

"CLINICAL PROFILE OF DENGUE
FEVER IN ADULTS - A ONE YEAR
HOSPITAL BASED CROSS-SECTIONAL
STUDY"

By

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KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

M. D. MEDICINE

Under the Guidance of

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I hereby declare that this dissertation entitled “**CLINICAL PROFILE OF DENGUE FEVER IN ADULTS - A ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY**” is a bonafide and genuine research work carried out by me under the guidance of **Dr. V. A. KOTHIWALE MD Ph.D** Professor and Head, Department of Medicine, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum – 10.

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LIST OF ABBREVIATIONS USED

-	-	Negative
+	-	Positive
+	-	Present
Alb	-	Albumin
aPTT	-	Activated partial thromboplastin time
C3a, C5a	-	Complement factor
CF	-	Complement fixation
CXR	-	Chest X-ray
D	-	Deranged
D/A	-	Date of admission
Den	-	Dengue
DF	-	Dengue fever
DHF	-	Dengue hemorrhagic fever
DIC	-	Dissiminated intravascular coagulation
DIS	-	Discharge
DOD	-	Date of discharge
DSAS	-	Dengue with signs associated with shock
DSS	-	Dengue shock syndrome
ELISA	-	Enzyme linked immuno sorbent assay
Exp	-	Expired
F	-	Female
FFP	-	Fresh frozen plasma
Glb	-	Globulin
Hct	-	Hematocrit
HI	-	Hemagglutination inhibition
IL's	-	Interleukins

M	-	Male
MacELISA	-	IgM antibody capture ELISA
N	-	Normal
NM	-	Not mentioned
NS	-	Normal saline
PAF	-	Platelet activating factor
PCR	-	Polymerase chain reaction
PT	-	Prothrombin time
R	-	Raised
RL	-	Ringer's lactate
RNA	-	Ribose nucleic acid
SGOT	-	Serum glutamic oxaloacetic transaminase
SGPT	-	Serum glutamic puruvic transaminase
Sr proteins	-	Serum proteins
Sr. Bilirubin	-	Serum bilirubin
TNF	-	Tumor necrosis factor
USG	-	Ultrasonography
VHF	-	Viral hemorrhagic fever
WHO	-	World Health Organization

ABSTRACT

Background and Objectives

Dengue fever has emerged as one of the most important arthropod tropical infections in the recent years with an estimated 2.5 billion people at risk all over the world. The objective of present study was to assess clinical profile, complications and outcomes of dengue fever in adult patients.

Methods

The present cross sectional study was conducted in Department of Medicine, at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during January 2008 to December 2008 on 200 Adult patients with dengue fever proved by micro ELISA test. Detailed history, clinical examination, blood investigations were performed. All baseline investigations, liver function test, prothrombin time and activated partial thromboplastin time and tourniquet test were done at admission. Hematocrit was monitored every 12 hourly in DHF Grade III and Grade IV and 12 hourly in DF, Grade I and Grade II DHF till vital signs were stable.

Results

In this study, majority of the patients (70%) were in adult age group from 15 to 35 years, 22% were in 35 to 55 years age group and eight percent had more than 55 years of age. Out of 200 cases, tourniquet test was positive in 96 (48%) cases of DF. It was 100% positive in Grade I DHF, 40 (20%) patients in Grade II DHF, 32 (16%) patients in Grade III DHF. Test was negative with Grade IV DHF. Raised APTT was observed in 83 (41.5%) cases. In this study, 112 patients

had only IgM positive and 88 patients had both IgM and IgG were positive. Out of which 52 (26%) had bleeding manifestations and this correlation was statistically significant ($p=0.000$).

Conclusion

Dengue haemorrhagic fever is more common in younger age group with increased bleeding manifestation who had deranged aPTT, liver function test, low plate count ($<20,000$ /cmm) and IgM and IgG positive patients.

Key words

Classical Dengue Fever; Dengue Haemorrhagic fever; Dengue Shock Syndrome.

CONTENTS

SL. NO.	TOPIC	PAGE NO.
1.	INTRODUCTION	1
2.	OBJECTIVES	4
3.	REVIEW OF LITERATURE	5
4	METHODOLOGY	29
5.	RESULTS	36
6.	DISCUSSION	52
7.	CONCLUSION	62
8.	SUMMARY	64
9.	BIBLIOGRAPHY	67
10.	ANNEXURE I – CONSENT FORM	72
11.	ANNEXURE II – PROFORMA	74
12.	ANNEXURE III – MASTER CHART	78

LIST OF TABLES

TABLE. NO.	DESCRIPTION	PAGE NO.
1	Gender distribution	36
2	Age Distribution	37
3	Distribution of patients according to symptoms	38
4	Distribution of cases according to signs	40
5	Grading of dengue virus infections	41
6	Distribution of cases according to hematological laboratory findings	42
7	Distribution of patient according to liver function test and coagulation profile	44
8	Correlation between thrombocytopenia and bleeding	45
9	Correlation between coagulation profile and bleeding	47
10	Correlation of IGM and IGG with bleeding manifestations	48
11	Temperature trend during the hospital stay	49
12	: Platelet trend during the hospital stay	50
13	Distribution of dengue patients according to treatment	51
14	Mortality rate	51

LIST OF GRAPHS

GRAPH NO.	DESCRIPTION	PAGE NO.
1	Gender distribution	36
2	Age Distribution	37
3	Distribution of patients according to symptoms	39
4	Distribution of cases according to signs	40
5	Grading of dengue virus infections	41
6	Distribution of patient according to liver function test and coagulation profile	44
7	Correlation between thrombocytopenia and bleeding	45
8	Correlation between coagulation profile and bleeding	47
9	Temperature trend during the hospital stay	49
10	: Platelet trend during the hospital stay	50

LIST OF FIGURES

PHOTO NO.	DESCRIPTION	PAGE NO.
1	Spectrum of clinical features of dengue virus infection	16
2	Flow chart of the treatment for DHF Grade – I and Grade – II	23
3	Flow chart of the treatment for DSS	24

INTRODUCTION

Dengue fever and dengue haemorrhagic fevers are self limiting mosquito born, viral diseases caused by the bite of female anophele Aedes aegypti mosquito. Dengue fever has been known for more than a century in the tropical countries. Dengue fever has emerged as one of the most important arthropod tropical infections in the recent years with an estimated 2.5 billion people at risk all over the world.

Infection with dengue virus is witnessing a global resurgence over last 15 to 20 years. Of the estimated 50 to 100 million cases occurring annually, about 5,00,000 cases require hospitalization. Among children of South East Asian Region Countries, these infections including dengue fever (DF), dengue haemorrhagic fever (DHF) and dengue shock syndrome (DSS) are the leading causes of hospitalization and death was preceded in prevalence by diarrhoeal disease and acute respiratory infections only. The resurgence of infections is attributed to decay in public health infrastructure, lack of mosquito control, unplanned urbanization and global population explosion. Increase in air travel and excellent mode of transport of pathogens also contributes spread. A total of 2500 million people worldwide are at risk of dengue virus infection. Dengue affects more than 100 countries in all continents except Europe.

Dengue as a disease has been poorly studied and important lacunae remain in the understanding of the presentations, the complications and the treatment of the disease.

The disease is caused by four serotypes of the dengue virus, any of which may be responsible for an epidemic. The clinical manifestation of dengue infection varies from asymptomatic to severe life threatening illness in the form of DHF/DSS. Dengue haemorrhagic fever or DSS may be fatal in 40% to 50% of untreated patients, however, with appropriate treatment the mortality can be brought down to one to five percent. Epidemic transmission requires a favourable temperature ($< 20^{\circ}$ C) and stagnant water for the breeding of *Aedes aegypti*. Outbreaks in urban areas infected with the *Aedes* mosquito may be explosive with attack rates reaching upto 70% of the population. A higher temperature within range of mosquito viability leads to more infectious mosquitoes, which bite more frequently. The DHF / DSS remained a disease of children and young adults for two decades after its identification in the 1950s. The epidemics from Malaysia and Delhi, which occurred recently, show more affection of adults than children. The reasons for this change in epidemiology of DHF are not clear.¹

Dengue fever, DHF are caused by dengue virus, RNA virus of flavi virus group. There are four serotypes of the dengue virus that is 1, 2, 3, 4. The disease is transmitted by the bite of *Aedes* mosquito infected with virus. The illness encompasses febrile hemorrhagic disease with capillary fragility, leading to acute severe shock.

Laboratory diagnosis of dengue virus infection depends upon demonstration of specific antibodies in serum samples by haemagglutination inhibition, complement fixation, neutralization test or ELISA. Virus isolation methods are expensive, time consuming and not widely available. Reverse transcriptase PCR and hybridization probes for nucleic acid are other newer tests

for diagnosis. As no specific antiviral therapy is available supportive therapy is of utmost importance. Cases with DF are treated with antipyretics (paracetamol is the preferred antipyretic, aspirin and ibuprofen are avoided as they may precipitate bleeding), rest, good diet and fluid intake.

Dengue is viral disease which often presents with a confusing clinical profile. There are very less established guidelines for management of dengue fever. The dilemmas in the treatment of dengue have resulted in increased morbid periods and higher mortality. Management of all patients DHF / DSS includes administration of parenteral fluids like crystalloids initially, colloids, blood and blood products (FFP, platelets) as required. With the help of this energetic fluid therapy along with supportive measures most cases with DHF/DSS are successfully managed in hospital settings.

With advent of sophisticated diagnostic test it is now possible to identify the disease early in settings which would have been confusing in the past. Early recognition of shock, careful monitoring and appropriate fluid therapy has resulted in considerable reduction of mortality to one to five percent.²

Dengue is now widely prevalent in the areas in and around Belgaum and no studies have been documented in adults in the present settings. Hence the present study was undertaken to assess clinical profile, manifestations, complications and outcomes of dengue fever in adult patients in and around Belgaum.

OBJECTIVES

The objective of the study was to assess clinical profile, complications and outcomes of dengue fever in adult patients.

REVIEW OF LITERATURE

Historical Aspects

The term “Dengue” was introduced into the English medical literature from the West Indies during the 1827 – 28 Caribbean epidemic of an exanthema with arthralgia. Dengue is a Spanish homonym for the Swahili “Ki denga Pepo” (a sudden cramp like seizure caused by an evil spirit).

The term “Break bone fever” for the modern dengue was known in Philadelphia since 1780.

Shock cases and deaths accompanied a dengue epidemic in Queensland, Australia in 1897, while nearly 1250 persons died during the explosive Greek dengue epidemic of 1928. The later epidemic was related to substandard living conditions among refugees repatriated from Turkey following the Greco-Turkish war of 1922.

The dengue viruses for the first time adapted to laboratory animals in the 1940’s (type 2) and 1950’s (type 3 and 4).

In 1954, Filipino paediatricians and shortly thereafter, Physicians in South East Asian countries, described the DHF/DSS syndrome and it was associated with dengue virus infection by Hammon et al in 1956.³

The first case of dengue hemorrhagic fever in Southeast Asia were noted in Manila in 1953 to 1954 and outbreaks have since been reported throughout the Indo-China Peninsula and the Indian sub-continent.⁴

Epidemic and pandemic of dengue virus infections⁵

The tip of the 18th century pandemic can be identified from the classic description of dengue fever in Philadelphia in 1780 by Benjamin rush. The causal virus and mosquito were introduced into Philadelphia by ship, an unwelcome consequence of the sugar, rum and slave trade between African, colonial American and Caribbean ports. This first pandemic produced reports of sporadic dengue outbreaks in the United States of America, Caribbean and South American coastal cities during the 19th century and first three decades of 20th century.

Second pandemic occurred in semitropical Northern Queensland where due to gold and sugar generated, rapid population growth occurred.

The first outbreak of a disease resembling DHF/DSS was reported from Charters Lowers and nearby towns in 1897. Dengue, like epidemics were also reported from the Eastern Mediterranean in the late 19th century, culminating in the explosive and severe Greek epidemic of 1928. Effective mosquito control in Greece and in many cities of tropical Asia and anti Aedes campaigns of America, produced a global interregnum in dengue transmission in the mid 20th century.

The great 20th century pandemic grew after World War – II in which dengue strains were carried by combatants from South East Asia to Japan and Pacific Islands. Destruction of city water supplies, temporary housing for war refugees, the explosive post – war growth of populations through high fertility, rural to urban migration and the steady deterioration of urban environments, have led to sustained growth in density and the area occupied by Aedes aegypti.

Together these factors have resulted in the endemic transmission of all four dengue serotypes in most of the Asian tropics.

Mean while, the remarkable gains achieved towards the eradication of *Aedes aegypti* in the American tropics have been eroded and reversed. This was followed by the introduction and spread of dengue viruses beginning in the 1960s. Where once they were absent, dengue viruses have invaded Cuba, Caribbean Islands, Mexico, the United States, Central America, Colombia, Ecuador, Peru, Paraguay, Bolivia, Argentina and Brazil.

By the 1990s dengue had spread north to China. Taiwan, south to Queensland (Australia) and eastward to nearly all of the Pacific Islands. In Africa and the Middle East, areas of epidemic activity include outbreaks in Kenya, Mozambique, Somalia and Yemen. Major recent outbreaks occurred in Cuba (1981), Southern China, Sri Lanka, India, Maldives, Tahiti and Venezuela in mid to late 1980s.

Epidemics of dengue fever in India⁶

Dengue fever is endemic in many parts of India except the Himalayan and other mountainous regions where conditions are not conducive to the propagation of its vector.

Outbreaks of dengue fever occur mostly in India, during or after the rainy season, but outbreaks during summer season have also been reported due to storage of water for domestic purposes causing a rise in vector population,

Some of the epidemics of DHF/DSS which occurred in India are as follows :

Place	Year	Dengue virus serotype incriminated
Calcutta	1963	Den -2
Vishakapatnam	1964	Den – 2
Kanpur	1968	Den – 4
Vellore	1968	Den – 3, 4
Ajmer	1969	Den – 1, 3
Kanpur	1969	Den – 2
Delhi	1970	Den – 1,3
Jalore	1985	Den -2
Delhi	1988	Den -2
Vellore	1990	Not established

**ETIOLOGY / PATHOGENESIS / PATHOLOGY / CLINICAL FEATRUES
/ LABORATORY DIABNOSIS AND MANAGEMENT**

Etiology

Arboviruses (Arthropod borne viruses) are viruses of vertebrates, biologically transmitted by hematophagous insect vector. They multiply in blood sucking insects and are transmitted by bite to vertebrate hosts. Taxonomically, Arboviruses belong to families as diverse as Togaviridae, Bunyaviridae, Reoviridae, Arenaviridae and Rhabdoviridae.

Togaviruses are spherical viruses, 40-70 nm in diameter with lipoprotein envelope and single stranded RNA genome. The Togavirus family contains three genera of medically important viruses.⁷

Alphavirus : Arbovirus Group A

Flavivirus : Arbovirus Group B. The name being derived from flavi meaning yellow

Rubivirus : Rubella virus. It is antigenically and epidemiologically unrelated to arboviruses

Dengue virus, a species of flavivirus genus belonging to Togaviridae family, is a single stranded RNA virus. There are four serotypes of dengue virus, which are 1, 2, 3 and 4, and all serotypes can cause DF and DHF. All four types of dengue viruses have been isolated in this country and occasionally more than one type of dengue virus have been isolated from the same patient. The virus can survive at 4°C for several weeks and at 70°C for years.

Transmission and vector

Dengue viruses are transmitted by the mosquitoes of “*Stegomyia* family”. *Aedes aegypti* is the principal vector other vectors, which are also responsible for outbreaks of dengue infections are *Aedes albopictus*, *Aedes polynesiensis* and *Aedes scutellaris* complex.⁸

These are peridomestic mosquitoes and are most abundant during the rainy season. Females are fearless biters and bite during daytime. Two peak biting activity periods being two to three hours after dawn and in the afternoon few hours before dark. They do not fly over long distances and epidemic transmission of dengue requires a favourable temperature (>20°C).

The reservoir of infection is both man and mosquito. The transmission cycle is “man-mosquito-man”, although in jungle setting, probably the monkeys are also responsible for maintaining this infection cycle. The *Aedes* mosquito becomes infective by feeding on a patient during viremia i.e. from a day before onset to the fifth day of illness. The virus multiplies in its salivary glands. After an incubation period of 8 to 10 days the mosquito becomes infective and is able to transmit the disease. Once the mosquito become infective, it remains so for life.

Pathology and Pathogenesis

Certain terms, which are come across during the pathogenesis of dengue fever are

Homotypic infection

Refers to the infection caused by dengue virus strains of a single serotype.

Heterotypic infection

Refers to the infection caused by different virus serotype.

Primary infection

Is infection caused by any serotype in non-immune individual.

Secondary infection

Is heterotypic infection in a monotypic immune individual.

Tertiary infection

Is heterotypic infection in a multitypic immune individual (two infection).

The most significant pathophysiologic changes among DV infections are seen in DHF/DSS, due to plasma leakage from intravascular to extravascular compartments. The leakage of plasma leads to hemoconcentration, hypotension, hypoproteinemia and collection of fluid in serious cavities. The plasma leakage occurs as a result of acute increase in vascular permeability which is attributed to transient functional disturbance due to action of short acting chemical mediators as no significant inflammatory or destructive vascular lesions are seen on histological examination.

Most accepted hypothesis explaining the pathogenesis of DHF/DSS is immune enhancement hypothesis. According to this hypothesis presence of non-

neutralizing heterologous antibody is necessary for occurrence of serious manifestations due to vessel wall dysfunction. This heterologous antibody acquired either transplacentally from mothers or as a result of first infection binds to DV and facilitate the entry of virus into the cells of monocyte macrophage lineage. Within these cells, rapid viral replication occurs through a processes called antibody dependent enhancement. These cells produce various vasoactive mediators e.g. tumour necrosis factor, interleukins (IL-1, IL-2, IL-6 etc.), platelet activating factor, complement activation products (C3a, C5a) and histamine. Simultaneously CD4 + T-Lymphocytes are also induced to produce gamma interferon, lymphotoxins and various interleukins. These cytokines have a complex interplay and act synergistically on vessel wall to produce increased vascular permeability.⁹

Though immunopathogenesis is important in the severity of DHF/DSS, certain viral factors may also be important determinant of severity, genetic changes might be occurring in the virus leading to variation in virulence and epidemic potential. Certain host factors like age, state of nutrition, sequence of infection for example serotype 1 followed by serotype 2 is more dangerous than serotype 4 followed by serotype 2 are also important in the severity of disease.¹⁰

These are four serologically related dengue viruses that parenterally enter human hosts. After a short period of cross protection, individuals infected with one serotype are fully susceptible to infection with other types, in contrast there is life long immunity to reinfection by the homologous serotype. Primary and heterologous infections can be distinguished by their characteristic serological responses. In primary dengue infections antibody responses are largely of IgM

class and predominantly directed against type specific determinant. In secondary infections antibodies are largely of IgG class and directed against the antigens of flavivirus group on the dengue virus complex or sub complex.¹¹

Three major hemostatic factors appear to be involved in the bleeding diathesis in DHF/DSS, which are;¹²

Vascular injury

Vasculopathy is manifested by petechiae, positive tourniquet test and leakage of fluid and protein into extravascular spaces. This cause an acute increase in vascular permeability leading to loss of plasma from the vascular compartment, clinically producing pleural effusion, ascites, hemoconcentration, hypoproteinemia and shock. It is said that chemical mediators, histamine and not endothelial infection generate vascular permeability.

Coagulopathy

Weiss and Halstead et al observed a moderate prolongation of the prothrombin time due to decrease in factors II, V, VII and X.

In WHO collaborative study, platelet counts and average minimum fibrinogen level fell in correlation with severity of illness, while fibrin degradation products (FDP) rose correspondingly.

Suratte et al, Bokish et al and Srichaikul et al confirmed the mild increase in FDP but since euglobulin clot lysis times were normal, the authors concluded that there is evidence of mild to moderate consumptive coagulopathy, but no

DIC, also it contributes neither to shock nor to bleeding nor was therapy with heparin justified.

Thrombocytopenia

The cause of thrombocytopenia is controversial, but the possibilities include impaired megakaryocyte production earlier in the disease, platelet injury by virus itself, platelet specific antibodies, immune complexes or DIC.

Mitrakul et al in a study of ten patient observed that platelet half survival ranges form 6.5 to 53 hours in comparison of normal 72 hours to 96 hours and restudied after 20 days to two years later and shows normal platelet survival times.

Weiss also demonstrated that discrepancies between platelet counts and bleeding times and clot retraction point to the possibility that platelets are qualitatively abnormal.

Hematological abnormalities¹²

Carpenter and Sutton and Vedder came to a conclusions that –

In DF cases leukopenia begins on day two of infection, reaching low point on fourth to sixth day along with early absolute neutropenia and lymphopenia, gradually returning to normal by ninth to tenth day with lymphocyte count returning to normal before neutrophils. Contrast to it in DHF/DSS cases where early absolute leukopenia was observed in a few cases while moderate leukocytosis between days four to none along with early relative lymphocytosis

was observed in most. In both syndromes there occurred marked degeneration of mature neutrophils and “shift to left” during febrile phases of illness.

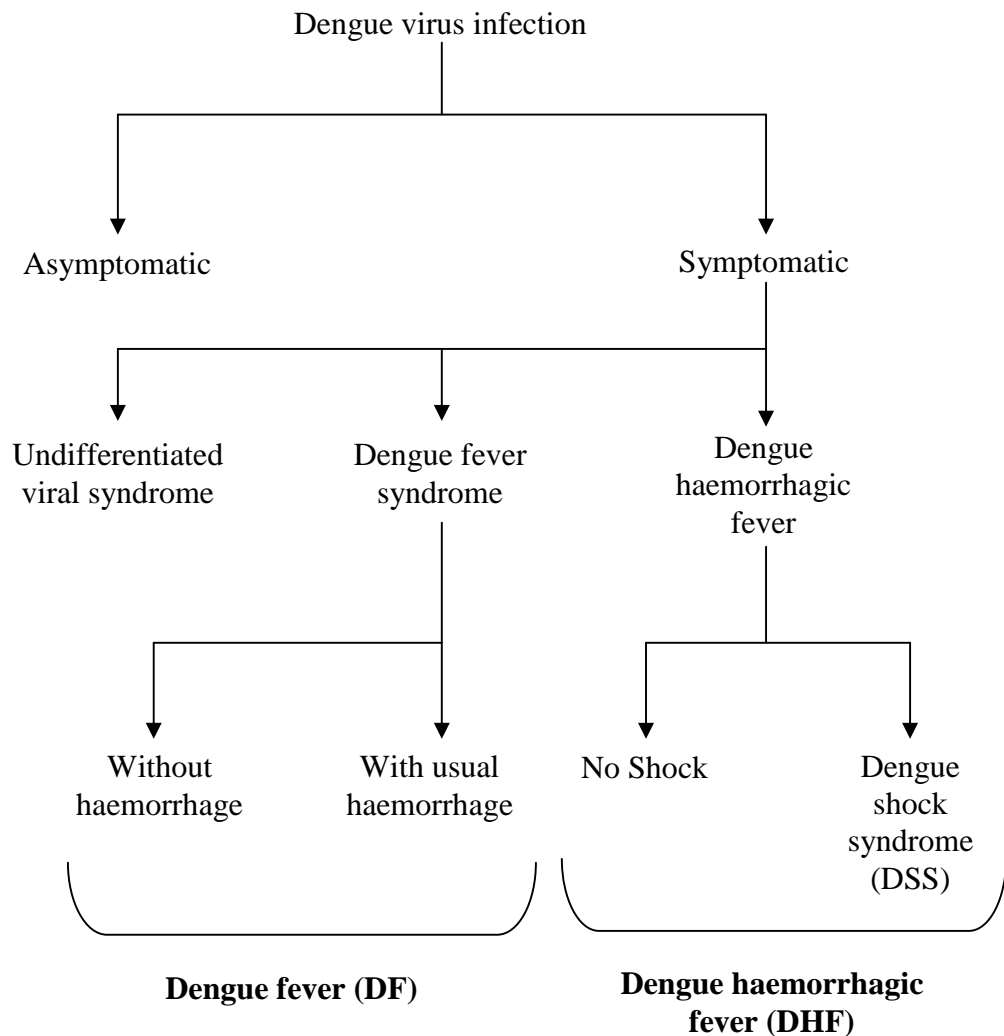
Suratte and Longsamon found atypical or transformed lymphocytes on day fifth of illness, while have large nuclei with fine, homogenous nuclear chromatin and azurophilic cytoplasm.

Bierman and Nelson, who had done bone marrow biopsies on fourth day of fever in dengue fever and found that the bone marrow was hypocellular with diminished megakaryocytes, diminished erythropoiesis and totally absent granulocytogenesis, on day seven and ten the bone marrow cellularity returns to normal. In DHF/DSS, early in febrile course, the bone marrow is hypocellular with maturation arrest of all elements. At the time of shock or defervescence marrow are usually normocellular or hyper cellular with an unusual incidence of phagocytic reticulum cells.

Clinical features

The dengue virus infection may be manifested as asymptomatic to symptomatic disease as classical dengue fever, dengue haemorrhagic fever/dengue shock syndrome.

Figure 1: Spectrum of clinical features of dengue virus infection⁸



Dengue fever

Dengue fever is an acute viral infection caused by at least four serotype (1,2,3 and 4) of dengue virus. All ages and both sexes are susceptible to dengue fever. The illness is characterized by an incubation period of three to ten days. The onset is sudden with chills and high fever, intense headache muscle pain, joint or bony pain (Break bone fever), retro orbital pain and photophobia. Other

common symptoms include weakness, abdominal pain sore throat and general depression.

Fever is usually between 39°C and 40°C, followed by a remission of a few hours to two days (biphasic fever or saddle back fever).

The skin eruptions in 80 percent of case appear during the remission or during second febrile phase, which lasts for one to two days. The rash may be diffuse flushing, mottling or fleeting pinpoint eruptions or the rash may be maculopapular or scarlatiniform.

Some patients with dengue fever have evidence of mucosal or cutaneous bleeding without other evidence of DHF/DSS like hemoconcentration or fluid leak; such patients are classified as dengue fever with unusual bleeding.¹³

Fever lasts for about five to seven days after which recovery is usually complete although convalescence may be protracted.

Dengue hemorrhagic fever/dengue shock syndrome (DHF/DSS)

DHF/DSS is a severe form of dengue fever, caused by infection with more than one dengue virus and may be fatal in 40-50% of untreated patients. The disease is confined exclusively to children less than 15 years of age, but due to change in epidemiological trend the disease may occur in adult population.

After an incubation period of four to six days the patient develops clinical features like of dengue fever. There may be varying degree of tender hepatomegaly or less commonly splenomegaly. All patients have some degree of

haemorrhagic phenomenon like positive tourniquet test, petechial spots, bruising at venepuncture site, bleeding from gums, epistaxis, hematemesis or melena, muscle hematoma, hematuria and rarely intracranial haemorrhage may occur.

Fever may subside after two to seven days. At this stage patient may develop varying degree of peripheral circulatory failure. With progressive peripheral circulatory failure patient may have sweating, restlessness, cold extremities, pulse pressure gets narrow, blood pressure starts falling ultimately leading to unrecordable blood pressure and irreversible shock.

Unusual manifestations of DHF/DSS include hepatitis, encephalitis and glomerulonephritis.¹³

Laboratory diagnosis

Virus isolation

For virus isolation, the blood should be collected in the acute phase of illness i.e. from day one to five of illness.⁷ These samples were inoculated into the cell cultures: LLC – MK2 or vero cells, cells of *Ae. Albopictus*, *Ae. Seudocultellaris* or live mosquitoes, inoculated intrathoracically and examined seven to 14 days after. Then dengue serotypes can be identified by complement fixation, plaque reduction neutralization test, or immunofluorescence with type specific monoclonal antibodies.¹⁴

This method of virus isolation are not routinely used for diagnosis as they are expensive, time consuming and not widely available.

Serological diagnosis

Detection of antibodies against serospecific dengue infection is less time consuming, less costly and easily available.

The various tests available are IgM antibody capture ELISA (MACELISA), haemagglutination inhibition test (HI), complement fixation test (CFT) and neutralization test.¹⁵

WHO recommends collection of blood from suspected cases at first contact during first week of illness and second sample at the time of discharge from the hospital. A convalescent sample should be obtained between 14 – 21 days of illness. This sample should be stored, transported and then processed according to specific recommendation led down by WHO.

Categories of serological response and criteria for interpretation are as follows:¹⁶

1. Proven dengue

- I. Conversion from negative to positive or a four fold or higher rise in HI and / or CF antibody titre to one or more of the dengue virus types, in paired sera and
- II. Conversion or rise in titre of HI and / or CF antibodies to JE and WN viruses was either absent or when present, antibody titres were at least four fold lower than those to dengue viruses.

2. Presumptive dengue

- I. High titres of HI (1:160 or higher) and or CF (1:32 or higher) antibodies to one or more of the dengue virus types, in single sera obtained later than five days post onset of illness. HI and /or CF antibody titres were at least four fold higher than those JE and WN viruses and /or

- II. IgM antibody in high titre to DEN-2 virus, which was higher than that to JE and WN viruses.

3. Recent dengue virus infection but not related to the present illness

Same criteria as in 2(i) and 2(ii) except that single sera obtained early in the illness (day one to five) hence high antibody titres do not relate to the present illness.

4. Proven flavivirus infection

Antibody titres to the JE-WN complex being similar to the antibody titres to the dengue viruses.

Reverse transcriptase PCR and hybridization probes for nucleic acid are other newer tests for diagnosis. Immuno-histochemistry for virus particle demonstration in histological sections is useful for confirmation of fatal cases for epidemiological purposes.

Management

General principles

As there is no specific antiviral treatment, management is essentially supportive and symptomatic. Key to the success is frequent monitoring and strategy changes depending on clinical and laboratory evaluation. As there is plasma leakage in DHF/DSS, intravenous fluid therapy, in the form of crystalloid and colloid therapy.

As the plasma leakage is not constant in rate, the volume and rate of fluid therapy should be adjusted accordingly. However even there is massive plasma loss judicious fluid replacement is necessary to avoid overhydration.

Indications of hospitalization¹⁷

- Restlessness or lethargy
- Cold extremities or circumoral cyanosis
- Bleeding in any form
- Oliguria or reluctance to take fluid orally
- Rapid and weak pulse
- Capillary refill time 2 seconds
- Narrowing of pulse pressure (<20mm of Hg) or hypotension)
- Hematocrit of 40 or rising hematocrit
- Platelet count of less than 1,00,000/mm³
- Acute abdominal pain
- Evidence of plasma leakage eg. Pleural effusion, ascites.

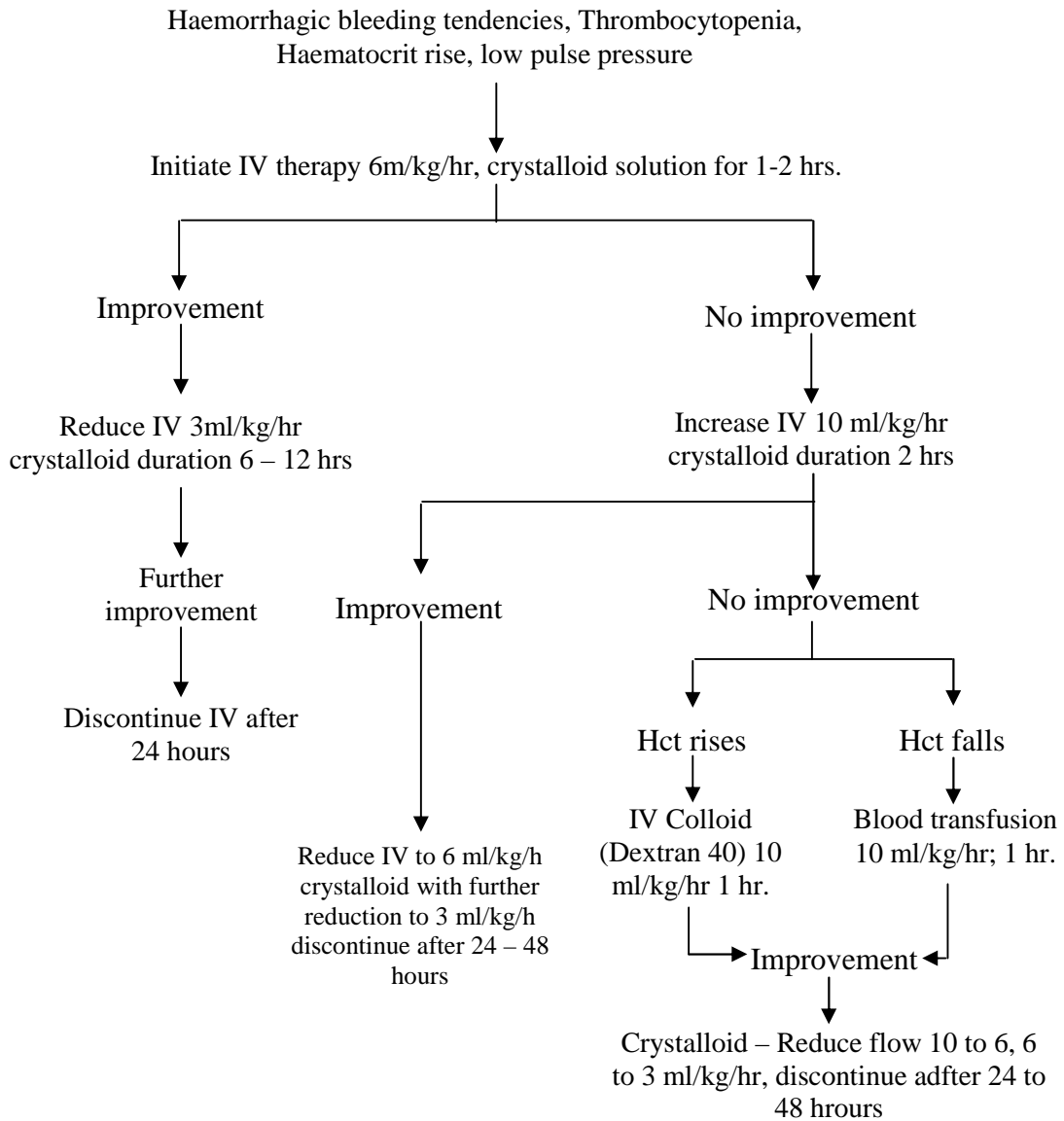
Dengue fever

Patient with dengue fever require rest, oral fluids to compensate for losses via diarrhea or vomiting, analgesics and antipyretics preferably paracetamol. Antibiotics are not indicated in uncomplicated patients.¹⁸

Dengue hemorrhagic fever Grade I and Grade II

In DHF Grade – I and Grade – II, administered intravenous fluid in the form of isotonic fluid like N.S or R. L at 6 – 7 ml/kg/hr for an hour. After one hour if Hct has decreased and vital parameter are improving fluid infusion rate should be decreased step wise to 3ml kg/hr and maintained for 24-48 hours. If Hematocrit is rising and vitals are deteriorating, stepwise increase in intravenous fluid should be made.

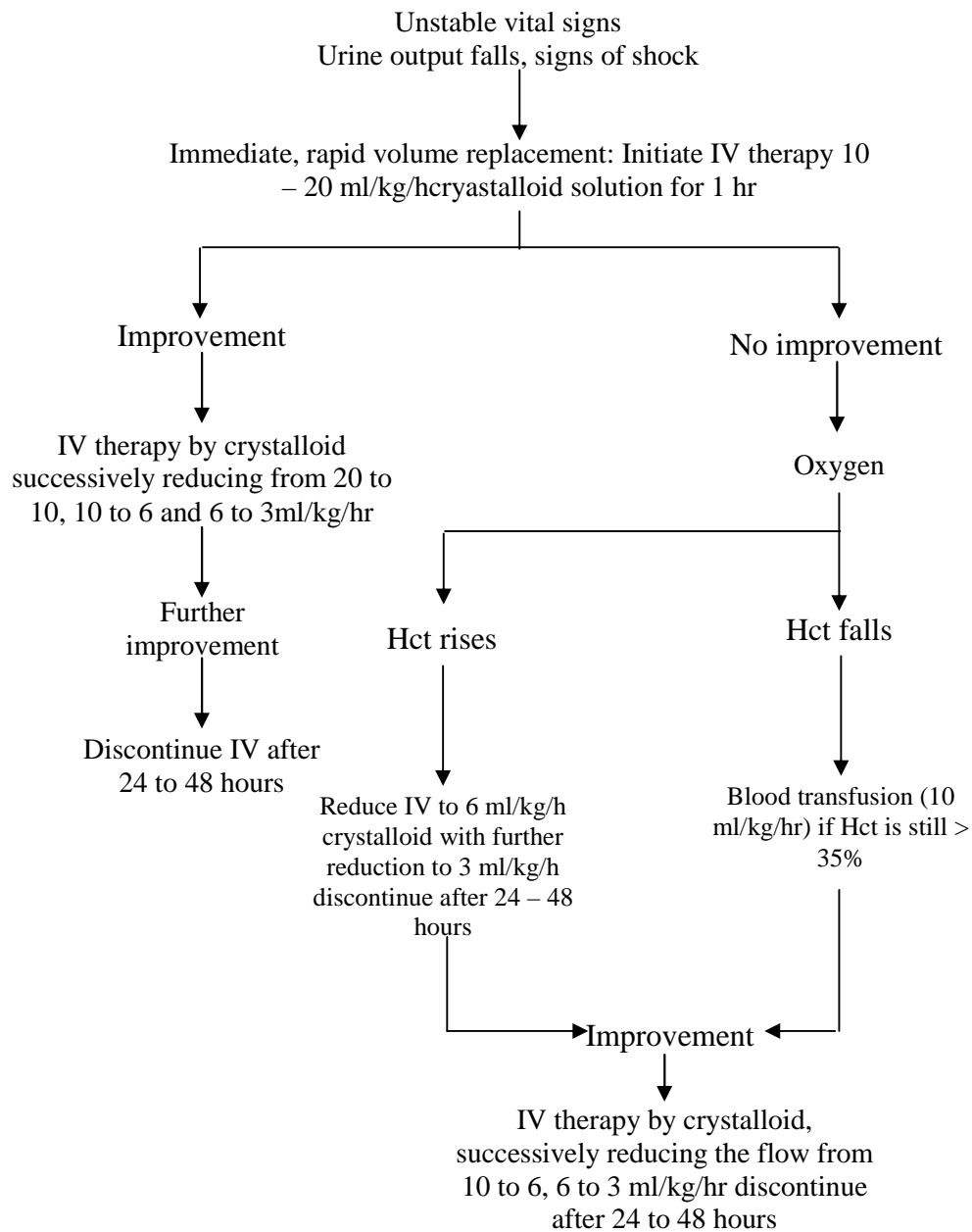
Figure 2: Flow chart of the treatment for DHF Grade – I and Grade – II⁹



DHF Grade – III and Grade – IV¹⁹

This is life threatening situation in which rapid and massive plasma loss occurring through increased capillary permeability leading to hypotension or shock, for what is required is prompt and adequate fluid replacement with crystalloids or colloids (plasma expanders).

Figure 3: Flow chart of the treatment for DSS⁹



Delayed or inadequate fluid resuscitation can cause multisystem organ dysfunction that may lead to death. Electrolyte and acid base disturbance may occur. There is a high potential for developing disseminated intravascular coagulopathy (DIC) in cases with prolonged shock.

Immediate Replacement of Plasma Loss

Fluid used for rapid volume expansion include physiological saline or Ringers lactate or Ringer's acetate. N.S or R.L. should be given at 10-20 ml per kg body weight boluses as rapidly as possible, repeat boluses 2-3 times until vital signs return to normal. Oxygen should be given to all patients in shock. If vitals are improving change fluids to 0.45% dextrose saline at rate of 3-6ml/kg body weight.

If hematocrit is still high and if there is no clinical improvement, plasma substitutes or 5% albumin (10-20ml/kg body weight) should be given, repeated if necessary for a total dose of 20-30ml/kg body weight of colloidal solution.

If shock still persists, hematocrit values should be revived for any evidence of a decline, which may indicate internal bleeding. Fresh whole blood transfusion (10ml/kg) may be necessary in such cases.

Continued Replacement of Further Plasma Loss

Plasma loss may continue for 24-48 hours requiring continued fluid administration with 5% dextrose in 0.45% normal saline. Decrease in infusion should be done stepwise and in general intravenous fluid therapy is not needed for more than 48 hours after termination of shock.

Replacement of extravasted plasma and hypervolemia, pulmonary oedema or heart failure may occur if more fluid is given during the recovery phase. At this stage, drop in haematocrit should not be interpreted as a sign of

internal bleeding. Strong pulse and blood pressure and adequate diuresis are good signs of recovery.

Use of Blood and Blood Products

Fresh Whole Blood

A drop in hematocrit with no clinical improvement despite adequate fluid administration indicates significant internal hemorrhage. Transfusion with fresh whole blood is preferable and the amount to be given such as normal red blood cell concentration should not be exceeded.

Fresh Frozen Plasma

It is indicated in cases where consumptive coagulopathy causes massive bleeding. DIC is usual in severe shock and may play an important part in the development of massive bleeding or lethal shock.

Platelet Transfusion

It is surrounded with controversies in DHF/DSS. Mild thrombocytopenia usually not associated with significant bleeding. Secondly, thrombocytopenia in DHF/DSS is a short lived phenomenon with platelets returning to normal by 7-9 days.

Platelet transfusion is indicated in adults when platelet count is less than 20,000/mm³ and have severe hemorrhage.¹ In children, prophylactic platelet transfusion indicated when platelet count is less than 20,000/mm³ with evidence of significant bleedings.¹⁹

Kebra SK et al (1998) analyzed patients of DHF/DSS with severe thrombocytopenia for the benefit of platelet transfusion, they conclude that number of days of hemorrhage and outcome in these patients were not improved compared to control.

Use of steroids

At patients WHO is not recommending the use of steroids in the management of DHF/DSS.

There are two clinical trails in paediatric age group namely Sumarmo et al¹⁷ which used hydrocortisone and Sampson Tassniyom et al²⁰ who used methyl prednisolone and found that the response in terms of mortality, duration of shock and amount of replacement fluids required same in both the study and control group.

Newer Drugs

The use of intravenous immunoglobulin in DSS and efficacy of heparin in DIC have not yet been documented.

Prognosis²

Most of the dengue virus infections are asymptomatic while some present with nonspecific constitutional symptoms undifferentiated from other viral infections.

The mortality in DHF/DSS may be as high as 40-50 percent if left untreated. Early recognition of illness, careful monitoring and appropriate fluid

therapy alone has resulted in considerable reduction of mortality to 1-5 percent. Early recognition of shock is of paramount importance as the outcome of patient with DSS depends on the duration of shock.

With proper treatment recovery is fast and majority of the patients recover completely in 24-48 hours without any residual sequelae.

Prevention

Attenuated dengue viruses type 1,2,3 and 4 vaccines are under development. The possibility is that dengue vaccination may sensitize recipient so that ensuing dengue infection could result in hemorrhagic fever.

The basic preventive measures consists of control of *Aedes aegypti* mosquitoes, which breeds in and around human dwellings and flourish in water.

WHO global control programme recommends the followings:²¹

- I. Selective integrated vector control with community and intersectoral participation.
- II. Active surveillance based on a strong health information system,
- III. Emergency preparedness.
- IV. Capacity building and training.
- V. Vector control.

METHODOLOGY

The present study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on adult patients with dengue fever during the period of January 2008 to December 2008.

Study design

One year cross-sectional study.

Study period

The present study was conducted during January 2008 to December 2009.

Method of collection of data

Source of Data

Adult patients with dengue fever proved by micro ELISA test admitted at medicine wards of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Sample size and sampling procedure

All the patients with dengue fever admitted at medicine wards of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the study period that is 200 admissions were recorded during the study and same considered as sample size.

Selection criteria

Inclusion Criteria

- All the adult patients with dengue fever proved using IgM micro ELISA test.

Exclusion Criteria

- Patients who were negative for dengue IgM micro ELISA test.

Procedure

The study was approved by the Ethical and Research Committee of J. N. Medical College, Belgaum. During the study period, all patients presenting with symptoms suggestive of dengue fever were approached to participate in the study and patients proved positive on IgM micro ELISA test were included in this study after obtaining informed written consent (Annexure–I).

Demographic data like age, gender, address and occupation was recorded on predesigned and pretested proforma (Annexure-II). At admission detailed history was taken in every patient and thorough general and systemic examinations were carried out with special attention to the symptoms of the patient, conscious level, temperature, pulse, blood pressure, pallor, icterus, skin lesion bleeding manifestations, hepatosplenomegaly, ascites and pleural effusion.

Vital signs

Temperature, pulse rate, blood pressure, respiratory rate, pulse oximetry were monitored every hourly in Grade III and Grade IV DHF, every four hourly

in Grade I and Grade II DHF every eight hourly in patients with DF. Patient with grade III and grade IV DHF were admitted in critical care unit and patients with DF, DHF Grade I and Grade II in the general ward.

Tourniquet test was carried out in all patients on admission. The test was performed by inflating the blood pressure cuff on the upper arm to the pressure level halfway between systolic and diastolic and maintained for five minutes and then examined the forearm for number of petechiae in a fixed template area. Test was considered positive if more than 20 petechiae were observed in a 2.5 cm² circular area.⁸

Intake and output chart

Meticulous fluid chart consisting of amount and type of intravenous fluid given and accurate urine output was maintained in all patients.

All baseline investigations as per the predesigned and pretest proforma like complete blood count, platelet count, hematocrit, coagulation profile like prothrombin time, activated partial thromboplastin time, bleeding time, clotting time, liver function test, kidney function test, X-ray chest, ultrasound abdomen were done at admission.

Hematocrit was monitored every 12 hourly in DHF Grade III and Grade IV and 12 hourly in DF, Grade I and Grade II DHF till vital signs were stable. Then depending upon patients clinical condition in DHF/DSS frequency was reduced to 24 hourly till patient was discharged.

The level at which the hematocrit and hemodynamic stabilized during recovery was used as an index of each patient baseline (normal) hematocrit.

The ratio was calculated to evaluate the degree of hemoconcentration in patients as follows.²²

$$[(\text{Highest HCT} - \text{Recovery HCT}) / \text{Recovery HCT}] \times 100$$

A value of 20% or more was considered as evidence of significant hemoconcentration. The patient who bleed enough to lower his hematocrit were excluded from these calculations.

Absolute platelet count in less than $< 1,00,000 /\text{mm}^3$ was defined as thrombocytopenia.¹⁰ Absolute platelet count was done every 24 hourly till it became normal or more than $1,00,000 /\text{mm}^3$, counts less than 20,000 to 50,000 $/\text{mm}^3$ and 50,000 to $1,00,000 /\text{mm}^3$ were considered as mild, moderate and severe grades of thrombocytopenia respectively.

Total differential white blood cell count was done daily till it became normal. Count between $4000 /\text{mm}^3$ to $11,000 /\text{mm}^3$ was considered normal. Total count $< 4000 /\text{mm}^3$ and $11,000 /\text{mm}^3$ were considered as leukopenia and leukocytosis respectively.

Liver function test was done in all patients at admission, repeated as and when indicated. Serum levels of SGOT and SGPT of more than 40 U/L were considered elevated. Hypoproteinemia was defined as total serum protein of less than 5.5 gm/dL. Hyperbilirubinemia was defined as total serum bilirubin of more than 1.2 gm/dL.

Prothrombin time and activated partial thromboplastin time (APTT) were done in all patients at admission and repeated as and when required. Prothrombin time and APTT values more than 1.5 times of control values were considered prolonged.

Chest X-ray PA / AP was done in all patients on admission and repeated as and when clinically indicated to confirm presence of pleural effusion.

Ultrasonography of abdomen and or chest was done in selected patients as and when clinically indicated to confirm presence of ascites and/or pleural effusion.

Serum electrolytes (Sr Na⁺, K⁺), blood urea and serum creatinine was done in all patients on admission and repeated as and when required.

Arterial blood gases parameter was done in selected patient depending on clinical condition of patient.

Blood sample for serological testing was collected after seven days of fever. Blood samples were centrifuged for detection of IgM antibodies by MAC ELISA test. Interpretation as “Recent dengue infection” was made if IgM antibodies to dengue virus was positive.

All cases were graded according to severity criteria based on the technical guidelines from the WHO which is as follows.^{23,24,25}

Dengue fever

Fever (Biphasic) accompanied by nonspecific constitutional symptoms like headache, retro orbital pain, myalgia, arthralgia with occasional leucopenia or thrombocytopenia with no evidence of plasma loss.

Dengue haemorrhagic fever

Grade I

Fever accompanied by non specific constitutional symptoms with only hemorrhagic manifestation was a positive tourniquet test with evidence of thrombocytopenia, leucopenia or rise in hematocrit.

Grade II

Above manifestations with evidence of spontaneous bleeding usually in the form of skin or mucosal bleed.

Grade III

Above manifestations with evidence of circulatory failure manifested by rapid and weak pulse, pulse pressure less than 20 mm Hg or hypotension with the presence of cold, clammy skin and restlessness.

Grade IV

Above manifestations with evidence of profound shock with undetectable blood pressure and pulse. Grade III and IV are collectively called as Dengue Shock Syndrome.

Statistical methods

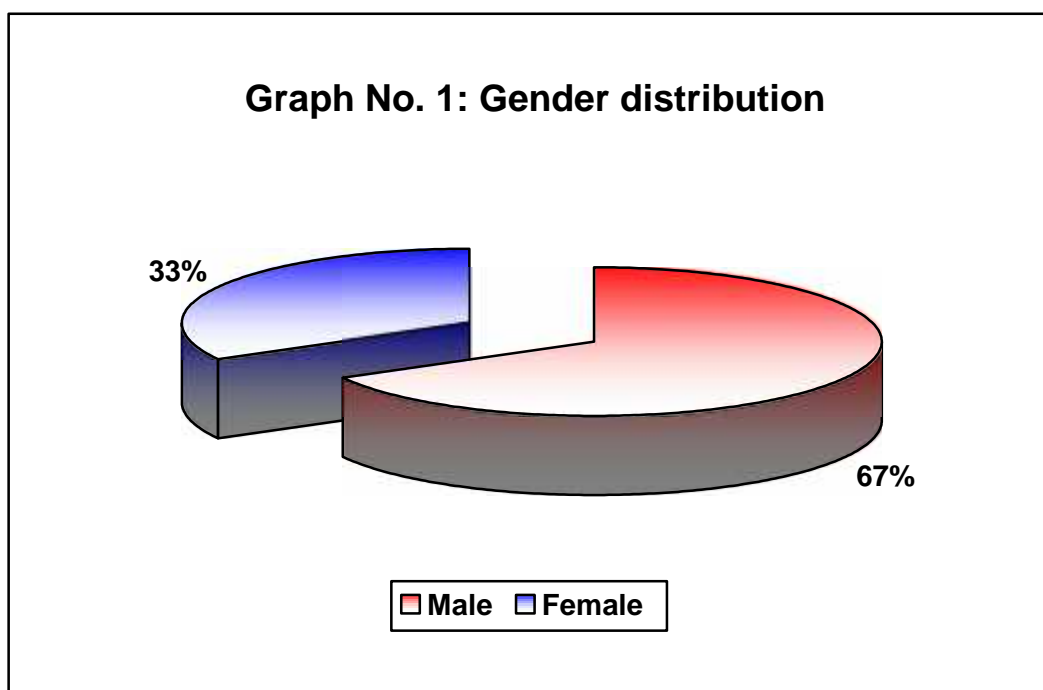
The data was tabulated and analysed using rates, ratios and percentages.

RESULTS

The present study was conducted in the Department of Medicine, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on 200 dengue IgM positive patients. The observations recorded are tabulated as below.

Table No. 1: Gender distribution

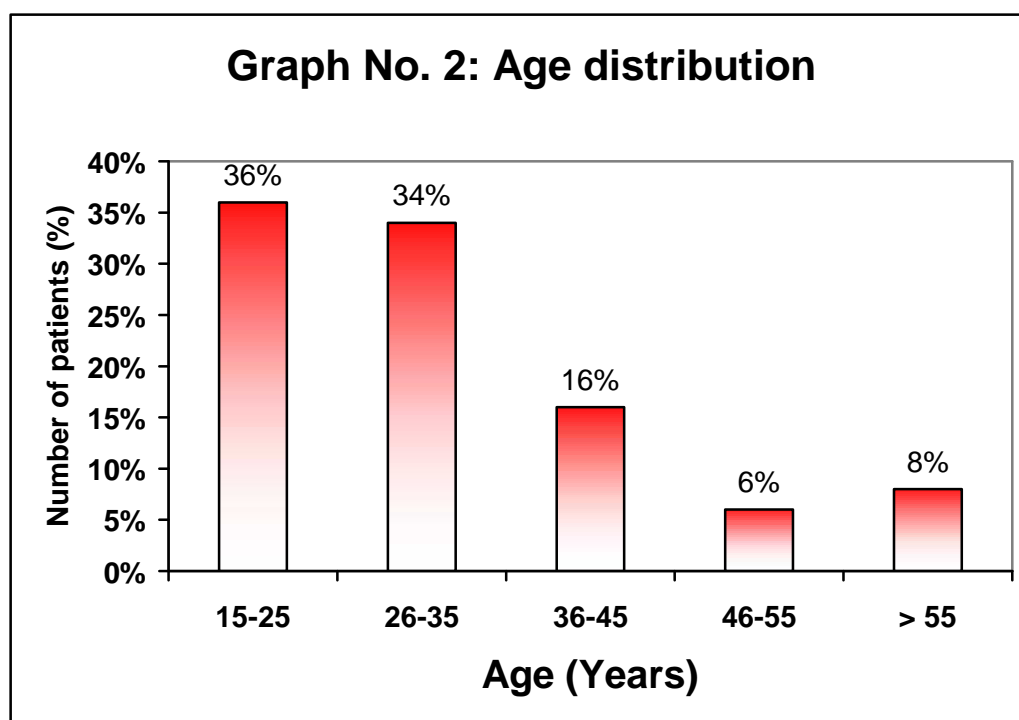
Sex	No. of patients	Percentage
Male	134	67%
Female	66	33%
Total	200	100%



In the present study out of 200 patients, 134 (67%) were males and 66 (33%) were females. The male : female ratio was 2:1.

Table No. 2: Age Distribution

Age groups (Years)	No. of patients	Percentage
15 – 25	72	36%
26 – 35	68	34%
36 – 45	32	16%
46 – 55	12	6%
> 55	16	8%
Total	200	100%



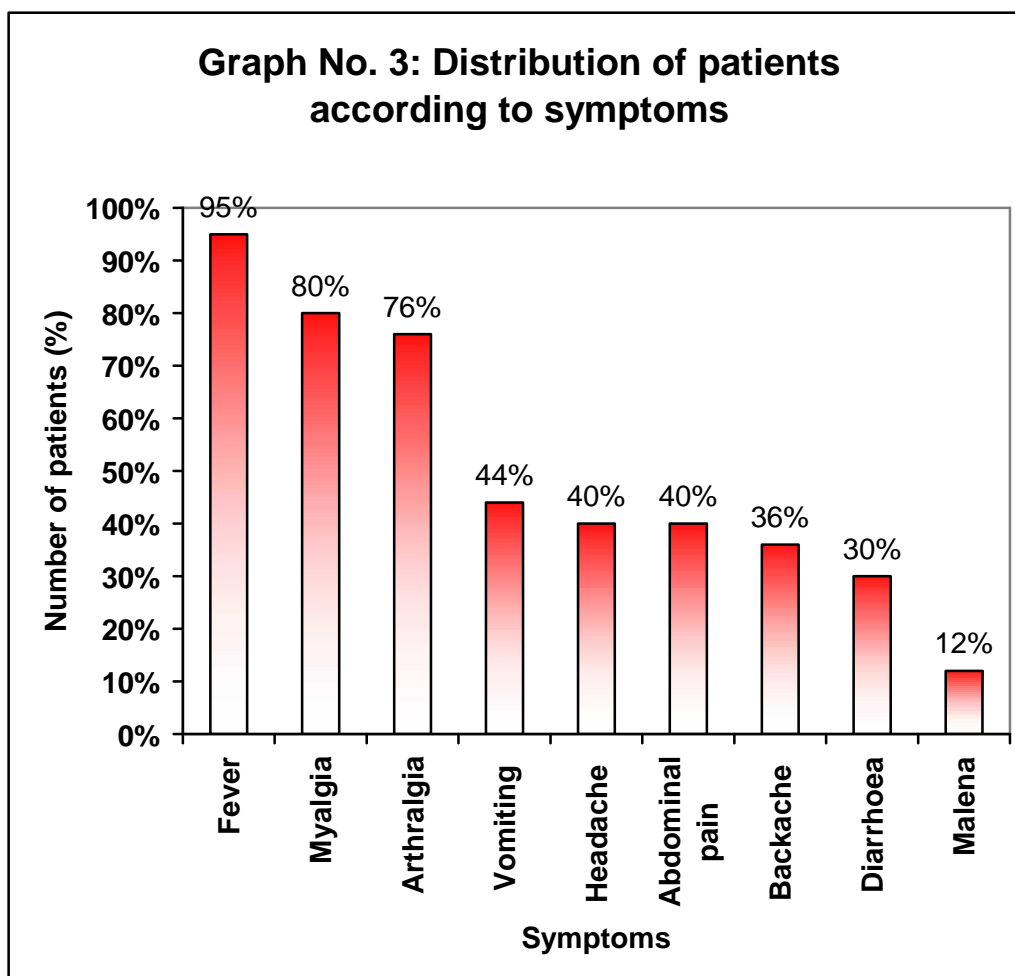
Out of 200 patients, majority that is 72 (36%) were in age group of 15 to 25 years followed by 68 (34%) in the age group of 26 to 34 years, 32 (16%) in

the age group of 36 to 45 years and 16 (8%) with age more than 55 years.

Twelve cases (6%) were recorded in the age group of 46 to 55 years.

Table No. 3: Distribution of patients according to symptoms

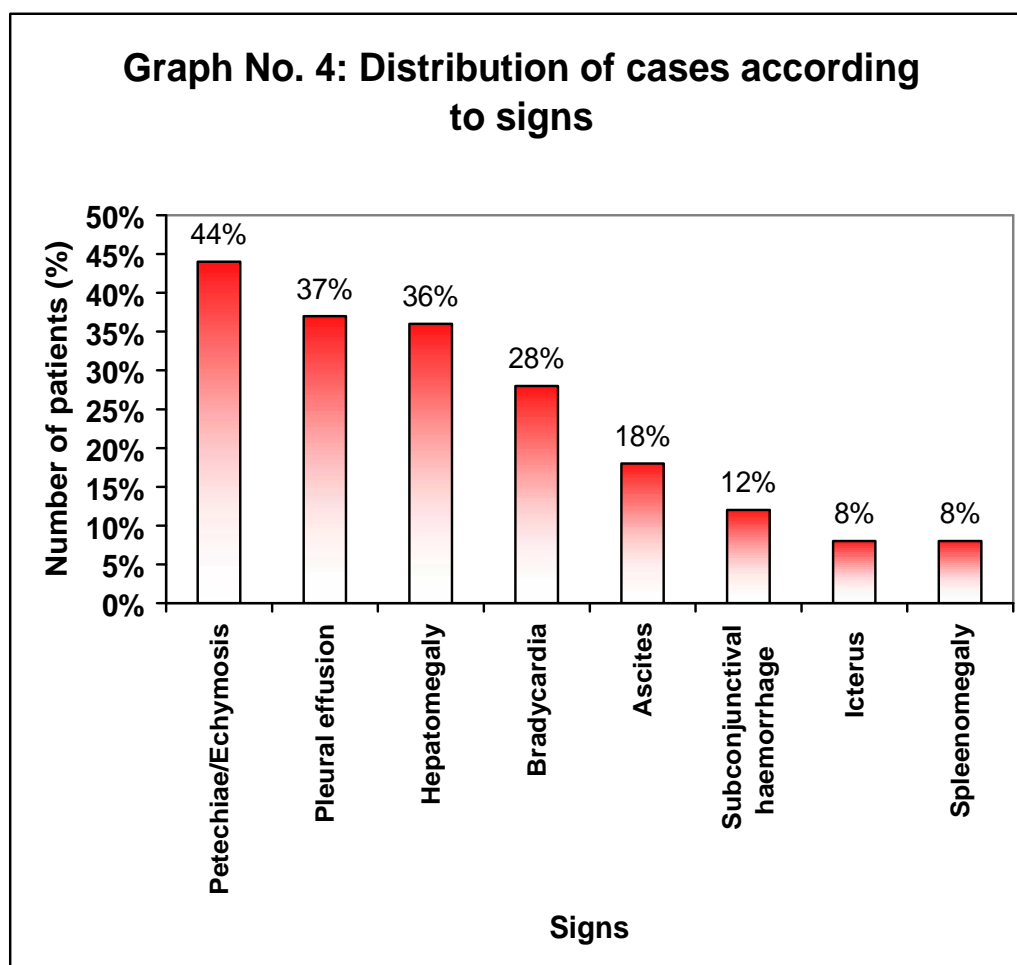
Symptoms	Number	Percentage
Fever	190	95%
Myalgia	160	80%
Arthralgia	152	76%
Vomiting	88	44%
Headache	80	40%
Abdominal pain	80	40%
Backache	72	36%
Diarrhea	60	30%
Malena	24	12%
Altered sensorium	4	2%
Seizure	2	1%



In this study majority that is 190 (95%) of the patients had fever as presenting symptom. Other symptoms like myalgia in 160 (80%), arthralgia in 152 (76%), vomiting in 88 (44%), abdominal pain and headache in 80 (40%), backache in 72 (36%), diarrhea in 60 (30%) and malena in 24 (12%) patients. Four (2%) patients presented with symptoms of altered sensorium and two (1%) patients presented seizures as presenting symptoms.

Table No. 4: Distribution of cases according to signs

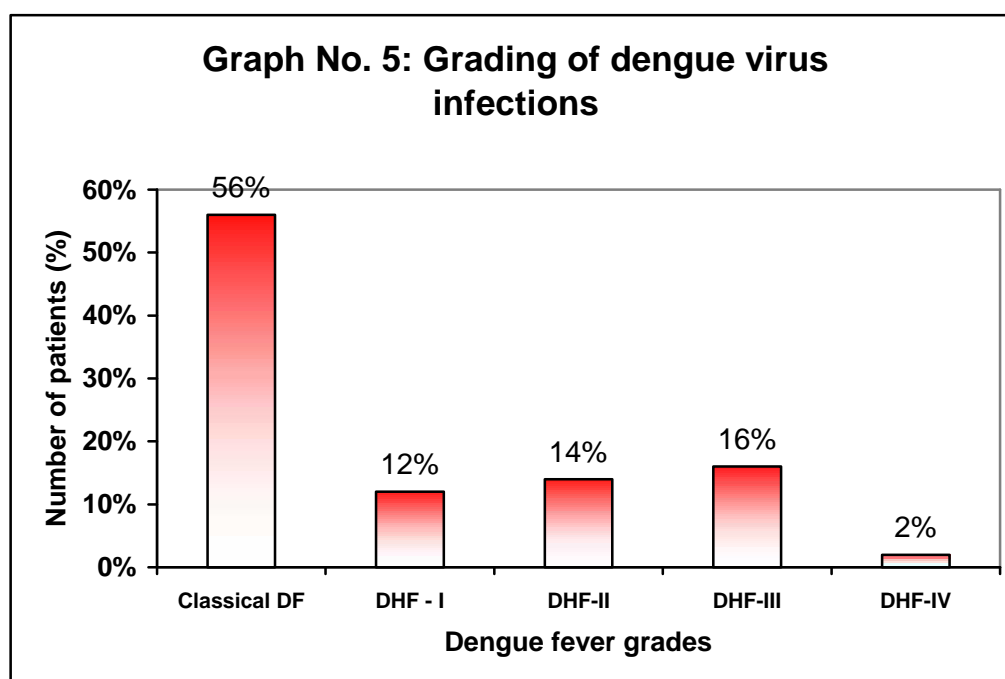
Signs	Number	Percentage
Petechiae / Echymosis	88	44%
Pleural effusion	74	37%
Hepatomegaly	72	36%
Bradycardia	56	28%
Ascites	36	18%
Subconjunctival haemorrhage	24	12%
Icterus	16	8%
Splenomegaly	16	8%



Out of 200 patients, 88 (44%) had petchial/echyonosis, 74 (37%) had pleural effusion, 72 (36%) had hepatomegaly, 56 (28%) had bradycardia, 36 had ascites, 24 (12%) had subconjunctival haemorrhage, 16 (8%) had icterus and 16 (8%) patients had splenomegaly.

Table No. 5: Grading of dengue virus infections

Grading of DV infection	No. of patients	Percentage
Classical DF	112	56%
DHF Grade – I	24	12%
DHF Grade – II	28	14%
DHF Grade – III	32	16%
DHF – IV	4	2%
Total	200	100%



Out of 200 patients, majority that is 112 (56%) were dengue fever, 24 (12%) were DHF Grade I, 28 (14%) were DHF Grade II, 36 (18%) were in DSS that is DHF Grade III + Grade IV.

Table No. 6: Distribution of cases according to hematological laboratory findings

Investigation	No. of patients	Percentage
Rise in hematocrit (%)	> 20%	39 19.5%
	< 20%	114 57%
	Normal	47 23.5%
Platelet count (/mm ³)	< 10000	30 15%
	10,000 – 20,000	41 20.5%
	20,000 – 50,000	73 36.5%
	50,000 – 1,00,000	33 16.5%
	> 1,00,000	23 11.5%
Leucocyte count (/mm ³)	<4,000	44 22%
	4,000 – 11,000	132 66%
	> 11,000	24 12%

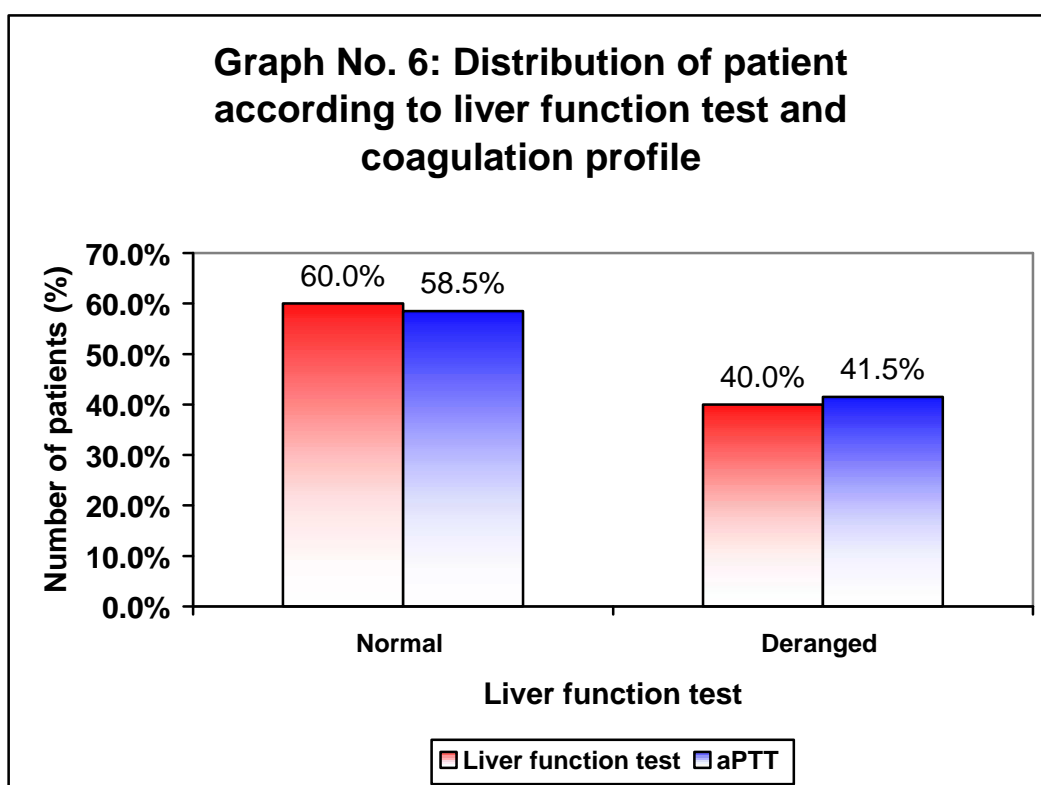
Out of 200 patients, 39 (19.50%) had hematocrit levels $> 20\%$, 114 (57%) had $< 20\%$ and 47 (23.50%) had normal hematocrit.

In this study, 71 (35.5%) had severe thrombocytopenia that is platelet count $< 20,000 /\text{mm}^3$, 73 (36.5%) had 20,000 to 50,000 $/\text{mm}^3$, 33 (16.5%) (19.40%) had platelet count 50,000 to 1,00,000 $/\text{mm}^3$ and 23 (11.5%) had more than 1,00,000 $/\text{mm}^3$.

Leukocyte count in 44 (22%) patients was $< 4,000/\text{mm}^3$, 132 (66%) patients had 4,000 to 11,000 $/\text{mm}^3$ and 24 (12%) had leukocyte count more than 11,000 $/\text{mm}^3$.

Table No. 7: Distribution of patient according to liver function test and coagulation profile

	No. of patients	Percentage
Liver function tests		
Normal	120	60%
Deranged	80	40%
aPTT		
Normal	117	58.5%
Raised	83	41.5%



In the present study, out of 200 patients, 120 (60%) patients had normal liver function test and 80 (40%) had deranged liver function. Activated partial thromboplastin time in 117 (58.5%) patients was normal and in 83 (41.5%) patients aPTT was raised.

Table No. 8: Correlation between thrombocytopenia and bleeding

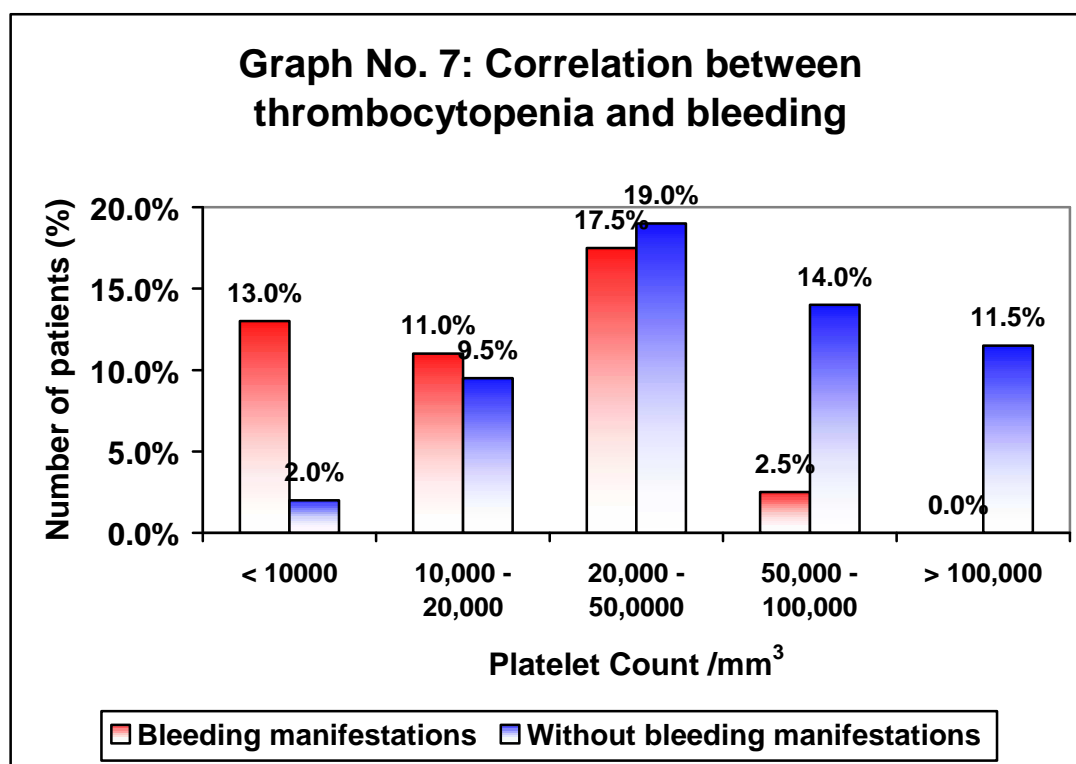
Platelet count	Bleeding Manifestations		Without bleeding manifestations		Total	
	No	Percentage	No	Percentage	No	Percentage
<10,000	26	13.0%	4	2.0%	30	15%
10,000 – 20,000	22	11.0%	19	9.5%	41	20.5%
20,000 – 50,000	35	17.5%	38	19.0%	73	36.5%
50,000 – 1,00,000	5	2.5%	28	14.0%	33	16.5%
>1,00,000	0	0.0%	23	11.5%	23	11.5%
Total	88	44.0%	112	56.0%	200	100.0%

 $\chi^2 = 53.345$

p=0.000

 $\chi^2 = 52.133$

p=0.000



In the present study out of 200 cases, 30 (15%) patients had platelet count $< 10,000 /\text{mm}^3$. Among them 26 (13%) had bleeding manifestations. Forty one patients had platelet count between 10,000 to 20,000 $/\text{mm}^3$ out of which 22 (11%) had bleeding manifestations. There were 73 (36.5%) patients who had platelet count ranged from 50,000 to 1,00,000 $/\text{mm}^3$ and among them five had bleeding manifestations whereas 23 (11.5%) patients had platelet count more than 1,00,000 and there were no bleeding manifestations and this correlation was statistically significant (0.000).

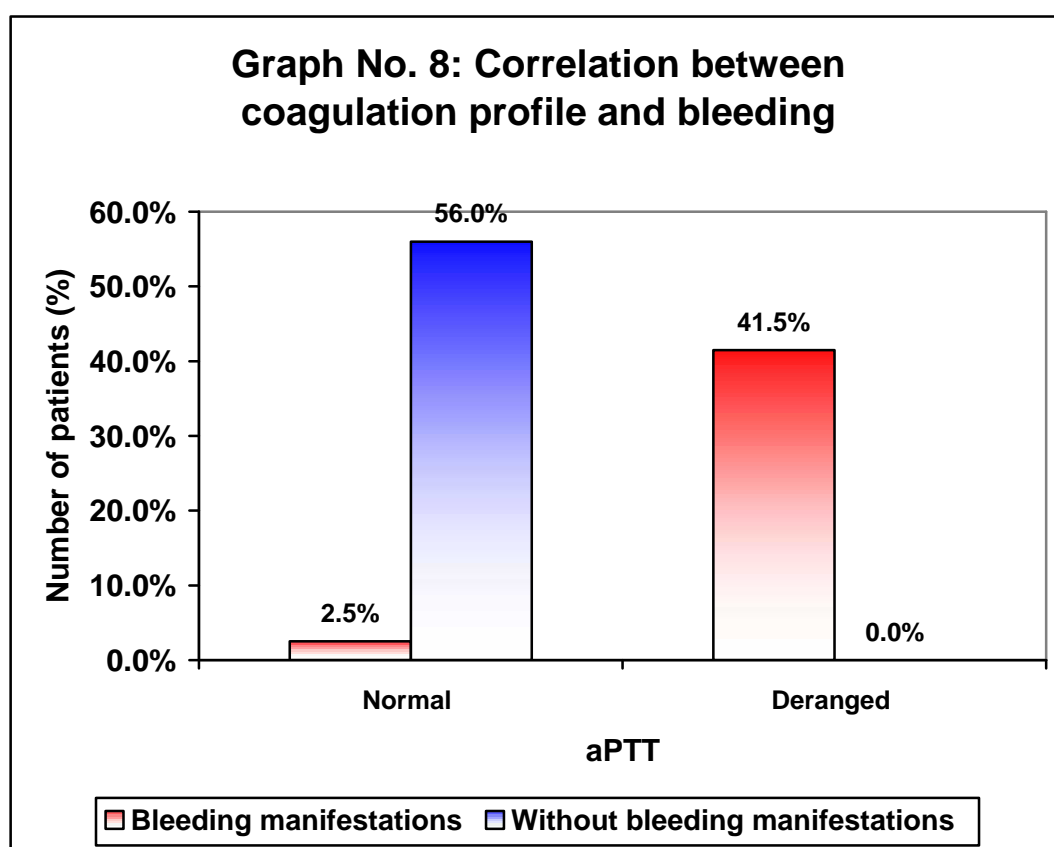
Table No. 9: Correlation between coagulation profile and bleeding

aPTT	Bleeding Manifestations		Without bleeding manifestations		Total	
	No	Percentage	No	Percentage	No	Percentage
Normal	05	2.5%	112	56.0%	117	58.5%
Deranged	83	41.5%	00	0.00%	83	41.5%
Total	88	44.0%	112	56.0%	200	100.0%

$$X^2=180.574$$

$$DF=1$$

$$p=0.000$$



In the present of 200 cases, 117 (58.5%) patients had normal aPTT. Among them five (2.5%) patients had bleeding manifestations. There were 83 (41.5%) with deranged aPTT function and all had bleeding manifestations and this correlation was statistically significant (0.000).

Table No. 10: Correlation of IGM and IGG with bleeding manifestations

Serology positive	Bleeding Manifestations		Without bleeding manifestations		Total	
	No	Percentage	No	Percentage	No	Percentage
IgM	36	18.0%	76	38.0%	112	56.0%
IgM and IgG	52	26.0%	36	18.0%	88	44.0%
Total	88	44.0%	112	56.0%	200	100.0%

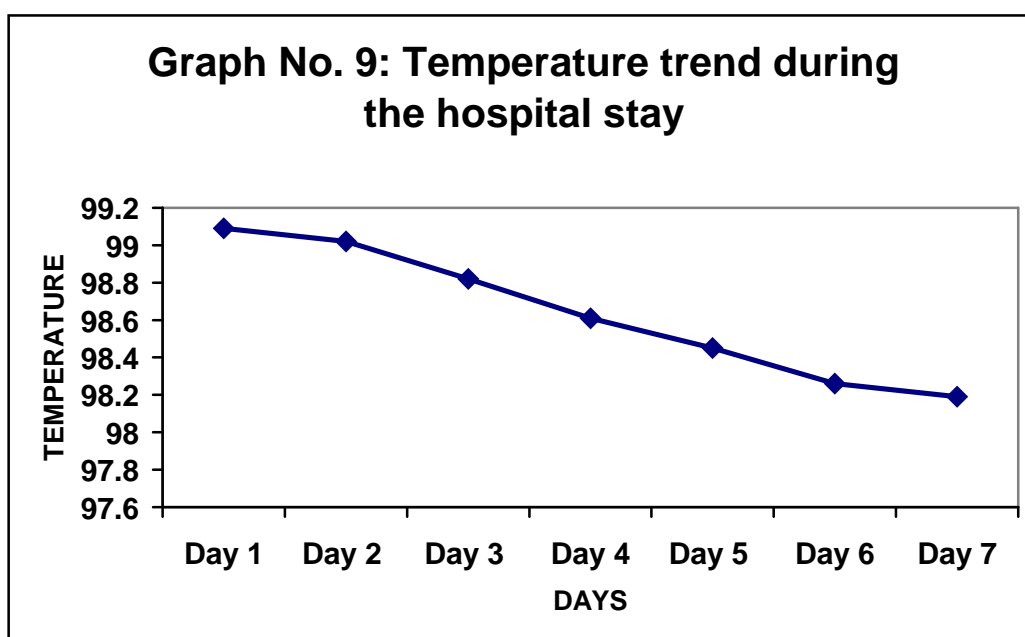
$$X^2=14.524$$

$$p=0.000$$

In the present study out of 200 patients 112 (56.0%) were only IgM positive. Among them 36 (18.0%) had bleeding manifestations. Remaining 88 patients had IgM and IgG positive dengue serology out of which 52 (26.0%) patients had bleeding manifestations and this correlation was statistically significant (0.000).

Table No. 11: Temperature trend during the hospital stay

Duration	Temperature ($^{\circ}\text{C}$)		p value
	Mean	S.D.	
Day 1	99.09	1.14	0.254
Day 2	99.02	1.18	0.000
Day 3	98.82	1.01	0.000
Day 4	98.60	0.74	0.000
Day 5	98.45	0.55	0.000
Day 6	98.26	0.39	0.000
Day 7	98.19	0.32	0.007

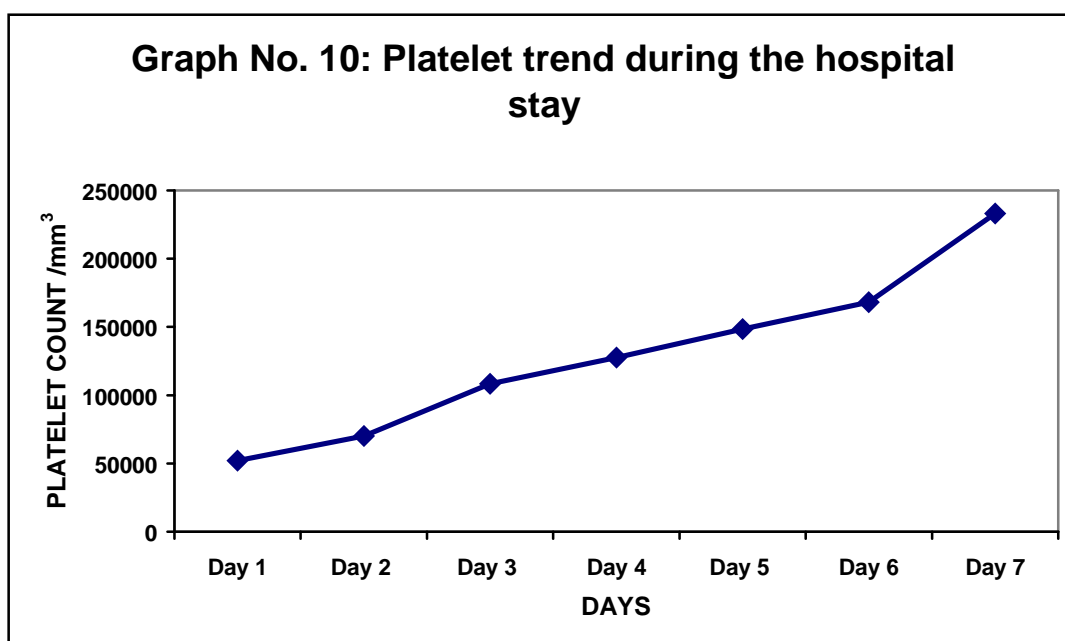


The mean temperature among the dengue patients during the hospital stay had decreasing trend. On the Day 1 mean temperature was 99.09 ± 1.14 and on

day 7 it was 98.19 ± 0.32 . However this reduction between temperature from day two to day seven was statistically significant ($p < 0.050$).

Table No. 12: Platelet trend during the hospital stay

Duration	Platelet count (/mm ³)	
	Mean	S.D.
Day 1	52135	52885.43
Day 2	70060	34173.81
Day 3	108350	35107.64
Day 4	127585	25042.45
Day 5	148536	13588.35
Day 6	168048	162917.12
Day 7	233000	45254.83



The mean temperature among the dengue patients during the hospital stay had decreasing trend. On the Day 1 mean temperature was 99.09 ± 1.14 and on

day 7 it was 98.19 ± 0.32 . However this reduction between temperature from day two to day seven was statistically significant ($p < 0.050$).

Table No. 13: Distribution of dengue patients according to treatment

Treatment	Number of patients	Percentage
Vitamin K	84	42.0%
Single donor platelet	108	54.0%
Steroids	14	7.0%
Fresh frozen plasma	68	34.0%

In the present study majority (54%) of the patients received single donor platelet followed by 42% received vitamin K, 34% received fresh frozen plasma and least being (Seven percent) received steroid.

Table No. 14: Mortality rate

Grading of DV infections	Total No. of patients	Mortality		
		Number	Percentage	
Dengue fever	112	00	0.0%	
Dengue haemorrhagic fever	Grade – I	24	00	0.0%
	Grade – II	28	00	0.0%
	Grade – III	32	02	1.0%
	Grade – IV	04	03	1.5%

In present study there was no mortality in dengue fever and grade-I and II dengue haemorrhagic fever. Two (1%) patients with dengue haemorrhagic fever Grade III and three (1.5%) patients with grade IV succumbed to death.

DISCUSSION

Total of 200 serologically confirmed cases of DF, DHF/DSS admitted in Department of Medicine.

In this study, out of 200 patients, majority that is 140 (70%) were in age group of 15 to 35 years. Nine (13.43%) patients were in age group 35 to 55 years and (10.44%) were more than 55 years of age. Most of these patients were adults, this may be due to the fact that adults are not immune to all strains of the dengue virus.²⁶

A study conducted by Sarkar JK et al²⁷ showed 84% of the cases in age group of 11 to 30 years. Another study conducted by Fu Xi Qiu et al²⁸ found 81% of the patients were among more than 20 years. In this study 70% of the cases were between age group of 15 to 35 years.

In the present study out of 200 patients, 134 (67%) patients were male and 66 (33%) were females with a male to female ratio of 2:1. These findings were comparable with a study conducted by Agarwal et al²⁹ in which male to female ratio was 1.9:1. Another study conducted by Sharma et al²⁶ showed that male to female ratio was 3:1.

In the present study, out of 200 patients, majority that is 112 (56%) cases were classical DF, 24 (12%) cases were DHF Grade – I, 28 (14%) were DHF Grade – II and 36 (18%) cases were DSS (DHF Grade – III and Grade IV).

Eva Harris et al³⁰ studied 614 cases of dengue viral infections, of these 268 (44%) were classified as dengue fever (DF), 267 (43%) as DF with hemorrhagic manifestations, 40 (7%) as dengue hemorrhagic fever (DHF), 20 (3%) as dengue shock syndrome (DSS) and 17 (3%) as dengue with signs associated with shock (DSAS).

Kalayanarooj S et al³¹ studied 60 serologically confirmed cases of dengue viral infection in child age group and found 32 (53%) cases were DF, 5 (8%) cases were DHF Grade – I, 14 (23%) as DHF Grade – II, 9 (15%) were DHF Grade – III and no subject with Grade IV DHF.

In the present majority (95%) of the patients presented with fever All patients were admitted with history of fever in the hospital in this study. Among them 80% of patients had history of biphasic fever associated with chills and rigors. The other symptoms observed were bodyache (80%) (in the form of myalgia, headache, backache, retro-orbital pain), vomiting (44%) and abdominal pain (diffuse, sometimes localized in the epigastrium) (40%). The skin rash in the form of erythematous maculopapular rash, blanching, over trunk and extremities, presented in 60 (30%) cases. Altered sensorium or history of convulsion due to intracranial bleed, or hypoxia of brain due to hypotension were present in four (2%) cases.

A study conducted by Sharma et al²⁶ showed that most commonest presenting symptom among the dengue cases was fever (100%). The symptoms reported included bodyache (98%), vomiting (28%) loose motion (12.7%), abdominal pain (10.5%), skin rash (43.1%) and altered sensorium (0.5%)

In another study by Eva Harris et al³⁰ showed 99% of the cases had a fever, 91% had bodyache, 83% had vomiting, 15% had loose motion 48% had abdominal pain and 56% had skin rash as presenting symptoms.

In the present study out of 200 patients, 44% had petchial/ echyonsis, 37% had pleural effusion, 36% had hepatomegaly, 28% had bradycardia, 18% had ascites, 12% had subconjunctival haemorrhage, eight percent each had icterus and splenomegaly.

A study conducted by Krishnamurthy K et al³² showed 16.8% each had hepatomegaly and splenomegaly, 12% had bradycardia, 13.1% had pleural effusion and eight percent had ascites as presenting signs. Another study conducted by Sharma S et al²⁶ showed 40% of the patients had petechial / ecchymosis, 15% had pleural effusion, 20.4% had hepatomegaly, 8.2% had splenomegaly as presenting signs of the patients.

In the present study, 20 (10%) had one or the other evidence of spontaneous hemorrhagic manifestations. Among 24 (12%) cases gastrointestinal tract bleed in the form of hematemesis or melena was present. In 88 (44%) patients skin bleed in the form of petechiae, echymoses or bleeding at venepuncture sites was noticed, 12 (6%) patients had epistaxis or gum bleed and 8 (4%) cases had hematuria. One patient had an intracranial bleed, which is a very rare bleeding manifestation of viral hemorrhagic fever.

A study conducted by Eva Harris et al³⁰ on epidemic of dengue in Nicaragua (1998) among 216 cases in adults more than 15 years of age showed that

haemorrhagic manifestations in the form of petechiae in 35% patients, epistaxis in 11% and melena in nine percent of cases.

Fu-Xi Qiu et al²⁸ conducted study on 154 laboratory confirmed cases of dengue hemorrhagic fever which was carried out during 1978's epidemic in South China showed the evidence of epistaxis (35.7%), petechiae/purpura/ecchymoses (26.6%), hematemesis/melena (26.6%), gum bleeding (17.5%), uterine bleeding (17.5%), bloody stool (8.4%), hematuria (5.8%) conjunctival hemorrhage (2.6%), haemoptysis (1.3%), intracranial haemorrhage (0.7%) as bleeding manifestations.

A study conducted by Krishnamurthy K et al³² during 1964's epidemic of dengue fever at Visakhapatnam (AP) found hemorrhagic manifestation in 23 (21.5%) case out of 107. vaginal bleeding was observed in 9.34%, hematemesis and melena 4.6% petechiae, ecchymosis and purpura (skin bleed) in 7.47%, gum bleeding in two hemoptysis in one and epistaxis in one.

In this study the tourniquet test was positive in 48 (24%) cases out of 200. The test was 100% positive in DHF grade – I, 20% in grade – II DHF, 16.6% in grade – III DHF and the test was negative in grade – IV DHF.

Fu-Xi Qiu et al²⁸ a study of 220 patient, age group 10 – 29 years, from a epidemic of Zhan country, 1985, shows positive tourniquet test in 66.9% of cases. Another study by Eva Harris et al³⁰ (1998) showed 33% positivity of tourniquet test among the patients with dengue fever.

The less positivity of tourniquet test in the present study as compared to the other two studies was because both DF and DHF was included in the present study while both the other studies included only DHF and as the test is positive only in DHF cases.

In the present study, out of 200 patients, 72 (36%) had hepatomegaly and 16 (8%) cases had splenomegaly. Percentage of cases with hepatomegaly was slightly more than splenomegaly.

In the study conducted by Krishnamurthy K et al³² 16.8% of patients were splenomegaly and 16.8% were hepatomegaly. In another study by Sharma S et al²⁶ 20.4% had hepatomegaly and 8.2% had splenomegaly.

In majority of the studies, hepatomegaly was more than splenomegaly, except in studies of Krishnamurthy K. et al³² which was conducted during epidemic of dengue fever in Visakhapatnam during 1964, which showed equal occurrence of both hepatomegaly and splenomegaly.

In this study, 110 (55%) cases had evidence of either pleural effusion or ascites, out of which 74 (37%) had pleural effusion, 36 had ascites. In the 40 (20%) patients in which we had done diagnostic tapping, serous fluid was transudative. The diagnosis of pleural effusion was done by X-ray chest and ultrasound of chest and diagnosis of ascites was done by ultrasound of abdomen.

A study conducted by Sharma et al²⁶ showed 10.2% had pleural effusion and 5.10% had ascites.

In the present study, out of 200 patients, 12 (6%) cases had evidence of circulatory failure, out of which 10 patients had hypotension and two patient had shock (pulse and blood pressure not recordable).

Eva Harris et al³⁰ studied virologic features of dengue in the 1998 epidemic in Nicaragua (US). Of the 614 confirmed case, 268 (44%) presented with dengue fever, 267 (43%) with dengue fever with hemorrhagic manifestations, 40 (7%) with dengue hemorrhagic fever, 20 (3%) with DSS and 17 (3%) dengue with signs associated with shock (DASS). Another study by Tripathi BK et al³³ showed 2.5% cases with circulatory failure.

Circulatory failure in different studies^{30,33} was due to differences in the inclusion of cases from DF to DHF. The studies which included patients of DF and low percentages of circulatory failure and the studies which included only DHF had higher percentages of circulatory failure.

In the present study, calculation of hematocrit was possible in 200 patients. One patient expired without correction of shock, thus recovery Hct could not be obtained and the baseline Hct was not known.

In the present study, Hct > 20% was present in 39 (19.40%) case. Sharma S et al²⁶ showed a epidemic of DHF in adults in Delhi during 1996 that raised Hct > 48% were present in 6 (6.12%) cases and Hct > 20% were present in 14 (14.28%) cases.

In the present study, thrombocytopenia (<1,00,000/mm³) was observed in 88.5% of the patients. Among them seven (35.5%) had severe thrombocytopenia

(<20,000/mm³), 73 patients (36.5%) had moderate thrombocytopenia (20,000 to 50,000/mm³), while 33 patients (16.50%) had mild thrombocytopenia (50,000 to 1,00,000/mm³).

Sharma S. et al²⁶ from a outbreak of dengue hemorrhagic fever in Delhi (1998), observed out of 98 patients only four patients had platelet count >1,00,000/mm³, 94 (95.91%) had platelet count < 1,00,000/m³ and 43.8% had severe thrombocytopenia that is platelet count <20,000/mm³.

In our study, out of 200 patients 44 (22%) cases had leukocyte count <4,000/ mm³ 132 (66%) cases had leukocyte count 4,000 – 11,000/ mm³ and 24 (12%) had leukocyte count >11,000 mm³. Leukopenia was more common in DF (34.37%) and DHF Grade I and II (42.85%) as compared to DSS (16.66%).

Krishnamurthy K. et al³² in 1964, from a outbreak of dengue like illness in Visakhapatnam observed that out of 89 cases, 28 (31.46%) had leukopenia (<5,000/ mm³), remaining 61 (68.54%) cases had normal leukocyte count and there was no leukocytosis in any of the cases.

Fu-Xi Qiu et al²⁸ observed leucopenia in 94% cases and S. Sharma et al observed leukopenia in 30.13% of cases.

Results in present study, out of 200 patients 120 (60%) had normal liver function tests (LFT'S) and 80 (40%) had deranged LFT'S (deranged LFT's when liver enzymes that is SGOT or SGPT are elevated or serum proteins levels fall below 5.5 gm/dl).

Sharma S et al²⁶ observed elevation of SGOT in 88.4% of cases and SGPT in 76.7% of the cases. Fu-Xi Qiu et al²⁸ observed mild to moderate elevation of SGOT in 29.4% of cases and overall abnormal LFT in 68 (44.2%). Cohen S et al²² in their study of 12 patients with DHF/DSS reported hypoproteinemia in 49 (40%) patients.

In our study out of 200 patients, 117 (58.5%) had normal PT/aPTT and 83 (41.5%) patients had raised PT/APTT (raised PT/aPTT when values raised more than 1.5 times of the normal values).

In a study conducted by Fu Xi Qiu et al²⁸ out of 154 serologically confirmed cases of DHF, prolonged bleeding time was observed in 108 (76.7%) cases.

In the present study out of 200 cases, 30 (15%) patients had platelet count $< 10,000 /\text{mm}^3$. Among them 26 (13%) had bleeding manifestations. Forty one patients had platelet count between 10,000 to 20,000 $/\text{mm}^3$ out of which 22 (11%) had bleeding manifestations. There were 73 (36.5%) patients who had platelet count ranged from 50,000 to 1,00,000 $/\text{mm}^3$ and among them five had bleeding manifestations whereas 23 (11.5%) patients had platelet count more than 1,00,000 and there were no bleeding manifestations and this correlation was statistically significant (0.000).

A study³⁴ conducted in Indonesia in 2003 observed 59 cases with platelet count less than 25,000 $/\text{mm}^3$ out of which 11 had bleeding manifestations. 164 patients had platelet count 25000 to 50000 $/\text{mm}^3$ out of which 14 had bleeding manifestations, 205 patients had platelet count of 50,000 to 74,000 out of which

22 cases had bleeding manifestations, 209 cases had platelet count 75,000 to 100,000 /mm³ out of which 16 had bleeding manifestations and 663 patients had platelet count more than 100,000 /mm³ out of which nine had bleeding manifestations.

In the present of 200 cases, 117 (58.5%) patients had normal aPTT. Among them five (2.5%) patients had bleeding manifestations. There were 83 (41.5%) with deranged aPTT function and all had bleeding manifestations and this correlation was statistically significant (0.000).

A study³⁵ conducted in Taiwan in 2003 showed that 77 patients out 79 (97.5%) patients with DHF had prolonged aPTT and 33 out of 48 (68.8%) had prolonged aPTT in classical dengue fever patients.

In the present study out of 200 patients 112 (56.0%) were only IgM positive. Among them 36 (18.0%) had bleeding manifestations. Remaining 88 patients had IgM and IgG positive dengue serology out of which 52 (26.0%) patients had bleeding manifestations and this correlation was statistically significant (0.000).

A study³⁶ conducted in Thailand in 2000 recorded 32 only IgM positive patients with dengue fever out of which 23 had dengue haemorrhagic fever and 133 patients with both IgM and IgG positive out of which 53 had dengue haemorrhagic fever.

There is no specific treatment indicated for dengue fever. Despite majority (54%) of the patients with the platelet count being less than 20,000

/mm³ improved with single donor platelet and combination with vitamin K and FFP. Out of 88 patients with DHF 83 (41.5%) had deranged aPTT and most of them required vitamin K and FFP for stabilization of bleeding manifestations apart from fluid therapy. Even though steroids are not indicated in dengue fever some of the patients (7%) responded to steroid therapy in the form of rapid recovery of platelet count and stabilizing blood pressure.

In the present study, overall out of 200 patients of DF, DHF/DSS, 5 (2.5%) patients died.

- i) There was no mortality in DF and DHF Grade I and Grade II.
- ii) In DSS, 5 (2.5%) patients died, due to intracranial hemorrhage, acute fulminant hepatic failure and disseminated intravascular coagulation (DIC) respectively.

So, early recognition of the disease and prevention rather than treatment of complications are most important for the favourable outcome of the disease.

Tripathi BK et al³³ observed out of 560 patients, eleven patients died, three due to DIC, one of intracranial hemorrhage and seven due to massive gastric hemorrhage.

Fu-Xi Qiu et al²⁸ observed that out of 154 patients of DHF, 10 patients died. The causes of death were: shock (3 cases), pulmonary or cerebral hemorrhage (3), DIC (2), massive gastrointestinal hemorrhage (1) and pan hematocytopenia syndrome (1).

CONCLUSION

- In our study 200 serologically confirmed cases of dengue viral infection, majority of the patients were in adult age group.
- There was slight male preponderance with male to female ratio of 2:1.
- Half of the cases belonged to classical dengue fever with the other half to dengue hemorrhagic fever and only 10% cases had dengue shock syndrome.
- All the patients had fever and constitutional symptoms like bodyache, headache, vomiting, abdominal pain etc.
- Thirty percent patients had evidence of spontaneous hemorrhagic manifestations with hematemesis and melena at the top with one patient of intracranial bleed.
- Most important sign of tourniquet test was positive in 48% evidence of plasma leakage in the form of ascites and pleural effusion was present in 55% cases and circulatory failure in 6% cases.
- Average time taken to rise platelet count to near normal was six to seven days.
- Mortality in DHF was mainly related to the complications of the DHF, so early recognition of the disease and prevention rather than treatment of

complications are most important for the favourable outcome of the disease.

- Severity of deranged aPTT had significant correlation with severity of bleeding manifestations and also IgM and IgG positive cases had more severe disease compared to only IgM positive cases.

SUMMARY

This hospital based cross sectional study of “clinical profile of Dengue – fever in Adults” includes 200 serologically confirmed cases of Dengue viral infections.

All patients were classified and managed with the help of WHO guidelines.

During one year of study period almost 450 patients were admitted with clinical evidence of fever, skin rash, bleeding manifestations, thrombocytopenia or hypotension. Certain conditions were ruled out like malaria, typhoid, acute ITP and other viral infections by performing particular tests. Out of 450 serum samples 200 were positive for Dengue IgM Antibodies and were included in this study.

Findings from the present study are summarized below

1. In this study, majority of the patients (70%) were in adult age group from 15 to 35 years, 22% were in 35 to 55 years age group and eight percent had more than 55 years of age.
2. There was a slight male preponderance (67%).
3. Majority of patients (56%) had dengue fever, 26% had DHF Grade I and II and 18% had evidence of hypotension.

4. Out of 200 patients, 95% had fever and constitutional symptoms in the form of body ache (80%), vomiting (44%), diarrhea (30%) and abdominal pain (40%).
5. Altered sensorium, a sign of either intracranial hemorrhage or brain hypoxia due to hypotension was present in one percent of cases.
6. Forty four (44%) percent patients had petechial echymosis and 12% had malena as bleeding manifestations.
7. Out of 200 cases, tourniquet test was positive in 96 (48%) cases of DF. It was 100% positive in Grade I DHF, 40 (20%) patients in Grade II DHF, 32 (16%) patients in Grade III DHF. Test was negative with Grade IV DHF.
8. Objective evidence of plasma leak in the form of pleural effusion or ascites was present in 110 (55%) cases.
9. Out of 88 cases of DHF, evidence of circulatory failure was present in 12 (6%) cases.
10. Rise in hemetocrit more than 20% was present in 39 (19.50%) cases and less than 20% rise of hematocrit was present in 114 (57%) cases.
11. Platelet count less than 20,000 /cmm was observed in 71 patients (35.5%), 20,000 to 50,000 /cmm in 73 (36.5%) and 50,000 to one lakh in 33 (16.5%) cases. Twenty three (11.5%) cases had more than one lakh /cmm platelet count.

12. Leukocyte count less than 4,000 /cmm was found in 44 (22%) cases, 4000 to 11,000 /cmm was noted in 132 (66%) cases and more than 11,000 /cmm was recorded in 24 (12%) patients.
13. Out of 200 patients hepatomegaly was present in 71 (35.5%) and splenomegaly was present in 16 (8%) cases. Hepatosplenomegaly was more common in DHF and DF.
14. Eighty (40%) cases had deranged LFT.
15. Raised APTT was observed in 83 (41.5%) cases.
16. In this study, 112 patients had only IgM positive and 88 patients had both IgM and IgG were positive. Out of which 52 (26%) had bleeding manifestations and this correlation was statistically significant ($p=0.000$).
17. Majority of patients with DF/DHF/DSS were managed with supportive therapy like bed rest, antipyretics and parenteral crystalloid fluid, four patients of DSS received FFP and 108 patients received platelet transfers.
18. Mean duration of hospital stay or in other words time taken to raise platelet count to normal, in DF was six days, in DHF Grade I was eight days in Grade III seven days and Grade IV eight days.
19. Overall mortality observed in DF and DHF was five (2.5%) cases, in DHF III two (1%) cases and in DHF IV three (1.5%) cases.

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ANNEXURE I - CONSENT FORM

STUDY: CLINICAL PROFILE OF DENGUE FEVER IN ADULTS - A ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY

Principal Investigator: Dr. Ravi Solabannavar

Guide: Dr. V. A. Kothiwale

VOLUNTARY PARTICIPATION / WITHDRAWAL

Taking part in this study is voluntary. I may choose not to take part in this study, or if I decide to take part I can later change my mind and withdraw from the study. My decision is will not change the present or future health care or other services that I receive. The study doctor or sponsor may stop my participation in this study any time. If I choose not to take part in the study I will receive the standard treatment for patients with my condition.

COSTS

Costs for investigations

COMPENSATION

The study is purely voluntary and no compensation will be given in any form.

CONFIDENTIALITY

All information collected about me during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify

me in this research record. Information from this study may be published but my identity will be confidential in any publication.

This research is intended to study clinical profile of dengue fever. I agree to be part of the study and hence requested to participate in the same.

If I agree to be part of the research study I will be asked the relevant history and will be subjected to relevant clinical examination and investigations. My co-operation will be of great help to patients with dengue fever.

CONSENT OF THE SUBJECT TO PARTICIPATE IN THE STUDY

I voluntarily agree to take part in this study by signing below. I may withdraw at any time. I am not giving up any of my legal rights by signing this form. My signature below indicates that I have read, or it has been read to me, this entire consent form, and have had all my questions answered.

Name of the Participant or legally authorised representative: _____

Signature _____

In case of any queries during the study or in future, I may contact following person

Principal investigator: Dr. Ravi Solabannavar

Guide: Dr. V. A. Kothiwale

Name of the legally authorised representative _____

Signature _____

Name of the Witness _____

Signature _____

Investigator Name and Signature _____

Date:

Place:

ANNEXURE II

PROFOMA

I.P number:

Patient Name:

Age:

Sex:

Address:

Date of admission:

Date of discharge:

SYMPTOMS AND SIGNS:

1. Fever
2. Chills
3. Headache
4. Neck rigidity
5. Vomiting
6. Nausea
7. Giddiness
8. Backache
9. Myalgia
10. Joint pains
11. Retroorbital pain
12. Abdominal pain
13. Loss of appetite
14. Loose stools
15. Jaundice

Duration:

16. High coloured urine
17. Photophobia
18. Oliguria
19. Cough
20. Sore throat
21. Breathlessness
22. Haemoptysis
23. Epistaxis
24. Diarrohoea / Dysentery
25. Malena
26. Hematemesis
27. Subconjunctival haemorrhage of purpura
28. Bleeding gums
29. Petechiae
30. Urticaria

PAST HISTORY:

1. Previous hospitalisation
2. Similar illnesses

PERSONAL HISTORY:

(Menstrual history in females)

PHYSICAL EXAMINATION:

General condition:

Pallor:

Icterus:

Lymphadenopathy:

Conjunctiva:

Tongue:

Rashes:

Temperature:

Pulse:

Blood pressure:

SYSTEMIC EXAMINATION:

R.S:

C.V.S:

P/A:

CNS:

LABORATORY INVESTIGATIONS:

- CBC
- ESR
- Platelet count
- Peripheral smear for morphology
- BT, CT
- Urine routine and microscopy
- PCV
- MR
- LFT
- Dengue IgM Micro ELISA
- aPTT

- PT, INR
- D-dimer
- FDP
- Chest X-ray
- USG Abdomen
- Lipid profile

Sr. No.	IP No.	Age (Years)	Sex	Symptoms													Signs							Temperature (°F)							Investigations													Number of SDP transfused	Steroids	Dengue grade
				Fever	Myalgia	Arthralgia	Vomiting	Headache	Abdominal pain	Backache	Diarrhoea	Malena	Altered sensorium	Seizure	Petechiae/Echymosis	Pleural effusion	Hepatomegaly	Bradycardia	Ascites	Subconjunctival haemorrhage	Icterus	Splenomegaly	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	TC	HCT	Platelet Count (/cmm)							aPTT	SGOT>>SGPT	IgM	IgG				
																																Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7					Day 1			
118	318861	57	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0	98.0	98.0	7400	L	96000			145000				32	N	+	+	0	N	CDF				
119	315595	25	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	99.6	98.6	98.0	98.0			5200	I	46000	66000	87000	110000				32	N	+	+	0	N	CDF				
120	312744	24	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.6	98.6	98.0	98.0	98.0	98.0	3200	L	6000	66000	98000	269000				49	Y	+	+	2	Y	DHF4					
121	306228	58	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.2	98.2	98.2	98.2			6450	NM	85000	97000	1E+05				30	N	+	+	0	N	CDF					
122	323992	23	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	98.6	98.2	98.0	98.0			8800	L	39000	57000	78000	95000	123000				32	N	+	+	0	N	CDF			
123	322256	22	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.6	98.2	98.6	98.2			10400	I	77000	1E+05				32	N	+	+	0	N	CDF						
124	325569	20	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.2	98.6	98.0	98.0				6450	L	45000	78000	1E+05				30	N	+	+	0	N	CDF					
125	319756	59	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.4	98.6	98.6					4900	L	9000	56000	1E+05				44.6	Y	+	+	1	N	DHF2					
126	323834	16	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.6	98.6	98.6	99.0	98.0	98.0			9800	I	98000	1E+05				30	N	+	+	0	N	CDF					
127	322046	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0				4500	L	1E+05	2E+05				32	N	+	+	0	N	CDF						
128	324072	62	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0				5800	L	1E+05	1E+05				30	N	+	+	0	N	CDF						
129	305016	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0	98.0	98.0	4400	I	48000	96000	1E+05	166000	198000	3E+05				47.2	Y	+	+	0	N	DHF3		
130	302132	25	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	100.0	102.0	99.0	98.6			6700	NM	89000	94000	1E+05				32	N	+	+	0	N	CDF					
131	324875	58	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	100.0	101.0	99.6	99.9	98.0			8900	NM	2E+05	2E+05				30	N	+	+	0	N	CDF					
132	302407	24	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.6	98.2	98.0	99.0	98.0			7800	L	97000	1E+05				32	N	+	+	0	N	CDF						
133	318857	22	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.2	99.0	99.0	99.2	99.6			9600	L	44000	78000	92000	110000	165000				44	Y	+	+	0	N	DHF2			
134	313579	59	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	98.2	98.2	98.6	99.0			9800	NM	25000	45000	78000	165000				30	N	+	+	0	N	CDF				
135	319657	25	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.2	98.6	98.2	99.0				4500	L	1E+05	2E+05				32	N	+	+	0	N	CDF						
136	308387	56	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	99.0	99.0					3200	I	18000	59000	2E+05				49.2	Y	+	+	1	N	DHF3					
137	325168	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.2	98.6				6800	L	33000	59000	77000	142000				29	N	+	+	0	N	CDF				
138	326100	24	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	102.0	101.0	100.6	99.0	98.6			6500	NM	5000	46000	78000	100400	185000				48.2	Y	+	+	1	N	DHF2			
139	326273	57	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	99.0	98.6	98.2	98.6			7200	I	85000	1E+05				29	N	+	+	0	N	CDF						
140	326091	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.2			8500	L	2E+05	2E+05				29	N	+	+	0	N	CDF						
141	325633	69	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	101.0	102.0	99.9	99.0			2600	NM	42000	66000	88000	128000	152000				41	Y	+	+	0	N	DHF3			
142	303897	22	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	98.0	98.0	98.0	98.0	98.0			9300	L	49000	78000	1E+05				29	N	+	+	0	N	CDF				
143	311244	25	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	101.0	100.0	99.0	99.9	98.6	98.0			6900	NM	12000	36000	69000	100000				48.2	Y	+	+	1	N	DHF3			
144	320760	68	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.2	99.0	98.6	98.2	98.6	98.2			7600	L	96000	1E+05				29	N	+	+	0	N	DHF2					
145	303893	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	98.6	98.4	99.0				4900	I	2E+05		256000					30	N	+	+	0	N	CDF				
146	311926	25	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0	98.0			4200	L	1E+05	2E+05				29	N	+	+	0	N	CDF					
147	323885	67	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	99.0	98.6	98.2	98.0			5900	NM	44000		88000	132000				29	N	+	+	0	N	CDF				
148	305759	18	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	100.0	101.0	99.0	98.6	98.6			3400	L	8800	36000	96000	112000	158000	2E+05				47.2	Y	+	+	1	N	DHF3	
149	306369	59	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0			6900	I	79000	97000	1E+05				30	N	+	+	0	N	CDF					
150	312758	17	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	102.0	100.0	98.2				3900	NM	15000	64000	1E+05	169000				44	Y	+	+	1	N	DHF2				
151	305126	16	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	98.0	98.0	98.0	98.0	98.0			9800	L	2E+05		166000					30	N	+	+	0	N	CDF			
152	316820	66	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0	98.6			10200	I	33000	49000	97000	144000				29	N	+	+	0	N	CDF			
153	320772	24	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0	98.0			10800	L	78000	95000	1E+05				28	N	+	+	0	N	CDF				
154	320365	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0			3200	NM	9600	32000	65000	88000	110000				45	Y	+	+	1	N	DHF3			
156	320955	22	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	98.6	98.2	100.0	99.6	99.0			8800	I	2E+05							29	N	+	+	0	N	CDF			
157	320393	16	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	99.0	99.6	98.0	98.0			9900	L	42000		69000		132000					30	N	+	+	0	N	CDF		
158	316845	25	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0					3300	L	50000	1E+05	2E+05							39	Y	+	+	0	N	DHF3		
159	316821	18	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	99.0	98.6	98.6				3600	NM	19000	88000	1E+05	196000				42	Y	+	+	1	N	DHF3				
160	323900	23	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0	98.0			7500	L	95000	2E+05						28	N	+	+	0	N	CDF			
161	315978	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	99.0	99.0					5200	L	9200	64000	1E+05				46.6	Y	+	+	1	Y	DHF2					

Sr. No.	IP No.	Age (Years)	Sex	Symptoms										Signs							Temperature (°F)							Investigations														Number of SDP transfused	Steroids	Dengue grade								
				Fever	Myalgia	Arthralgia	Vomiting	Headache	Abdominal pain	Backache	Diarrhoea	Malena	Altered sensorium	Seizure	Petechiae/Echymosis	Pleural effusion	Hepatomegaly	Bradycardia	Ascites	Subconjunctival haemorrhage	Icterus	Splenomegaly	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	TC	HCT	Platelet Count (/cmm)							aPTT	SGOT>>SGPT	IgM				IgM and IgG							
																																Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7								Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
178	319537	25	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	99.0	98.6	98.0	98.0			9600	L	2E+05								30	N	+	0	N	CDF										
179	321578	22	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	98.2	98.6	98.6				7400	NM	6000	1E+05	2E+05							30	N	+	1	N	CDF									
180	318857	25	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	102.0	101.0	100.0	98.0	98.0			8200	L	45000	69000	79000	140000							32	N	+	0	N	CDF								
181	320784	16	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	98.0	98.0	98.0	99.0			6900	NM	2E+05	2E+05									29	N	+	0	N	CDF								
182	326398	18	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	102.0	100.0	100.0	100.0	98.6	98.0	98.0	4900	L	92000			133000								28	N	+	0	N	CDF							
183	305016	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.6	98.6	99.0					3600	I	8200	64000	1E+05									39.6	Y	+	1	Y	DHF2							
184	323497	36	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0			9700	L	25000	52000	69000	120000									30	N	+	0	N	CDF						
185	323888	37	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	101.6	102.0	101.3	99.0	98.6	98.6	98.0	3900	L	16000	69000	65000	122000	136000									46.3	Y	+	+	1	N	DHF4				
186	322044	44	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	98.6	98.2	98.0	98.0	98.0			10200	NM	66000	89000	1E+05											29	N	+	0	N	CDF				
187	322186	45	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0			10800	L	2E+05	2E+05													28	N	+	0	N	CDF				
18	323106	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	102.0	101.0	100.0	99.0	98.0	98.0			10400	L	9000	21000	36000	147000											30	N	+	+	1	N	CDF		
189	320336	24	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0					2400	NM	18000	40000	1E+05													46.8	Y	+	1	N	DHF2			
190	303893	20	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.2	99.6	99.2	98.6	98.6	98.0			9400	I	49000	78000		125000											32	N	+	0	N	CDF			
191	316085	22	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	100.0	99.0	98.6	98.0	98.0			8700	L	33000	52000	75000	92000	147000											32	N	+	0	N	CDF			
192	311244	21	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.2	99.0	96.0	98.6	98.0			6900	L	76000	1E+05		165000	250000											44	Y	+	0	N	DHF3		
193	315595	23	M	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.6	98.2	98.0	98.6			9900	L	2E+05																30	N	+	0	N	CDF		
194	323350	24	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	101.0	102.0	100.0	99.0	99.2	98.0			6900	L	96000	1E+05															32	N	+	0	N	CDF	
195	320377	25	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0	98.0			5800	NM	1E+05	2E+05															30	N	+	0	N	CDF	
196	322119	17	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0	98.0	98.0			8800	L	8900	32000	69000	88000	1E+06													32	N	+	1	N	CDF
197	319874	23	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	101.0	100.0	99.0	98.6				3900	I	82000	94000	2E+05														40.2	Y	+	0	N	DHF2		
198	319938	22	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0				6600	L	9400	88000	1E+05	163000														44	Y	+	1	N	DHF2	
199	324040	24	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	99.0	98.0	98.0					9600	L	57000	1E+05																32	N	+	0	N	CDF	
200	323900	16	F	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	98.0	98.0	98.0	98.0				8900	L	1E+05	2E+05																	30	N	+	0	N	CDF

ANNEXURE III - KEY TO MASTER CHART

-	-	Absent
+	-	Present
⁰ F	-	Degree Fahrenheit
aPTT	-	Activated partial thromboplastin time
CDF	-	Classical dengue fever
cmm	-	Cubic centimeter
DHF	-	Dengue haemorrhagic fever
F	-	Female
HCT	-	Hematocrit
I	-	Increased
IgG	-	Immunoglobulin G
IgM	-	Immunoglobulin M
IP. No.	-	In patient number
L	-	Low
M	-	Male
N	-	No
NM	-	Normal
SDP	-	Single donor platelet
SGOT	-	Serum glutamic oxaloacetic transaminase
SGPT	-	Serum glutamic puruvic transaminase
Sr. No.	-	Serial number
TC	-	Total count
Y	-	Yes