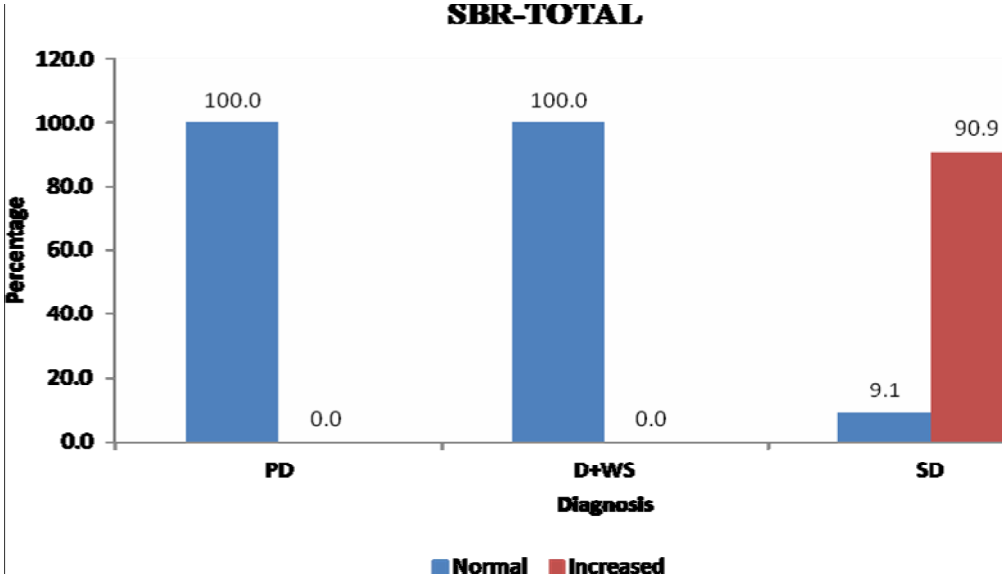
Thrombocytopenia occurred in 74% of patients with probable dengue , 98% with warning signs and 100% in severe dengue

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERM BILIRUBIN :

SBR-TOTAL	Diagnosis						Total	Chi square	p
	PD		D+WS		SD				
	N	%	N	%	N	%			
Normal	42	100.0	47	100.0	1	9.1	90	89.90	< 0.001**
Increased	-	-	-	-	10	90.9	10		
Total	42	100.0	47	100.0	11	100.0	100		

** Significant at 1 % (Highly significant)

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERM BILIRUBIN :



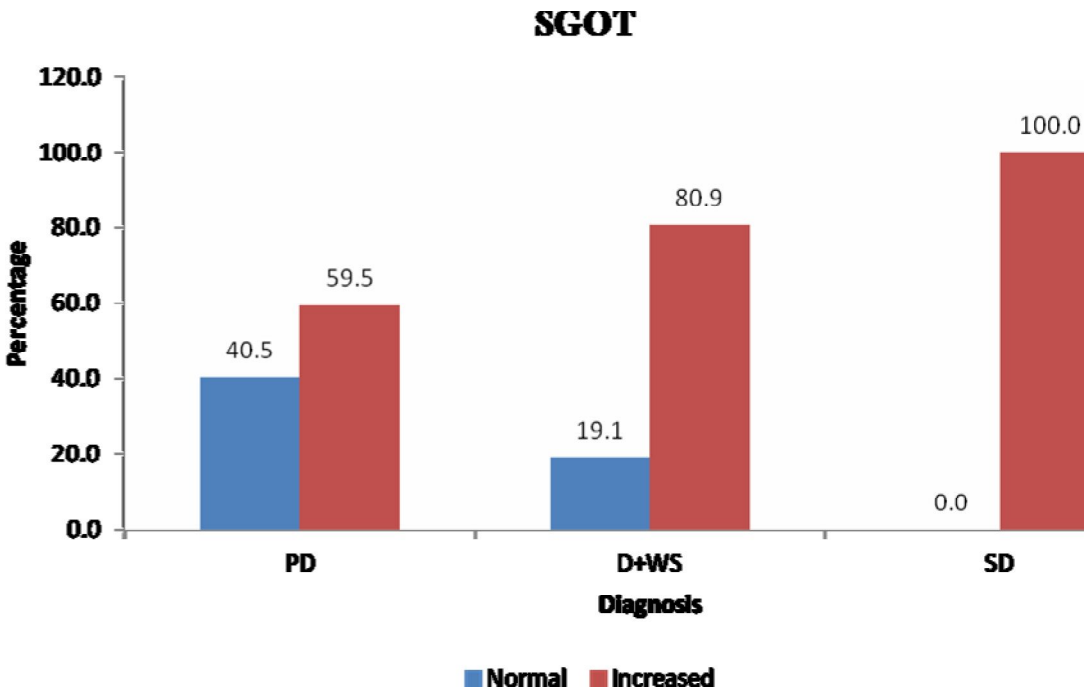
In this study, serum total bilirubin was raised in 10% of subjects with severe dengue infection.

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERUM SGOT :

SGOT	Diagnosis						Total	Chi square	p
	PD		D+WS		SD				
	N	%	N	%	N	%			
Normal	17	40.5	9	19.1	-	-	26	9.59	0.008**
Increased	25	59.5	38	80.9	11	100.0	74		
Total	42	100.0	47	100.0	11	100.0	100		

** Significant at 1 % (Highly significant)

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERUM SGOT



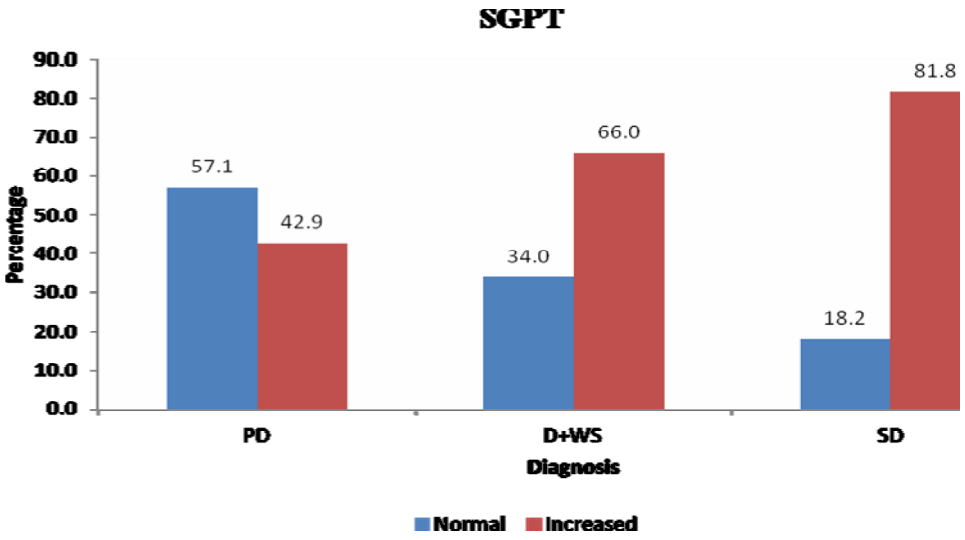
Serum SGOT was raised in 74 % of patients with dengue. When compared between the groups ,rise in SGOT occurred in 59.5% of patients with probable dengue , 80.9% with warning signs and 100% in severe dengue .

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERUM SGPT :

SGPT	Diagnosis						Total	Chi square	p
	PD		D+WS		SD				
	N	%	N	%	N	%			
Normal	24	57.1	16	34.0	2	18.2	42	7.74	0.021*
Increased	18	42.9	31	66.0	9	81.8	58		
Total	42	100.0	47	100.0	11	100.0	100		

* Significant at 5 %

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERUM ALKALINE PHOSPHATASE



SGPT was raised in 58% of patients with dengue infection.When compared between the groups ,rise in SGPT occurred in 43% of patients with probable dengue , 66% with warning signs and 82% in severe dengue .

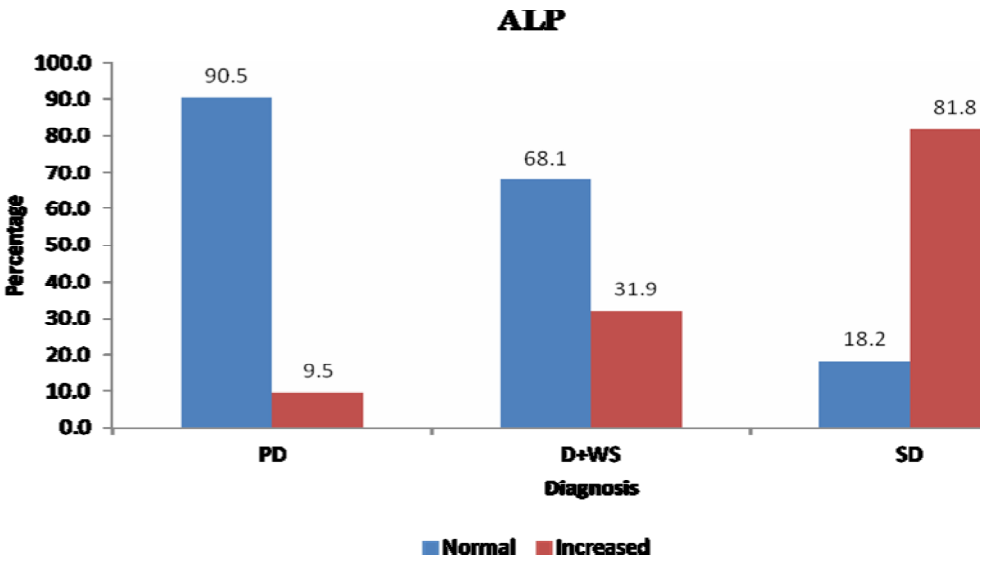
COMPARISION BETWEEN GROUPS WITH RESPECT TO

SERUM ALKALINE PHOSPHATASE

ALP	Diagnosis						Total	Chi square	p
	PD		D+WS		SD				
	N	%	N	%	N	%			
Normal	38	90.5	32	68.1	2	18.2	72	23.27	< 0.001**
Increased	4	9.5	15	31.9	9	81.8	28		
Total	42	100.0	47	100.0	11	100.0	100		

** Significant at 1 % (Highly significant)

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERUM ALKALINE PHOSPHATASE



Serum Alkaline Phosphatase was raised in 58% of patients with dengue infection.When compared between the groups ,rise in SGPT occurred in 9.5% of patients with probable dengue , 32% with warning signs and 82% in severe dengue .

COMPARISION BETWEEN GROUPS WITH RESPECT TO

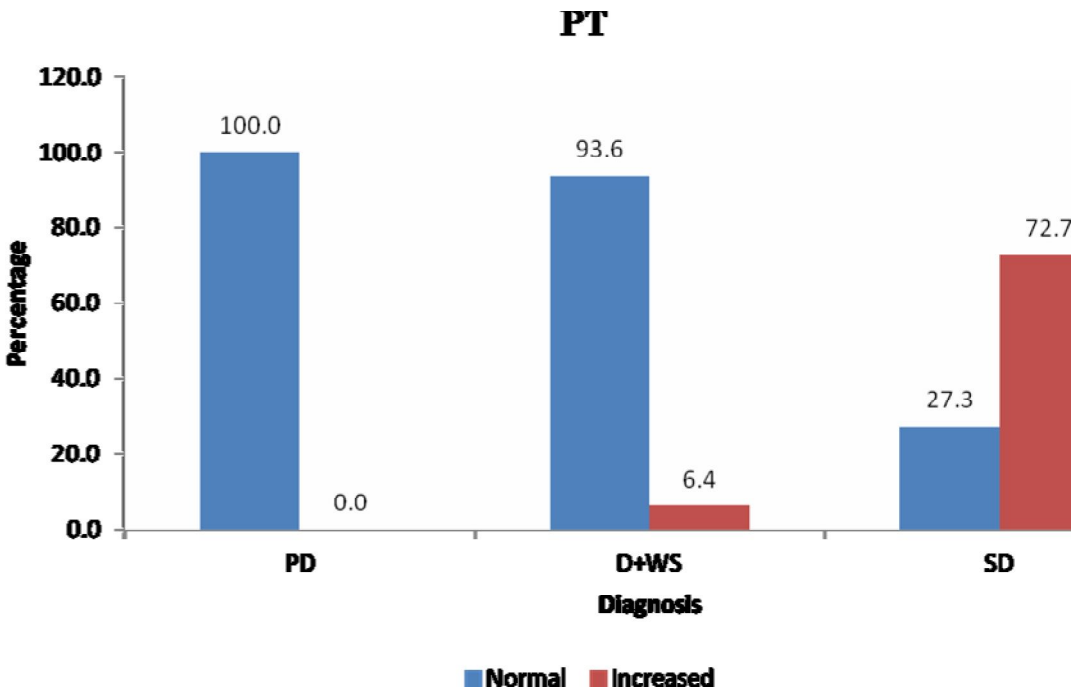
PROTHROMBIN TIME :

PT	Diagnosis						Total	Chi square	p
	PD		D+WS		SD				
	N	%	N	%	N	%			
Normal	42	100.0	44	93.6	3	27.3	89	49.03	< 0.001**
Increased	-	-	3	6.4	8	72.7	11		
Total	42	100.0	47	100.0	11	100.0	100		

** Significant at 1 % (Highly significant)

COMPARISION BETWEEN GROUPS WITH RESPECT TO

PROTHROMBIN TIME :



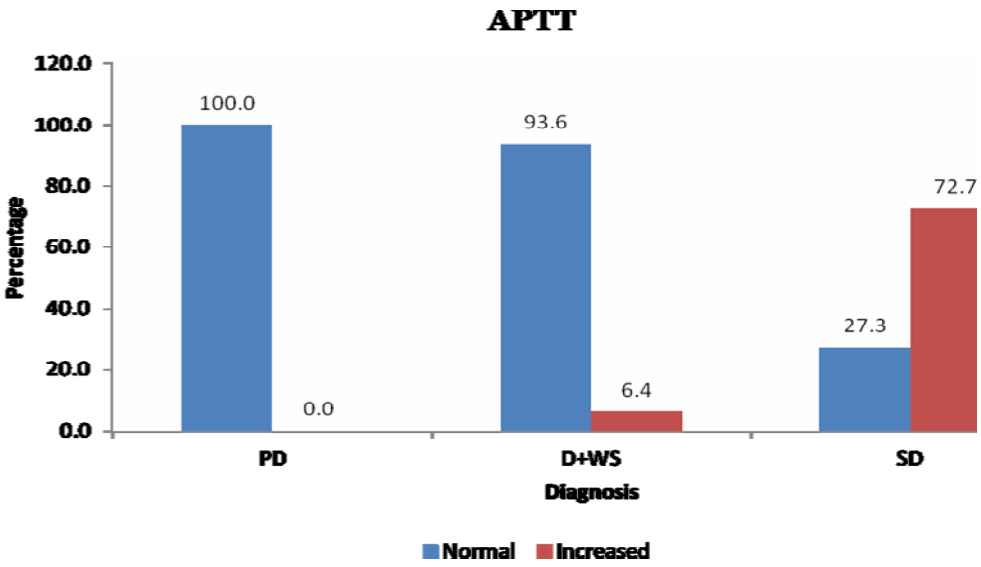
Prothrombin time was raised in 11% of patients with dengue infection.When compared between the groups , rise in PT occurred in 6.4% with warning signs and 73% in severe dengue.

COMPARISION BETWEEN GROUPS WITH RESPECT TO APTT

APTT	Diagnosis						Total	Chi square	p
	PD		D+WS		SD				
	N	%	N	%	N	%			
Normal	42	100.0	44	93.6	3	27.3	89	49.03	< 0.001**
Increased	-	-	3	6.4	8	72.7	11		
Total	42	100.0	47	100.0	11	100.0	100		

** Significant at 1 % (Highly significant)

COMPARISION BETWEEN GROUPS WITH RESPECT TO APTT



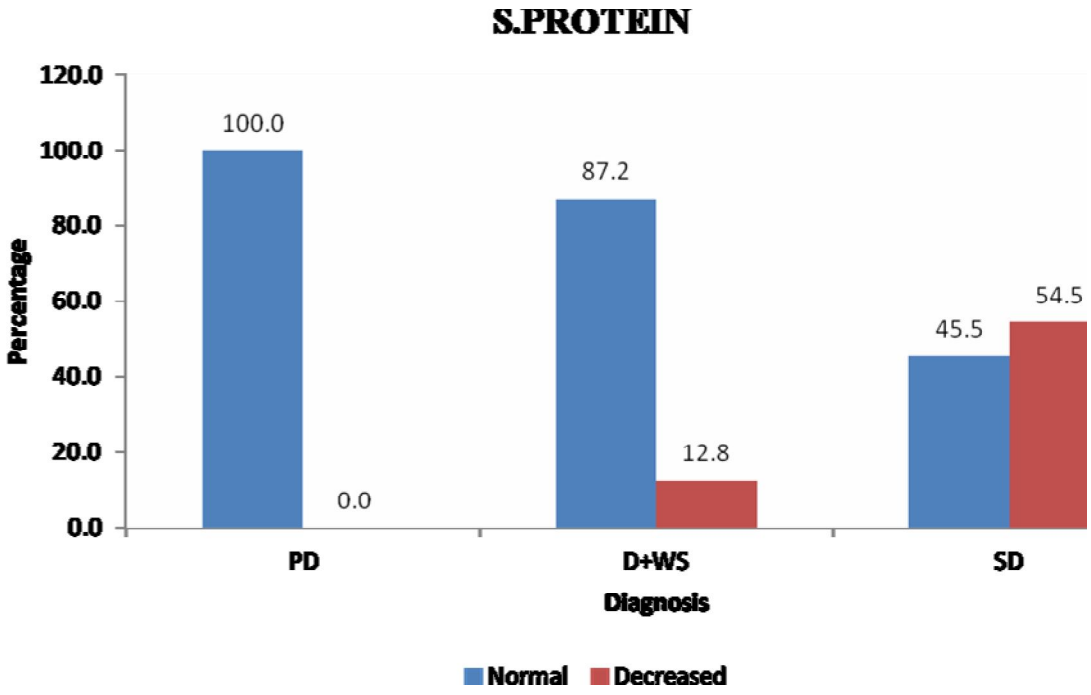
Activated Partial Thromboplastin Time was raised in 11% of patients with dengue infection.When compared between the groups ,rise in APTT occurred in 6.4% of patients with warning signs and 73% in severe dengue .

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERUM TOTAL PROTEIN :

S.PROTEIN	Diagnosis						Total	Chi square	p
	PD		D+WS		SD				
	N	%	N	%	N	%			
Normal	42	100.0	41	87.2	5	45.5	88	24.61	< 0.001**
Decreased	-	-	6	12.8	6	54.5	12		
Total	42	100.0	47	100.0	11	100.0	100		

** Significant at 1 % (Highly significant)

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERUM TOTAL PROTEIN :

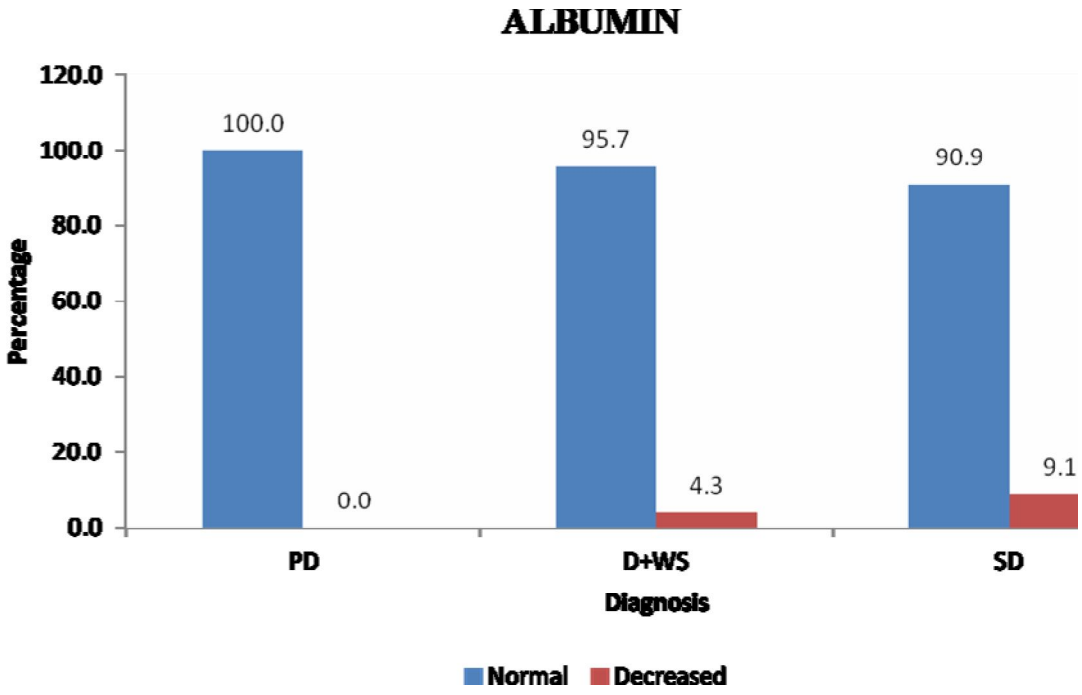


Serum total protein was reduced in 12% of patients with dengue infection. When compared between the groups fall in serum protein occurred in 12.7% with warning signs and 54.5% in severe dengue .

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERUM ALBUMIN :

ALBUMIN	Diagnosis						Total	Chi square	p
	PD		D+WS		SD				
	N	%	N	%	N	%			
Normal	42	100.0	45	95.7	10	90.9	97	2.96	0.228
Decreased	-	-	2	4.3	1	9.1	3		
Total	42	100.0	47	100.0	11	100.0	100		

COMPARISION BETWEEN GROUPS WITH RESPECT TO SERUM ALBUMIN :

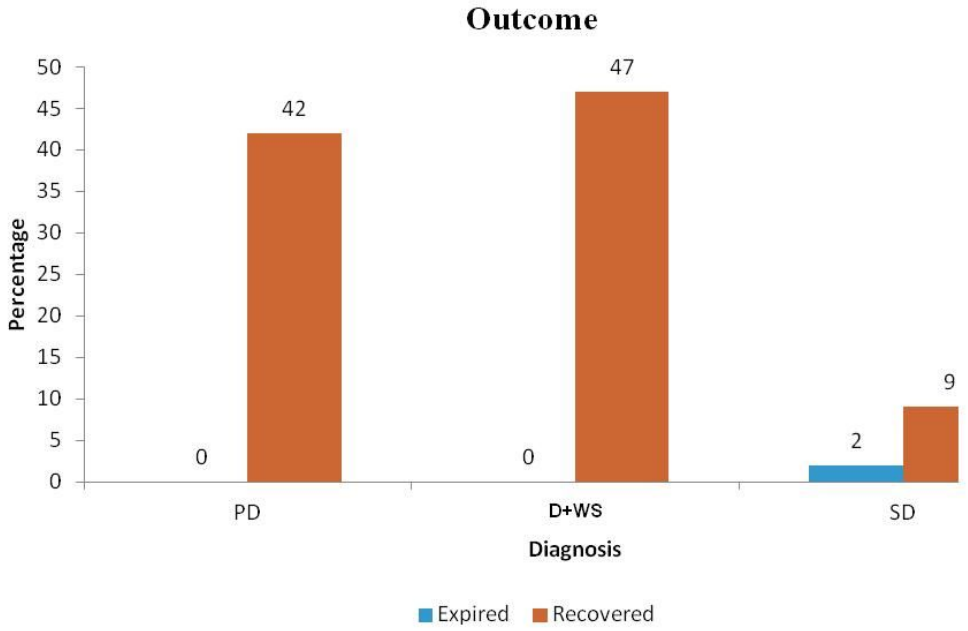


Serum Albumin was reduced in 3% of patients with dengue infection. When compared between the groups , fall in serum albumin occurred in 4.3% with warning signs and 9% with severe dengue.

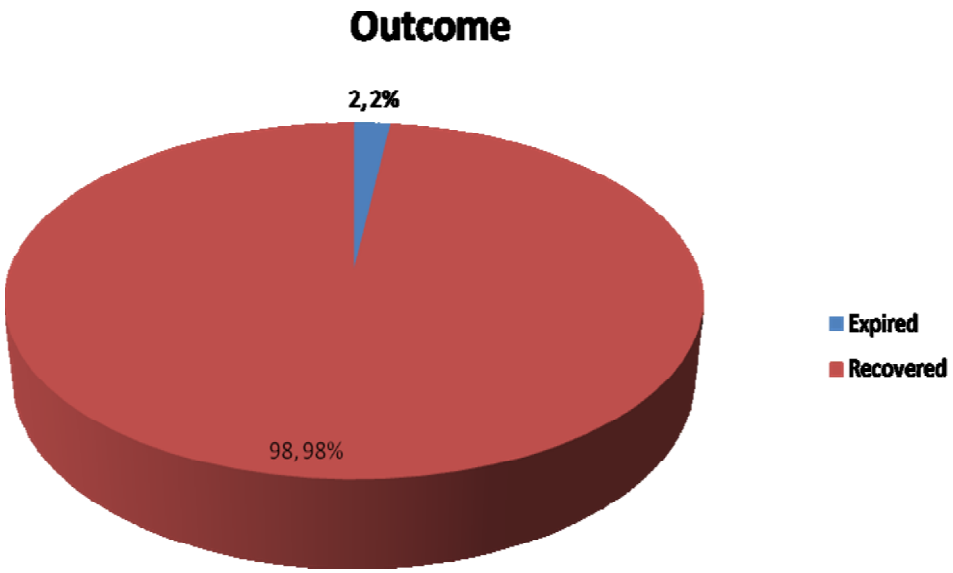
OUTCOME :

	Diagnosis			Total	Chi square	p
Outcome	**PD**	**D+WS**	**SD**			
Expired	-	-	2	2	16.51	< 0.001**
Recovered	42	47	9	98		
Total	42	47.0	11	100		

** Significant at 1 % (Highly significant)



In our study 2 cases suffering from severe dengue expired. In these two cases , the enzymes level were very high.



DISCUSSION

Dengue infection is one of the most common mosquito borne disease of the world. The causative agent is dengue virus, mainly of four serotypes DEN 1, DEN 2, DEN 3, DEN 4 . It has a protean of manifestations ranging from asymptomatic to life threatening complications. Recent data suggests that, there is an upsurge in incidence among children. [5-7]

HEPATIC INVOLVEMENT IN DENGUE:[3, 14-19]

HEPATOMEGALY:

Luiz Jose de Souza [23] et al	74%
Nimmantya et al[12]	98%
Gurdeep S. Dhoria et al[14]	60%
Ole Wichmann et al[19]	43%
Brij Mohan et al[16]	74%
Kuo et al[15]	74%
Present study	55%

Hepatomegaly in dengue occurred more commonly in patients with severe dengue and those with warning signs. In present study 55% with probable dengue, % with warning signs and % with severe dengue had hepatomegaly. Thus hepatomegaly may be used as a tool to indicate the severity of the disease.

LIVER ENZYMES:

The degereeof rise in liver enzymes might be used as a tool to predict the severity of the disease. Higher is the level of liver enzymes, poorer is the prognosis. Study by Brij Mohan et al[16] reported that levels of SGOT raised during first week and SGPT raised during second week. The levels of both began to decline by third week. Serum alkaline phosphatase also was seen to rise in a similar trend. Nimmannitya et al[12] found that SGOT raised from day 3, peaks by day 7 and became normal by 3 to 8 weeks. SGPT is primarily associated with hepatocytes and is raised due to liver damage. SGPT is found in cardiac and skeletal muscle, hepatocytes, renal and brain tissue and is raised due to damage to these structures. Liver enzymes can be a potential marker for dengue during early febrile phase.

SGOT/AST:

Serum SGOT was raised in 74 % of patients with dengue. When compared between the groups, rise in SGOT occurred in 74% of patients with probable dengue, 98% with warning signs and 100% in severe dengue .

Study by M Narayanan et al[20], SrivenuItha et al[21] and Brij Mohan et al[16]also observed deranged liver enzyme levels. Souza et al reported elevation of SGOT in 63.4% cases and Kuo et al[15] observed rise in SGOT in 97.9% of cases.

SGPT/ALT:

SGPT was raised in 58% of patients with dengue infection. When compared between the groups, rise in SGPT occurred in 42% of patients with probable dengue, 66% with warning signs and 81% in severe dengue .Study by M Narayanan et al[20], SrivenuItha et al[21] and Brij Mohan et al [16]also observed elevation in SGPT. Luiz Jose Souza et al[23] found rise in ALT in 45% of cases. Kuo et al[15] observed rise in ALT in 82% of cases. MMA Faridi et al [22]reported 64.6% rise in ALT levels. Patients with severe dengue had higher level of enzymes.

ALKALINE PHOSPHATASE:

SGPT was raised in 28% of patients with dengue infection. When compared between the groups, rise in SGOT occurred in 9.5% of patients with probable dengue, 32% with warning signs and 82% in severe dengue .

STUDY	%OF ELEVATION IN ALP
Kuo et al[15]	16%
MMA Faridi et al[22]	35.3%
Present study	28%

JAUNDICE:

Jaundice is associated with poor prognosis. It is associated with fulminant hepatic failure.In this study, serum total bilirubin was raised in 10% of subjects with severe dengue infection.

STUDY	% WITH JAUNDICE
Dhin The Thrung et al [24]	2%
SrivenuItha et al[21]	16%
Patware et al[16]	25%
Present study	10%

SERUM ALBUMIN:

Hypoalbuminemia may be due to liver injury and capillary leakage. In current study 12% had hypoalbuminemia.

Study by Manzhi Wong et al [25]with 16.5% had hypoalbuminemia.

PROTHROMBIN TIME:

Prothrombin time depends on vitamin K dependant clotting factors. Abnormal PT is seen severe dengue.

In current study Prothrombin time was raised in 11% of patients with dengue infection. When compared between the groups, rise in PT occurred in 6.4% with warning signs and 72% in severe dengue. Sri Venutha et al[21] observed prolongation of PT in 16% of patients.

ACTIVATED PARTIAL THROMBOPLASTIN TIME:

Activated Partial Thromboplastin Time was raised in 11% of patients with dengue infection. When compared between the groups, rise in APTT occurred in 6.4% of patients with warning signs and 72% in severe dengue .

PLATELET COUNT:

Thrombocytopenia occurred in 74% of patients with pbobabledengue, 98% with warning signs and 100% in severe dengue.

CLINICAL OUTCOME:

Of 100 serologically confirmed cases hospitalized with dengue, 42 were classified as having Probable Dengue, 47 were with Warning Signs and 11 were suffering from Severe Dengue. Two cases of severe dengue expired secondary to DIC. The enzyme levels in these cases were very high.

SUMMARY

This study was done to know the hepatic dysfunction in children with dengue infection and its clinical correlates like clinical features, laboratory parametres, morbidity and mortality.

Of 100 serologically confirmed cases hospitalized with dengue, were classified into Probable Dengue, Dengue with Warning Signs and Severe Dengue as per WHO guidelines.

The following were observed:

- All patients presented with fever
- Most commonly occurred in age group of 5 to 7 years
- Hepatomegaly was the commonest clinical sign seen
- Thrombocytopenia was seen in 88% of cases
- Serum total bilirubin was raised in 10% of subjects with severe dengue infection.
- Serum SGOT was raised in 74 % of patients with dengue. When compared between the groups, rise in SGOT occurred in 74% of patients with probable dengue, 98% with warning signs and 100% in severe dengue .
- SGPT was raised in 58% of patients with dengue infection. When compared between the groups, rise in SGPT occurred in 42% of patients with probable dengue, 66% with warning signs and 81% in severe dengue
- SGPT was raised in 28% of patients with dengue infection. When compared between the groups, rise in SGOT occurred in 9.5% of patients with probable dengue, 32% with warning signs and 82% in severe dengue

- Prothrombin time was raised in 11% of patients with dengue infection. When compared between the groups, rise in PT occurred in 6.4% with warning signs and 72% in severe dengue.
- Activated Partial Thromboplastin Time was raised in 11% of patients with dengue infection. When compared between the groups, rise in APTT occurred in 6.4% of patients with warning signs and 72% in severe dengue
- Serum total protein was reduced in 12% of patients with dengue infection. When compared between the groups fall in serum protein occurred in 12.7% with warning signs and 54.5% in severe dengue .
- Serum Albumin was reduced in 3% of patients with dengue infection.
- In our study 2 cases suffering from severe dengue expired. The enzyme levels in these cases were very high.

CONCLUSION

In developing country like India, incidence of dengue outbreaks is increasing. Hepatic involvement of varying degrees have been reported. As hepatic dysfunction in dengue is transient and reversible, early identification of the same would help to reduce life threatening complications. This can help to reduce the morbidity and mortality due to dengue infection. The role of hepato protective drugs in reducing morbidity and mortality should be analysed by further studies.

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BIBLIOGRAPHY

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PROFORMA

PROFORMA

- **NAME:**
- **AGE:**
- **SEX:**
- **ADDRESS:**

- **IP NO:**
- **D.O.A:**
- **D.O.D:**
- **DIAGNOSIS:**
- **CHIEF COMPLAINTS:**

- **ULTRASOUND ABDOMEN:**
- **IgM DENGUE POSITIVE ON:**
- **COMPLETE BLOOD COUNT:**

  TC :

  DC:

  PLATELET:

  HB:

  PCV:

- **LIVER FUNCTION TEST:**

  SBR TOTAL :

  DIRECT :

  INDIRECT:

  SGOT:

  SGPT:

  ALP:

  S.PROTEIN:

  ALBUMIN:

  GLOBULIN:

- PSS FOR MALARIAL PARASITE:
- IgM ANTI HAV:
- HBsAg:
- HCV:
- WIDAL:
- MAT FOR LEPTOSPIROSIS:

PATIENT CONSENT FORM

STUDY TITLE:

"A STUDY ON OUTCOME OF CHILDREN WITH DENGUE INFECTION AND ITS CLINICAL CORELATION WITH HEPATIC DYSFUNCTION IN GMKMCH"

Department of PAEDIATRICS , GMKMCH, SALEM

PARTICIPANT NAME : AGE : SEX:

I.P. NO :

I confirm that I have understood the purpose of the above study. I have the opportunity to ask the question and all my questions and doubts have been answered to my satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving any reason.

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my child's health records both in respect to the current study and any further research that may be conducted in relation to it, even if I withdraw from the study. I understand that my child's identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study.

I hereby consent to participate in this study..

Time :

Date : Signature/Thumb Impression of Patient's Care taker

Place :

Patient's name:

Signature of the investigator: _____

Name of the investigator :_____

ABBREVIATIONS

LIST OF ABBREVIATIONS

DF	Dengue fever
DHF	Dengue hamorrhage fever
D+WS	Dengue with warning signs
DSS	Dengue shock syndrome
SD	Severe syndrome
ALT /SGPT	Alanine Trasaminase / Serum Glutomic –Pyruvic Transaminase
AST /SGOT	Aspartate Transaminase / Serum Glutamic – Oxaloacetic Transaminase
ALP	Alkaline phosphatase
HCT	Haematrocrit
GB	Gall Bladder
PT	Prothrombin time
APTT	Activated partial thromboplastin time
USG	Ultrasonography
W.H.O	World Health Organization
SGOT	Serum glutamate oxalate transminase
SGPT	Serum glutamate pyruvate transminase

KEY TO MASTER CHART

USG	-ULTRASOUND ABDOMEN
TC	-TOTAL COUNT
HB	-HAEMOGLOBIN
SBR	-SERUM BILIRUBIN
SGOT	-SERUM
SGPT	-SERUM
ALP	-SERUM ALKALINE PHOSPHATASE
PT	-PROTHOMBIN TIME
APTT	-ACTIVATED PARTIAL THROMBOPLASTIN TIME
Ig M	-IMMUNOGLOBULIN M AGAINT DENGUE
	PSSPERIPHERAL SMEAR STUDY
MP	-MALARIAL PARASITE
S.PROTEIN	-SERUM PROTEIN
IgM Anti HAV	-IMMUNOGLOBULIN M AGAINST HEPATITIS A
VIRUS	
HBsAg	-HEPATITIS B ANTIGEN
HCV	-HEPATITIS C ANTIGEN
MAT	-MACROSCOPIC AGGLUTINATION TEST
SL.NO.	-SERIAL NUMBER

MASTER CHART

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	IG M	USG ABDOMEN	TC	PLATELET	HB
1	Thirupathi	2y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	9000	45000	9.5
2	Nithish	7y	M	PD	POSITIVE	NORMAL STUDY	11000	100000	11
3	Vishwa	12y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	4500	30000	12.6
4	Paramakudi	7y	M	PD	POSITIVE	NORMAL STUDY	12400	90000	9.8
5	Pradeep	8y	M	PD	POSITIVE	NORMAL STUDY	10000	60000	12
6	Sathya priya	4y	F	PD	POSITIVE	GB WALL EDEMA	7500	115000	11.9
7	Deepika	2y	F	D + WS	POSITIVE	HEPATOMEGALY	8700	60000	8.5
8	Hasini	6y	F	PD	POSITIVE	GB WALL EDEMA	5300	30000	9
9	Kishore	6y	M	PD	POSITIVE	GB WALL EDEMA	5900	70000	8.8
10	Aparna	4y	F	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	9800	40000	10.5
11	Kousika	4y	F	PD	POSITIVE	GB WALL EDEMA	4200	90000	12.2
12	Giridharan	11y	F	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	1050	10000	10.9
13	Haneesh	4y	M	PD	POSITIVE	GB WALL EDEMA	4000	50000	12.6
14	Logith	2y	F	PD	POSITIVE	GB WALL EDEMA	8600	80000	10.5
15	Jothika	7y	F	D + WS	POSITIVE	HEPATOMEGALY	5800	60000	9.8
16	Akilan	5y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	2600	75000	8.9
17	Vishali	3y	F	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	7800	15000	10.9
18	Gokul raj	6y	M	PD	POSITIVE	GB WALL EDEMA	4500	60000	12.1
19	Parameshwari	8y	F	PD	POSITIVE	GB WALL EDEMA	8000	75000	10
20	Arun kumar	7y	M	PD	POSITIVE	GB WALL EDEMA	5800	90000	10.9
21	Satheesh	10y	M	PD	POSITIVE	NORMAL STUDY	7800	150000	12.5
22	Ayyadurai	8y	M	PD	POSITIVE	GB WALL EDEMA	6900	250000	11
23	Sabari	11y	M	PD	POSITIVE	NORMAL STUDY	5600	160000	10.8
24	Hema	7y	F	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	9000	20000	9
25	Suchithra	4y	F	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	12400	60000	11
26	Suresh	6y	M	PD	POSITIVE	GB WALL EDEMA	3800	50000	10.8
27	Priyadharshini	4y	F	PD	POSITIVE	NORMAL STUDY	4900	75000	11

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	IG M	USG ABDOMEN	TC	PLATELET	HB
28	Jeya shree	10y	F	PD	POSITIVE	GB WALL EDEMA	5700	30000	9
29	Dhanush	5y	M	D + WS	POSITIVE	HEPATOMEGALY	4700	75000	9.5
30	Boopathy	5y	M	D + WS	POSITIVE	HEPATOMEGALY	9700	50000	9.8
31	Nandhini	9y	F	D + WS	POSITIVE	HEPATOMEGALY	8000	20000	12
32	Vijay	5y	M	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	5900	5000	11
33	Kavyashree	3y	F	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	4800	60000	10.5
34	Soumya	4y	F	D + WS	POSITIVE	HEPATOMEGALY	3900	50000	11
35	Bharath kumar	10y	M	PD	POSITIVE	NORMAL STUDY	4900	60000	7.9
36	Shruthika	3y	F	D + WS	POSITIVE	HEPATOMEGALY	4800	170000	8.5
37	Iniyan	3y	M	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	1100	30000	9.5
38	Lavanya	4y	F	PD	POSITIVE	GB WALL EDEMA	8700	70000	11
39	Leenashree	6y	F	PD	POSITIVE	GB WALL EDEMA	6200	60000	10
40	Rajkumar	5y	M	PD	POSITIVE	GB WALL EDEMA	8100	40000	10.5
41	Monika	6y	F	D + WS	POSITIVE	HEPATOMEGALY	6400	75000	11
42	Gowtham	3y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	9600	80000	12
43	Sakthi	2y	M	D + WS	POSITIVE	HEPATOMEGALY	4900	45000	9.9
44	Samaya	5y	F	D + WS	POSITIVE	HEPATOMEGALY	6900	90000	9
45	Hariharasudhan	3y	F	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	8900	75000	8.7
46	Poornima	2y	F	PD	POSITIVE	NORMAL STUDY	7900	65000	11
47	Kamalesh	4y	M	PD	POSITIVE	NORMAL STUDY	5800	30000	7.9
48	Desigan	5y	M	PD	POSITIVE	GB WALL EDEMA	4800	80000	8.8
49	Raghul	11y	M	PD	POSITIVE	GB WALL EDEMA	9100	150000	10
50	Santhosh	12y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	8200	60000	11
51	Santhosh kumar	4y	M	D + WS	POSITIVE	HEPATOMEGALY	8300	25000	11.5
52	Anbarasan	5y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	5400	40000	11
53	Mahammed siddi	2y	M	PD	POSITIVE	GB WALL EDEMA	7300	180000	11.9
54	Sandhiya	9y	F	PD	POSITIVE	NORMAL STUDY	7900	250000	9.8

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	IG M	USG ABDOMEN	TC	PLATELET	HB
55	Jeevitha	5y	F	PD	POSITIVE	NORMAL STUDY	8100	360000	8.8
56	Sindhu	10y	F	PD	POSITIVE	NORMAL STUDY	11000	160000	7.9
57	Amudhini	6y	F	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	9100	20000	9
58	Sri gugan	3y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	8700	50000	9.4
59	Dharaneeshwarar	12y	M	D + WS	POSITIVE	HEPATOMEGALY	9600	60000	10
60	Susi baskar	4y	M	D + WS	POSITIVE	HEPATOMEGALY	5100	35000	11.6
61	Madhi malar	9y	F	D + WS	POSITIVE	HEPATOMEGALY	7900	30000	12
62	Manikandan	10y	M	PD	POSITIVE	NORMAL STUDY	6800	100000	11.8
63	Pranesh	8y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	4900	40000	13
64	Soundharya	6y	F	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	7500	70000	10.7
65	Vadivazhagan	5y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	8000	50000	12
66	Soundararajan	9y	M	PD	POSITIVE	GB WALL EDEMA	7900	90000	9.5
67	Yazhini	6y	F	PD	POSITIVE	NORMAL STUDY	10500	65000	9.9
68	Yogaprakash	4y	M	D + WS	POSITIVE	HEPATOMEGALY	4900	75000	11
69	Sankar	3y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	9000	70000	10.5
70	Muthukumar	3y	M	PD	POSITIVE	GB WALL EDEMA	6900	150000	10
71	Jana	2y	M	PD	POSITIVE	NORMAL STUDY	4900	250000	9.9
72	Manikandan	9y	M	PD	POSITIVE	NORMAL STUDY	6900	175000	11
73	Srimathy	5y	F	PD	POSITIVE	NORMAL STUDY	9000	90000	13
74	Dharshini	7y	F	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	5900	30000	12.9
75	Pavithra	3y	F	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	8900	5000	10
76	Prabakar	2y	M	D + WS	POSITIVE	HEPATOMEGALY	4900	25000	9.9
77	Nisha shree	5y	F	D + WS	POSITIVE	HEPATOMEGALY	5100	75000	8.5
78	Rani	12y	F	D + WS	POSITIVE	HEPATOMEGALY	7100	80000	11
79	Gokul	1y	M	D + WS	POSITIVE	HEPATOMEGALY	6900	45000	10.5
80	Neha	2y	F	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	8100	60000	8.8
81	Mubarak	7y	M	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	5100	10000	9

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	IG M	USG ABDOMEN	TC	PLATELET	HB
82	Sadhasivam	5y	M	D + WS	POSITIVE	HEPATOMEGALY	7800	90000	10.5
83	Akil	7y	M	PD	POSITIVE	NORMAL STUDY	6900	150000	12
84	Rasika	8y	F	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	6000	70000	11
85	Kamal raj	6y	M	D + WS	POSITIVE	HEPATOMEGALY	8100	35000	9.5
86	Vignesh	8y	M	D + WS	POSITIVE	NORMAL STUDY	7100	90000	9.9
87	Abbas	10y	M	D + WS	POSITIVE	NORMAL STUDY	9000	75000	12
88	Geetha shri	1y	F	PD	POSITIVE	NORMAL STUDY	5000	150000	10.5
89	Praveen	8y	M	D + WS	POSITIVE	HEPATOMEGALY	9100	60000	9.5
90	Muthusamy	5y	M	SD	POSITIVE	HEPATOMEGALY	5900	10000	11
91	Eswaran	8y	M	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	8100	60000	10.9
92	Madhumitha	6y	F	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	7100	80000	9.5
93	Shanmugapriya	8y	F	D + WS	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	6000	50000	12
94	Kavi barathy	5y	F	D + WS	POSITIVE	HEPATOMEGALY	5100	65000	11
95	Valliyappan	3y	M	SD	POSITIVE	HEPATOMEGALY,GB WALL EDEMA	9100	45000	8.9
96	Afreen	11y	M	PD	POSITIVE	NORMAL STUDY	8000	160000	9.5
97	Vanmathy	6y	F	PD	POSITIVE	GB WALL EDEMA	7100	250000	10
98	Surya	10y	M	PD	POSITIVE	NORMAL STUDY	6800	90000	12
99	Diwakar	9y	M	D + WS	POSITIVE	NORMAL STUDY	9800	45000	10.5
100	Siva shankar	4y	M	D + WS	POSITIVE	HEPATOMEGALY	10900	80000	11

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	SBR-TOTAL	SBR-DIRECT	SGOT	SGPT	ALP
1	Thirupathi	2y	M	D + WS	0.8	0.3	34	20	50
2	Nithish	7y	M	PD	0.7	0.1	20	39	45
3	Vishwa	12y	M	D + WS	0.6	0.2	98	110	90
4	Paramakudi	7y	M	PD	0.8	0.3	45	32	40
5	Pradeep	8y	M	PD	0.7	0.2	30	20	60
6	Sathya priya	4y	F	PD	0.8	0.1	35	40	55
7	Deepika	2y	F	D + WS	0.7	0.3	90	78	45
8	Hasini	6y	F	PD	1	0.2	38	60	67
9	Kishore	6y	M	PD	1.1	0.2	27	20	58
10	Aparna	4y	F	D + WS	0.6	0.3	68	58	60
11	Kousika	4y	F	PD	0.8	0.3	68	38	89
12	Giridharan	11y	F	SD	1.7	0.5	1187	980	260
13	Haneesh	4y	M	PD	0.7	0.2	57	45	68
14	Logith	2y	F	PD	0.8	0.1	35	30	56
15	Jothika	7y	F	D + WS	0.7	0.5	114	100	130
16	Akilan	5y	M	D + WS	0.8	0.3	230	98	100
17	Vishali	3y	F	SD	2.5	0.5	1200	890	390
18	Gokul raj	6y	M	PD	0.9	0.4	210	120	89
19	Parameshwari	8y	F	PD	0.6	0.1	65	70	55
20	Arun kumar	7y	M	PD	0.5	0.1	120	90	60
21	Satheesh	10y	M	PD	0.8	0.2	45	30	65
22	Ayyadurai	8y	M	PD	0.7	0.3	60	39	65
23	Sabari	11y	M	PD	0.7	0.2	35	20	88
24	Hema	7y	F	D + WS	0.6	0.1	60	30	78
25	Suchithra	4y	F	D + WS	0.8	0.2	110	90	69
26	Suresh	6y	M	PD	0.5	0.3	150	110	120

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	SBR-TOTAL	SBR-DIRECT	SGOT	SGPT	ALP
27	Priyadharshini	4y	F	PD	0.9	0.3	169	130	110
28	Jeya shree	10y	F	PD	0.7	0.2	157	178	230
29	Dhanush	5y	M	D + WS	0.8	0.4	467	390	240
30	Boopathy	5y	M	D + WS	0.6	0.2	55	40	55
31	Nandhini	9y	F	D + WS	0.5	0.1	110	59	80
32	Vijay	5y	M	SD	2.1	0.5	1150	1050	690
33	Kavyashree	3y	F	D + WS	0.6	0.2	48	35	60
34	Soumya	4y	F	D + WS	0.7	0.3	35	20	55
35	Bharath kumar	10y	M	PD	0.8	0.1	65	60	90
36	Shruthika	3y	F	D + WS	0.6	0.2	119	120	110
37	Iniyan	3y	M	SD	1.4	0.6	1500	900	980
38	Lavanya	4y	F	PD	0.6	0.3	120	95	230
39	Leenashree	6y	F	PD	0.5	0.2	50	38	55
40	Rajkumar	5y	M	PD	0.7	0.1	60	46	60
41	Monika	6y	F	D + WS	0.5	0.3	210	130	120
42	Gowtham	3y	M	D + WS	0.7	0.2	357	210	110
43	Sakthi	2y	M	D + WS	0.6	0.4	150	90	130
44	Samaya	5y	F	D + WS	0.6	0.1	170	140	90
45	Hariharasudhan	3y	F	D + WS	0.7	0.2	60	50	56
46	Poornima	2y	F	PD	0.6	0.3	100	68	88
47	Kamalesh	4y	M	PD	0.7	0.2	120	45	78
48	Desigan	5y	M	PD	0.8	0.1	138	120	69
49	Raghul	11y	M	PD	0.6	0.2	120	110	82
50	Santhosh	12y	M	D + WS	0.7	0.3	678	460	360
51	Santhosh kumar	4y	M	D + WS	0.6	0.2	786	670	990
52	Anbarasan	5y	M	D + WS	0.5	0.1	399	422	560

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	SBR-TOTAL	SBR-DIRECT	SGOT	SGPT	ALP
53	Mahammed siddiq	2y	M	PD	0.8	0.2	120	124	97
54	Sandhiya	9y	F	PD	0.6	0.2	356	280	88
55	Jeevitha	5y	F	PD	0.7	0.4	130	96	65
56	Sindhu	10y	F	PD	0.6	0.3	34	33	76
57	Amudhini	6y	F	SD	1.7	0.6	1200	994	680
58	Sri gugan	3y	M	D + WS	0.8	0.1	64	120	90
59	Dharaneeshwaran	12y	M	D + WS	0.7	0.1	28	33	56
60	Susi baskar	4y	M	D + WS	0.8	0.2	55	45	67
61	Madhi malar	9y	F	D + WS	0.6	0.1	35	20	82
62	Manikandan	10y	M	PD	0.5	0.3	42	34	56
63	Pranesh	8y	M	D + WS	0.7	0.2	50	43	66
64	Soundharya	6y	F	D + WS	0.6	0.2	39	40	87
65	Vadivazhagan	5y	M	D + WS	0.6	0.1	165	120	59
66	Soundararajan	9y	M	PD	0.7	0.3	210	190	120
67	Yazhini	6y	F	PD	0.6	0.2	45	40	61
68	Yogaprakash	4y	M	D + WS	0.5	0.3	68	60	76
69	Sankar	3y	M	D + WS	0.8	0.2	409	380	158
70	Muthukumar	3y	M	PD	0.7	0.1	100	90	90
71	Jana	2y	M	PD	0.7	0.3	68	45	84
72	Manikandan	9y	M	PD	0.6	0.2	42	30	69
73	Srimathy	5y	F	PD	0.6	0.3	36	26	76
74	Dharshini	7y	F	SD	1.6	0.6	115	40	90
75	Pavithra	3y	F	SD	1.5	0.5	630	560	390
76	Prabakar	2y	M	D + WS	0.8	0.2	150	120	100
77	Nisha shree	5y	F	D + WS	0.6	0.1	120	100	150
78	Rani	12y	F	D + WS	0.7	0.3	290	180	180

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	SBR-TOTAL	SBR-DIRECT	SGOT	SGPT	ALP
79	Gokul	1y	M	D + WS	0.8	0.2	260	210	150
80	Neha	2y	F	D + WS	0.6	0.1	110	95	99
81	Mubarak	7y	M	SD	1.9	0.5	1390	1100	590
82	Sadhasivam	5y	M	D + WS	0.7	0.2	60	30	55
83	Akil	7y	M	PD	0.8	0.3	27	23	60
84	Rasika	8y	F	SD	0.7	0.2	980	890	780
85	Kamal raj	6y	M	D + WS	0.8	0.1	450	360	210
86	Vignesh	8y	M	D + WS	0.6	0.2	38	29	55
87	Abbas	10y	M	D + WS	0.7	0.1	58	35	68
88	Geetha shri	1y	F	PD	0.6	0.3	60	45	73
89	Praveen	8y	M	D + WS	0.5	0.2	150	90	88
90	Muthusamy	5y	M	SD	1.6	0.5	890	780	680
91	Eswaran	8y	M	D + WS	0.6	0.2	356	210	130
92	Madhumitha	6y	F	D + WS	0.7	0.1	124	98	110
93	Shanmugapriya	8y	F	D + WS	0.6	0.3	560	390	260
94	Kavi barathy	5y	F	D + WS	0.6	0.2	35	20	56
95	Valliyappan	3y	M	SD	1.1	0.5	55	34	87
96	Afreen	11y	M	PD	0.6	0.3	123	78	90
97	Vanmathy	6y	F	PD	0.8	0.2	45	40	76
98	Surya	10y	M	PD	0.7	0.1	34	30	85
99	Diwakar	9y	M	D + WS	0.6	0.2	40	28	59
100	Siva shankar	4y	M	D + WS	0.7	0.3	120	90	72

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	PT	APTT	S.PROTEIN	ALBUMIN	GLOBULIN
1	Thirupathi	2y	M	D + WS	NORMAL	NORMAL	6.8	4	2.8
2	Nithish	7y	M	PD	NORMAL	NORMAL	7.2	4.5	2.7
3	Vishwa	12y	M	D + WS	NORMAL	NORMAL	6.9	4.4	2.5
4	Paramakudi	7y	M	PD	NORMAL	NORMAL	6.5	4.6	1.9
5	Pradeep	8y	M	PD	NORMAL	NORMAL	7.3	5	2.3
6	Sathya priya	4y	F	PD	NORMAL	NORMAL	8.1	3.8	4.3
7	Deepika	2y	F	D + WS	NORMAL	NORMAL	7.5	3.9	3.6
8	Hasini	6y	F	PD	NORMAL	NORMAL	7.3	4.1	3.2
9	Kishore	6y	M	PD	NORMAL	NORMAL	6.9	4.9	2
10	Aparna	4y	F	D + WS	NORMAL	NORMAL	6.8	4	2.8
11	Kousika	4y	F	PD	NORMAL	NORMAL	7.1	3.9	3.2
12	Giridharan	11y	F	SD	INCREASED	INCREASED	8.2	3.5	4.7
13	Haneesh	4y	M	PD	NORMAL	NORMAL	7.8	3.8	4
14	Logith	2y	F	PD	NORMAL	NORMAL	6.6	4	2.6
15	Jothika	7y	F	D + WS	NORMAL	NORMAL	7.4	4.5	2.9
16	Akilan	5y	M	D + WS	NORMAL	NORMAL	7.7	4	3.7
17	Vishali	3y	F	SD	INCREASED	INCREASED	5.9	4.2	1.7
18	Gokul raj	6y	M	PD	NORMAL	NORMAL	8	4.9	3.1
19	Parameshwari	8y	F	PD	NORMAL	NORMAL	7.8	4.5	3.3
20	Arun kumar	7y	M	PD	NORMAL	NORMAL	7.44	5	2.44
21	Satheesh	10y	M	PD	NORMAL	NORMAL	6.9	3.9	3
22	Ayyadurai	8y	M	PD	NORMAL	NORMAL	6.6	4.1	2.5
23	Sabari	11y	M	PD	NORMAL	NORMAL	8.1	4	4.1
24	Hema	7y	F	D + WS	NORMAL	NORMAL	7.8	4.3	3.5
25	Suchithra	4y	F	D + WS	NORMAL	NORMAL	7.9	5	2.9

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	PT	APTT	S.PROTEIN	ALBUMIN	GLOBULIN
26	Suresh	6y	M	PD	NORMAL	NORMAL	7.5	4.9	2.6
27	Priyadharshini	4y	F	PD	NORMAL	NORMAL	8	5.2	2.8
28	Jeya shree	10y	F	PD	NORMAL	NORMAL	8	5	3
29	Dhanush	5y	M	D + WS	NORMAL	NORMAL	7.8	4.8	3
30	Boopathy	5y	M	D + WS	NORMAL	NORMAL	7.1	4.9	2.2
31	Nandhini	9y	F	D + WS	NORMAL	NORMAL	7.5	4.4	3.1
32	Vijay	5y	M	SD	INCREASED	INCREASED	8	3.5	4.5
33	Kavyashree	3y	F	D + WS	NORMAL	NORMAL	6.9	3.6	3.3
34	Soumya	4y	F	D + WS	NORMAL	NORMAL	7.5	3.9	3.6
35	Bharath kumar	10y	M	PD	NORMAL	NORMAL	6.9	4.1	2.8
36	Shruthika	3y	F	D + WS	NORMAL	NORMAL	7	4.4	2.6
37	Iniyan	3y	M	SD	INCREASED	INCREASED	4.9	3.9	1
38	Lavanya	4y	F	PD	NORMAL	NORMAL	7.5	3.8	3.7
39	Leenashree	6y	F	PD	NORMAL	NORMAL	7.4	4.1	3.3
40	Rajkumar	5y	M	PD	NORMAL	NORMAL	6.8	4.5	2.3
41	Monika	6y	F	D + WS	NORMAL	NORMAL	6.6	3.6	3
42	Gowtham	3y	M	D + WS	NORMAL	NORMAL	7.1	3.9	3.2
43	Sakthi	2y	M	D + WS	NORMAL	NORMAL	7.7	4	3.7
44	Samaya	5y	F	D + WS	NORMAL	NORMAL	6.9	4.2	2.5
45	Hariharasudhan	3y	F	D + WS	NORMAL	NORMAL	6.6	4.4	2.2
46	Poornima	2y	F	PD	NORMAL	NORMAL	8	5.1	2.9
47	Kamalesh	4y	M	PD	NORMAL	NORMAL	7.5	4.8	2.7
48	Desigan	5y	M	PD	NORMAL	NORMAL	7.8	4.6	3.2
49	Raghul	11y	M	PD	NORMAL	NORMAL	7	4.9	2.1
50	Santhosh	12y	M	D + WS	INCREASED	INCREASED	5.2	3.8	1.4

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	PT	APTT	S.PROTEIN	ALBUMIN	GLOBULIN
51	Santhosh kumar	4y	M	D + WS	INCREASED	INCREASED	5.5	3.2	2.3
52	Anbarasan	5y	M	D + WS	INCREASED	INCREASED	7.2	3.5	3.7
53	Mahammed siddiq	2y	M	PD	NORMAL	NORMAL	6.9	4	2.9
54	Sandhiya	9y	F	PD	NORMAL	NORMAL	6.6	3.6	3
55	Jeevitha	5y	F	PD	NORMAL	NORMAL	7.5	3.8	3.7
56	Sindhu	10y	F	PD	NORMAL	NORMAL	7	3.9	3.1
57	Amudhini	6y	F	SD	INCREASED	INCREASED	5.9	4.2	1.7
58	Sri gugan	3y	M	D + WS	NORMAL	NORMAL	6.9	4.4	2.5
59	Dharaneeshwaran	12y	M	D + WS	NORMAL	NORMAL	7.2	3.9	3.3
60	Susi baskar	4y	M	D + WS	NORMAL	NORMAL	7	3.8	3.2
61	Madhi malar	9y	F	D + WS	NORMAL	NORMAL	6.1	4.2	1.9
62	Manikandan	10y	M	PD	NORMAL	NORMAL	8.2	4.8	3.4
63	Pranesh	8y	M	D + WS	NORMAL	NORMAL	6.8	3.9	2.9
64	Soundharya	6y	F	D + WS	NORMAL	NORMAL	7.5	4.2	3.3
65	Vadivazhagan	5y	M	D + WS	NORMAL	NORMAL	8	4.4	3.6
66	Soundararajan	9y	M	PD	NORMAL	NORMAL	7.9	4.8	3.1
67	Yazhini	6y	F	PD	NORMAL	NORMAL	6.9	3.9	3
68	Yogaprakash	4y	M	D + WS	NORMAL	NORMAL	7	3.5	3.5
69	Sankar	3y	M	D + WS	NORMAL	NORMAL	5.2	3.3	1.9
70	Muthukumar	3y	M	PD	NORMAL	NORMAL	6.6	4	2.6
71	Jana	2y	M	PD	NORMAL	NORMAL	6.8	4.1	2.7
72	Manikandan	9y	M	PD	NORMAL	NORMAL	7.8	4.7	3.1
73	Srimathy	5y	F	PD	NORMAL	NORMAL	7.5	3.8	3.7
74	Dharshini	7y	F	SD	NORMAL	NORMAL	6.4	4.2	2.2
75	Pavithra	3y	F	SD	NORMAL	NORMAL	5.5	3.5	2

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	PT	APTT	S.PROTEIN	ALBUMIN	GLOBULIN
76	Prabakar	2y	M	D + WS	NORMAL	NORMAL	7	3.6	3.4
77	Nisha shree	5y	F	D + WS	NORMAL	NORMAL	8.2	3.9	4.3
78	Rani	12y	F	D + WS	NORMAL	NORMAL	7.8	4.2	3.6
79	Gokul	1y	M	D + WS	NORMAL	NORMAL	7.7	4.4	3.3
80	Neha	2y	F	D + WS	NORMAL	NORMAL	6.9	4.9	2
81	Mubarak	7y	M	SD	INCREASED	INCREASED	5.8	3.1	2.7
82	Sadhasivam	5y	M	D + WS	NORMAL	NORMAL	8	3.9	4.1
83	Akil	7y	M	PD	NORMAL	NORMAL	7.6	3.5	4.1
84	Rasika	8y	F	SD	INCREASED	INCREASED	6.2	3.9	2.3
85	Kamal raj	6y	M	D + WS	NORMAL	NORMAL	7.9	3.5	4.4
86	Vignesh	8y	M	D + WS	NORMAL	NORMAL	7.8	4	3.8
87	Abbas	10y	M	D + WS	NORMAL	NORMAL	8	4.1	3.9
88	Geetha shri	1y	F	PD	NORMAL	NORMAL	6.8	4.2	2.6
89	Praveen	8y	M	D + WS	NORMAL	NORMAL	6	3.8	2.2
90	Muthusamy	5y	M	SD	INCREASED	INCREASED	6.9	3.6	3.3
91	Eswaran	8y	M	D + WS	NORMAL	NORMAL	6.8	3.5	3.3
92	Madhumitha	6y	F	D + WS	NORMAL	NORMAL	7.1	4	3.1
93	Shanmugapriya	8y	F	D + WS	NORMAL	NORMAL	7.5	4.2	3.3
94	Kavi barathy	5y	F	D + WS	NORMAL	NORMAL	8.1	4.4	3.7
95	Valliyappan	3y	M	SD	NORMAL	NORMAL	7.8	4.1	3.7
96	Afreen	11y	M	PD	NORMAL	NORMAL	7.7	3.9	3.8
97	Vanmathy	6y	F	PD	NORMAL	NORMAL	7.8	3.8	4
98	Surya	10y	M	PD	NORMAL	NORMAL	6.9	4.2	2.7
99	Diwakar	9y	M	D + WS	NORMAL	NORMAL	6.5	4.4	2.1
100	Siva shankar	4y	M	D + WS	NORMAL	NORMAL	7.1	3.9	3.2

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	PSS FOR MP	IG M ANTI HAV	HBsAG STATUS	HCV STATUS	WIDAL	MAT FOR LEPTO-SPIROSIS	OUTCOME
1	Thirupathi	2y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
2	Nithish	7y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
3	Vishwa	12y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
4	Paramakudi	7y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
5	Pradeep	8y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
6	Sathya priya	4y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
7	Deepika	2y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
8	Hasini	6y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
9	Kishore	6y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
10	Aparna	4y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
11	Kousika	4y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
12	Giridharan	11y	F	SD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
13	Haneesh	4y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
14	Logith	2y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
15	Jothika	7y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
16	Akilan	5y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
17	Vishali	3y	F	SD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
18	Gokul raj	6y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
19	Parameshwari	8y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
20	Arun kumar	7y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
21	Satheesh	10y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
22	Ayyadurai	8y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
23	Sabari	11y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
24	Hema	7y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	PSS FOR MP	IG M ANTI HAV	HBsAG STATUS	HCV STATUS	WIDAL	MAT FOR LEPTO-SPIROSIS	OUTCOME
25	Suchithra	4y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
26	Suresh	6y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
27	Priyadharshini	4y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
28	Jeya shree	10y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
29	Dhanush	5y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
30	Boopathy	5y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
31	Nandhini	9y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
32	Vijay	5y	M	SD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
33	Kavyashree	3y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
34	Soumya	4y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
35	Bharath kumar	10y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
36	Shruthika	3y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
37	Iniyan	3y	M	SD	Negative	Negative	Negative	Negative	Negative	Negative	Expired
38	Lavanya	4y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
39	Leenashree	6y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
40	Rajkumar	5y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
41	Monika	6y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
42	Gowtham	3y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
43	Sakthi	2y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
44	Samaya	5y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
45	Hariharasudhan	3y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
46	Poornima	2y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
47	Kamalesh	4y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
48	Desigan	5y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	PSS FOR MP	IG M ANTI HAV	HBsAG STATUS	HCV STATUS	WIDAL	MAT FOR LEPTO-SPIROSIS	OUTCOME
49	Raghul	11y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
50	Santhosh	12y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
51	Santhosh kuma	4y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
52	Anbarasan	5y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
53	Mahammed sic	2y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
54	Sandhiya	9y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
55	Jeevitha	5y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
56	Sindhu	10y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
57	Amudhini	6y	F	SD	Negative	Negative	Negative	Negative	Negative	Negative	Expired
58	Sri gugan	3y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
59	Dharaneeshwa	12y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
60	Susi baskar	4y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
61	Madhi malar	9y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
62	Manikandan	10y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
63	Pranesh	8y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
64	Soundharya	6y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
65	Vadivazhagan	5y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
66	Soundararajan	9y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
67	Yazhini	6y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
68	Yogaprakash	4y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
69	Sankar	3y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
70	Muthukumar	3y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
71	Jana	2y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
72	Manikandan	9y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	PSS FOR MP	IG M ANTI HAV	HBsAG STATUS	HCV STATUS	WIDAL	MAT FOR LEPTO-SPIROSIS	OUTCOME
73	Srimathy	5y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
74	Dharshini	7y	F	SD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
75	Pavithra	3y	F	SD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
76	Prabakar	2y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
77	Nisha shree	5y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
78	Rani	12y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
79	Gokul	1y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
80	Neha	2y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
81	Mubarak	7y	M	SD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
82	Sadhasivam	5y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
83	Akil	7y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
84	Rasika	8y	F	SD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
85	Kamal raj	6y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
86	Vignesh	8y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
87	Abbas	10y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
88	Geetha shri	1y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
89	Praveen	8y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
90	Muthusamy	5y	M	SD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
91	Eswaran	8y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
92	Madhumitha	6y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
93	Shanmugapriya	8y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
94	Kavi barathy	5y	F	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
95	Valliyappan	3y	M	SD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
96	Afreen	11y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered

SL. NO.	NAME OF THE CHILD	AGE	GENDER	DIAGNOSIS	PSS FOR MP	IG M ANTI HAV	HBsAG STATUS	HCV STATUS	WIDAL	MAT FOR LEPTO-SPIROSIS	OUTCOME
97	Vanmathy	6y	F	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
98	Surya	10y	M	PD	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
99	Diwakar	9y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered
100	Siva shankar	4y	M	D + WS	Negative	Negative	Negative	Negative	Negative	Negative	Recovered