CLINICAL PROFILE OF DENGUE IN ADULT PATIENTS AGED MORE THAN 13 YEARS

Dissertation submitted to

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

CHENNAI – TAMILNADU

In partial fulfillment for the Degree of

DOCTOR OF MEDICINE

BRANCH I - M.D., (General Medicine)

APRIL - 2014

DEPARTMENT OF MEDICINE

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI – 627011

TAMILNADU

CERTIFICATE

This is to certify that this dissertation titled **"CLINICAL PROFILE OF DENGUE IN ADULT PATIENTS AGED MORE THAN 13 YEARS"** submitted by **DR. N.R. RIA MOL** to the Tamil Nadu Dr. M.G.R. Medical University, Chennai is in partial fulfillment of the requirement for 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.

DR.L.RAJAGOPALA MARTHANDAM M.D.,
Professor,
Department of Medicine,
Tirunelveli Medical College,
Tirunelveli.

DR. R.GEETHA RANI M.D.,
Professor and Head,
Department of Medicine,
Tirunelveli Medical College,
Tirunelveli.

The DEAN,
Tirunelveli Medical College,
Tirunelveli.

Tirunelveli
Date:

TIRUNELVELI MEDICAL COLLEGE

TIRUNELVELI,

STATE OF TAMILNADU, INDIA

PIN CODE:627011

Tel: 91-462-2572733, 2572734 Fax: 91-462-2572944

Estd:1965

Under the Directorate of Medical Education, Government of Tamilnadu.

Institutional Ethical Committee

Certificate of Approval

This is to certify that the Institutional Ethical Committee of this College unanimously approves the Thesis /Dissertation/ Research Proposal submitted before this committee by Dr. N.R.RIAMOL, **A POST GRADUATE IN MD MEDICINE,** Department of **MEDICINE** of Tirunelveli Medical College /Hospital, Tirunelveli titled **"STUDY ON CLINICAL PROFILE OF DENGUE FEVER IN ADULT MORE THAN 13 YEARS"** registered by the IEC as 195/G.M./IEC/2012 dated. 11.07.2012. The Investigator is hereby advised to adhere to all the stipulated norms and conditions of this ethical committee.

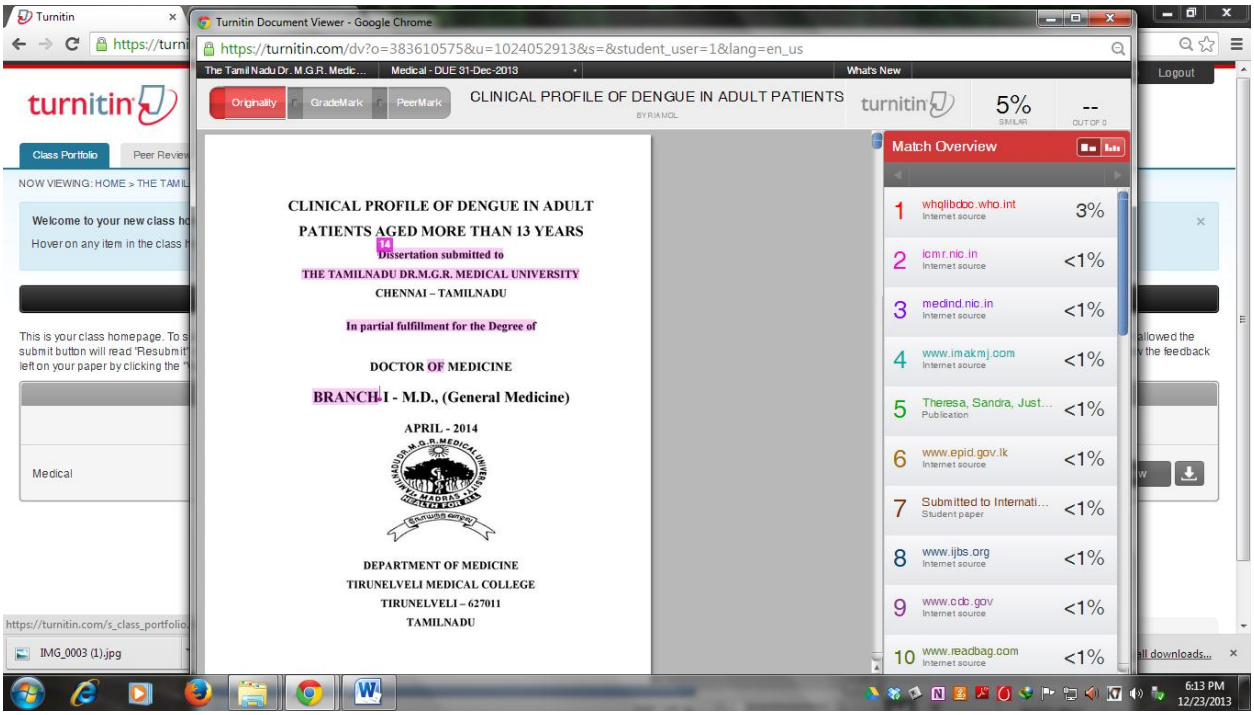
Secretary

Issued on this Date

11.07.2012

Under Seal

TIRUNELVELI MEDICAL COLLEGE ★ TAMILNADU INDIA ★ TIRUNELVELI-11


Turnitin
Turnitin Document Viewer - Google Chrome
https://turnitin.com/dv?o=383610575&u=1024052913&s=&student_user=1&lang=en_us
CLINICAL PROFILE OF DENGUE IN ADULT PATIENTS
5%
Match Overview
CLINICAL PROFILE OF DENGUE IN ADULT PATIENTS AGED MORE THAN 13 YEARS
Dissertation submitted to
THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY
CHENNAI – TAMILNADU
In partial fulfillment for the Degree of
DOCTOR OF MEDICINE
BRANCH I - M.D., (General Medicine)
APRIL - 2014
DEPARTMENT OF MEDICINE
TIRUNELVELI MEDICAL COLLEGE
TIRUNELVELI – 627011
TAMILNADU
1 whqlibdoc.who.int 3%
2 icmr.nic.in <1%
3 medind.nic.in <1%
4 www.imakmj.com <1%
5 Theresa, Sandra, Just... <1%
6 www.epid.gov.lk <1%
7 Submitted to Internati... <1%
8 www.ijbs.org <1%
9 www.cdc.gov <1%
10 www.readbag.com <1%
6:13 PM
12/23/2013

DECLARATION

I **DR. RIA MOL N.R** declare that I carried out this work on **"CLINICAL PROFILE OF DENGUE IN ADULT PATIENTS AGED MORE THAN 13 YEARS**" at Department of General Medicine, Tirunelveli Medical College and Hospital during the period of April 2012 – March 2013. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, and diploma to any university, board either in India or abroad. This is submitted to the Tamilnadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulation for the M.D. Degree examination in General Medicine.

Tirunelveli Medical College, **DR. RIA MOL N.R**

Tirunelveli.

Date:

ACKNOWLEDGEMENT

I owe my thanks to the Dean, Government Tirunelveli Medical College and Hospital, **DR.S.SOUNDARARAJAN M.S.** for allowing me to avail the facilities needed for my dissertation work.

I am grateful to **Dr.R.Geetharani M.D**, Professor and Head of the Department of Medicine, Government Tirunelveli Medical College and Hospital, for her constant support, guidance and encouragement during every step of this study.

I express my gratitude to my Guide and Unit Chief **Prof. Dr.L.Rajagopala Marthandam M.D.,** for his valuable time, guidance and for being a constant source of inspiration and support.

I sincerely thank my unit Assistant Professors **Dr.S.Madhavan M.D., Dr.J.Bharath M.D.,** and **Dr.A.Thomas Edwin Raj M.D.,** for their encouragement and invaluable support in completing this study.

I am extremely thankful to my fellow postgraduates and friends in the Department of Medicine for their understanding, support and encouragement.

I thank God Almighty for his blessings and my family that I could not be where I am without their encouragement, unconditional love and support.

I am also thankful to my colleagues for their full cooperation in this study and my sincere thanks to all the patients and their family who co-operated for this study.

CONTENTS

LIST OF ABBREVIATIONS

AST	**- Serum Aspartate Transaminase**
ALT	**- Serum Alanine Transaminase**
CBC	**- Complete Blood Count**
CT	**- Computed Tomography**
CNS	**- Central Nervous System**
CXR	**- Chest X-Ray**
DCF	**- Dengue Classical Fever**
DF	**- Dengue Fever**
DIC	**- Disseminated intravascular coagulation**
DSS	**- Dengue Shock Syndrome**
DHF	**- Dengue Hemorrhagic Fever**
ELISA	**- Enzyme-Linked Immunosorbent Assay**
GTCS	**- Generalized Tonic Clonic Seizures**
HCT	**- Haematocrit**
IgG	**- Immunoglobulin G**
IgM	**- Immunoglobulin M**
LFT	**- Liver function Test**
NS1	**- Non structural Protein 1**
RFT	**- Renal Function Test**
USG	**- Ultra SonoGraphy**

ABSTRACT

Dengue Fever, known commonly as Break bone fever is the most common Arboviral mosquito borne disease in the world. This dengue virus is spread by the bite of infected Aedes mosquito, most commonly Ae. aegypti. Many countries especially the countries of the Indian subcontinent have suffered at the hands of this disease Dengue has a varied and wide spectrum of clinical presentations, often with unpredictable clinical evolution and outcome. This study is an attempt to derive the clinical profile of Dengue infection from the current epidemic that stormed Tirunelveli district. To identify some peculiar features that may help in early recognition and appropriate case management. The present study is an observational study where we studied the clinical profile of 251 serologically proven dengue patients admitted in the Department of Medicine, Tirunelveli Medical College from April 2012-March 2013.

Out of 251 cases, 149 patients (59.40%) belonged to DCF, DHF in 76 patients (30.30%), where as 26 patients (10.40%) belonged to more severe variety of DSS. Majority of cases 40.20% occurred in young adult <20 years of age. The incidence appeared to reduce with advancing age with least number of cases seen in the age group >60 years of age. DCF (44.30%) and DSS (42.30%) was more common in the younger age group <20 years.DHF (39.50%) was more common in age group of 20-29 years where as DSS is not observed in patients >60 years in this study. Distribution of dengue in females was slightly

higher 51.40% when compared to males 48.60%. In this study all patients had fever 100%. Followed by headache 61%, Myalgia 54.60%, chills 46.20%, abdominal pain 43.40%, vomiting 42.20%.The characteristic feature of dengue like bone pain and retro-orbital pain was present in only 3.20% and 32.70% respectively. Atypical clinical feature like seizure was present in 1.20%. Most common sign observed in this study was conjunctival congestion 23.90%, followed by Hepatomegaly 12%, Ascites 10%, Rashes 10%, Pleural effusion 9.60%, Puffiness of the face and Splenomegaly 8.40%. Most common bleeding manifestation encountered was malena 27.50% followed by petechiae 8.40% and gum bleeding in 4.80% less frequent was bleeding manifestation hemoptysis 0.40%. BP is the most important clinical monitor in a case of dengue for identifying onset of complications like shock. Pulse pressure is more important than BP in identifying early stage shock. Narrowed pulse pressure (< 20 mm of Hg) is the most sensitive sign.

87 cases showed enzyme abnormalities in 100% cases with LFT abnormality which was similar to our study. This could be due to direct injury to liver cells by the virus or due to immunological response. Ischemic hepatitis in patients especially in shock could be another possible etiology USG was more sensitive in picking up Pleural effusion.

Out of 251 patients mortality rate was 0.8% Recovery rate was 99.20%. The cause of death was intra cerebral bleed/ encephalopathy/seizures / DSS/

DIC/Refractory shock in one case. The other case succumbed due to massive malena/encephalopathy/DSS/DIC/refractory shock.

Key Words: Dengue, DHF, DSS.

Dengue Fever, known commonly as Break bone fever is the most common Arboviral mosquito borne disease in the world. Many countries especially the countries of the Indian subcontinent have suffered at the hands of this disease. Epidemiology of Dengue Fever in Indian subcontinent is very complex. It has changed over the last few years with regard to the strains, affected regions and disease severity.

Dengue has a varied and wide spectrum of clinical presentations, often with unpredictable clinical evolution and outcome. Most of the patients will recover following a self-limiting, less severe clinical course, where as a small proportion of patients with dengue infection, progress to severe disease, characterized by plasma leakage, with or without hemorrhagic manifestations.

Intravenous rehydration of the patient is the treatment of choice. By this simple intervention, case fatality rate is reduced to less than 1% even in severe cases.

Clinical profile of dengue fever may vary with each epidemic because of the numerous strains available, varied possibilities of co-infections according to the geography and also due to the vector density of the particular area of outbreak.

This study is an attempt to derive the clinical profile of Dengue infection from the current epidemic that stormed Tirunelveli district. An effort is also made to identify some peculiar clinical features that may help us to identify those seemingly simple cases that worsen without any warning signs, so that we may reduce the serious morbidity and mortality associated with this disease.

Fig: 1 Aedes aegypti (Tiger Mosquito)

AIM

1) To study the clinical profile of serologically proven dengue fever in Tirunelveli Medical College hospital.

2) To identify some peculiar features that may help in early recognition and appropriate case management which all together helps in bringing out a good clinical outcome.

REVIEW OF LITERATURE

HISTORY:

Dengue is one of the most common mosquito-borne arbo viral diseases. History of dengue dates back from the period of Jin dynasty. Initially it was referred as WATER POISON as it was thought to be spread by flying insects. The word DENGUE is derived from the Swahili phrase KA-DINGA PEPO which means "Cramps like seizure". Later Benjamin Rush called it "BREAK BONE FEVER "as it causes severe arthralgia and myalgia[1].

INCIDENCE:

Its incidence has been in increasing trend in the recent years. About 2.5 billion people reside in dengue endemic countries and its annual incidence is estimated to be around fifty million[2].

Dengue has shifted its focus from urban and peri urban areas to rural areas[3,4]. And also it is said to exhibit seasonal variations[5] which is determined by the characteristics of the vector, agent and host.

DENGUE IN INDIA:

The epidemiology of Dengue Fever in Indian subcontinent is very complex. It has changed over the last few years with regard to the strains, affected regions and disease[6] severity.

Dengue was first reported in India in 1946[7].Then for the next 18 years, there was no further epidemic. In 1963 to 1964, an outbreak was reported in the Eastern coast of our country, which then spread and reached Delhi by 1967[8]. Gradually it involved the entire country.

The Kanpur epidemic[9] in 1967 was due to DV-4 and in 1969[10] epidemic, both DV-2 and 4 were isolated. The Vellore epidemic [11,12] of 1966 was reported to be due to DV-3 strain.DV-3 was also reported from Calcutta[13] in 1983, Gwalior In 2003, 2004[14,15] and at Tirupur, Tamilnadu in 2010[16].

The outbreaks in Delhi during 1996 reported DV-2 and in 2003 it was due to DV-2 and DV-3 strains. At Delhi till 2003[15] DV-2 was the predominant serotype reported, which was then replaced by DV1 strain for about a period of three years from 2007 to 2009[17]. Concurrent infection by Chikungunya and DV-2 was reported from Vellore[18] and Delhi[19].

The emergence of DV-4 which was related to increased severity was reported in Andhra Pradesh in 2007[20] and in Pune 2009 to 2010[21]. Phylogenetic analysis revealed that the strains isolated from South India were closely related to Gwalior and Delhi isolates.

Approximate number of Dengue cases and death reported during 2012[22] were

Table: 1

States, Union Territories that were most affected in 2012		
States, Union Territories	No of Cases	Deaths
Tamilnadu	12826	66
West Bengal	6456	11
Kerala	4172	15
Karnataka	3924	21
Delhi	2093	04
Puducherry	3506	05

Case fatality rate in India approximately ranges from 3-5% [23]

AGENT:

Dengue virus is a single-stranded RNA virus. It has four serotypes Dengue1,2,3,4. They belong to the Flavivirus genus of family "Flaviviridae"[24,25].

This spherical shaped virus is of 50 nm diameter. It has multiple copies of 3 structural proteins, a bilayered membrane derived from host and a single copy of single stranded RNA.

This RNA is cleaved by host and viral proteases into 3 structural proteins: a capsid C, a prM (the precursor of membrane M) and a protein and envelope E. It also contains 7 nonstructural proteins. Each serotype has many distinct genotypes within. Among them genotypes DEN-2 and DEN-3 are known to cause serious infections[26,27,28]. Intra host viral diversity (QUASISPECIES) is also seen in human hosts.

VECTOR:

This dengue virus is spread by the bite of infected Aedes mosquito, most commonly Ae. aegypti [29, 30]. This mosquito is mostly seen in tropical and sub tropical countries. Since it cannot withstand lower temperatures, it is uncommon above 1000 meters[31]. The immature stages inhabit artificial collection of water nearby human dwellings. Dengue epidemics have also been occurred due to Aedes polynesiensis, Aedes albopictus and Aedes cutellaris complex[32]. The eggs can remain viable for long period even without water[33].

HOST:

Incubation period of dengue fever is about 4-10 days. The dengue virus enters the host through cutaneous route when an infected mosquito is sucking its blood meal. Following infection with dengue virus, the patient can have varied clinical presentations, although many remain asymptomatic. Primary infection with any one of the serotype will result in lifelong immunity for that particular serotype[34].

Following primary infection, the infected individuals are protected from other serotypes for about 2-3 months but there will be no long-term cross-protectivity[35,36]. Monocyte/macrophages play a key role in the pathogenesis of DF and then to the origin of DHF and DSS. Infection with a heterologous dengue virus may lead to the production of antibodies that are non protective. These sub neutralizing levels of antibodies enhances the infectivity of virus [37-40].

Cross-reactive memory T cells that are rapidly activated during the course of secondary infection. This activated T cells proliferate, express cytokines and undergo apoptosis in a manner producing plasma leakage from capillaries, thereby causing haemoconcentration and abnormalities in homeostatic mileau, and thus leading to severe dengue [41].

In the course of secondary infection, antibody-dependent enhancement (ADE) of infection was proposed as a mechanism in the causation of severe dengue[42,43]. This explains the reason why severe dengue is regularly found during primary infection in infants born to dengue-immune mothers. In this model, non-neutralizing cross-reactive antibodies that are formed during primary infection, or that are acquired passively at birth, bind to the epitopes present on the surface of the heterologous infecting virus and this in turn facilitates virus entry into Fc-receptor-bearing cells[44,45,46].

The number of infected cells is therefore increased and this results in a high viral burden and induction of a profound host immune response that includes release of inflammatory mediators, some of which are attributed to cause capillary leakage.

The clinical outcome of infection in an individual is also said to be influenced by the host genetic determinants but this issue is not adequately addressed in most of the studies[47,48]. The severity of the disease is also influenced by the time interval between the infections and the specific viral sequence of the infection.

TRANSMISSION OF THE DENGUE VIRUS:

The main amplifying hosts of the virus are humans. Dengue virus circulating in the blood of infected humans during the acute phase of illness is ingested by female Aedes mosquitoes during feeding. The ingested virus then infects the mosquito's mid-gut and over a period of 8-12 days, systemic spread occurs. After this period of extrinsic incubation, the dengue virus can be transmitted to other humans during subsequent probing or feeding. Environmental conditions, especially ambient temperature influence this extrinsic incubation period.

Infected mosquito spreads the disease throughout its lifetime; its distribution is an important factor in determining the epidemic. Aedes mosquitoes bites several times before completing the oogenesis, and thrive in close proximity to humans. Being highly anthropophilic, Ae. aegypti is one of the most efficient vectors associated with arboviral transmission.

Vertical transmission[49] (transovarial transmission) of dengue virus has also been demonstrated in the laboratory but not very often in the field. The concept of vertical transmission in the maintenance of the dengue virus is also not well understood. Apart from the environmental and climatic conditions, the dynamics of viral transmission is also influenced by host-

pathogen interactions and the immunological factors that are prevalent in a particular population[50].

Fig: 2

Fig: 3

Fig: 4

Breeding Source of Aedes Mosquito

THROMBOCYTOPENIA IN DENGUE INFECTION:

Thrombocytopenia occurs in dengue as a result of

1. Alterations in megakaryocytopoiesis when human haematopoietic cells are infected with dengue virus, resulting in impaired progenitor cell growth thereby causing platelet dysfunction.

2. Enhanced destruction of platelets.

3. Increased consumption (due to peripheral sequestration) of platelets.

Hemorrhage can occur as a consequence of the thrombocytopenic state and associated platelet dysfunction or it can be due to disseminated intravascular coagulation[51,52,53].

CLINICAL FEATURES:

DCF:

Incubation period is usually 4-6 days. It is followed by abrupt onset of high grade fever (39 to 40 degree) followed by remission for about two days. Next comes second febrile phase (SADDLE BACK), chills, headache, retro orbital pain, myalgia, arthralgia etc[54]. Rarely sore throat and conjunctival suffusion (fig 6) may occur, Constitutional symptoms like Anorexia, nausea, disguise, vomiting are common. Transient generalized erythematous

Fig: 5 Erythmatous Rashes

Fig: 6 Conjunctival Suffusion

rash appears on the trunk which may spread centripetally to face, trunk and limbs which at times resolves with desquamation[55]. (Fig 5)

Physical examination may reveal relative bradycardia, hepatomegaly, splenomegaly, lymphadenopathy[56]. Monitoring for warning signs like petechiae and mucosal membrane bleeding[56, 57] should be done. Massive haemorrhagic phenomena like vaginal bleeding, gastrointestinal bleeding, epistaxis and other bleeding may occur during this phase[57]. The earliest alarm is a progressive decrease in white blood cells[56]. Clinically, it may be very difficult to distinguish dengue from other febrile diseases in the early phase. A positive tourniquet test during this phase increases the probability of diagnosis of dengue[56,58].

DHF:

Most common in young adults around 12 years of age. This is characterized by fever, major /minor bleeding manifestation and evidence of plasma leakage like pleural effusion and ascites supported by thrombocytopenia and hemoconcentration[59,60]. Development of DHF with progressively decreasing platelet count and rising haematocrit is an alarm for impending shock.

Fig :7 Gum Bleeding

Fig 8 Palatal Petechiae

Fig: 9 Lower Limb Petechiae

Fig: 10 Conjunctival Hemorrhage

DSS:

DSS is defined as DHF along with signs of circulatory failure like narrow pulse pressure, hypotension and frank shock with warning signs of sustained abdominal pain, persistent vomiting, restlessness, lethargy, hypothermia and sweating. Physical signs may include blotchy skin, cyanosis, tachypnoea, hepatomegaly, pleural effusion, edema and oliguria[61].

In the initial stage of shock there will be a compensatory mechanism which maintains the systolic pressure and may misguide the clinician. There will be peripheral vasoconstriction, tachycardia, cold extremities and narrow pulse pressure because of high vascular resistance. This is followed by stage of decompensatory shock which is characterized by hypotension, hypoxia and finally multi-organ failure, acidosis and DIC. Patient may recover after volume correction but there is chance of recurrence due to excessive capillary permeability. During the recovery phases, excessive fluid therapy may subject to pulmonary edema or congestive heart failure[62,63].

RARE PRESENTATION:

Rare presentations of dengue like Acute liverfailure[64], encephalopathy[65,66], Myocarditis[67,68,69], ARDS and Renal failure[70] should also be thought of.

Reducing dengue mortality requires early recognition of the disease, simple but effective triage principles and timely decisions regarding the management, which may all helps in identifying those people at risk of developing severe disease.

In a study conducted at a teaching hospital in North India[71] regarding clinical profile of dengue fever. It was shown that out of 356 patients with suspected dengue fever 138 patients with serologically confirmed dengue was included in the study.

Of them 58% patients were males and 42% were females. 70% patients had classical dengue fever; where as 30% had dengue hemorrhagic fever. The most common symptoms observed in the study were headache 76%, abdominal pain in 63%, vomiting in 58%, where as rash and cutaneous hypersensitivity were present in 26% and 16% respectively.

Hemorrhagic manifestations were present in 40% patients. Atypical manifestations were also recorded in 14% of patients who had neurological involvement[72] and liver enzymes elevation, were also noted in dengue infection [73,74] . In this present study, AST levels were equal to or greater than those of ALT levels in all of dengue infected patients, a finding that has also been reported earlier[75] 4% of them had acute hepatic failure[76]. Overall

mortality was recorded to be 6% and death was observed due to multi-organ failure.

In another prospective study conducted in 100 confirmed cases of dengue fever in North Karnataka[77] from May 2012 to April 2013, it was observed that majority were males 54%, and the most common age group affected was between16-40 years.

The most common presentation was fever which was present in all the 100 cases, followed by headache in 90%, myalgia in 81%, vomiting in 56% and abdominal pain in 48%. 22% of them had dengue hemorrhagic fever and among the hemorrhagic manifestations observed, the most common was petechiae 21%. Dengue shock syndrome (DSS) was present in16%. Pleural effusion was found in 70% of patients and right sided pleural effusion was found in 52% of patients. USG abdomen had revealed ascites in 54% of patients.

The most atypical clinical feature noticed was encephalitis and very rarely GBS[78,79]. The other unusual features observed were observed were hepatic dysfunction (34%), renal failure (26%), multi organ dysfunction (18%), encephalopathy (13%), ARDS (12%), and finally 11% death were reported.

Hepatic Dysfunction was studied in 70 serologically proven dengue patients[80]. SGOT levels were higher than SGPT levels in them. Hepatosplenomegaly and ascites were also present.

Usually involvement of liver in dengue has been reported frequently in children.[81,82] But in adults there are only few studies supporting this evidence [83-86.] In the present study, it was noted that SGOT levels were elevated than that of SGPT levels. The reversal of this will be seen in malaria and enteric fever[87]. According to Srivenu Itha et al, the study conducted had showed no significant elevation of liver enzymes in dengue[88].

- SGOT- elevated in 100%
- SGPT level- elevated in 91%
- SGOT level> twice the upper limit of normal-85%
- SGPT levels> twice the upper limit of normal-48%.
- Hepatomegaly-50%
- Splenomegaly-21%[89]
- Ascites-60%[90]
- Pleural effusion- 15%
- Leucopenia- 10%

Though the mechanisms involved in derangement of liver enzymes in dengue was not very clear it was believed that it may be due to direct injury to liver cells or due to an immunological response. None of their patients had DSS. Therefore shock as a major cause of liver injury was ruled out.

Dengue haemorrhagic fever among adults – an observational study was conducted in Chennai, here 128 cases who were fullfilled the diagnostic criteria for DHF according to WHO case definitions were included[91].

In that study, males and females were affected in equal proportion in the ratio of 1:1. The average duration of fever was 4 days with the range of 2-6 days. The most common symptom was fever, present in 100%, followed by myalgia in 68%, gingival bleeding in 63%, headache in 57%, fatigability in 42% and arthralgia in 20%.

Among the bleeding manifestations, 46% had petechiae or purpura, 12% had sub-conjuctival bleed, 11% had ecchymosis and 10% had rashes. 9% of them had malena and 8 of them had menorrhagia, 22% of them had hepatomegaly and 5% had splenomegaly. Also, 37% showed hypotension. Leucopenia was present in 57% patients.

Thrombocytopenia was present in 18% patients and hepatitis was noted in 27.3% patients. Of them, 18.6% of patients with elevated transaminase values (> 1000 IU/l) were suspected to have ischemic hepatitis

since they were in shock. When USG is done 62.5% were diagnosed to have pleural effusion whereas only 27% people showed pleural effusion in chest X-ray, 14% had ultrasound evidence of gall bladder wall thickening and Dengue IgM antibodies was positive in 46% cases.

NS1 ANTIGEN:

NS1(non-structural protein 1) Antigen testing also known as Platelia Dengue NS1 Ag assay, is a test for dengue that allows rapid detection of dengue infection on the first day of fever, even before the appearance of antibodies. Recently, NS1 antigen has gained interest as a new and early biomarker for the early diagnosis of DENV infection. Dengue NS1 antigen is a highly conserved glycoprotein, produced both in membrane-associated and secretory forms [92].

MAC-ELISA:

IgM antibody capture ELISA is especially useful in the diagnosis of recent infection. These are relatively specific for dengue but do not distinguish between various serotypes. Rising titer of IgM antibodies is much more specific.

Regarding IgG ELISA, this can be used for the diagnosis of a previous dengue illness. Those patients with first degree dengue infection

will have negative IgG test in the acute phase of illness and then become positive for IgG antibodies in the convalescent phase. Whereas patients with second degree dengue infection show positive IgG test in the acute phase with a fourfold increase in IgG titer in the convalescent phase.[93]

RT-PCR:

Reverse transcriptase PCR is a specific and sensitive test to detect viral RNA. Hybridisation probes helps to identify viral nucleic acid[94].

BLOOD CHEMISTRY:

Platelet count and haematocrit should be monitored 6^{th} hourly. Thrombocytopenia and hemoconcentration are constantly observed in DHF. Platelet count usually drops below one lakh between the third and eighth day of illness. 20% increase in haematocrit from the baseline value is consistent with plasma leakage when there is drop in the haematocrit, it is suggestive of bleeding.[95]

Leucopenia with reduction in the number of neutrophils and relative lymphocytosis may also be observed[96].

Other investigations like Liver function tests, renal function tests, blood sugar levels, serum electrolytes, bicarbonate or lactate, cardiac enzymes, ECG and urine specific gravity can be done as indicated.

Management decisions

Depending on the clinical manifestations, patients may grouped into three categories[97] as follows,

1. **Group A**-includes those who can be sent home
2. **Group B-** those who require hospital management
3. **Group C**- those who need emergency treatment and urgent referral.

Group A includes patients who can be managed at home. In the absence of signs of dehydration or bleeding, they should be reviewed every day for disease progression, until they are out of the danger. Patients should be alert.

Group B includes those patients who need in-hospital management**.**

Admission criteria

Presence of warning signs related to hypotension, dehydration, profuse sweating, cold extremities, bleeding manifestation or co-morbid condition warrants admission. In these patients, careful and repeated estimation of volume status and fluid replacement are the cornerstone of management.[23]

If the patient has dengue with warning signs, the action plan should be as follows:

Baseline haematocrit should be observed before fluid therapy. Isotonic solutions like 0.9% saline or RL or Hartmann's solution should be preferred.

5-7ml/kg/hr for1-2hours,

⇩ Assess if vitals are stable

3-5 ml/kg/hr for 2-4 hours,

⇩ Assess if vitals are stable

2-3 ml/kg/hr or less depending upon clinical response.

Following fluid therapy, reassess the clinical status and repeat the total blood count and haematocrit. Here, it is very important to change the rate of fluid infusion frequently, with the aim to maintain good perfusion and adequate urine output of about 0.5 ml/kg/hr[23].

Group C:

Patients who require emergency treatment and urgent referral are those who have evidence of severe plasma leakage, leading to dengue shock and fluid accumulation with complication like, profuse hemorrhages, multiple organ failure, and respiratory distress.

In cases of hypotensive shock, colloid solutions are preferred. Continuous replacement of further plasma losses is essential to maintain effective circulation for 24-48 hours. Blood grouping and cross matching should be done meanwhile for all patients in shock. Blood transfusion is carried out only in cases with suspected/severe bleeding.

Treatment of compensated shock:

For those patients with compensated shock,

Isotonic fluid should be administered

5–10ml/kg/hr

⇩ Reassess, if vitals stable

5-7ml/kg/hr for 1-2 hours,

⇩

3-5ml/kg/hr for 2-4hours,

⇩

2-3ml/kg/hr maintenance

Further infusion depends on hemodynamic status, and can be maintained for up to 24-48 hours.

If shock persists or if the vitals are unstable, check the haematocrit after the first bolus. If the haematocrit is persistently high (>50%), repeat a second bolus of crystalloids at 10-20 ml/kg/hr for one hour. After the second

bolus, if there is improvement, reduce the infusion, and then continue to reduce as above. The goals of fluid resuscitation are to improve central and peripheral circulation and to enhance end-organ perfusion.

If haematocrit decreases with respect to the initial reference haematocrit (<40% in children and females, <45% in males), then there is internal bleeding and so transfuse blood as soon as possible. Patients with hypotensive shock should be managed vigorously

Treatment for patients in decompensated shock:

Initiate intravenous fluid resuscitation with crystalloid or colloid solution at the rate of

20 ml/kg as a bolus, over 15 minutes.

⇩ Patient improves give crystalloid/colloid

10 ml/kg/hr over one hour.

⇩ If haematocrit was high, switch to colloids

10–20ml/kg as a second bolus over 30-60minutes

Then to follow the protocol of compensated shock [99-103], if the vitals were stable.

If vital signs are still unstable, review the haematocrit, before the second bolus. If the haematocrit has reduced, suspect internal bleeding and consider blood transfusion.

The higher the fluid infusion rate, more frequently the patient should be monitored and reviewed, so as to avoid fluid overload.

Treatment of hemorrhagic complications:

Mucosal bleeding should be considered as minor, if the patient remains stable with fluid resuscitation/replacement and there is no need for any blood transfusion. Bleeding usually improves rapidly during the recovery phase. Intramuscular injections should be avoided for fear of formation of hematoma. In otherwise haemodynamically stable patients, prophylactic platelet transfusions for severe thrombocytopenia have not been shown to be effective and not necessary.

Severe bleeding is recognized by:

- Persistent / overt bleeding in the presence of unstable haemodynamic status, irrespective of the haematocrit level.
- A decrease in haematocrit after fluid resuscitation together with unstable hemodynamic status.
- Refractory shock not responding to consecutive fluid resuscitation of 40-60 ml/kg.
- Hypotensive shock with low/normal haematocrit prior fluid resuscitation

- Persistent/worsening metabolic acidosis ± a well-maintained systolic blood pressure, particularly in those with severe abdominal tenderness and distension.
- Haematocrit of <30% as a trigger for blood transfusion, as recommended by the Surviving Sepsis Campaign Guideline [104] ,is inapplicable to severe dengue, as in dengue, bleeding usually occurs after a period of prolonged shock, preceded by plasma leakage.

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

- Infuse 5-10ml/kg of fresh-packed red cells or 10-20 ml/kg of fresh whole blood at a suitable rate and observe the clinical response. It is important to give fresh whole blood or fresh red cells as Oxygen delivery at tissue level is optimal when levels of 2,3 di-phosphoglycerate are high and stored blood loses 2,3 DPG, which impede the oxygen-releasing capacity of hemoglobin, leading to functional tissue hypoxia. A positive clinical response includes improving haemodynamic status and acid-base balance.
- Repeat blood transfusion if there is further blood loss or no appropriate rise in haematocrit following the initial blood transfusion. There is no proper evidence to support the practice of

transfusing platelet concentrates and fresh-frozen plasma in cases of severe bleeding. Those can be given when massive bleeding cannot be managed with just fresh whole blood/fresh-packed cells alone. However, it may exacerbate the fluid overload.

The management of fluid overload varies with respect to the phase of the disease.

Complications of dengue:

- Both hyperglycemia and hypoglycemia are prone to occur, even in the absence of diabetes mellitus or hypoglycemic agents.
- Electrolyte and acid-base imbalances are also common complications in severe dengue and are probably due to gastrointestinal losses through vomiting and diarrhoea or due to the use of hypotonic solutions for resuscitation and dehydration correction.
- Hyponatraemia, hypo/hyperkalaemia, serum calcium imbalances and metabolic acidosis can also occur.
- Co-infections and nosocomial infections are not uncommon.

Differential diagnosis of dengue fever:

Flu-like syndromes Influenza, measles, Leptospirosis, infectious mononucleosis, HIV, seroconversion illness, Illnesses with a rash Rubella,

scarlet fever, meningococcal infection, drug reactions, diarrhoeal diseases, Rotavirus or other enteric infections, Illnesses with neurological manifestations, Meningo/encephalitis, Febrile seizures.

METHODOLOGY

Settings:	Department of Medicine, Tirunelveli Medical College and Hospital.
Collaborative Department:	Department of Biochemistry, Department of Microbiology, Department of Pathology, Department of Radiology.
Study Design:	Hospital based cross sectional Observational Study.
Period of Study:	April 2012 to March 2013
Sample size:	251 cases
Ethical committee approval:	The present study was approved by the Institutional Ethical committee.

Inclusion Criteria:

- Fever with thrombocytopenia with Dengue antigen(NS1) or antibody(IgG or IgM) positivity
- Adults with age more than 13 years.

Exclusion Criteria:

- Fever with thrombocytopenia due to other causes

Materials:

Total of 251 serologically proven dengue cases, who satisfied the inclusion and exclusion criteria were taken up for the study.

Methods:

Complete history, signs, symptoms and laboratory data were recorded as per the Performa.

Investigations:

The following investigations were performed in all the patients.

Blood:

TC, Platelet Count, Haematocrit, LFT, RFT, Electrolytes

Radiology

X-ray chest,

USG Abdomen

Statistical analysis:

The data was entered in the Microsoft Excel, spread sheet and analyzed statistically using standard statistical software, SPSS for windows. Chi Square test used for categorical variables. Significance was considered if the 'p' value was below 0.05.

Cases were defined as per WHO guidelines:-

Dengue Classical Fever (DCF):

An acute febrile illness with two or more of the following

- Headache
- Retro orbital pain

- Myalgia and Arthralgia
- Skin rash
- Hemorrhagic manifestation
- Nausea, vomiting and
- Supportive serology.

Dengue Hemorrhagic fever (DHF): (all 4 criteria required)

- Fever or history of fever lasting for 2-7 days
- Bleeding tendencies indicated by either a positive tourniquet test

  Petechiae, ecchymoses and purpura

  Bleeding per mucosa, Haematemesis, malena etc.
- Thrombocytopenia less than one lakh
- Plasma leakage evidenced by

  Rise in haematocrit more than 20% or fall in haematocrit more than 20% after iv fluids

  Pleural effusion, ascites, hypoalbuminemia

Dengue Shock Syndrome (DSS):

DSS require all DHF criteria in addition to circulatory failure manifested by

- Rapid and weak pulse
- Narrow pulse pressure < 20mm of Hg
- Hypotension
- Cold dry skin, restlessness.

Normal Values:

Hemoglobin

Adult male	13.3 - 16.2 gm/dl
Adult female	12 - 15.8 gm/dl
Leucopenia	4000 cells /dl

Haematocrit

Adult Male	38.8 - 46.4
Adult Female	35.4 - 44.4

LFT

Bilirubin

Total	0.3 - 1.3mg/dl
Direct	0.1- 0.4 mg/dl
Indirect	0.2 – 0.9mg/dl
ALT	7-41 U/L
AST	12 – 38 U/L

RFT

Serum Urea	15 - 40 mg/dl

Reference values are from

(Harrison laboratory values of clinical importance).

RESULTS

The present study is an observational study where we studied the clinical profile of 251 serologically proven dengue patients admitted in the Department of Medicine, Tirunelveli Medical College from April 2012 - March 2013.

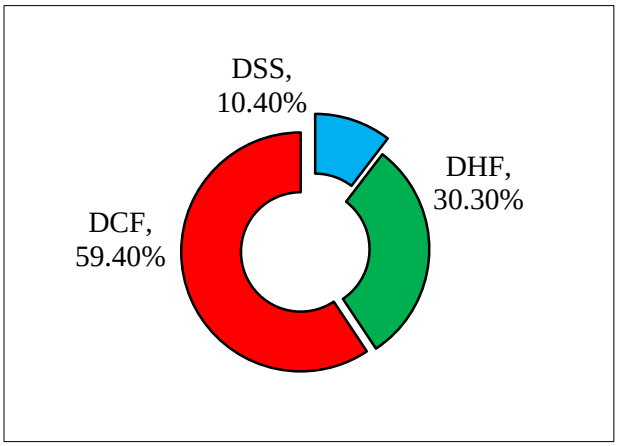
The study had revealed much observation that concurred with traditional teaching about dengue, there were other findings that were peculiar to this epidemic. The observations are as follows,

Clinical classification of cases

Table 2: Clinical Spectrum of Cases

Clinical spectrum of cases	No. of cases	Percent
DSS	26	10.4
DHF	76	30.3
DCF	149	59.4
Total	251	100

Fig: 11 Clinical Spectrum Of Cases

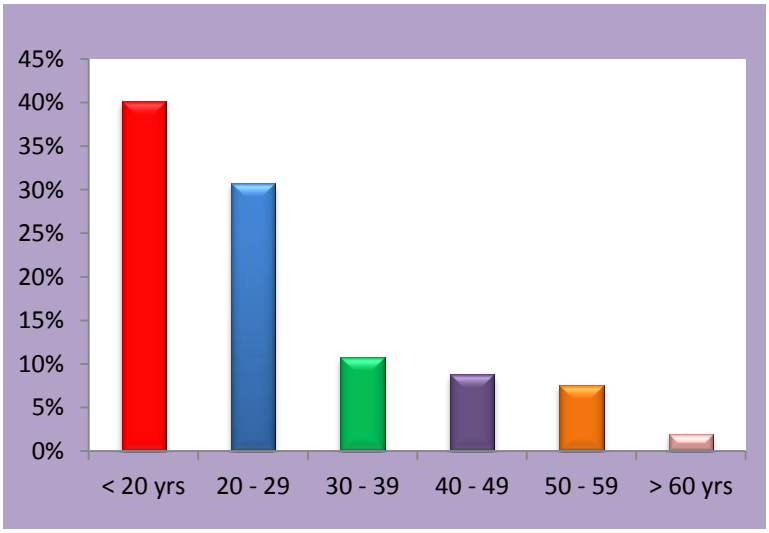


Out of 251 cases, 149 patients (59.40%) belonged to DCF, DHF in 76 patients (30.30%), where as 26 patients (10.40%) belonged to more severe variety of DSS. The details are given in the above table.

Table: 3 Distribution of Age

Age	Frequency	Percent
< 20 yrs	101	40.2
20 - 29	77	30.7
30 - 39	27	10.8
40 - 49	22	8.8
50 - 59	19	7.6
> 60 yrs	5	2.0
Total	251	100

Fig 12 Distribution of Age

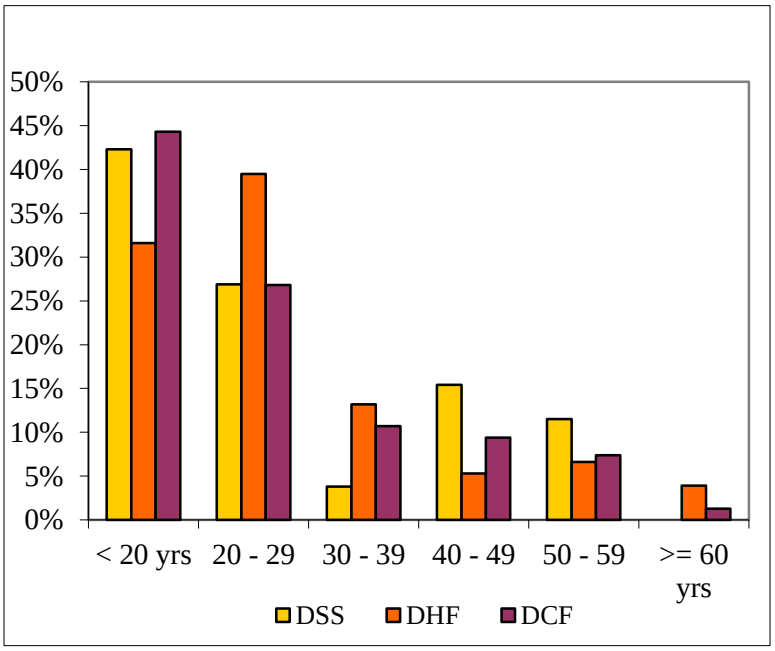


Majority of cases 40.20% occurred in young adult <20 years of age. The incidence appeared to reduce with advancing age with least number of cases seen in the age group >60 years of age.

Table: 4 Associations between Dengue type and age

Age (Code)	Dengue Clinical Type		
	DSS	DHF	DCF
< 20 yrs	42.30%	31.60%	44.30%
20 - 29	26.90%	39.50%	26.80%
30 - 39	3.80%	13.20%	10.70%
40 - 49	15.40%	5.30%	9.40%
50 - 59	11.50%	6.60%	7.40%
> 60 yrs	-	3.90%	1.30%

Fig:13 Associations between Dengue type and age

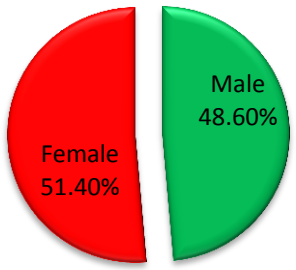


DCF (44.30%) and DSS (42.30%) was more common in the younger age group <20 years.DHF (39.50%) was more common in age group of 20-29 years where as DSS is not observed in patients >60 years in this study.

Table: 5 Analysis in respect to gender distribution

Gender	Frequency	Percent
Male	122	48.6
Female	129	51.4
Total	251	100

Fig :14 Gender Distribution

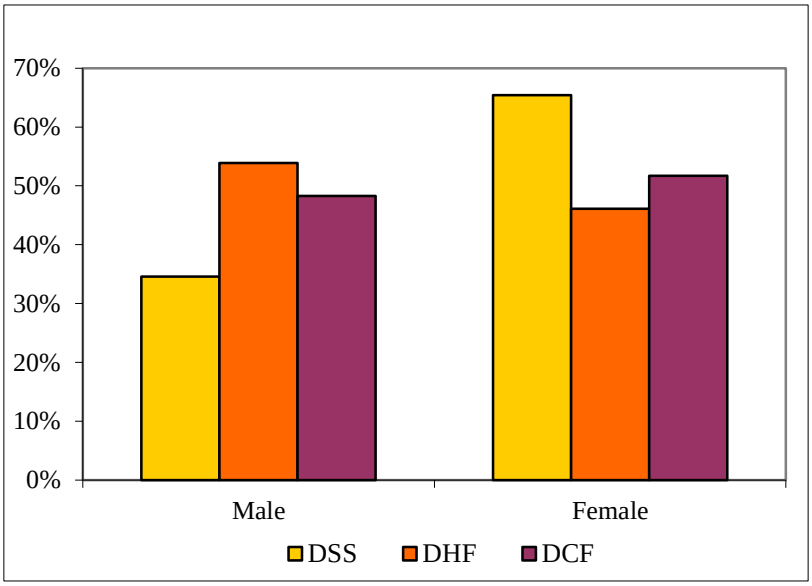


Distribution of dengue in females was slightly higher 51.40% when compared to males 48.60%.

Table: 6 Analysis of clinical spectrum with respect to gender

Gender	DSS	DHF	DCF
Male	34.60%	53.90%	48.30%
Female	65.40%	46.10%	51.70%

Fig:15 Associations between Dengue type and Gender



DSS was more common in females 65.40%, when compared to males 34.60% where as DHF in men53.90% when compared to women 46.10%.

Table :7 Duration of hospital stay

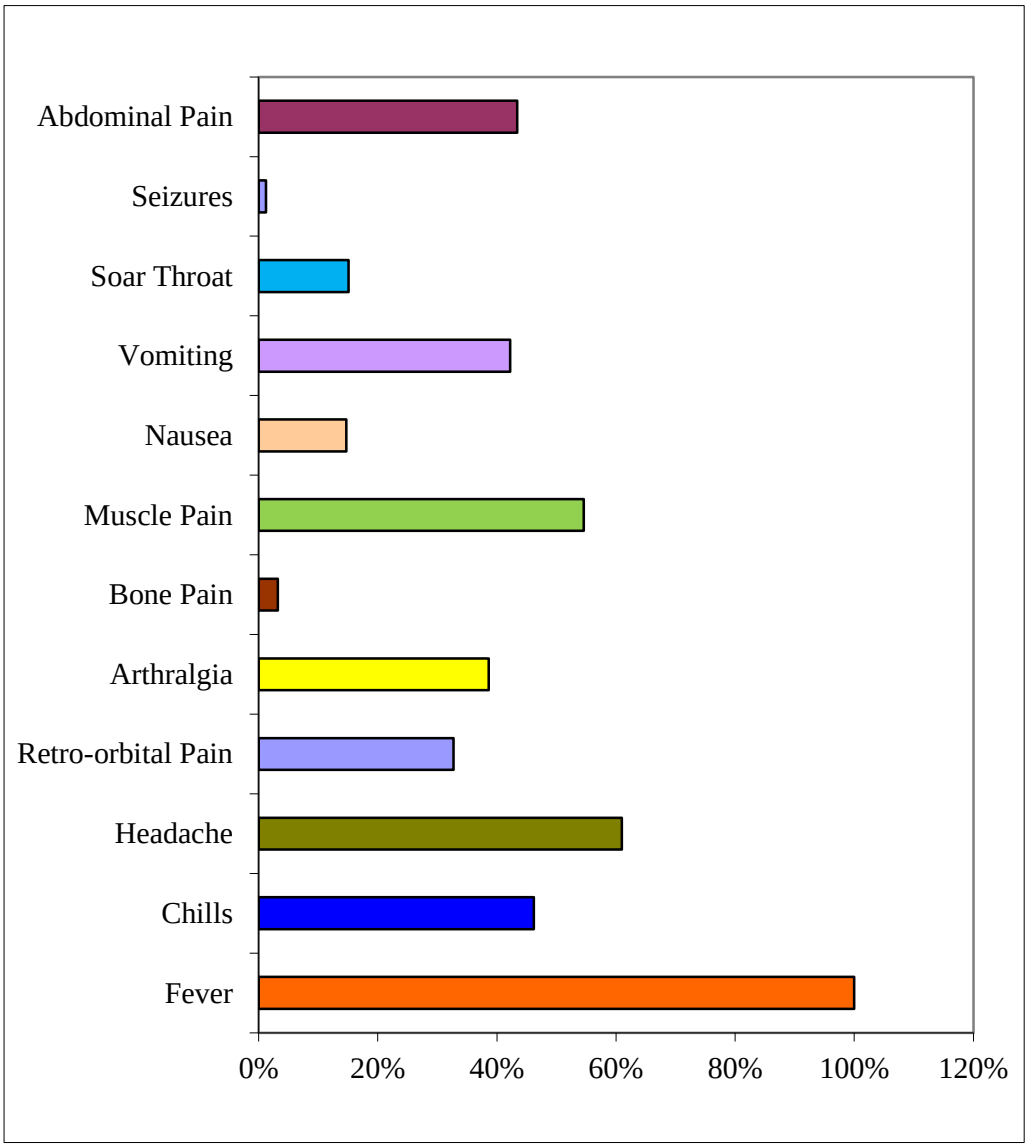
Duration of Hospital Stay	Frequency	Percent
Upto 1 Week	165	65.7
7 - 14 Days	86	34.3
Total	251	100

The average duration of hospital stay of patients in this study was less than 1 week.

Table:8 Analysis of symptoms:

Symptoms	Frequency	Percent
Fever	251	100
Chills	116	46.2
Headache	153	61.0
Retro-orbital Pain	82	32.7
Arthralgia	97	38.6
Bone Pain	8	3.2
Muscle Pain	137	54.6
Nausea	37	14.7
Vomiting	106	42.2
Soar Throat	38	15.1
Seizures	3	1.2
Abdominal Pain	109	43.4

Fig :16 Analysis of Symptoms

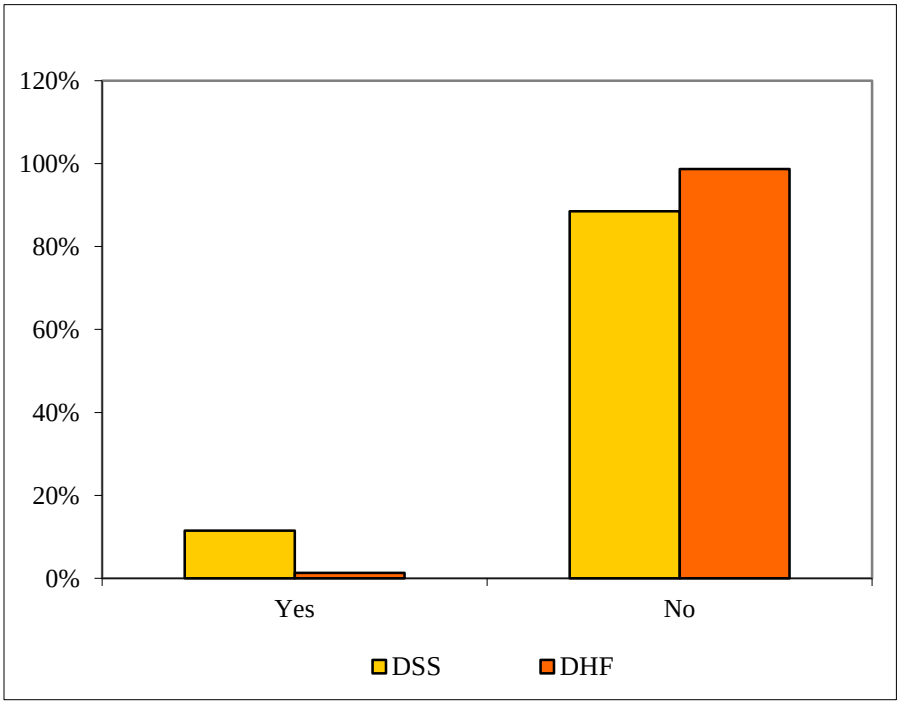


In this study all patients had fever 100%. Followed by headache 61%, Myalgia 54.60%, chills 46.20%, abdominal pain 43.40%, vomiting 42.20%.The characteristic feature of dengue like bone pain and retro-orbital pain was present in only 3.20% and 32.70% respectively. Atypical clinical feature like seizure was present in1.20%

Table:9 Association between Dengue type and Bone Pain

Bone Pain	Dengue Type			Total
	DSS	DHF	DCF	
Yes	3	1	4	8
	11.50%	1.30%	2.70%	3.20%
No	23	75	145	243
	88.50%	98.70%	97.30%	96.80%
Total	26	76	149	251
Chi Square: 6.861; P < 0.05				

Fig 18 Association between Dengue type and Bone Pain



Even though the bone pain was present in 3.20% it has got statistically significant correlation with Dengue classical fever.

Table: 10 Analysis of clinical signs

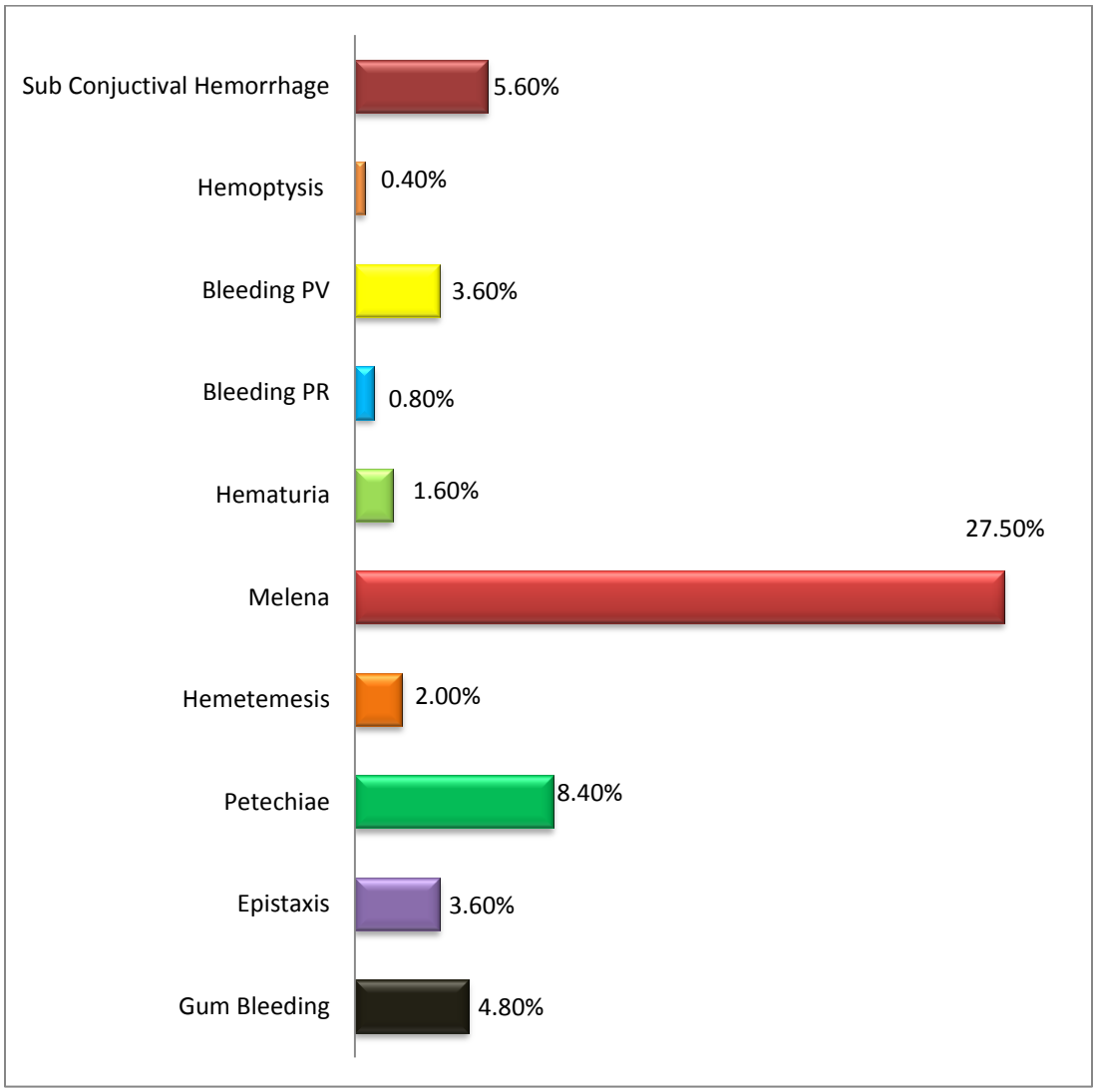
Signs	Frequency	Percent
Icterus	16	6.4
Pallor	13	5.2
Conjunctival Congestion	60	23.9
Hepatomegaly	30	12
Splenomegaly	21	8.4
Myocarditis	4	1.6
Pleural Effusion	24	9.6
Ascites	25	10.0
Encephalopathy	4	1.6
Rashes	25	10.0
Exfoliative Dermatitis	1	0.4
Diarrhoea	16	6.4
Neck Stiffness	1	0.4
Puffiness of Face	21	8.4
Breathlessness	1	0.4
Pneumonia	4	1.6
Loss of Appetite	1	0.4
Pelvic Abscess	1	0.4
Herpes Labialis	1	0.4

Most common sign observed in this study was conjunctival congestion 23.90%, followed by Hepatomegaly 12%, Ascites 10%, Rashes 10%, Pleural effusion 9.60%, Puffiness of the face & Splenomegaly 8.40%

Table: 11 Analysis of bleeding manifestation

Bleeding	Frequency	Percent
Gum Bleeding	12	4.8
Epistaxis	9	3.6
Petechiae	21	8.4
Hemetemesis	5	2.0
Melena	69	27.5
Hematuria	4	1.6
Bleeding PR	2	0.8
Bleeding PV	9	3.6
Hemoptysis	1	0.4
Sub conjunctival hemorrhage	14	5.6

Fig: 19 Bleeding manifestation



Most common bleeding manifestation encountered was malena 27.50% followed by petechiae 8.40% and gum bleeding in 4.80% less frequent was bleeding manifestation hemoptysis 0.40%

Table: 12 Associations between Dengue type and Bleeding

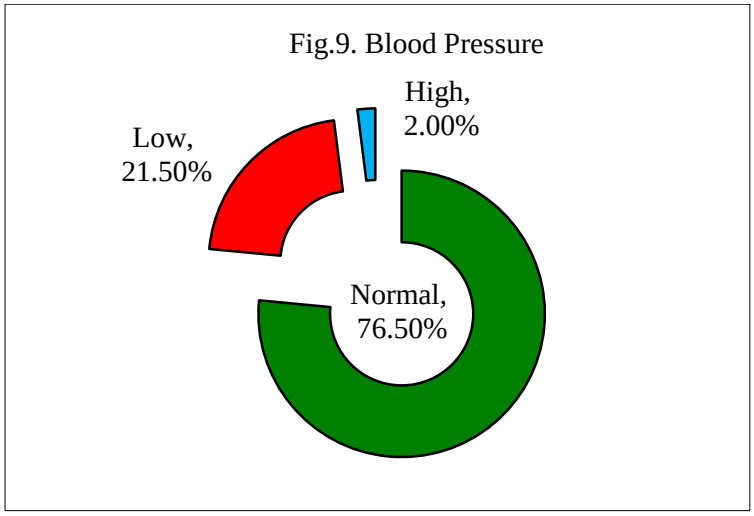
Melena	Dengue Type			Total
	DSS	DHF	DCF	
Yes	5	28	36	69
	19.20%	36.80%	24.20%	27.50%
No	21	48	113	182
	80.80%	63.20%	75.80%	72.50%
Total	26	76	149	251
Chi Square: 5.987; $P < 0.05$				

Malena has got statistically significant association with DHF

Table: 13 Analysis of blood pressure in dengue

Blood Pressure	Frequency	Percent
Normal	192	76.5
Low	54	21.5
High	5	2.0
Total	251	100

Fig: 20 Analysis of blood pressure in dengue

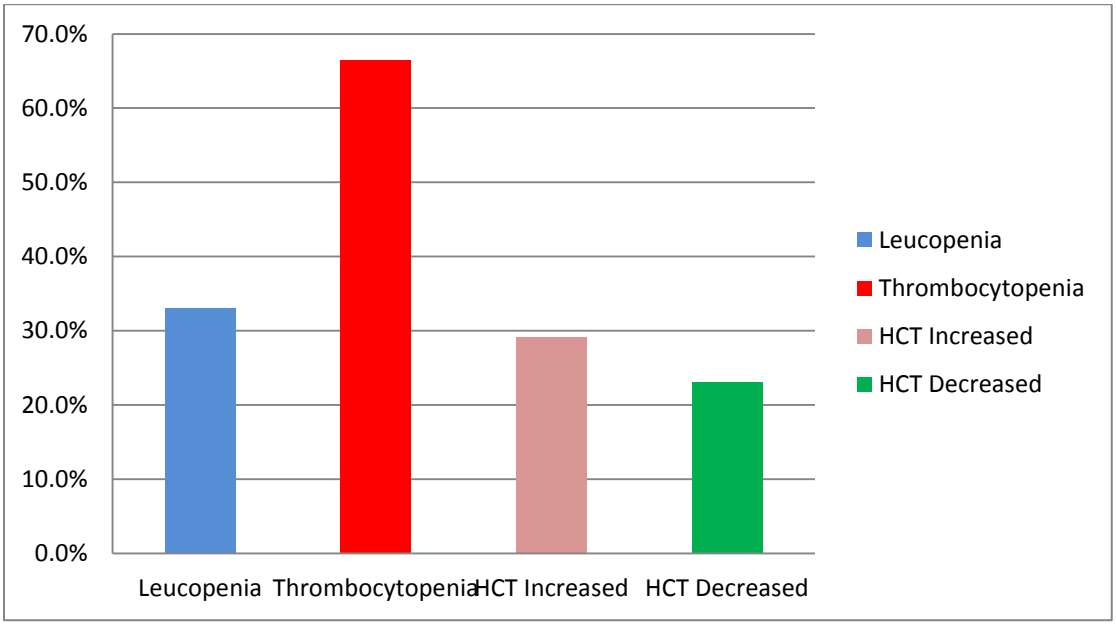


Blood pressure was normal in 76.50%, eventhough 21.50% of patient presented with hypotension, DSS had occurred in 10.40%

Table: 14 Analysis of blood count

CBC	Frequency	Percent
Leucopenia	83	33.1
Thrombocytopenia	167	66.5
HCT Increased	73	29.1
HCT Decreased	58	23.1

Fig: 21 Analysis of blood count

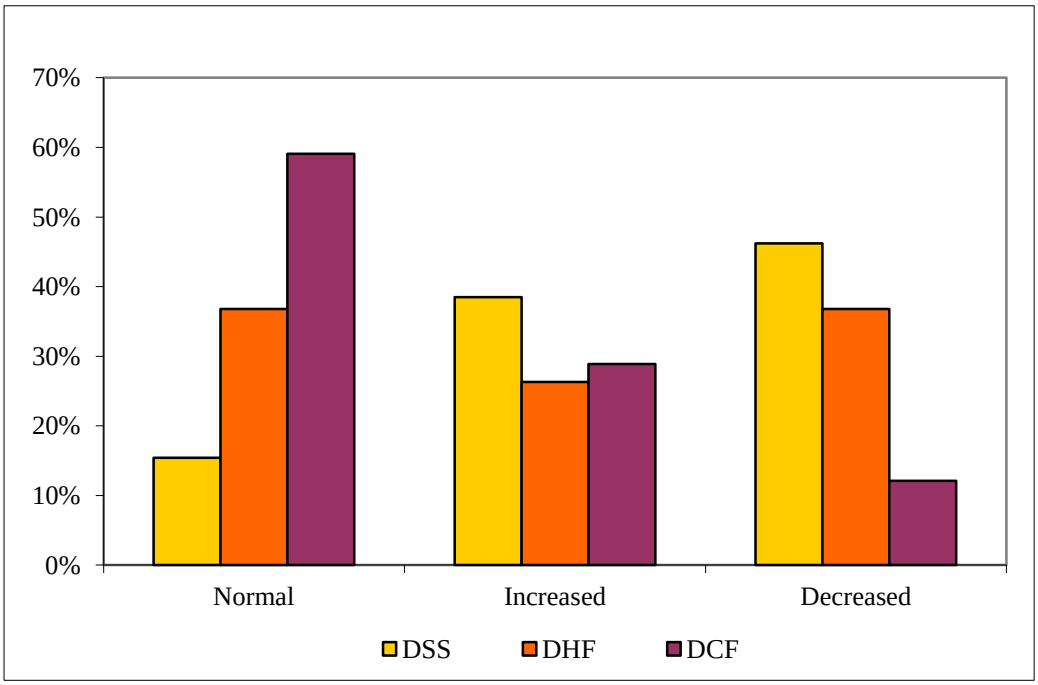


CBC, thrombocytopenia was seen in 66.50%, leucopenia in 33.10% haematocrit increased and decreased in 29.10% and 23.10% respectively.

Table: 15 Associations between Dengue types and haematocrit

Haematocrit	Dengue Type			Total
	DSS	DHF	DCF	
Normal	4	28	88	120
	15.40%	36.80%	59.10%	47.80%
Increased	10	20	43	73
	38.50%	26.30%	28.90%	29.10%
Decreased	12	28	18	58
	46.20%	36.80%	12.10%	23.10%
Total	26	76	149	251
Chi Square: 32.585; P < 0.001				

Fig : 22 Associations between Dengue types and haematocrit

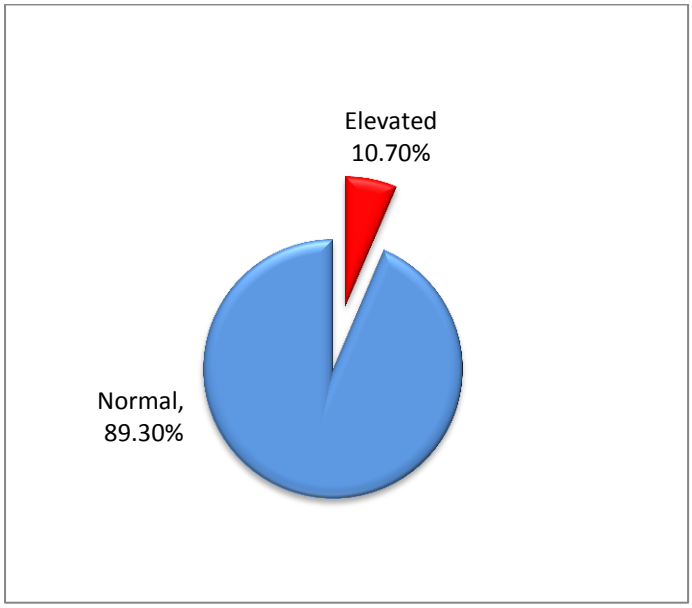


Increased haematocrit has got statistical correlation with DSS

Table: 16 Analysis of Elevation of Bilirubin

LFT: Bilirubin	Frequency	Percent
Elevated	27	10.7
Normal	224	89.3
Total	251	100

Fig: 23 Analysis of Elevation of Bilirubin

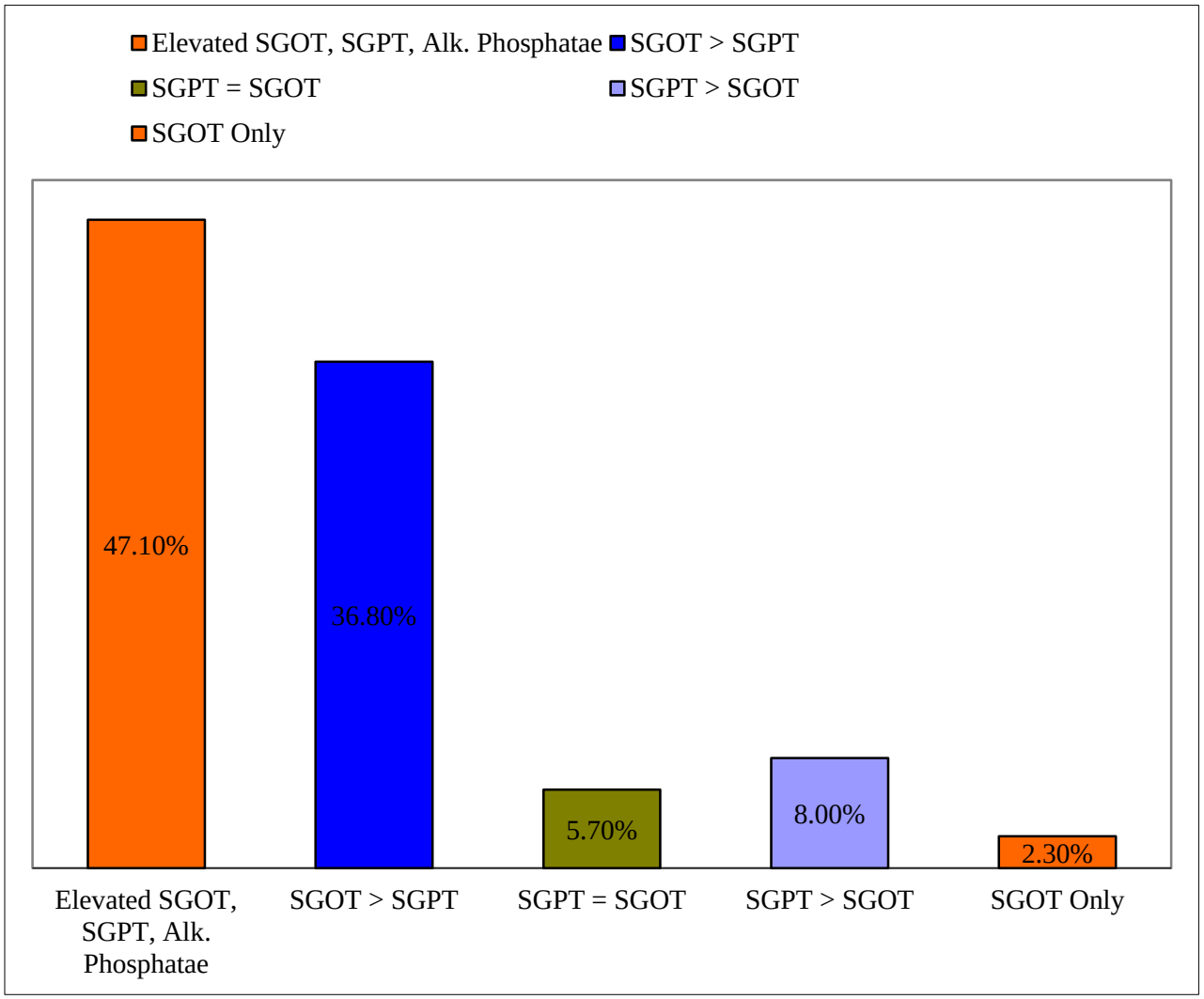


Serum bilirubin was elevated in 10.70%, where as it was normal in 89.30%.

Table: 17 Analyses of Liver Enzymes

LFT: Liver Enzymes	Frequency	Percent
Elevated SGOT, SGPT, Alk. Phosphatae	41	47.1
SGOT > SGPT	32	36.8
SGPT = SGOT	5	5.7
SGPT > SGOT	7	8.0
SGOT Only	2	2.3
Total	87	100

Fig 24Analysis of Liver Enzymes

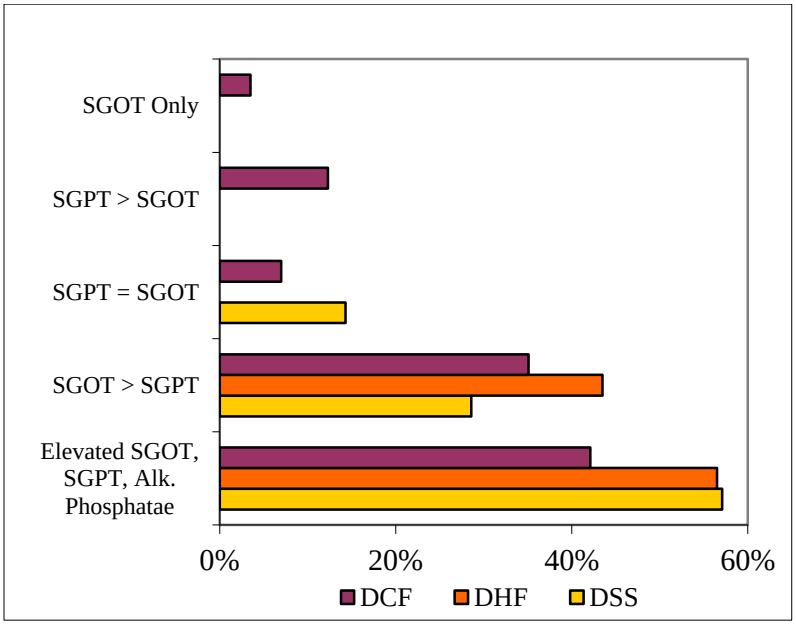


In LFT, All the three enzymes ALT,AST and ALP were elevated in 47.10%.SGOT more than SGPT in 36.80%;SGPT more than SGOT; both AST and ALP were almost equally elevated in 5.70%.where as SGOT only in 2.30%.

Table: 18 Association of Dengue type and Liver Enzymes

Liver Enzymes	Dengue Type			Total
	DSS	DHF	DCF	
Elevated SGOT, SGPT, Alk. Phosphatase	4	13	24	41
	57.10%	56.50%	42.10%	47.10%
SGOT > SGPT	2	10	20	32
	28.60%	43.50%	35.10%	36.80%
SGPT = SGOT	1		4	5
	14.30%		7.00%	5.70%
SGPT > SGOT			7	7
			12.30%	8.00%
SGOT Only			2	2
			3.50%	2.30%
Total	7	23	57	87
Chi Square: 8.445; P > 0.05				

Fig:25 Association of Dengue type and Liver Enzymes

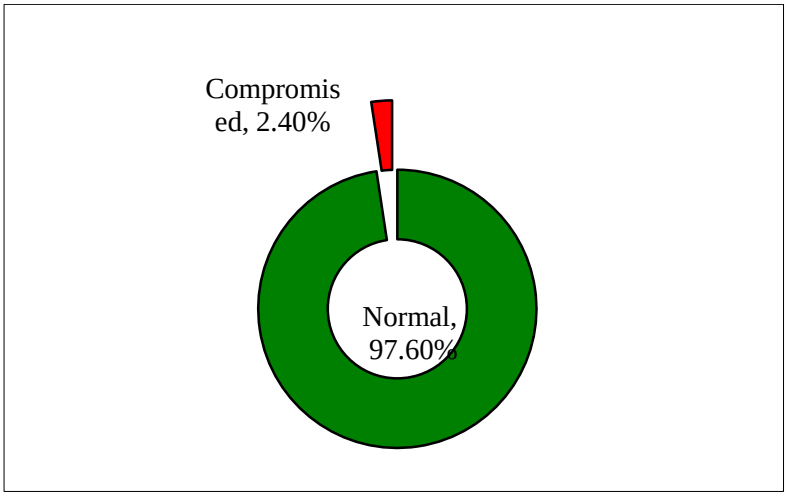


The elevation of liver enzymes has no statistical correlation with any particular type of dengue.

Table: 19 Analysis of Renal function test

	Frequency	Percent
Normal	245	97.6
Compromised	6	2.4
Total	251	100

Fig: 26 Analyses of Renal Function Test

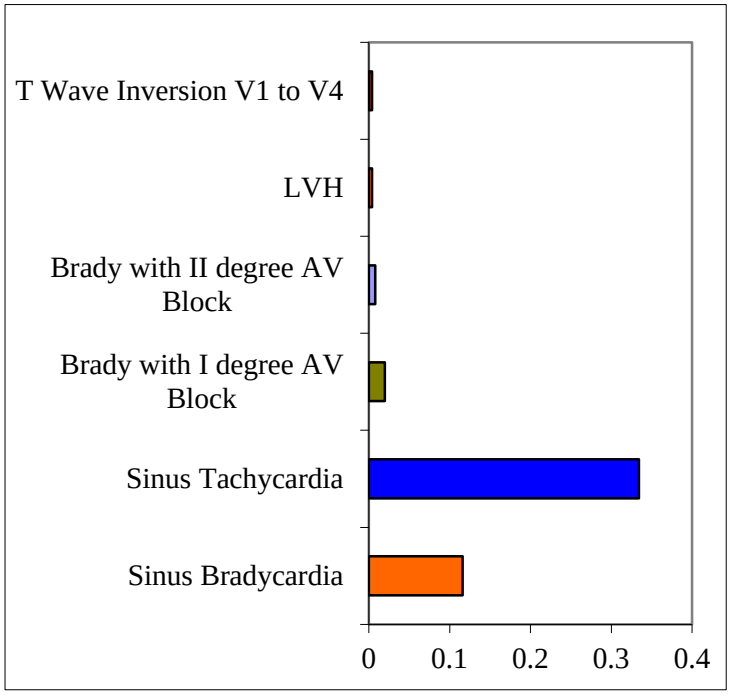


Renal function test was normal in 97.60%, elevated in 2.40% but it has no statistically correlation.

Table: 20 Analysis of ECG

ECG	Frequency	Percent
Sinus Bradycardia	29	11.6
Sinus Tachycardia	84	33.4
Brady with I degree AV Block	5	2.0
Brady with II degree AV Block	2	0.8
LVH	1	0.4
T Wave Inversion V1 to V4	1	0.4
Normal	129	51.4
Total	251	100

Fig: 27 Analyses of ECG

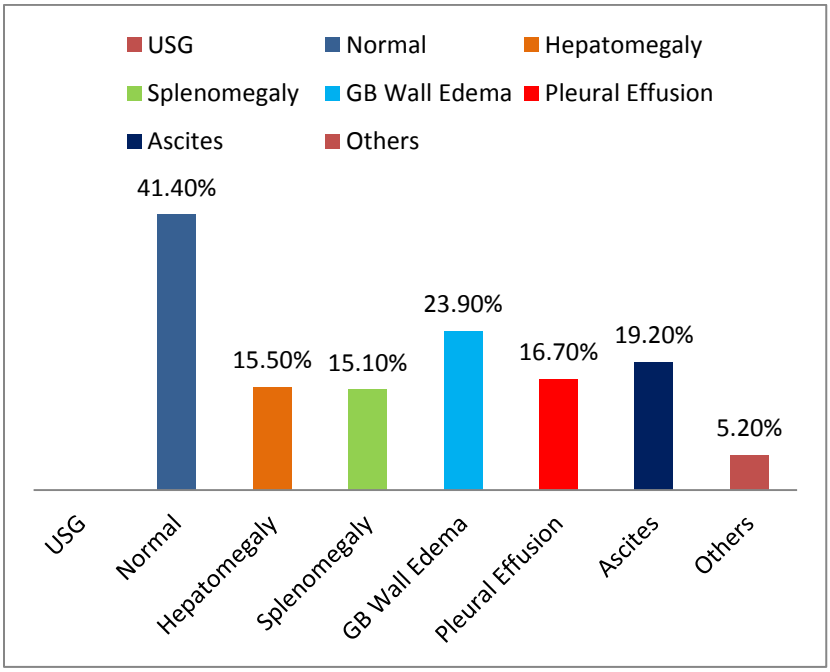


ECG was normal in 51.40%. Most frequent ECG sign was sinus Tachycardia 33.40%, where as sinus bradycardia in 11.60%, First degrees AV block in 2.0%, Second degrees AV block 0.80%.

Table: 21 Analysis of USG

USG	Frequency	Percent
Normal	104	41.4
Hepatomegaly	39	15.5
Splenomegaly	38	15.1
GB Wall Edema	60	23.9
Pleural Effusion	42	16.7
Ascites	48	19.2
Others	13	5.2

Fig: 28 Analyses of USG Abdomen

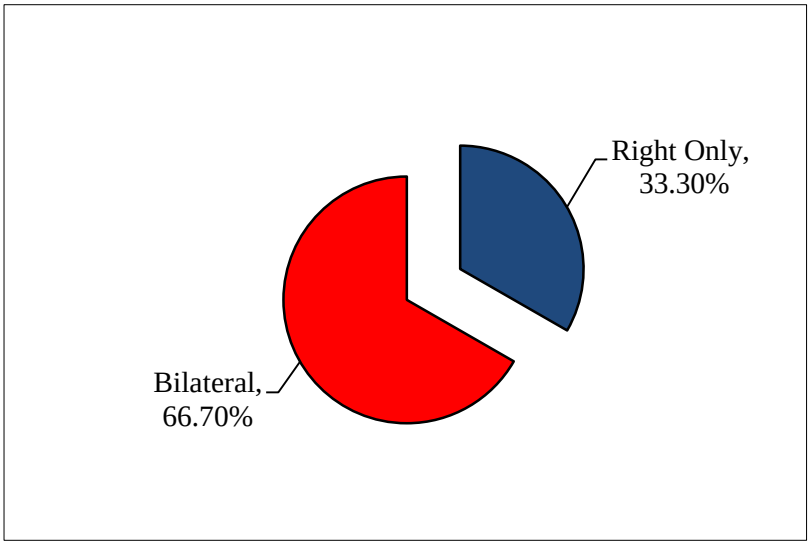


USG Abdomen was normal in 104 patients. GB wall edema was present in 23.90%, Ascites was present in 19.20%, and Pleural effusion was seen in 16.70%, Hepatomegaly15.50% and Splenomegaly 15.10%.

Table: 22 Analyses of USG Pleural effusion

USG: Pleural Effusion	Frequency	Percent
Right Only	14	33.3
Bilateral	28	66.7
Total	42	100

Fig: 29 Analyses of USG Pleural Effusion

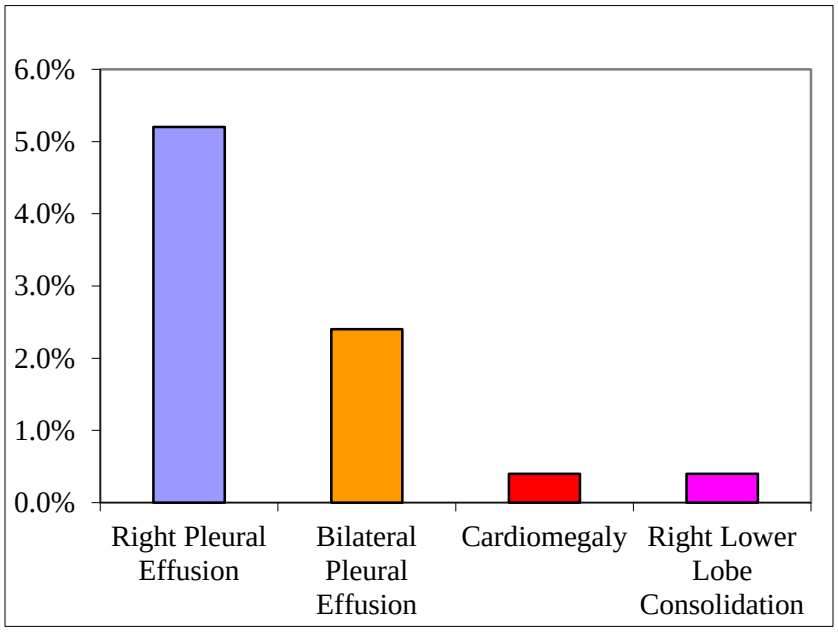


In USG Right side Pleural Effusion was seen in 33.30% where as bilateral effusion was more common and it was present in 66.70%.

Table: 24 Analysis of Chest X-Ray PA View

Chest X Ray P/A	Frequency	Percent
Normal	230	91.6
Right Pleural Effusion	13	5.2
Bilateral Pleural Effusion	6	2.4
Cardiomegaly	1	0.4
Right Lower Lobe Consolidation	1	0.4
Total	251	100

Fig: 31 Analyses of Chest X-Ray



X-ray chest P/A view was normal in 91.60%, here right sided pleural was more common 5.20% when compared with bilateral pleural effusion 2.40%.

Table: 25 Analyses of CT Brain

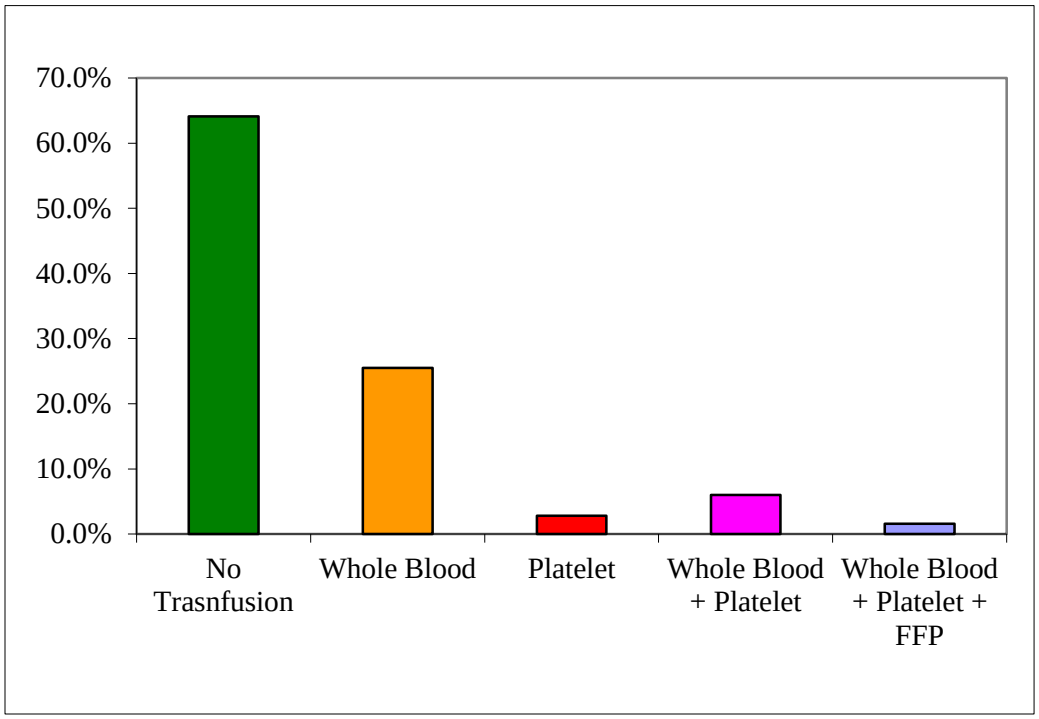
CT Brain	Frequency	Percent
Normal	2	50
Minimal White Matter Hypo density	1	25
Intracerebral Bleed	1	25
Total	4	100

CT brain was taken for 4 patients presented with encephalopathy. It was normal in 2 patients, 1 patient with Seizure had Intracerebral bleed.

Table: 26 Analyses of Blood Transfusion

Blood Transfusion	Frequency	Percent
No Transfusion	161	64.1
Whole Blood	64	25.5
Platelet	7	2.8
Whole Blood + Platelet	15	6.0
Whole Blood + Platelet + FFP	4	1.6
Total	251	100

Fig: 32 Analysis of Blood Transfusion

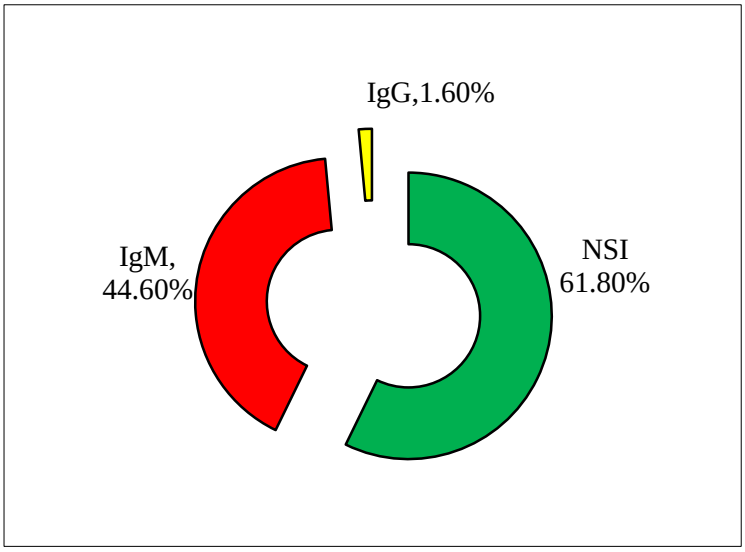


Blood transfusion was not needed for 161 patients. 64 (25.50%) was transfused with whole blood, only platelet in 7 patients, whole blood along with platelet in 6.0%.whole blood along with platelet and FFP in 1.60%.

Table: 23 Analysis of Serology

Serology	Frequency	Percent
NS 1	155	61.8
IgM	112	44.6
IgG	4	1.6

Fig: 30 Analyses of Serology

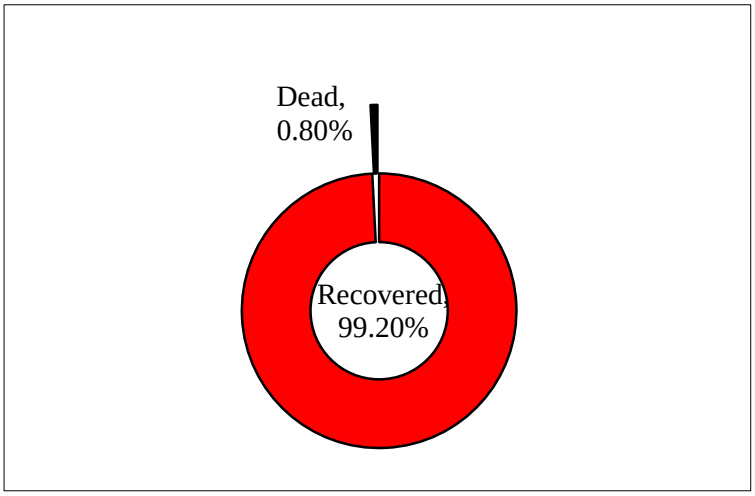


In serology NSI antigen was positive in 61.80%, IgM in 44.60%, IgG in 1.60%.

Table: 27 Analyses of Out come

Outcome	Frequency	Percent
Recovered	249	99.2
Dead	2	0.8
Total	251	100

Fig: 33 Analyses of Out Come

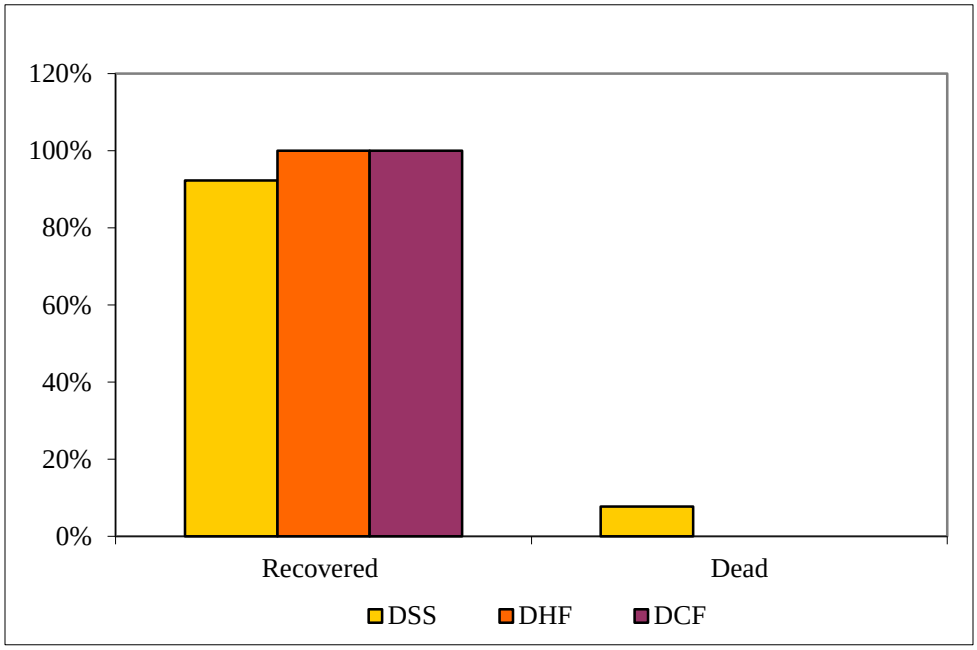


Regarding the Outcome, patient's recovery rate achieved was 99.20%. We lost 2 cases (0.80%) among the 251 cases studied.

Table: 28 Associations between Dengue type and Outcome

Outcome	Dengue Type			Total
	DSS	DHF	DCF	
Recovered	24	76	149	249
	92.30%	100.00%	100.00%	99.20%
Dead	2			2
	7.70%			0.80%
Total	26	76	149	251
Chi Square: 17.447; P < 0.01				

Fig: 34 Associations between Dengue type and Outcome



The death has got statistical correlation with DSS.

DISCUSSION

This study describes the clinical profile of serologically proven dengue cases in adults aged more than 13 years admitted in our hospital during the current Dengue epidemic of 2012.

A total of 251 cases admitted between April 2012 – March 2013 were studied by analyzing the symptoms, signs, hematological and laboratory findings, and outcome.

The findings are discussed in detail with reference to other studies conducted in clinical profile of dengue in various parts of the country. Also an attempt is made to find out the significance of various parameters, in order to help us diagnose promptly and treat effectively the cases thereby reducing morbidity and mortality.

Clinical spectrum of Cases:

The cases studied were classified according to the WHO Diagnostic criteria for Dengue published in 2009.

Of the total 251 cases studied, 149 cases (59.40%) were diagnosed to have DCF, 76 cases (30.30%) were diagnosed with DHF. More severe form of dengue DSS constituted 26 cases which was 10.4% of the cases studied.

This clinical distribution of cases as DCF, DHF and DSS in our study followed similar pattern that was observed in other studies conducted in Uttar Pradesh in 2010 (RituKaroli et al) and Karnataka during the recent epidemic of May 2012 – April 2013(Dr Mohan SDMCMS). In these studies, DCF constituted around 70% of cases and DHF and DSS constituted the rest.

Average hospital stay calculated was, 1 week (65.70%) depending on the severity ranged from 1 - 15 days.

Age Distribution

Age group of the cases ranged from 13 – 65 years. Majority of cases in this study were young adults belonging to the age group of 20 – 39 years of age. Whereas it was less common among age group > 60 years .Incidence of dengue showed a decreasing trend with increasing age. The study revealed that younger age group was more susceptible to dengue infection in our population.

DCF showed a highest incidence among cases < 20 years of age (44.30%) whereas DHF was common in 20 -29 years age group(39.50%), which was slightly more than in cases < 20 years (31.60%).

Incidence of DSS was high among cases < 20 years of age whereas no cases with DSS belonged to age group > 60 years.

According to WHO, DSS was more prone in children and young adults because the compensatory mechanism for capillary leakage was weak when compared to adults.

Gender Distribution

51.40% of the cases were females and males constituted 48.60%. There was no statistical significance for this finding. DSS was more common among females (65.40%) when compared to males (34.60%). Similar observation was made by WHO, this might be due to difference in genetic background and immune response among males and females.

Analysis of presenting symptoms

Presenting symptoms were analyzed and categorized into common presentation and atypical presentation.

The following were the observations:

1**. Fever**: 251 cases (100%) had fever at the time of presentation which was similar to the studies conducted in Chennai and North Karnataka. Traditional teaching of Saddle back fever in dengue was not observed during our

epidemic. Most of the cases had high grade intermittent fever. Fever was associated with chills and rigor in 46.20% of cases. A particular periodicity was not observed.

2**. Headache and Retro-orbital pain**: Headache was the second most common symptom (61.0%) similar to the study from North Karnataka (2013) and Uttar Pradesh (2010).

Retro orbital pain was observed in 32.70% of cases. This was not specific for DCF, DHF or DSS.

3**. Myalgia, arthralgia and bone pain:** 54.60% of the cases had moderate to severe myalgia. Most common sites involved were the back, hips and lower limbs. The incidence of bone pain which is supposed to be characteristic of Dengue, which even gave it the synonym of" Break bone fever", was surprisingly low (3.20%) and was more common in DCF and was statistically significant. Arthralgia had occurred in 38.60% of the cases and was not specific to DCF, DHF or DSS.

4. **Abdominal Pain**: 43.40% cases had abdominal pain which was diffuse, non-colicky and vague in character. The severity of abdominal pain was more in cases who had associated vomiting.

5. Vomiting: 42.20% (106 cases) had vomiting. Vomiting lasted for an average of 1.7 days. All the patients with vomiting were treated symptomatically.

Atypical presentation:

Seizures:

Apart from all the above mentioned presenting symptoms, one of the most dreadful presentations of dengue during this epidemic was seizure (1.20%). Out of 4 patients with dengue encephalopathy 2 patients had developed GTCS and 1 patient had seizures following CNS bleed and the patient had later succumbed to the disease.

Analysis of Signs

Physical examination in Dengue was done with utmost seriousness so that no critical signs that aid in prompt diagnosis of the disease and its complications was not missed. The signs were analyzed in detail in the present study. The observations are as follows:

1**. Conjunctival congestion** due to dilatation of the superficial capillaries was the most common sign (23.90 %) noted and was most striking too. If present, it was used as a sign for clinical suspicion of Dengue.

2. Rashes: Transient erythematous morbiliform rash (Exanthem) was associated in 10% of the cases (25 cases). The spectrum of rash varied from erythematous flush that blanched on pressure to exfoliative dermatitis in 1 case. The rash was associated with itching in 11 cases

3. Hepatomegaly and Splenomegaly: Clinically hepatomegaly was present in 30 cases (12%) and splenomegaly in 21 cases (8.40%). No significance was present in statistical analysis and hence these findings could not be specifically attributed to DCF, DHF, and DSS.

4. Ascites: Free fluid abdomen (ascites) was clinically apparent in 30 cases (10%). Ascites was a part of third space volume loss due to increased capillary leakage more during the later phase of the disease. However ascitic fluid analysis was not attempted in these cases for proving the etiology and ascites reduced and disappeared with clinical recovery of the patient. Ascites was observed in DCF, DHF and DSS.

5. Pleural effusion: Clinically apparent pleural effusion was present in 24 cases (9.6%). Clinically right sided pleural effusion was detected more frequently followed by bilateral pleural effusion. Pleural fluid analysis was not performed in these cases and hence could not be classified as exudative

or transudative type . With recovery from the disease, pleural effusion subsided and cleared completely.

6. Puffiness of face: A typical dengue facies resulted from facial puffiness especially eye lid edema associated with erythema of the surrounding areas. This kind of facial puffiness was present in 21 cases (8.4%)

7. Jaundice: 16 cases (6.4%) had icterus associated with dark yellow discoloration of urine. However no features suggestive of obstruction of the biliary tree were noticed like clay colored stool and pruritis. LFT was done, the details of which will be discussed in a later section.

8. Pneumonia: 4 cases of dengue also had developed pneumonia. This pneumonia would have been caused by either a co infection or nosocomial infection. Similar cases were observed in the previous studies and WHO had opined about the same as above.

9. Myocarditis: One of the atypical presentation of dengue observed in the present study was myocarditis. Young adults (4 cases) were diagnosed to have myocarditis and confirmed with ECHO and treated accordingly. 3 cases recovered and 1 case succumbed after deteriorating to refractory shock.

Analysis of Bleeding Manifestations:

Studying the bleeding manifestation among the cases admitted was undertaken separately. A detailed analysis of various bleeding manifestations is as follows.

A) Melena: 69 cases (27.50%) presented with melena. This was the most common bleeding manifestation encountered. DHF and melena were linked statistically with significant p value. The severity of melena ranged from mild to severe. Majority were self limiting. 5 (2.0%) of them had hemetamesis. Massive episodes were treated with blood and blood product transfusions. 2 cases who succumbed to the disease had massive episodes of melena as a part of the clinical spectrum and later progressed to DIC and refractory shock.

B) Petechiae: 21 cases had petechiae especially over the extremities. Lower limbs were commonly involved than the upper limbs may be due to capillary damage aggravated on standing by gravity. Petechiae were also noted over palate which is an uncommon site for the same. Petechiae were statistically significant in DCF.

C) Sub-conjunctival hemorrhage: Noted in 14 cases (5.60%), subconjuntival hemorrhage was commonly precipated by repeated bouts of cough in patients .

D) Gum bleeding and epistaxis : Mucosal bleeding were common during this epidemic.12 cases (4.80%) had gum bleeding and 9 cases (3.60%) had epistaxis. All these cases had spontaneous bleeding and some cases had to be transfused with blood for replacing the lost volume and for prophylaxis against further bleed they were given blood product transfusions.

E) Menorrhagia: 9 cases of young females admitted with dengue had developed menorrhagia during the menstrual cycles and some of them had required blood transfusion.

F) Other bleeding: 4 cases (1.60%) had developed hematuria.1 had hemopysis.

Analysis of Blood Pressure

BP is the most important clinical monitor in a case of dengue for identifying onset of complications like shock. Pulse pressure is more important than BP in identifying early stage shock. Narrowed pulse pressure (< 20 mm of Hg) is the most sensitive sign.

In this study hypotension was noted at presentation among 54 cases and the majority was stabilized using IV fluid support. Hypotension was persistent in cases of DSS (26 cases). They had to manage with intense fluid therapy according to WHO treatment guidelines. 2 cases that were known hypertensives on treatment but admitted in shock had elevated BP at the time of discharge and was started on antihypertensives for the same.

In our ICU setup we used pulse pressure as the main monitor for identifying and treating patients with DSS.

Analysis of Blood count among cases

Thrombocytopenia was the most common abnormality observed. 167 cases (66.50%) had thrombocytopenia ranging from mild to severe thrombocytopenia. Leucopenia was observed in 83 cases (33.10%) and strengthened the suspicion of dengue infection and later was confirmed with dengue serology. Eosinophilia was observed among many cases mostly in patients who had associated dermatological manifestations like rash and itching may be as an effect of some allergic response.

Haematocrit was elevated in 73 cases studied and they had to be treated with IV fluid therapy in addition to oral fluids. Haematocrit was

followed up every 6 hours and fluids were titrated accordingly. Haematocrit was persistently elevated in 27 cases where the duration of fluid therapy was extended to > 48 hours and even 72 hours to normalize the haematocrit. WHO recommends fluid therapy only for 48 hours but we had to give IVF for an average of 72 hours according to haematocrit. This was a significant change in the fluid management strategy that we followed in this epidemic and was found to be more effective in treating our population with dengue. Further studies dealing with fluid therapy in our population with dengue is needed to validate our finding.

Low haematocrit was noticed among 58 cases but was significantly low in cases who presented with significant bleeding. However a low haematocrit was also noted among the cases without bleeding manifestations. This may be due to the presence of anemia in these patients that might have resulted in low baseline values of haematocrit. Increased haematocrit was noted among 38.50% of cases with DSS and was statistically significant.

Analysis of LFT

LFT was done in all cases. Normal results were obtained in 137 cases. Elevated bilirubin was noted in 27 cases (10.70%). Both direct and indirect

bilirubin was increased. However clinically manifest jaundice was present in only 16 cases.

87 cases showed enzyme abnormalities. All the three enzymes AST, ALT and ALP were elevated in 41 cases (47.10%). AST was more elevated than ALT in 32 cases and in 5 cases both AST and ALT were elevated in almost same proportion. ALT was elevated more than AST in 7 cases. Isolated elevation on AST was noticed only in 2 cases.

In a study conducted at CMC Vellore, elevation of AST was in 100% cases with LFT abnormality which was similar to our study.

This elevation of liver enzymes could be due to direct injury to liver cells by the virus or due to immunological response. Ischemic hepatitis in patients especially in shock could be another possible etiology.

Analysis of ECG findings:

Normal recordings were seen in 169 cases. Sinus tachycardia was seen in 84 cases mostly due to fever itself and many of them in hypotension or shock and tachycardia seemed to resolve with normalization of BP. Sinus bradycardia was observed in 29 cases. First degree AV block was seen in 5

cases and second degree in 2 cases. No case studied had third degree AV block.

Analysis of RFT:

Compromised renal function was observed only in 6 cases out of the 251 cases studied. They belonged to age group < 20 years. However this finding was not statistically significant.

Analysis of USG findings:

USG abdomen was performed in all cases. The findings are as follows

USG abdomen was essentially normal in 104 cases (41.40%) of the cases.

Gall bladder wall edema was the most common USG finding (60 cases, 23.90% cases).Hepatomegaly and ascites were present in 39 cases and 48 cases respectively.

Splenomegaly was observed in 38 % cases. Pleural effusion was detected in 42 cases (16.70%).

Analysis of X-Ray findings:

Chest X-ray PA View was normal in 230 cases. Right sided pleural effusion was detected in 13 cases and bilateral pleural effusion noted in 6 cases

Cardiomegaly was noted only in 1 case.

Consolidation was present in 1 case whereas pneumonitis with infiltration was reported in 3 cases.

On comparing USG and X-ray in diagnosing pleural effusion, USG proved to be more sensitive than X-ray. Cases with even mild pleural effusion were picked up using USG whereas X-ray revealed normal study.

Bilateral pleural effusion was common in USG whereas right sided pleural effusion was commonly reported in X-rays. This may be because even the minimal pleural effusion on the left side was picked up in USG. No patient had isolated left sided pleural effusion.

CT brain was performed in patients who had presented with encephalopathy and seizures. CT brain was normal in 2 cases. Intracerebral bleed was noted in 1 case and minimal white matter hypo density was noted in the other case.

Dengue cases were confirmed by serology. NS1 antigen ELISA was the most important test used for diagnosing dengue cases in our hospital. Dengue IgM and IgG were performed in cases according to the availability of test kits. NS1 was found to be positive in 155 cases (61.80%) and IgM positive in 112 cases (44.60%). IgG was positive only in 4 cases.

Analysis of outcome:

The cases were admitted and treated in special wards. Cases with DSS and DHF were managed in IMCU with intensive fluid therapy.

Transfusion with blood and blood products were needed in 190 cases. Platelets were not transfused routinely for thrombocytopenia. We used fresh whole blood for cases with uncontrolled bleeding.

Platelets were transfused in cases with platelet count < 10000/ml or in cases with uncontrolled bleeding not responding to whole blood.

Regarding the Outcome of dengue epidemic cases admitted in our department, Cure rate achieved was 99.20% .We lost 2 cases among the 251 cases studied. The cause of death was I.C bleed/ encephalopathy/seizures / DSS/ DIC / Refractory shock . The other case succumbed due to massive malena/encephalopathy/DSS/DIC/refractory shock.

SUMMARY

- 251 serologically proven dengue cases were studied from April 2012 to March 2013.
- Most of the patient 59.40% belonged to DCF were DHF 30.30% and DSS in 10.40%.
- The most common age group affected was the young adult<20 years
- Distribution of Dengue & DSS was more common in female gender.
- Fever was the most prominent symptom followed by headache myalgia, abdominal pain and vomiting.
- Characteristic of dengue like bone pain was present only in 3.2% but it has got statistical correlation with DCF.
- Conjunctival congestion was the most common sign.
- Atypical feature like encephalopathy, seizures, Myocarditis was also observed.
- Malena was the common bleeding manifestation followed by Petechiae, subconjunctival bleeding and Gum bleeding.
- BP is the most important clinical monitor in a case of Dengue. Narrowed pulse pressure (< 20 mm of Hg) is the most sensitive sign for identifying early signs of shock.
- Elevated serum bilirubin was observe in 27 patients and clinical jaundice was present in 16 patients.
- 87 patients showed abnormal liver enzymes were SGOT was elevated in all 87 patients.
- USG was more sensitive in picking up Pleural effusion.

- Sinus Tachycardia (84) was predominant finding in ECG but sinus Bradycardia and various degree of AV blocks was observed.
- Out of 251 patients mortality rate was 0.8%
- Recovery rate was 99.20%.

The cause of death was intra cerebral bleed/ encephalopathy/seizures / DSS/ DIC / Refractory shock in one case. The other case succumbed due to massive malena/encephalopathy/DSS/DIC/refractory shock.

CONCLUSION

In this study the Clinical Profile of serologically proven Dengue fever was studied from April 2012 – March 2013 particularly during the epidemic that stormed Tirunelveli district. With this clinical profile it is easy to recognize and understand the clinical problem. The application of clinical spectrum of WHO classification system is not as very simple and straightforward as it seems because clinical features may overlap among different categories. The WHO classification system of dengue does not include unusual manifestations like encephalopathy, seizures, myocarditis, etc, which might be life-threatening. Although these manifestations are rare, clinicians should always have a high index of suspicion and knowledge of these atypical manifestations, particularly in view of the increasing burden of dengue in recent years. Blood pressure and haematocrit should be monitored for evaluating for the progress of the disease. Bleeding tendency should be closely watched. Management of patients with dengue is mainly supportive simple inexpensive and very effective in saving lives prophylactic FFP and platelets are not necessary for treating DHF, DSS, but early and meticulous monitoring are the corner stone for positive outcome.

BIBLIOGRAPHY

1. Dengue in India Indian J Med Res 136, September 2012, pp 373-390
2. WHO Dengue and dengue haemorrhagic fever. Factsheet No 117, revised May 2008. Geneva, World Health Organization, 2008
3. Abdul Kader MS, Paul Kandaswamy, Appavoo NC & Anuradha L(1997) Outbreak and control of dengue in a village inDharmapuri, Tamil Nadu.Journal ofCommunicable Disease29, 69–7
4. Halstead SB (1980) Dengue hemorrhagic fever – public healthproblem and a field for research.Bulletin of World HealthOrganization58, 1–21.
5. Distribution, seasonal variation & dengue transmission prediction in Sisaket, Thailand. Indian J Med Res 138, September 2013, pp 347-353
6. Nathan MB, Dayal-Drager Recent epidemiological trends the global strategy and public health advances in dengue report of the Scientific Working Group on Dengue Geneva World Health Organization; 2006 (TDR/SWG/08).
7. Karamchandani PV. Dengue group of fevers in India. Lancet1946; 1:92.

8. Singh UB, Maitra A, Broor S, Rai A, Pasha ST, Seth P. Partial nucleotide sequencing and molecular evolution of epidemic causing dengue 2 strains. J Infect Dis 1999; 180 : 959-65.
9. Chaturvedi UC, Mathur A, Kapoor AK, Mehrotra NK, Mehrotra RML. Virological study of an epidemic of febrile illness with haemorrhagic manifestations at Kanpur, India, during 1968. Bull World Health Organ 1970; 43 : 289-93.
10. Chaturvedi UC, Mathur A, Kapoor AK, Tandon HO, MehrotraRML. Clinicovirological study of the recurrence of dengue epidemic with haemorrhagic manifestation at Kanpur, during 1969. Indian J Med Res 1972; 60 : 329-33.
11. Myers RM, Carey DE, Banerjee K, Reuben R, Ramamurti DV. Recovery of dengue type 3 virus from human serum and Aedes aegypti in South India. Indian J Med Res 1968; 56 :781-7.
12. Myers RM, Carey DE, De Ranitz CM, Reuben R, Bennet B. Virological investigations of the 1966 outbreak of Dengue type 3 in Vellore, Southern India. Indian J Med Res 1969; 57: 1392-401.
13. Mukherjee KK, Chakravarti SK, Dey PN, Dey S, Chakraborty MS. Outbreak of febrile illness due to dengue virus type 3 in Calcutta during 1983. Trans R Soc Trop Med Hyg 1987; 81 :1008-10.

14. Dash PK, Saxena P, Abhyankar A, Bhargava R, Jana AM. Emergence of dengue virus type-3 in northern India. Southeast Asian J Trop Med Public Health 2005; 36 : 370-7.
15. Dash PK, Saxena P, Abhyankar A, Bhargava R, Jana AM. Reemergence of dengue virus type-3 (subtype-III) in India: implications for increased incidence of DHF & DSS. Virol J 2006; 3 : 55-65.
16. Paramasivan R, Thenmozhi V, Thangaratham PS, RajendranR, Tewari SC, Dhananjeyan KJ, et al. An outbreak of dengue fever in Tirupur, Coimbatore district, Tamil Nadu. Indian J Med Res 2010; 132 : 105-7.
17. Chakravarti A, Kumar A, Matlani M. Displacement of dengue virus type 3 and type 2 by dengue virus type 1 in Delhi during 2008. Indian J Med Microbiol2010;28 : 412-3.
18. Myers RM, Carey DE. Concurrent isolation from patient of two arboviruses, Chikungunya and dengue type 2. Science 1967; 157 : 1307-8.
19. Chahar HS, Bharaj P, Dar L, Guleria R, Kabra SK, Broor S.Co-infections with chikungunya virus and dengue virus in Delhi, India. Emerg Infect Dis 2009; 15 : 1077-80

20. Dash PK, Sharma S, Srivastava A, Santhosh SR, Parida MM, Neeraja M, et al. Emergence of dengue virus type 4 (genotypeI) in India. Epidemiol Infect 2011; 139 : 857-61.
21. Dayaraj C, Kakade MB, Bhagat AB, Vallentyne J, Singh A, Patil JA, et al. Detection of dengue-4 virus in Pune, western India after an absence of 30 years - its association with two severe cases. Virol J 2011; 8 : 46-9.
22. National vector bourn control programme ,Directorate general of health service ministry of health and family welfare. Government of India .
23. WHO Dengue Guidelines for management, treatment, Prevention and control new edition 2009. Chapter 1 Page 17.
24. Gubler DJ The global emergence/resurgence of arboviral diseases as public health problems. Arch Med Res 2002;33:330-422.
25. Westaway EG, Blok JTaxonomy and evolutionary relationships of flaviviruses. In: Gubler DJ, Kuno G, editors. Dengue and dengue hemorrhagic fever. New York: CAB International; 1997. p. 147
26. Leitmeyer KC. Dengue virus structural differences that correlate with pathogenesis. Journal of Virology, 1999, 73(6):4738--4747.

27. Lanciotti RS et al. Molecular evolution and epidemiology of dengue-3 viruses.Journal of General Virology, 1994, 75(Pt 1):65--75.
28. Messer WB. Emergence and global spread of a dengue serotype 3, subtype III virus. Emerging Infectious Diseases, 2003, 9(7):800—809
29. Wilder-Smith A, Schwartz E. Dengue in travelers. N Engl J Med. 2005;353:924–932.]
30. Guzman MG, Kouri G. Dengue: an update. Lancet Infect Dis. 2002;2:33–42]
31. Aedes aegypti: size, reserves, survival, and flight potential. J Vector Ecol. 2001 Jun;26(1):21-31
32. Chang MS et al. Challenges and future perspective for dengue vector control in the Western Pacific Region. Western Pacific Surveillance and Response Journal, 2011, 2(2):9-16. doi:10.5365/ wpsar. 2010.1.1.012
33. Kuno, G. "Factors Influencing the Transmission of Dengue Viruses." In Dengue and Dengue Hemorrhagic Fever, eds. D. J. Gubler & G. Kuno (Cambridge: CABI, 2001):
34. Halstead SB. Etiologies of the experimental dengues of Siler and Simmons. American Journal of Tropical Medicine and Hygiene, 1974, 23:974--982.

35. Martina, B. E. E., Koraka, P., & Osterhaus, A. D. M. E. Dengue virus pathogenesis: An integrated view. Clinical Microbiology Reviews 22, 564–581 (2009). doi:10.1128/CMR.00035-09

36. Diamond, M. S. Evasion of innate and adaptive immunity by flaviviruses. Immunology and Cell Biology 81, 196–206 (2003). doi:10.1046/j.1440-1711.2003.

37. Halstead SB, Nimmannitya S, Cohen SN. Observations related to pathogenesis of dengue hemorrhagic fever. IV. Relation of disease severity to antibody response and virus recovered. Yale Journal of Biology and Medicine, 1970, 42:311–328.

38. Sangkawibha N et al. Risk factors in dengue shock syndrome: a prospective epidemiologic study in Rayong, Thailand. I. The 1980 outbreak. American Journal of Epidemiology, 1984;120:653--669.

39. Guzman MG et al. Epidemiologic studies on dengue in Santiago de Cuba, 1997. American Journal of Epidemiology, 2000, 152(9):793--799.

40. Halstead SB. Pathophysiology and pathogenesis of dengue haemorrhagic fever. In:Thongchareon P, ed. Monograph on dengue/dengue haemorrhagic fever. New Delhi, World Health

Organization, Regional Office for South-East Asia, 1993 (pp 80--103)

41. Role of T cells, cytokines and antibody in dengue fever and dengue haemorrhagic fever. Fink J, Gu F, Vasudevan SG. Rev Med Virol. 2006 Jul-Aug;16(4):263-75. Review.
42. Halstead SB. Antibody, macrophages, dengue virus infection, shock, and hemorrhage: a pathogenetic cascade. Reviews of Infectious Diseases, 1989, 11(Suppl 4):S830--S839.
43. Halstead SB, Heinz FX. Dengue virus: molecular basis of cell entry and pathogenesis, 25-27 June 2003, Vienna, Austria. Vaccine, 2005, 23(7):849--856.
44. Kliks SC, Nisalak A, Brandt WE, Wahl L, Burke DS Antibody-dependent enhancement of dengue virus growth in human monocytes as a risk factor for dengue hemorrhagic fever. Am J Trop Med Hyg 1989;40:444-51
45. Halstead SBPathogenesis of dengue: challenges to molecular biology. Science 1988;239:476-81
46. Sullivan NJAntibody-mediated enhancement of viral disease. Curr Top Microbiol Immunol 2001;260:145-69

47. Kouri GP, Guzman MG. Dengue haemorrhagic fever/dengue shock syndrome: lessons from the Cuban epidemic, 1981. Bulletin of the World Health Organization, 1989, 67(4):375--380.
48. Sierra B, Kouri G, Guzman MG. Race: a risk factor for dengue hemorrhagic fever.Archives of Virology, 2007, 152(3):533—54.
49. Joshi V, Singhi M, Chaudhary RC. Transovarial transmission of dengue 3 virus by Aedes aegypti. Trans R Soc Trop Med Hyg 1996; 90 :643-4.
50. Trpis M, Hausermann W. Genetics of house - entering behavior in East African populations of Aedes aegypti (L) (Diptera: Culicidae) and its relevance to speciation. Bull Entomol Res 1978; 8: 521-32. ..
51. Schexneider, K.I. and Reedy, E.A. (2005) Thrombocytopenia in dengue fever.Curr. Hematol. Rep 145–1484 Kalayanarooj, S.et al.(2002) Dengue patients at the children's hospital,Bangkok: 1995–1999. Review. Dengue Bull.26, 33–43
52. Mourao, M.P.et al.(2007) Thrombocytopenia in patients with denguevirus infection in the Brazilian Amazon. Platelets 18, 605–612

53. Honda, S.et al.(2009) Increased phagocytosis of platelets from patientswith secondary dengue virus infection by human macrophages.Am. J.Trop. Med. Hyg.80, 841–845...
54. Rigau-Perez JG et al. Dengue and dengue haemorrhagic fever. Lancet, 1998,352:971–97
55. MUCOCUTANEOUS MANIFESTATIONS OF DENGUE FEVERIndian J Dermatol. 2010 Jan-Mar; 55(1): 79–85. doi: 10.4103/0019-5154.60359
56. Kalayanarooj S et al. Early clinical and laboratory indicators of acute dengue illness.Journal of Infectious Diseases, 1997, 176:313–321.
57. Balmaseda A et al. Assessment of the World Health Organization scheme for classification of dengue severity in Nicaragua. American Journal of Tropical Medicineand Hygiene, 2005, 73:1059–1062
58. Phuong CXT et al. Evaluation of the World Health Organization standard tourniquet test in the diagnosis of dengue infection in Vietnam. Tropical Medicine and International
59. Srikiatkhachorn A et al. Natural history of plasma leakage in dengue hemorrhagic fever: a serial ultrasonic study. Pediatric Infectious Disease Journal, 2007, 26(4):283–290

60. Nimmannitya S et al. Dengue and chikungunya virus infection in man in Thailand,1962–64. Observations on hospitalized patients with haemorrhagic fever. American Journal of Tropical Medicine and Hygiene, 1969, 18(6):954–971.
61. Martinez-Torres E, Polanco-Anaya AC, Pleites-Sandoval EB. Why and how children with dengue die? Revista cubana de medicina tropical, 2008, 60(1):40–47.
62. World Health Organization. Geneva, Switzerland: WHO; 2009. Dengue: Guidelines for Diagnosis, Treatment, Prevention and Control.
63. World Health Organization. Geneva, Switzerland: WHO; 1997. Dengue Haemorrhagic Fever: Diagnosis, Treatment, Prevention and Control, 2nd edn.
64. De Souza LJ, Alves JG, Nogueira RMRet al.(2004) Amino-transferase changes and acute hepatitis in patients with Denguefever: analysis of 1585 cases.Brazilian Journal of InfectiousDiseases8 156–163
65. BMJ Case Reports 2013; doi:10.1136/bcr-2012-008229 CASE REPORTA rare case of dengue encephalitis

66. Cam BV, Fonsmark L, Hue NBet al(2001) Prospective case-control study of encephalopathy in children with dengue
67. Horta Velosa H, Ferreira Junior JA , Braga de Pawa JM, Faria Honorio J, Vicenzo de Paola AA. Acute atrial fibrillation during dengue haemorrhagic fever. Braz J Infect Dis. 2003;7(6):418–22.
68. Nagaratnam N, Siripala K, de Silvia N. Arbovirus (dengue type) as a cause of acute myocarditis and pericardium. Br Heart J. 1973;35(2):204–6
69. Wali JP, Biswas A, Chandra S, et al. Cardiac involvement in dengue haemorrhagic fever. Int J Cardiol. 1998;64(1):31–6.,
70. Hommel D, Talarmin A, Reynes JMet al.(1999) Acute renalfailure associated with dengue fever in French Guiana.Nephron83, 183
71. Clinical profile of dengue infection at a teaching hospital in North India Ritu Karoli, Jalees Fatima, Zeba Siddiqi, Khursheed I. Kazmi, Amit R. Sultania Department of Medicine, Era's Lucknow Medical College, Sarfarazganj, Hardoi Road, Lucknow, Uttar Pradesh, India J Infect Dev Ctries 2012; 6(7):551-554. Received 01 April 2011--Accepted 26 July 2011

72. Gupta P, Khare V, Tripathi S, Nag VL, Kumar R, Khan MY, Dhole TK (2010) Assessment of WHO definition of dengue hemorrhagic fever in North India. J Infect Dev Ctries 4: 150-155

73. Parkash O, Almas A, Jafri Wasmin SM, Hamid S, Akhtar J, Alishah H (2010) Severity of acute hepatitis and its outcome in patients with dengue fever in a tertiary care hospital Karachi, Pakistan (South Asia). BMC Gastroenterology 10: 43.

74. Itha S, Kashyap R Krishnani N, Saraswat VA, Choudhri G, Aggarwal R (2005) Profile of liver involvement in dengue virus infection. Natl Med J India 18: 127-130.

75. de Souza LJ, Goncalves Carneiro H, Souto Filho JT, Ferreira de Souza T, Azevedo Cortes V, Neto CG, Bastos Assed D, Wallace da Silva Siqueira E (2002) Hepatitis in dengue shock syndrome. Braz J Infect Dis 6: 322-327

76. Kumar R, Tripathi P, Tripathi S, Kanodia A, Venkatesh V (2008) Prevalence of dengue infection in north Indian children with acute hepatic failure. Ann Hepatol 7: 59-62.

77. Clinical Profile and Outcome of Dengue Fever Cases Vinod H. Ratageri, T.A. Shepur, P.K. Wari, S.C. Chavan, I.B. Mujahid1 and P.N. Yergolkar2 Department of Pediatrics, Karnataka Institute of

Medical Sciences, Hubli, 1PICU, Hubli Children Hospital, Hubli2National Institute of Virology, Field Station, Bangalore, Karnataka, India. Indian J Pediatr 2005; 72 (8) : 705-706

78. Garacia-Rivera EJ, Rigan-Perez JG. Encephalitis and Dengue.Lancet 2002; 360 : 261.
79. Gubler DJ. Kuno G, Waterman SH.Neurologic disorders associated with Dengue infection: Proceedings of the international conference on Dengue/DHF, Kuala Lumpur, Malaysia,1983; 290-301.
80. A Study of Hepatic Dysfunction in Dengue ,Vaibhav Shukla*, Ashok Chandra** Journal of the association of physicians of india • july 2013 • VOL.61.
81. Brij Mohan, Patwari A.K., Anand V.K. Hepatic Dysfunction in childhood Dengue infection Journal of Tropical Pediatrics 2000;46:40-45.
82. Manjith Narayanan, Aravind M.A., Thilothammal N, PreRna R, Rex C.S., Sarguram and Nalini Ramamurthy. Dengue fever Epidemic in Chennai- A Study of Clinical Profile and outcome. Indian Pediatrics 2002;39:1027-1030B.

83. Ooi ET, Ganesanathan S, Anil R, Kwok FY, Sinniah M, Gastrointestinal manifestations of Dengue infection in adults. Med J Malaysia 2008;63;401-05.

84. de Soza LJ, Nogueira RM, So9 9. Dinh The Trung, Le Thi Thu Thao ares LC, Soares CE, Ribas BF, Alves FP,Vieira FR,Pessanha FE. The impact of dengue on liver function asevaluated by aninotransferase levels. Braz J Infect Dis 2007;11:407-10.

85. Lt. Col M Banerjee, Lt Col T. Chatterjee, Lt. Col GS Choudhary, Col V Srinivas, Brig VK Kataria Dengue: A clinco Hematological profile. MJAFI 2008;64:333-36.

86. S Fadilah S Abdul Wahid, Sahrir Samsi, MM Zawawi, Raymond Azman Ali. A Comparison of the patients of liver with classic dengue hemorrhagic fever with classic dengue fever. South East Asian J Trop Med Public Health 2000;31:259-63.

87. Tran Tinh Hien, Nguyen The Hung, Nguyen Ngoc Vinh, Pham Tran Dieu Hien, Nguyen Tran Chinh, Cameron Simmons and Bridget Wills Liver InvolvementAssociated with Dengue Infection in Adults in Vietnam. Am J TropMed Hyg 2010;774-780.

88. Srivenu Itha, Rajesh Kashyap, Narendra Krishnani Vivek A, Saraswat, Profile of Liver Involvement National Medical Journal of India 2005;18
89. PM Venkata Sai R. Krishnan Role of Ultra sound in Dengue fever. British Journal of Radiology 2005;78:416-418.
90. Srivenu Itha, Rajesh Kashyap, Narendra Krishnani Vivek A, Saraswat, Profile of Liver Involvement National Medical Journal of India 2005;18.
91. Dengue haemorrhagic fever among adults – An observational study in Chennai, south India Indian J Med Res 132, December 2010, pp 738-740
92. Use of dengue NS1 antigen for early diagnosis of dengue virus infection. Kassim FM, Izati MN, TgRogayah TA, Apandi YM, Saat Z. Southeast Asian J Trop Med Public Health. 2011 May;42(3):5629.
93. Martin DA, Muth DA, Brown T, Johnson AJ, Karabatsos N, Roehrig JT. (2000) Standardization of immunoglobulin M capture enzyme-linked immunosorbent assays for routine diagnosis of arboviral infections. J Clin Microbiol;38 (5):1823-1826

94. Johnson, B. W., Russell, B. J., Lanciotti, R. S. (2005). Serotype-Specific Detection of Dengue Viruses in a Fourplex Real-Time Reverse Transcriptase PCR Assay. J. Clin. Microbiol. 43: 4977-4983
95. Dengue fever with thrombocytopenia: studies towards defining vulnerability of bleeding.García S, Morales R, Hunter RF Bol Asoc Med P R. 1995 Jan-Feb;87(1-2):2-7.
96. Clinical Profile of Dengue Infection in Patients with Hematological Diseases ;Sanjeev Kumar Sharma[1], Tulika Seth[1], Pravas Mishra[1], Nitin Gupta[1], Narendra Agrawal[1], Shobha Broor[2], M Mahapatra[1] and Renu Saxena[1] [1]Department of Hematology and [2]Microbiology, All India Institute of Medical Sciences, New Delhi, India. Vol 3, No 1 (2011) Published: September 8, 2011
97. Martinez E. Preventing deaths from dengue: a space and challenge for primary health care. Pan American Journal of Public Health, 2006, 20:60.
98. Harris E et al. Fluid intake and decreased risk for hospitalization for dengue fever,Nicaragua. Emerging Infectious Diseases, 2003, 9:1003–1006.
99. Lum L et al. Preventive transfusion in dengue shock syndrome – is it necessary? Journal of Pediatrics, 2003, 143:682–684.

100. Dellinger RP, Levy MM, Carlet JM. Surviving Sepsis Campaign: internationalguidelines for management of severe sepsis and septic shock: 2008. Critical CareMedicine, 2008, 36:296–327.

101. Dung NM, Day NP, Tam DT. Fluid replacement in dengue shock syndrome: arandomized, double-blind comparison of four intravenous-fluid regimens. ClinicalInfectious Diseases, 1999, 29:787–794

102. Ngo NT, Cao XT, Kneen R. Acute management of dengue shock syndrome: arandomized double-blind comparison of 4 intravenous fluid regimens in the first hour.Clinical Infectious Diseases, 2001, 32:204–213.

103. Wills BA et al. Comparison of three fluid solutions for resuscitation in dengue shock syndrome. New England Journal of Medicine, 2005, 353:877–889

104. Dellinger RP, Levy MM, Carlet JM. Surviving Sepsis Campaign: international guidelines for management of severe sepsis and septic shock: 2008. Critical Care Medicine, 2008, 36:296–327

105. WHO. Dengue haemorrhagic fever: diagnosis, treatment, prevention and control, 2nd edition. Geneva, World Health Organization, 1997.cine and Hygiene, 1969, 18(6):954–971

KEY TO THE MASTER CHART

- Sex:

Male:	1
Female:	2

- Symptoms:

Fever :	Yes	1	No	2
Chills :	Yes	1	No	2
Retro-orbital pain:	Yes	1	No	2
Arthralgia	Yes	1	No	2
Bone pain	Yes	1	No	2
Muscular pain	Yes	1	No	2
Nausea	Yes	1	No	2
Vomiting	Yes	1	No	2
Sore Throat	Yes	1	No	2
Seizures	Yes	1	No	2
Abdominal pain	Yes	1	No	2

- **BP:**

Normal:	1
Low:	2
High:	3

- **Pulse Pressure:**

Normal:	1
Low:	2

- **Signs:**

Icterus :	Yes	1	No	2
Pallor :	Yes	1	No	2
ConjunctivalCongestion:	Yes	1	No	2
Gum Bleeding:	Yes	1	No	2

Epistaxis:	Yes	1	No	2
Petechiae:	Yes	1	No	2
Hemetemesis:	Yes	1	No	2
Melena:	Yes	1	No	2
Hematuria:	Yes	1	No	2
Bleeding PR:	Yes	1	No	2
Bleeding PV:	Yes	1	No	2
Hepatomegaly:	Yes	1	No	2
Splenomegaly:	Yes	1	No	2
Myocarditis:	Yes	1	No	2
Encephalopathy:	Yes	1	No	2

- **Pleural Effusion:**

Yes-right:	1
Yes-Bilateral:	2
No:	3

- **Others:**

Rashes :	1
Exfoliative Dermatitis:	2
Diarrhoea:	3
Neck stiffness, altered sensorium:	4
Puffiness of face:	5
Breathlessness:	6
Pneumonia:	7
Loss of apetite:	8
Pelvic abscess:	9
Herpes Labialis:	10
Hemoptysis:	11
Subconjunctival haemorrhage:	12

- Co morbid conditions:

CAD:	Yes	1	No	2
DM:	Yes	1	No	2
HT:	Yes	1	No	2

❖ **Others:**

Pneumonia:	1
HIV:	2
TB:	3
ANC:	4
No:	5

❖ **USG:**

- **Hepatomegaly**

  Yes: 1

- **Splenomegaly**

  Yes: 1

- **GB Wall Edema**

  Yes: 1

- **Ascites**

  Yes: 1

- **Normal**

  Yes: 1

- P**leural effusion**:

  Right only: 1
  Bilateral: 2

❖ **Others:**

Renal calculus:	1
Ovarian cyst:	2
Fatty liver:	3
Right Renal cyst:	4
Pericholicedema,:	5
Fluid-Morrisons pouch:	5
Cholelithiasis:	6
Cholecystitis:	7

Loculated pelvic collections: 8
Prostatomegaly: 9

- **CBC:**

Yes: 1
No: 2

- **LFT:**

  - **Enzymes:**

SGOT,SGPT: 1
Alkaline Phosphatase Elevated:1
SGOT>SGPT: 2
SGPT=SGOT: 3
SGPT>SGOT: 4
SGOT only: 5

  - **Elevated Bilirubin:**

No: 0
Yes : 1

- RFT:

Normal: 1
Compromised: 2

- **ECG:**

Sinus bradycardia: 1
Sinus tachycardia: 2
Brady with 1st degree AV block: 3
Brady with 2nd degree AV block: 4
LVH: 5
T wave inversion V1- V4:6
Normal: 7

- **CXR:**

Rt pleural effusion: 1
Bl pleural effusion: 2
Cardiomegaly: 3
Rt lower lobe consolidation: 4

- **CT BRAIN :**

Not taken:	0
Minimal white matter hypodensity:	1
Intracerebral bleed:	2
Normal:	3

- **SEROLOGY :**
  - **NS1**

No:	0
Yes:	1

  - **IgM;**

No:	0
Yes:	1

  - **IgG**

No:	0
Yes:	1

- **Rx**:
  - **TRANSFUSION** :

Whole blood

No:	0
Yes:	1

Platelet:

No:	0
Yes:	1

FFP:

No:	0
Yes:	1

- **DIAGNOSIS:**
  - **DCF**

No:	0
Yes:	1

- **DHF**
  - No: 0
  - Yes: 1
- **DSS**
  - No: 0
  - Yes: 1
- **DEATH**
  - No: 0
  - Yes: 1

PROFORMA

Name								
Age								
Sex								
Days of stay in hospital								
Symptoms	Fever							
	Chills							
	Head ache							
	Retro-orbital pain							
	Arthralgia							
	Bone pain							
	Muscular pain							
	Nausea							
	Vomiting							
	Soar Throat							
	Seizures							
	Abdominal pain							
Signs	Pulse Rate							
	BP	Normal						
		Hypotension						
	Pulse Pressure							
	Icterus							
	Pallor							
	Conjunctival Congestion							
	Gum Bleeding							
	Mucosal Bleeding							
	Petechiae							
	Hemetemesis							
	Melena							
	Bleeding PR							
	Bleeding PV							
	Hepatomegaly							
	Splenomegaly							
	Myocarditis							
	Pleural Effusion							
	Ascites							
	Encephalopathy							
Co-Morbid Conditions	CAD							
	DM							
	HT							
Investigations	USG Ab	Hepatomegaly						
		Splenomegaly						
		GB Wall Edema						
		Pleural Effusion						
		Ascites						
		Normal						
	CBC	Leucopenia						
		Thrombocytopenia						
		HCT	Increased					
			Decreased					
		Normal						
	RFT	Compromised						
		Normal						
	LFT	Elevated Liver Enzymes						
		Elevated Bilirubin						
		Elevated PT / APTT / INR						
		Normal						
	ECG Changes	Present						
		Absent						
	Serology	NS1						
		Ig M						
		Ig G						
		Negative						
Treatment	IVF	Bolus						
		Maintenance						
	Transfusion	Whole Blood						
		Platelet						
		FFP						
	Mech. Vent.	Needed						
		Not needed						
No. of Patients Discharged								
COD (if expired)	DSS							
	DHF							
	DIC							
	Sepsis							
	Myocarditis							
	Others							

Symptoms and signs

Name	Sl. No.	Age	Sex	Days of stay in hospital	Fever	Chills	Head ache	Retro-orbital pain	Arthralgia	Bone pain	Muscular pain	Nausea	Vomiting	Soar Throat	Seizures	Abdominal pain	Pulse Rate	BP	Pulse Pressure	Icterus	Pallor	Conjunctival Congestion	Gum Bleeding	Epistaxis	Petechiae	Hemetemesis	Melena	Hematuria	Bleeding PR	Bleeding PV	Hepatomegaly	Splenomegaly	Myocarditis
vinoth	1	13	1	7	1	1	1	2	1	1	1	2	1	2	2	1	106	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
sudalaimani	2	14	1	7	1	2	2	2	2	2	1	2	2	2	2	2	112	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
RAJAGOPAL	3	44	1	6	1	2	1	2	1	2	1	1	2	2	2	1	58	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2
PANEER	4	50	1	7	1	1	1	2	1	2	1	2	1	2	2	1	82	1	1	2	2	1	2	2	1	2	1	2	2	2	2	2	2
ANEES	5	28	2	12	1	2	2	2	1	2	1	2	1	2	2	1	74	1	1	2	2	1	2	2	2	2	1	2	2	2	2	2	2
AMBAL	6	42	2	7	1	1	1	2	1	2	1	2	2	2	2	1	110	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2
BAKYAM	7	34	2	6	1	1	2	1	2	2	1	1	1	2	2	2	76	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2
PECHIMANI	8	18	2	5	1	1	1	1	2	2	1	2	2	2	2	1	88	1	1	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2
MARI	9	25	2	7	1	2	1	1	2	2	1	1	2	2	2	1	82	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2
CHANDRA	10	55	2	7	1	2	1	1	2	2	1	2	2	2	2	1	100	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2
ESAKIAMMAL	11	29	2	6	1	2	2	2	2	2	1	1	1	2	2	1	74	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2
TAMILSELVI	12	23	2	6	1	2	1	1	2	2	1	2	2	2	2	2	112	2	2	2	2	1	1	2	2	2	2	2	2	2	2	2	2	2
USHA	13	27	2	7	1	1	1	1	2	2	1	2	2	2	2	2	106	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2
ARUMUGATHAI	14	40	2	7	1	2	1	1	2	2	1	2	2	2	2	2	104	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
RAJAKUMARI	18	26	2	7	1	2	2	2	2	2	1	1	2	2	2	1	107	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SADALI	19	55	2	6	1	1	1	1	1	1	1	1	1	2	2	1	60	3	1	2	1	1	2	2	2	2	2	2	2	2	2	2	2	2
mahalakshmi	20	30	2	4	1	1	1	2	2	2	1	1	1	2	2	2	84	1	1	2	2	1	2	2	1	2	2	2	2	2	2	2	2	2
SHAJAHAN	21	34	1	6	1	1	1	2	1	2	1	2	2	2	2	2	112	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1
SARATHY	22	17	1	6	1	1	1	1	1	2	2	2	1	2	2	2	110	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2
SARAVANAN	23	18	1	5	1	2	2	2	2	2	1	2	1	2	2	2	106	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
siriyapuspam	24	18	2	5	1	1	1	1	1	2	1	2	1	2	2	2	109	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
rajasekar	25	54	1	5	1	2	1	2	1	2	1	2	1	1	2	2	112	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
RENGANATHAN	27	20	1	6	1	1	1	2	1	2	1	2	2	2	2	1	60	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
PRIYA	29	26	2	6	1	1	2	2	2	2	2	1	2	1	2	1	100	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
PETCHIAMMAL	30	20	2	6	1	1	2	2	1	2	1	2	1	2	2	1	114	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
KARUNYA	31	22	2	6	1	2	2	2	2	2	2	2	2	2	2	1	88	1	1	2	2	1	2	2	2	2	1	1	1	1	2	2	2	2
JEYACHANDRAN	32	24	1	6	1	2	1	2	2	2	1	1	1	2	2	1	54	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2
KANNAN	33	19	1	7	1	1	1	2	2	2	2	2	2	2	2	2	90	1	1	2	2	2	2	2	2	2	2	1	2	2	2	1	2	2
mariyaraj	34	20	1	5	1	1	1	1	2	2	1	2	1	1	2	2	120	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ESAKIAMMAL	36	15	2	9	1	1	1	1	2	2	1	2	1	2	2	2	110	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2
PRIYA	38	16	2	5	1	1	1	2	2	2	1	2	2	1	2	1	84	1	1	2	2	2	2	1	2	2	2	2	2	2	2	2	2	2
RAMALAKSHMI	39	17	2	8	1	1	1	1	2	2	2	2	2	2	2	2	110	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2
GAYATHIRI	40	24	2	6	1	2	2	2	2	2	1	1	2	2	2	2	105	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2	2
SUMATHY	41	14	2	4	1	2	1	2	2	2	1	2	2	2	2	2	108	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
aasir samraj	43	14	1	5	1	1	1	2	2	2	2	1	1	2	2	1	109	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2
KRISHNA	44	19	1	8	1	1	1	1	2	2	2	2	1	1	2	1	60	1	1	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2
ANBARASI	45	16	2	7	1	1	2	2	1	2	1	2	2	2	2	1	114	1	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2

AJITH	46	15	1	6	1	1	1	2	2	2	2	1	1	2	2	2	118	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
LAKSHMI	47	37	2	10	1	2	2	2	1	2	1	2	2	2	2	2	104	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
BIRTHOUS	48	32	2	6	1	1	1	2	2	2	2	2	2	2	2	1	112	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
BALASUBRAMANIAN	49	22	1	4	1	1	1	1	2	2	2	2	2	2	2	2	56	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
DHARMAR	51	55	1	6	1	2	1	2	1	2	1	1	1	2	2	1	80	1	1	2	2	2	2	1	2	2	2	2	2	2	2	1	2
ESAKIRAJ	52	13	1	5	1	1	2	2	2	2	2	1	1	1	2	2	103	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SUDALAI SELVI	53	16	2	4	1	2	2	2	2	2	1	1	2	2	2	2	100	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
VANI	54	24	2	6	1	1	1	2	2	2	2	2	1	1	2	1	82	1	1	2	2	2	2	1	1	1	2	2	2	2	2	2	2
GOMATHI	55	60	2	12	1	2	1	2	2	2	1	2	2	2	2	1	103	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
KAVITHA	56	14	2	4	1	1	1	2	1	2	1	1	1	2	2	2	109	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
BALASUBRAMANI	57	13	1	7	1	2	2	2	1	2	2	2	2	2	2	2	102	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
KUMARESAN	59	19	1	6	1	1	2	2	1	2	1	2	2	2	2	2	100	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
KARTHIKEYAN	60	32	1	5	1	2	2	2	2	2	2	2	2	2	2	2	120	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
KOKILA	61	13	2	6	1	2	1	2	2	2	1	2	2	1	2	2	114	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SANDHYA	62	15	2	6	1	2	1	2	2	2	1	2	2	2	2	2	110	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2
LAKSHMI	63	55	2	5	1	2	1	2	2	2	1	2	2	2	2	2	112	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SUBBHU	64	50	2	6	1	2	2	2	2	2	1	2	2	2	2	1	62	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
GOMATHI	65	14	2	5	1	2	1	1	2	2	1	2	2	2	2	2	104	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
BHUVANESWARI	66	14	2	5	1	2	2	2	2	2	1	2	2	2	2	2	78	1	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2
ISAKI	67	30	1	3	1	2	1	1	1	2	1	2	2	2	2	2	100	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	1
KARUMARI	69	14	2	6	1	2	1	1	2	2	2	2	2	2	2	2	103	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
MAHARAJA	71	18	1	6	1	2	1	2	1	2	1	2	2	2	2	2	107	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
AYYADURAI	73	29	1	6	1	1	1	2	2	2	1	2	2	2	2	2	90	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
KODIMALAR	74	23	2	6	1	1	1	2	2	2	2	2	1	2	2	1	54	2	2	2	2	2	1	2	2	2	2	2	2	1	2	2	2
RAJKUMAR	75	16	1	7	1	2	2	2	2	2	2	2	2	1	2	1	106	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
PATTU	76	17	2	6	1	2	1	2	2	2	2	2	2	2	2	2	64	1	1	2	2	2	1	2	1	2	2	2	2	2	2	2	2
VELAMMAL	77	35	2	4	1	2	2	2	1	1	2	2	2	2	2	2	102	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2
AYYAMMAL	78	17	2	5	1	2	2	2	2	2	2	1	1	2	2	2	110	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2
MARIAMMAL	79	23	2	4	1	2	1	1	1	2	2	2	1	2	2	2	108	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
LAKSHMANAN	80	49	1	6	1	2	2	2	2	2	2	2	2	2	2	1	100	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ISAKIAMMAL	81	35	2	6	1	1	2	2	1	2	1	2	2	2	2	1	108	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ISWARIYA	83	14	2	7	1	1	2	2	2	2	2	2	1	2	2	2	100	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
INDHUMATHI	84	16	2	6	1	1	2	2	2	2	2	2	2	2	2	2	112	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2
ABRAHAM	85	50	2	10	1	1	2	2	2	2	2	2	2	2	2	2	62	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SHAKTHIMURUGAN	86	15	1	6	1	1	2	2	2	2	2	2	2	1	2	2	104	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
KATHIJA BEEVI	87	23	2	6	1	2	1	1	2	2	2	2	1	2	2	1	60	3	1	2	2	1	2	2	2	2	2	2	2	2	1	2	2
GNANAMOORTHY	88	14	1	5	1	1	1	1	1	2	2	2	1	2	2	1	90	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
RAMALAKSHMI	89	16	2	5	1	1	1	2	2	2	2	2	2	2	2	2	46	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
THANALAKSHMI	90	15	2	10	1	2	2	2	2	2	1	2	2	2	2	1	106	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
BHARATH	91	21	1	6	1	1	2	2	2	2	2	2	1	2	2	1	100	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
PATTAMUTHU	92	40	1	6	1	2	1	2	2	2	2	2	1	2	2	2	101	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
RAMALAKSHMI	93	28	2	5	1	2	1	2	1	2	1	2	1	2	2	2	82	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2
NARAYANAN	94	17	1	6	1	1	1	1	1	1	2	1	1	2	2	1	109	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
VEERAPANDI	95	35	1	6	1	1	1	2	1	2	1	2	2	2	2	1	101	2	1	2	2	1	2	2	2	2	2	2	2	2	1	2	2
KARUPPAIAH	96	42	1	6	1	1	1	1	2	2	2	2	1	2	2	2	109	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ISAKKIAMMAL	97	40	2	5	1	1	1	2	1	2	2	2	2	2	2	2	103	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ANITHA	98	18	2	6	1	1	2	2	2	2	2	2	1	2	2	2	102	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2

RAMKUMAR	99	14	1	5	1	2	2	2	1	2	1	2	1	2	2	1	109	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
KRISHNAMOORTHY	100	17	1	6	1	1	1	2	1	2	2	2	2	2	2	2	105	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
SHANMUGAVADIVU	101	38	2	5	1	1	1	1	1	1	2	2	2	2	2	1	120	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
MARISELVI	102	15	2	10	1	2	1	2	2	2	1	2	2	2	2	2	100	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
STALIN	103	17	1	5	1	2	2	2	1	1	2	2	2	2	2	2	109	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	1
SANKARESWARI	104	22	2	7	1	2	2	2	2	2	2	2	1	2	2	1	101	2	1	2	2	2	2	2	2	2	2	1	2	2	2	2	1	2
PARVATHI	106	27	2	7	1	1	1	2	2	2	1	2	2	2	2	1	72	1	1	2	2	2	2	2	1	2	1	2	2	2	2	1	2
NANDHINI	107	15	2	7	1	2	2	1	2	2	1	2	2	2	2	1	100	2	1	2	2	2	2	2	1	2	2	2	2	1	2	2	2
MERLIN	109	24	2	7	1	1	1	2	1	2	1	2	2	1	2	2	120	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
MURUGESH	112	15	1	10	1	2	2	1	1	2	1	2	2	2	2	2	118	2	2	2	2	1	2	2	2	2	1	2	2	2	2	2	2
MARIAMMAL	113	13	2	8	1	2	2	2	1	2	1	1	2	2	2	1	92	2	1	2	2	2	2	2	1	2	1	2	2	2	2	2	2
MICHAEL	114	27	1	10	1	1	1	2	1	2	1	2	2	2	2	2	110	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
RAJESHWARI	115	15	2	12	1	1	1	2	2	2	2	2	2	2	2	2	118	2	1	2	2	1	2	2	1	2	2	2	2	1	2	2	2
MAHALINGAM	116	75	1	8	1	1	1	1	2	2	2	2	2	2	2	2	86	1	1	2	2	2	2	2	1	2	1	2	2	2	2	2	2
PETCHIDURAI	117	17	1	9	1	1	2	2	2	2	2	2	2	2	2	2	103	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
MAYAKRISHNAN	118	35	1	7	1	2	2	2	2	2	1	2	2	2	2	1	48	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
PETER	120	54	1	5	1	1	1	2	2	2	2	1	1	2	2	1	110	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2
POOLIAH	121	30	1	7	1	2	1	1	2	2	2	2	2	2	2	2	113	2	2	2	2	2	2	2	2	2	1	2	2	2	1	2	2
MURUGAIAH	122	65	1	6	1	2	2	2	2	2	2	2	2	1	2	2	72	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
DEVI	124	25	2	5	1	1	2	2	2	2	2	2	1	1	2	1	52	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
PALANI	125	25	1	5	1	1	1	2	1	2	2	1	2	2	2	2	112	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
nallammal	126	48	2	4	1	2	1	2	1	2	1	1	2	2	2	1	119	2	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
PREMKUMAR	127	22	1	9	1	2	2	2	2	2	2	2	1	2	2	2	100	2	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2
PRAKASH	129	21	1	5	1	1	1	1	1	2	1	2	2	2	2	2	90	1	1	2	2	1	2	1	2	2	2	2	2	2	2	2	2
pitchammal	130	55	2	4	1	2	1	1	1	2	1	2	1	2	2	1	56	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
CHANDRALEKHA	131	24	2	4	1	2	1	1	1	2	1	2	2	2	2	2	72	1	1	2	2	1	2	2	1	2	2	2	2	2	2	2	2
CHINNATHAI	132	24	2	5	1	2	1	1	2	2	2	2	2	2	2	1	96	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SIVA	133	17	2	1	1	2	1	2	1	2	1	2	2	2	1	2	112	2	2	2	2	1	1	2	2	1	2	2	2	2	2	2	2
CHELLAMMAL	134	50	2	6	1	2	1	2	2	2	1	1	2	2	2	2	98	2	1	2	2	1	1	2	2	1	2	2	2	1	2	2	2
SUGANTHI	136	18	2	6	1	2	1	2	2	2	2	2	1	2	2	2	52	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SUBRAMANIYAN	138	21	1	5	1	2	1	1	2	2	2	2	1	2	2	1	82	1	1	2	2	2	2	1	2	2	1	2	2	2	2	2	2
NIYAS AHAMED	139	15	1	8	1	2	2	2	2	2	1	2	2	2	2	1	96	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
SELVAKUMAR	140	14	1	5	1	2	2	2	1	2	1	2	2	2	2	2	50	2	1	2	2	2	2	2	2	2	1	1	2	2	2	2	2
SHUNMUGAIAH	141	65	1	6	1	1	2	2	2	2	2	2	1	2	2	2	68	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
SORNAM	142	35	2	13	1	1	1	2	2	2	1	2	1	2	2	1	98	1	1	2	2	2	2	2	2	2	2	2	2	2	1	1	2
SARAVANAN	143	15	1	6	1	2	2	2	2	2	1	2	2	2	2	2	80	2	1	2	2	1	2	2	2	2	1	2	2	2	2	2	2
MARISELVI	144	29	2	7	1	1	2	2	2	2	2	2	2	2	2	2	72	1	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2
KARUPPASAMY	146	50	1	3	1	2	2	2	1	2	2	2	1	2	2	2	72	1	1	2	2	2	2	2	1	2	1	2	2	2	1	2	2
SHUNMUGASUNDARAM	147	19	1	7	1	1	2	2	2	2	2	2	1	2	2	1	110	2	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
ANITHA	148	20	2	5	1	1	2	2	2	2	1	2	1	1	2	2	112	2	1	2	2	2	1	2	2	2	2	2	2	1	2	2	2
MAYILMURUGAN	150	13	1	8	1	2	2	2	1	2	1	2	2	2	2	2	73	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
SIVARANCHANI	151	15	2	7	1	1	2	2	2	2	2	2	1	2	2	2	110	2	1	2	2	1	2	1	2	2	2	2	2	2	2	2	2
RAGHU	152	26	1	5	1	2	1	2	1	2	2	2	1	1	2	2	80	1	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2
RAMASAMY	153	45	1	4	1	1	2	2	2	2	2	2	2	2	2	2	80	1	1	2	1	2	2	2	2	2	1	2	2	2	2	2	2
SORIMUTHU	155	25	1	6	1	2	1	2	1	2	1	2	1	2	2	2	104	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
saamundeswari	156	22	2	4	1	2	2	2	1	2	1	1	2	2	2	2	98	1	1	2	1	2	2	2	2	2	1	2	2	2	2	2	2
DELSI	158	13	2	4	1	2	2	2	2	2	2	2	1	1	2	2	110	2	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2

SHUNMUGAM	159	60	1	7	1	2	2	2	2	2	1	2	1	2	2	1	84	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SHARMILABANU	160	31	2	11	1	2	2	2	2	2	2	2	1	2	2	2	112	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SASIKUMAR	161	19	1	8	1	2	1	2	2	2	1	2	1	2	2	1	80	1	1	1	2	2	2	2	2	2	2	2	2	2	1	2	2
SUGANYA	162	16	2	5	1	2	2	2	2	2	2	2	1	2	2	1	92	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
ILAIYAPERUMAL50	165	50	1	9	1	1	1	2	2	2	1	2	2	2	2	2	60	3	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
KAMALESH	166	13	1	4	1	1	2	2	1	2	1	2	2	2	2	2	100	2	1	2	2	1	2	2	2	1	2	2	2	2	1	2	2
MUTHULAKSHMI	167	27	2	10	1	2	2	2	2	2	2	2	1	2	2	2	84	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
GANDHI	168	47	1	6	1	2	1	2	2	2	1	2	1	2	2	2	82	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
VIGNESH	169	17	1	5	1	2	1	2	2	2	1	2	2	2	2	2	76	1	1	2	2	2	2	2	2	2	2	2	2	2	1	1	2
SELVI	171	16	2	6	1	1	1	2	2	2	1	2	1	2	2	2	78	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
SHEIKMOHAMMED	172	16	1	9	1	1	1	1	1	2	2	2	2	2	2	2	102	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
SAMDURAI	174	17	1	5	1	2	1	2	1	2	1	1	2	2	2	1	98	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ANAND	175	26	1	7	1	1	1	2	2	2	2	2	2	2	2	2	82	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2
ABDULKATHAR	176	45	1	5	1	2	1	2	1	2	2	2	1	2	2	2	74	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2
AMEEN	177	13	1	5	1	1	1	2	1	2	1	2	2	2	2	2	110	2	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
MYDEEN	179	24	1	4	1	2	2	2	2	2	2	2	2	2	2	2	98	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SELVARAJ	181	23	1	4	1	2	2	2	1	2	1	2	2	1	2	2	70	1	1	2	2	2	2	2	1	2	1	2	2	2	2	2	2
MANIKANDAN	182	40	1	6	1	2	1	1	2	2	2	2	2	1	2	1	50	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2
FASID	183	13	1	7	1	2	2	1	1	2	1	2	2	2	2	2	80	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
LAKSHMANAN	184	45	1	5	1	2	2	2	2	2	2	2	2	2	2	1	92	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
NAMEETHAL BEEVI	185	55	2	7	1	2	2	2	1	2	1	1	2	2	2	1	86	1	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2
ALAMEEN	186	22	1	5	1	2	1	2	1	2	2	2	2	2	2	2	88	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2
ABHINESH	189	21	1	8	1	2	1	1	1	2	2	2	1	2	2	2	92	2	2	2	2	2	2	2	2	2	2	2	2	2	2	1	2
SAMI	190	24	1	13	1	1	1	2	1	2	1	2	1	2	2	2	82	1	1	2	2	1	2	1	2	2	2	2	2	2	2	2	2
VALARMATHI	191	22	2	4	1	1	1	1	2	1	2	2	1	2	2	2	88	3	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2
KARPAGAM	192	30	2	4	1	1	1	2	2	2	2	2	1	2	2	1	82	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ESAKIMUTHU	193	27	1	5	1	1	1	1	2	2	2	2	1	1	2	1	80	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
MURUGAN	195	39	1	7	1	1	1	1	2	2	2	2	2	2	2	1	86	1	1	2	2	2	2	2	2	2	2	2	2	2	1	1	2
SYED MAZOODU	196	55	1	8	1	2	1	1	1	2	2	2	2	2	2	2	84	1	1	2	2	2	1	2	2	2	1	2	2	2	2	2	2
BASKARAN	197	29	1	4	1	1	1	2	2	2	2	2	2	2	2	2	82	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
PATTUKUMAR	201	17	1	8	1	2	1	1	2	2	2	2	1	2	2	1	78	1	1	2	2	2	2	2	2	2	1	2	2	2	1	1	2
SOLAI AMMAL	202	18	2	6	1	2	2	2	2	2	2	2	1	2	2	1	106	2	1	2	2	2	2	2	2	2	2	2	2	2	1	1	2
MURUGAN	203	47	1	7	1	1	1	1	1	2	2	2	2	1	2	1	82	1	1	2	2	2	2	2	2	2	1	2	2	2	1	1	2
BULA	204	19	2	7	1	2	1	1	2	2	1	2	2	2	2	1	108	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
KARTHIKEYAN	206	17	1	6	1	2	1	1	2	2	1	2	2	2	2	2	82	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
RAMAN	207	25	1	5	1	1	2	2	2	2	2	2	2	1	2	2	58	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
RAJENDRAN	209	50	1	9	1	1	2	2	1	2	1	2	2	2	2	1	60	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
SAMEERA BANU	210	18	2	4	1	1	1	1	2	2	2	1	1	2	2	1	96	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
GOPALA KRISHNAN	212	21	1	10	1	1	1	1	2	2	1	2	2	2	2	1	52	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
KOVIL RAJ	214	22	1	6	1	1	1	2	1	2	2	2	2	1	2	2	44	1	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2
ARUMUGAM	215	43	1	9	1	2	1	1	1	2	1	2	2	2	2	2	88	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
SORNAMALA	217	42	2	5	1	2	2	2	2	2	2	2	2	1	2	2	78	1	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2
USHA	218	23	2	9	1	2	1	1	2	2	2	2	2	1	2	2	76	1	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2
UDHAYAKUMAR	219	13	1	4	1	2	1	1	2	2	1	2	1	1	2	1	56	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
ANANDHI	222	17	2	10	1	2	2	2	1	2	1	2	2	2	2	1	78	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
UMA	223	37	2	5	1	1	1	1	1	2	1	2	2	2	2	2	82	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
KAJA MYDEEN	224	20	1	7	1	2	2	2	1	2	1	2	2	2	2	2	75	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2

SREEBANU	226	18	2	5	1	2	1	1	2	2	2	2	1	2	2	1	86	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
RAHEELA	227	17	2	7	1	1	1	1	1	2	1	1	1	1	1	1	82	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
MANIMARAN	229	16	1	5	1	2	2	2	1	2	1	2	1	2	2	2	72	2	2	1	1	1	2	2	2	2	2	2	2	2	2	2	2
SURESH	230	20	1	6	1	1	1	1	1	2	1	2	1	1	2	1	90	1	1	2	2	2	2	2	2	2	2	2	2	2	1	2	2
SINTHANISHA	231	15	2	4	1	1	1	2	1	2	1	2	2	2	2	1	84	1	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2
SARASWATHY	232	27	2	5	1	1	1	1	1	2	1	2	1	2	2	2	96	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2
MANIKANDAN	233	17	1	6	1	1	1	1	2	2	1	2	2	2	2	2	86	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
SANKARI	235	23	2	6	1	1	1	1	2	2	2	2	1	2	2	1	92	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2
SELVI	236	31	2	5	1	1	2	2	1	2	1	2	2	1	2	2	86	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SRIPAUL	237	23	1	8	1	2	1	2	1	2	1	2	2	2	2	2	50	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
NAJIMA	238	17	2	5	1	2	1	1	2	2	2	2	1	1	2	1	82	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
PREMRAJ	239	29	1	6	1	1	1	1	1	2	1	2	2	1	2	2	92	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ROHINI	240	14	2	8	1	1	2	2	1	2	1	2	1	1	2	1	44	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SARAVANAN	241	22	1	7	1	2	2	2	1	2	1	2	2	2	2	2	92	1	1	2	2	1	2	2	2	2	1	2	2	2	1	1	2
VELTHAI	244	23	2	3	1	2	1	1	1	2	1	2	2	2	2	2	80	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SAKTHI	245	23	2	3	1	1	1	1	1	2	1	1	2	2	2	2	48	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
KALIAMMAL	246	28	2	9	1	2	1	2	1	2	1	2	1	2	2	1	88	1	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2
MUTHUSELVI	249	21	2	6	1	2	2	2	2	2	1	2	1	2	2	1	82	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ANANDHA SELVI	250	14	2	5	1	1	1	1	2	2	2	2	2	2	2	1	48	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
JAMERLA	15	23	2	5	1	2	2	2	1	2	1	2	2	2	2	2	86	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
CHANDRASEKAR	26	20	1	5	1	1	1	2	2	2	2	2	1	2	2	2	74	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
VELAMMAL	28	45	2	6	1	1	1	2	2	2	1	2	2	2	2	1	78	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
MALLIKA	37	18	2	4	1	2	1	1	1	1	1	2	1	2	2	1	98	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ANU	42	28	2	6	1	2	1	1	1	2	1	1	2	1	2	1	54	1	1	2	2	2	2	2	2	2	2	1	2	2	2	2	2
KAPILKUMAR	58	16	1	6	1	2	2	2	2	2	2	1	1	1	2	1	90	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
PRABHAKARAN	68	22	1	5	1	1	2	2	2	2	1	1	2	2	2	2	86	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
DIVYA	72	18	2	5	1	2	1	2	2	2	2	2	1	2	2	2	78	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
MARI	111	17	2	10	1	2	2	2	2	2	2	2	1	2	2	2	82	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
prasanth	119	17	1	4	1	2	2	2	2	2	1	2	2	2	2	2	100	2	1	2	2	1	2	2	2	2	2	1	2	2	2	2	2
PALMANI	128	24	2	3	1	1	1	2	2	2	2	1	2	2	2	2	82	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SHAJAHAN	135	40	1	9	1	2	2	2	2	2	2	2	2	2	2	2	82	1	1	2	2	2	2	2	1	2	1	2	2	2	2	2	2
BRAHAMACHI	149	50	2	10	1	2	1	2	2	2	1	2	2	2	2	2	128	2	1	2	2	1	2	2	2	2	1	1	2	2	1	2	1
RAJAMMAL	154	45	2	6	1	2	2	2	2	2	2	2	2	2	2	1	116	2	1	2	2	2	1	2	2	2	2	2	2	2	2	2	2
AMALA ANTO	188	13	2	6	1	2	2	1	2	2	1	2	2	2	2	1	100	1	1	2	2	2	2	2	2	2	1	2	2	2	2	2	2
SENTHOOR KANI	194	30	2	5	1	2	1	1	2	2	2	2	1	2	2	2	60	2	1	2	1	2	2	2	2	2	1	2	2	2	2	2	2
VALLI	198	25	2	5	1	2	1	1	2	2	2	2	1	2	2	2	86	1	1	2	2	1	2	2	2	2	1	2	2	2	2	2	2
CHAKARAVARTHY	200	17	1	8	1	2	1	1	2	2	2	2	1	2	2	2	98	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
MURUGAN	205	30	1	7	1	1	1	2	1	2	1	2	1	2	2	1	82	1	1	2	2	1	2	2	1	2	2	2	2	2	2	2	2
FATHIMA	208	17	2	5	1	1	1	1	2	2	1	2	2	2	2	1	48	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
KULANTHAI RAJ	213	55	1	10	1	1	1	1	1	2	1	2	1	2	2	1	120	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
AJAY	216	14	1	6	1	1	2	2	1	2	1	2	2	2	2	2	106	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ISAKIRAJA	225	13	1	5	1	1	1	1	1	2	1	2	1	2	2	1	76	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2	2
MADASAMY	228	15	1	6	1	2	2	2	2	2	2	2	2	2	2	2	76	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
MARIAPPAN	251	19	1	5	1	2	1	1	2	2	1	2	2	2	2	2	72	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
GOHILA	16	25	2	4	1	2	2	2	1	2	1	2	2	2	2	1	90	1	1	2	1	1	2	2	2	2	2	2	2	2	2	2	2
MOHAMMED	17	17	1	8	1	2	1	1	2	2	1	2	2	2	2	1	82	1	1	2	2	1	2	2	1	2	2	2	2	2	2	2	2
MATHUMATHI	35	13	2	5	1	2	1	1	2	2	2	2	1	2	2	1	88	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2

CHANDRASEKAR	50	27	1	7	1	2	2	2	2	2	1	2	2	1	2	2	80	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2
ARUMUGAM	70	19	1	5	1	1	1	2	2	2	1	2	2	1	2	1	84	1	1	2	2	1	2	2	2	2	2	2	2	2	2	2	2
GNANAPOO	82	52	2	5	1	2	2	2	2	2	1	2	1	2	2	2	88	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
PATTAMMAL	105	35	2	7	1	1	1	1	2	2	1	2	2	2	2	2	76	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
MARI	123	25	2	6	1	2	2	2	2	2	1	2	1	2	2	2	80	1	1	2	2	2	2	2	1	2	1	2	2	2	2	2	2
ALAVUDEEN	137	17	1	5	1	2	1	2	1	2	1	2	1	2	2	1	86	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
SONIAGANDHI	170	23	2	6	1	1	2	2	2	2	2	2	2	2	2	1	88	1	1	2	2	1	2	2	2	2	1	2	2	2	2	2	2
SUMATHY	199	28	2	4	1	1	1	1	1	2	1	2	2	2	2	1	76	1	1	2	2	2	2	2	1	2	2	2	2	2	2	2	2
SUBASH	220	13	1	2	1	1	2	2	2	2	2	2	1	2	2	2	110	2	2	2	2	2	2	2	2	2	1	2	2	2	2	2	2
JASIMVICTOR	221	17	1	9	1	1	1	1	1	2	1	2	1	2	2	1	106	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SEKAR	234	40	1	6	1	2	1	1	2	2	2	2	1	2	2	1	90	2	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SUDALAIMANI	242	35	1	6	1	1	1	1	1	2	1	2	1	1	2	1	88	1	1	2	1	2	2	2	2	2	2	2	2	2	2	2	2
SANGEETHA	247	19	2	7	1	1	2	2	2	2	2	2	1	2	2	1	52	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
DEVIKALA	248	25	2	6	1	1	2	2	2	2	2	2	1	2	2	1	74	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
SARASWATHI	108	31	2	9	1	2	1	1	2	2	2	2	2	2	1	2	107	2	1	2	2	2	2	2	1	1	1	2	2	2	2	2	2
ARUN	164	20	1	6	1	1	1	2	1	2	1	1	1	2	2	1	80	1	1	2	2	2	2	2	2	2	1	2	2	2	1	2	2
MOHAMMEDRAHIM	163	30	1	4	1	1	2	2	2	2	2	1	1	2	2	1	62	3	1	1	2	1	2	2	2	2	1	2	2	2	2	2	2
MADASAMY	110	27	1	7	1	2	2	1	1	2	1	2	2	2	2	2	88	1	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2
RAJESWARI	157	36	2	7	1	2	1	2	1	2	1	2	2	2	2	1	86	1	1	2	2	1	2	2	2	2	1	2	2	2	2	2	2
SENTHIL	173	21	1	6	1	2	1	2	2	2	2	1	1	1	2	2	72	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
MALATHI	178	15	2	5	1	1	1	2	2	2	2	1	1	1	2	2	98	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
MURUGAN	180	42	1	6	1	2	2	2	1	2	2	2	2	2	2	1	100	1	1	1	2	2	2	2	2	2	1	2	2	2	2	2	2
LAKSHMI	187	16	2	14	1	2	1	2	2	2	2	2	1	2	2	1	120	2	1	1	1	2	2	2	2	2	1	2	2	1	2	2	2
KANAGA	211	18	2	7	1	1	1	1	1	2	2	2	1	2	2	1	94	1	1	2	2	2	2	2	2	2	2	2	2	2	2	2	2
ANGEL	243	48	2	10	1	2	1	2	2	2	2	2	2	2	2	2	85	1	1	2	2	2	2	2	2	2	2	2	2	2	2	1	2
NAMBIAMMAL	145	37	2	6	1	1	2	2	2	2	2	2	1	2	2	1	48	2	1	2	2	1	2	2	1	2	1	2	2	2	1	1	2

			Co-Morbid Conditions							USG Ab				CBC																	
Pleural Effusion	Ascites	Encephalopathy	Others	CAD	DM	HT	Others	Hepatomegaly	Splenomegaly	GB Wall Edema	Pleural Effusion	Ascites	Others	Normal	Leucopenia	Thrombocytopenia	HCT 1-Normal; 2-Increased; 3-	RFT (1-Normal; 2-Compromise	Elevated Liver Enz1mes	Elevated Bilirubin	ECG Changes	NS1	Ig M	Ig G	CXR PLAIN P/A	CT Brain	Whole Blood	Platelet	FFP	Transfusion (1-Whole Blood; 2	DSS	DHF	DCF	Death
3	2	2	5	2	2	2	5							1	2	1	1	1			2	1	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5			1					2	1	1	1			2	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	1	1	2	1	1	1	1	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5						1		1	1	3	1			7	1	1	0	0	0	1	1		12		1		0
3	1	2	0	2	2	2	5			1		1			1	1	2	1			7	1	0	0	0	0	1	1		12		1		0
3	2	2	0	2	2	2	1	1							1	1	3	1	2		2	1	0	0	4	0				0			1	0
3	2	2	0	2	2	2	5							1	1	1	3	1			7	0	1	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5							1	2	1	3	1			7	0	1	0	0	0				0		1		0
3	2	2	0	2	2	2	5							1	1	1	3	1			7	1	0	0	0	0	1	1		12		1		0
1	2	2	0	2	2	1	5			1	1				2	1	3	1		1	2	1	1	0	1	0		1		2			1	0
3	2	2	0	2	2	2	5		1						2	1	2	1			7	0	1	0	0	0	1	1	1	123		1		0
1	1	2	12	2	2	2	5				1	1			2	1	3	1	3		2	0	1	0	1	0	1	1		12	1	1		0
3	2	2	0	2	2	2	5							1	1	2	1	1			2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			2	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			2	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5						2		2	1	1	1			7	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5			1					2	1	2	1	3		2	1	0	0	0	0				0			1	0
3	2	2	12	2	2	2	5			1					2	1	3	1	1		2	0	1	0	0	0	1			1	1			0
2	1	2	0	2	2	2	5			1	2	1			2	1	2	1	4	1	2	0	1	0	2	0				0			1	0
3	2	2	5	2	2	2	5							1	1	1	1	1	2		2	1	0	0	0	0				0			1	0
3	2	2	0	2	1	1	5						3		2	1	2	1	4		2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5		1						2	1	2	1	2	1	1	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5			1					2	1	2	1	2		2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5						3		2	1	1	1	2		2	0	1	0	0	0				0			1	0
3	1	2	0	2	2	2	5			1		1			1	1	2	1	2		7	0	1	0	0	0	1	1		12		1		0
3	2	2	0	2	2	2	5							1	2	1	2	1			1	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5	1				1			2	1	2	1			7	1	0	0	0	0	1	1		12		1		0
3	2	2	5	2	2	2	5							1	2	1	1	1			2	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5			1					2	1	3	1			2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	1	3	1			7	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5							1	1	1	3	1	4		2	0	1	0	0	0	1			1			1	0
3	2	2	0	2	2	2	5			1					2	1	2	1			2	1	0	0	0	0				0			1	0
3	1	2	0	2	2	2	5			1		1			1	1	1	1	2	1	2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5	1				1			1	1	2	1			2	0	1	0	0	0				0			1	0
3	1	2	0	2	2	2	5	1				1			2	1	2	1			3	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5							1	1	1	2	1	2	1	2	1	1	0	0	0				0			1	0

3	2	2	0	2	2	2	5							1	1	1	1	2			2	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	1	1	1		1	2	1	0	0	0	0				0			1	0
3	2	2	5	2	2	2	5							1	2	1	1	1			2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	1	1	1	1			1	1	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5		1						2	1	2	1			7	0	1	0	0	0				0		1		0
3	2	2	0	2	2	2	5							1	1	1	1	1			2	1	0	0	0	0				0			1	0
2	2	2	5	2	2	2	5				2				2	1	2	1	3		2	0	1	0	2	0				0			1	0
3	2	2	0	2	2	2	5			1					2	1	2	1	2		7	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5							1	2	1	1	1	1		2	0	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	1	1	1	1		2	0	1	0	0	0				0			1	0
1	1	2	0	2	2	2	5			1	1	1			2	1	2	1	1		2	0	1	0	1	0		1		2			1	0
3	2	2	0	2	2	2	5				1	1			1	1	2	1			7	1	1	0	1	0	1	1		12		1		0
3	2	2	0	2	2	2	5				2	1			2	1	2	1			2	0	1	0	2	0	1			1			1	0
1	1	2	0	2	2	2	5				1	1			1	1	2	1	1		2	0	1	0	1	0				0			1	0
3	2	2	5	2	2	2	5							1	2	1	3	1	1		2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5			1					2	1	1	1			2	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	1	3	1		1	1	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	1	2	1	1			2	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5					1			1	1	2	1	1	1	7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5							1	1	1	2	1			2	1	1	1	0	0				0			1	0
3	2	2	0	2	2	2	5			1					1	1	1	1	1		2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5			1					2	1	1	1			2	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	1	1	3	1	1		7	1	0	0	0	0	1			1		1		0
3	2	2	12	2	2	2	5				1	1			1	1	2	1	1		1	0	1	0	1	0				0	1			0
3	2	2	0	2	2	2	5			1	1				1	1	2	1		1	2	1	0	0	1	0				0			1	0
3	2	2	12	2	2	2	5				1	1			1	1	2	1	1		1	0	1	0	1	0				0	1			0
3	2	2	0	2	2	2	5			1	1				1	1	2	1		1	2	1	0	0	1	0				0			1	0
3	2	2	0	2	2	2	5				2	1			1	1	3	1	1		7	1	1	0	2	0	1	1		12		1		0
3	2	2	0	2	2	2	5	1		1					2	1	1	1	1		2	1	0	0	0	0				0			1	0
3	2	2	5	2	2	2	5							1	1	1	3	1	1		2	1	0	0	0	0				0			1	0
1	2	2	0	2	2	2	5			1	1	1			1	1	2	1	2		2	1	0	0	1	0				0			1	0
1	2	2	0	2	2	2	5			1	1				2	1	2	1	1		2	1	0	0	1	0				0			1	0
3	2	2	5	2	2	2	5							1	1	1	1	1	1		2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	1	1	1	1	1		2	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5	1			1	1			1	1	2	1	1		2	1	0	0	1	0				0			1	0
3	2	2	0	2	2	2	5		1						2	1	2	1	1		1	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5		1						1	1	1	1	1		2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5	1	1						1	1	2	1		1	1	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	1	2	1	1	1		7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5							1	2	1	1	1			3	1	0	0	0	0				0			1	0
3	2	2	5	2	2	2	5							1	2	1	2	1			2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5			1	1				2	1	2	1			2	1	0	0	1	0				0			1	0
3	2	2	0	2	2	2	5							1	2	1	2	1			2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	1	1	1	1	1		7	0	1	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5		1						2	1	1	1	1		2	0	1	0	0	0				0			1	0
3	2	2	5	2	2	2	2	1							2	1	2	1	1		2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	1	1	1	1		1	2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	1	1	1	1		2	1	0	0	0	0				0			1	0
2	2	2	5	2	2	2	5				2				2	1	1	1	1		2	1	0	0	2	0				0			1	0

3	2	2	0	2	2	2	5							1	2	1	1	1			2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5		1						2	1	1	1		1	2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	1	2	1			2	1	0	0	0	0				0			1	0
3	2	2	5	2	2	2	5							1	1	1	1	1			2	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	1	2	1			2	1	0	0	0	0				0			1	0
3	2	2	5	2	2	2	5		1						2	1	3	1			2	1	0	0	0	0				0	1			0
3	2	2	0	2	2	2	5		1			1			1	1	3	1			7	0	1	0	0	0	1			1		1		0
3	2	2	12	2	2	2	5							1	2	1	3	1			2	0	1	0	0	0				0	1			0
3	2	2	0	2	2	2	5							1	1	1	3	1		1	2	1	0	0	0	0	1			1			1	0
3	2	2	5	2	2	2	5			1	2	1			2	1	3	1			2	1	0	0	0	0	1	1	1	123	1			0
3	2	2	0	2	2	2	5							1	1	1	2	1			7	1	0	0	0	0	1			1		1		0
3	2	2	5	2	2	2	5		1						2	1	2	1			2	1	0	0	0	0	1	1		12			1	0
3	2	2	12	2	2	2	5			1					1	1	2	1			2	0	1	0	0	0	1			1	1			0
3	2	2	0	2	2	2	5							1	1	1	2	2			7	1	0	0	0	0	1			1		1		0
3	1	2	0	2	2	2	5					1	5		1	1	2	1			2	1	0	0	0	0	1	1		12			1	0
3	2	2	0	2	2	2	5							1	1	1	3	1			3	1	0	0	0	0				0			1	0
3	2	2	12	2	2	2	5						6		2	1	2	1			2	1	0	0	0	0	1			1	1			0
3	2	2	12	2	2	2	5	1							2	1	2	1			2	1	0	0	0	0				0	1			0
3	2	2	0	2	2	2	5							1	2	1	3	1			7	1	0	0	0	0	1	1		12		1		0
3	2	2	0	2	2	2	5							1	2	1	2	1			1	0	1	0	0	0				0			1	0
3	2	2	5	2	2	2	5							1	1	1	2	1			2	1	0	0	0	0				0			1	0
3	2	2	12	2	2	2	5			1	2	1			2	1	3	1			2	0	1	0	0	0	1			1	1			0
2	2	2	5	2	2	2	5	1			2				2	1	2	1			2	1	0	0	0	0	1	1	1	123	1			0
3	2	2	0	2	2	2	5		1						1	1	2	1			7	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5		1						1	1	3	1	4		1	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5		1						1	1	2	1			7	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5		1						1	1	3	1	4		1	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	1	1	3	1			7	0	1	0	0	0				0		1		0
3	2	2	5	2	2	2	5		1						1	1	3	1			7	1	0	0	0	0	1			1			1	0
3	2	1	12	2	2	2	5	1		1		1			1	1	3	1	1		2	1	0	0	0	2	1	1		12	1			1
3	2	2	0	2	2	2	5			1					1	1	3	1			7	0	1	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5							1	1	1	2	1			3	1	0	0	0	0				0			1	0
2	2	2	0	2	2	2	5							1	2	2	2	1			7	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5			1					1	1	3	1			7	0	1	0	0	0				0		1		0
3	2	2	0	2	2	2	5							1	1	1	3	1			1	1	0	0	0	0	1			1		1		0
2	2	2	0	2	2	2	5	1		1					2	1	2	1			7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5	1	1	1	2	1			1	1	2	1			7	1	0	0	0	0	1			1			1	0
3	2	2	0	2	2	2	5							1	1	1	3	1			7	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5	1		1		1			1	1	2	1			7	0	1	0	0	0	1			1		1		0
2	1	2	0	2	2	2	5				2	1			2	1	2	1			7	1	0	0	0	0				0		1		0
3	2	2	5	2	2	2	5			1					1	1	2	1			2	1	0	0	0	0				0	1			0
3	2	2	12	2	2	2	5			1	2				2	1	2	1		1	2	0	1	1	0	0	1			1	1			0
3	2	2	0	2	2	2	5		1						2	1	3	1			7	0	1	0	0	0	1			1		1		0
3	2	2	12	2	2	2	5							1	2	1	3	1			2	1	0	0	0	0				0	1			0
3	2	2	0	2	2	2	5							1	2	1	2	1	2	1	7	0	1	0	0	0				0		1		0
3	2	2	0	2	2	2	5							1	1	2	3	1			7	0	1	0	0	0				0		1		0
3	2	2	0	2	2	2	5			1					2	1	3	1			7	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	1	3	1			7	0	1	0	0	0	1			1		1		0
3	2	2	5	2	2	2	5							1	2	1	3	1			2	1	0	0	0	0				0	1			0

3	2	2	0	2	2	2	5		1						2	1	2	1			7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5		1						2	1	3	1			7	1	0	0	0	0	1			1			1	0
3	2	2	0	2	2	2	5			1					2	1	2	1	1	1	7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5		1						1	1	3	1			7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5	1			2				1	1	2	1			7	1	0	0	0	0	1			1			1	0
3	2	2	12	2	2	2	5	1				1			2	1	3	1		1	4	0	1	0	0	0	1			1	1			0
2	1	2	0	2	2	2	5				2	1			1	1	3	1			7	1	0	0	0	0	1			1		1		0
2	2	2	0	2	2	2	5			1	2				2	1	2	1			7	1	0	0	0	0	1	1		12			1	0
3	2	2	0	2	2	2	5	1	1						1	1	2	1			7	1	0	0	0	0	1			1			1	0
3	2	2	0	2	2	2	5	1			2	1			1	1	3	1			7	0	1	0	0	0	1			1		1		0
3	1	2	0	2	2	2	5		1	1	2				2	1	3	1			7	0	1	0	0	0	1	1		12			1	0
3	2	2	0	2	2	2	5			1	2	1			2	2	1	1			7	0	1	0	0	0		1		2			1	0
3	2	2	0	2	2	2	5	1	1						2	2	1	1		1	7	0	1	0	0	0				0			1	0
3	2	2	0	2	1	2	5	1							2	2	1	1			7	0	1	1	0	0				0			1	0
3	2	2	12	2	2	2	5	1							2	2	1	1			2	0	1	0	0	0				0	1			0
3	2	2	0	2	2	2	5	1	1						2	2	1	1			7	0	1	0	0	0		1		2			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5							1	2	2	1	1	1	1	1	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5			1					2	2	1	1	1		7	1	0	0	0	0				0			1	0
2	2	2	0	2	1	2	5				2		7		2	2	1	1	1		7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5							1	2	2	1	1	1	1	3	0	1	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5							1	2	2	1	1	1		7	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5		1						2	2	1	1	5		7	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5	1	1						2	2	1	1			7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	1	5							1	2	2	1	2	1		7	0	1	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5	1	1						2	2	1	1			7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	1	5							1	2	2	1	2	1		7	0	1	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5				1	1			2	2	1	1	1		7	1	0	0	1	0				0			1	0
3	2	2	0	2	2	2	5			1		1			2	2	1	1			7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5								2	2	1	1	4	1	7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5						9		2	2	1	1			1	0	1	0	0	0	1	1	1	123		1		0
3	1	2	0	2	2	2	5			1		1			2	2	1	1			7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5	1	1						2	2	1	1			7	0	1	0	0	0	1			1		1		0
2	1	2	0	2	2	2	5	1				1			2	2	1	1	5		7	1	0	0	0	0	1			1			1	0
3	2	2	0	2	2	1	5	1	1						2	2	1	1	2		5	1	0	0	3	0				0		1		0
3	1	2	0	2	2	2	5					1			2	2	1	1	2		7	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5		1						2	2	1	1	2		7	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5		1						2	2	1	1	2		1	1	1	0	0	0	1			1			1	0
3	1	2	0	2	2	2	5			1		1			2	2	1	1	2		1	0	1	0	0	0	1			1		1		0
1	1	2	0	2	2	2	5			1		1			2	2	1	1	2		7	1	0	0	0	0				0			1	0
1	2	2	0	2	2	2	5			1	1				2	2	1	1	2		1	1	0	0	0	0	1			1		1		0
2	1	2	0	2	2	2	5			1	2	1			2	2	1	1	2		1	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5			1					2	2	1	1	2		7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5			1					2	2	1	1	4	1	7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	1	0	0	0	0				0		1		0
3	1	1	0	2	2	2	5			1					2	2	1	1	1		1	0	1	0	0	3	1			1		1		0
3	1	2	0	2	2	2	5	1				1			2	2	1	1	1		7	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5						2		2	2	1	1			7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	0	1	0	0	0				0			1	0

3	2	2	0	2	2	2	5							1	2	2	1	1	4		6	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	0	1	0	0	3				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	2	3	1	7	0	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5	1							2	2	1	1			7	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	1	0	0	0	0				0		1		0
3	2	2	0	2	2	2	5			1					2	2	1	1			7	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5						1		2	2	1	1			7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			1	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1			1	0	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5	1	1						2	2	1	1			7	1	1	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5							1	2	2	1	1			7	1	0	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1	2		1	1	0	0	0	0				0			1	0
3	1	2	0	2	2	2	5	1		1	2				2	2	1	1	2		7	1	0	0	0	0	1			1		1		0
3	2	2	0	2	2	2	5							1	2	2	1	1	2		7	1	1	0	0	0				0			1	0
3	2	2	0	2	2	2	5							1	2	2	1	1	2		1	0	1	0	0	0				0			1	0
3	2	2	1	2	2	2	5							1	1	1	2	1			7	0	1	0	0	0				0			1	0
3	2	2	1	2	2	2	5						4		1	1	2	1			7	1	0	0	0	0				0			1	0
3	2	2	1	2	2	2	5							1	1	2	1	1			7	0	1	0	0	0				0			1	0
3	2	2	1	2	2	2	5							1	2	1	1	1	4		7	1	0	0	0	0				0			1	0
3	1	2	1	2	2	2	5					1			2	1	3	1			1	1	0	0	0	0	1			1		1		0
3	2	2	1	2	2	2	5							1	1	1	2	1			7	1	1	0	0	0				0			1	0
3	2	2	1	2	2	2	5							1	2	1	1	1			7	0	1	0	0	0				0			1	0
3	2	2	1	2	2	2	5							1	1	1	1	1			7	1	1	1	0	0				0			1	0
3	2	2	1	2	2	2	5							1	1	1	1	1			7	1	0	0	0	0				0			1	0
3	2	2	1	2	2	2	5			1					1	1	2	1			2	1	0	0	0	0				0	1			0
3	2	2	1	2	2	2	5							1	2	1	3	1			7	0	1	0	0	0				0			1	0
3	2	2	1	2	2	2	5	1		1	2				2	1	2	1			7	0	1	0	0	0	1			1		1		0
2	2	2	1	2	2	2	5	1		1	2				2	1	2	1	1		2	0	1	0	0	0	1			1	1			0
3	2	2	1	2	2	2	5					1			2	1	3	1			2	1	0	0	0	0				0	1			0
2	2	2	1	2	2	2	5							1	2	2	1	1			7	0	1	0	0	0	1			1		1		0
3	2	2	1	2	2	2	5							1	2	2	1	1		1	1	0	1	0	0	0	1			1	1			0
3	2	2	1	2	2	2	4			1					2	2	1	1	1		7	1	0	0	0	0				0		1		0
3	2	2	1	2	2	2	5		1						2	2	1	1			7	1	0	0	0	0				0			1	0
3	1	2	1	2	2	2	5			1		1			2	2	1	1	2		7	0	1	0	0	0	1			1		1		0
3	2	2	1	2	2	2	5							1	2	2	1	1	3		1	0	1	0	0	0				0			1	0
3	2	2	1	2	2	2	5			1					2	2	1	2	2		4	1	0	0	0	0				0			1	0
3	2	2	1	2	2	2	5							1	2	2	1	1			7	1	0	0	0	0				0			1	0
3	1	2	1	2	2	2	5	1				1			2	2	1	1			7	1	0	0	0	0				0			1	0
3	1	2	1	2	2	2	5					1			2	2	1	2	2		7	1	0	0	0	0				0			1	0
3	2	2	1	2	2	2	5							1	2	2	1	1	2		7	0	1	0	0	0				0			1	0
3	2	2	2	2	2	2	5							1	1	1	3	1			7	0	1	0	0	0				0			1	0
3	2	2	3	2	2	2	5						1		1	1	3	1			7	1	1	0	0	0		1		2		1		0
3	2	2	3	2	2	2	5							1	1	1	1	1			7	0	1	0	0	0				0			1	0

3	2	2	3	2	2	2	5			1					2	1	2	1			7	1	0	0	0	0				0		1		0
3	2	2	3	2	2	2	5							1	1	1	2	1			7	0	1	0	0	0				0			1	0
3	2	2	3	2	2	2	5							1	1	1	1	1			7	0	1	0	0	0				0			1	0
3	2	2	3	2	2	2	5							1	2	1	1	1			7	1	0	0	0	0				0			1	0
3	2	2	3	2	2	2	5			1					1	1	3	1			7	1	0	0	0	0	1			1		1		0
3	2	2	3	2	2	2	5		1						1	1	1	1			7	1	0	0	0	0				0			1	0
3	2	2	3	2	2	2	5							1	1	1	3	1	1		7	1	0	0	0	0		1		2		1		0
3	2	2	3	2	2	2	5			1	1	1			2	2	1	1			7	0	1	0	1	0				0		1		0
3	2	1	3	2	2	2	5							1	2	2	1	1	2		2	1	1	0	0	0	1	1		12	1			1
3	2	2	3	2	2	2	5					1			2	2	1	1			7	1	1	0	0	0				0			1	0
3	2	2	3	2	2	2	5							1	2	2	1	1			7	1	0	0	0	0				0			1	0
3	2	2	3	2	2	2	3							1	2	2	1	1			7	1	0	0	0	0				0			1	0
3	2	2	3	2	2	2	5							1	2	2	1	1	2		1	1	1	0	0	0				0			1	0
3	2	2	3	2	2	2	5			1					2	2	1	1			7	1	0	0	0	0				0			1	0
3	2	1	4	2	2	2	5				2				2	1	2	1	2		2	1	0	0	0	1	1			1	1			0
3	2	2	5	2	2	2	5	1							1	1	3	1			7	1	0	0	0	0				0		1		0
3	2	2	6	2	2	2	5		1	1		1			2	1	3	1	1	1	1	1	1	0	0	0	1			1		1		0
3	2	2	7	2	2	2	5		1						1	1	3	1	2	1	7	0	1	0	0	0	1			1			1	0
3	2	2	7	2	2	2	5				2				2	1	3	1			7	1	0	0	0	0	1			1		1		0
3	2	2	7	2	2	2	5	1	1						1	1	2	1			7	0	1	0	0	0	1			1			1	0
3	2	2	7	2	2	2	5		1						2	2	1	1			7	0	1	0	0	0				0			1	0
3	2	2	8	2	2	2	5							1	2	2	1	1		1	7	0	1	0	0	0		1		2		1		0
2	2	2	9	2	2	2	5	1			2	1	8		2	2	1	1			2	1	0	0	0	0	1			1	1			0
3	2	2	10	2	2	2	5				2				2	2	1	1			7	1	0	0	2	0				0			1	0
3	2	2	11	2	2	2	5	1				1			2	2	1	1			7	1	0	0	0	0	1			1		1		0
3	2	2	12	2	2	2	5	1	1		2				2	1	3	1			1	0	1	0	0	0	1			1	1			0