"ONE YEAR CROSS SECTIONAL STUDY OF OCULAR MANIFESTATIONS IN DENGUE FEVER PATIENTS ADMITTED IN TERTIARY CARE HOSPITAL BELGAUM"

REGISTRATION NO. : BK 0111005

Dissertation

Submitted to the

KLE UNIVERSITY BELGAUM, Karnataka

In partial fulfillment of the requirements

For the degree of

MASTER OF SURGERY (M.S.)

IN

OPHTHALMOLOGY

DEPARTMENT OF OPHTHALMOLOGY,

JAWAHARLAL NEHRU MEDICAL COLLEGE,

BELGAUM - 590010.

APRIL- 2013

KLE UNIVERSITY BELGAUM,

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Endorsement by the HOD, Principal/Head of the Institution

This is to certify that the dissertation entitled **"ONE YEAR CROSS SECTIONAL STUDY OF OCULAR MANIFESTATIONS IN DENGUE FEVER PATIENTS ADMITTED IN TERTIARY CARE HOSPITAL BELGAUM"** is a bonafide research work done by **REGISTRATION NO. : BK 0111005**.

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ABBREVIATIONS

Ae	-	Aedes
aPTT	-	Activated partial thromboplastin time
C3a, C5a	-	Complement factor
CDF	-	Classic dengue fever
CF	-	Complement fixation
cmm	-	Cubic millimeter
c/o	-	Complaints of
DEN	-	Dengue serotype
DF	-	Dengue fever
DHF	-	Dengue haemorrhagic fever
DSS	-	Dengue shock syndrome
DIC	-	Disseminated intravascular coagulation
ELISA	-	Enzyme linked immuno sorbent assay
F	-	Female
FFP	-	Fresh frozen plasma
Hb	-	Haemoglobin
h/o	-	History of
Hct	-	Hematocrit
HI	-	Haemagglutination inhibition
IL’s	-	Interleukins
IgG	-	Immunoglobulin G
IgM	-	Immunoglobulin M
IP. No.	-	In patient number
L	-	Left eye

M	-	Male
MacELISA	-	IgM antibody capture ELISA
MH	-	Macular haemorrhage
N	-	Normal
NS	-	Normal saline
OP. NO	-	Out patient number
PAF	-	Platelet activating factor
PCV	-	Packed cell volume
PCR	-	Polymerase chain reaction
PL	-	Perception of light
PT	-	Prothrombin time
R	-	Right eye
RNA	-	Ribose nucleic acid
RL	-	Ringer’s lactate
SDP	-	Single donor platelet
Sr. No.	-	Serial number
T	-	Test
TC	-	Total count
TNF	-	Tumor necrosis factor
USG	-	Ultrasonography
VHF	-	Viral hemorrhagic fever
WHO	-	World Health Organisiation

ABSTRACT

TITLE: "ONE YEAR CROSS SECTIONAL STUDY OF OCULAR MANIFESTATIONS IN DENGUE FEVER PATIENTS ADMITTED IN TERTIARY CARE HOSPITAL BELGAUM"

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. Ocular manifestations in dengue fever are less studied. The objective of present study was to estimate the prevalence of ocular manifestations in a group of patients with dengue fever.

Methods

The present cross sectional study was conducted in Department of ophthalmology at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during October 2011 to September 2012 on 100 patients with dengue fever proved by micro ELISA test. Detailed ocular examination in all patients was carried out, including visual acuity, anterior segment examination with torch light while examining bed side, Slit lamp bio microscopy in ambulatory patients examined in OPD, and fundus examination with direct and/or indirect ophthalmoscope. Ppercentage of the total patients having ocular manifestation of DF was calculated and P value is calculated to study association of platelet count with the ocular complications.

Result

One hundred patients hospitalized with a diagnosis of dengue fever or DHF were included in the study. The patients ranged in age from 6 to 59 years with a mean of 22.32 years. All patients presented with a history of fever ranging from

37.8^0 C to 40.3^0 C. Fever was present in all 100 (100%) patients. Other common presenting symptoms were chills (84%), headache (52%), vomiting (50%), nausea (20%), giddiness (7%), backache (40%), myalgia (49%). The 60 % of the patients were not having any ocular manifestation. Only 40 % patient had ocular symptoms and sign. Ocular bleeding manifestations were present in 40 (40%) patients. Subconjunctival haemorrhage was the commonest haemorrhagic manifestation & was found in 37 (37%) patients, followed by superficial retinal haemorrhage in 2 (2%) patients and macular haemorrhage in 1(1%) patient. Marked thrombocytopenia (platelet count less than 50,000/µL) was present in 87.5 % of patients.35 out of 73 patients who had thrombocytopenia had ocular haemorrhage, and the association was statistically significant ($p<0.026$).

Conclusion

The study concluded that dengue is a significant cause of ocular disease with 40(40%) patients having ocular manifestations. The commonest ocular lesions observed were the anterior segment manifestations such as, subconjunctival petechial haehmorrhage and diffuse haemorrhage. The less common ocular manifestations seen were the posterior segment manifestations such as superficial retinal haemorrhage and macular haemorrhage. The prevalence of ocular manifestations correlated significantly with Dengue WHO clinical stage 2. We found close correlation with ocular manifestations of dengue and thrombocytopenia (platelet count <50,000cells/µl) which was statistically significant. The study conducted by our microbiology department on dengue patients, showed that majority have serotype 1 and 3 as causative virus by PCR method. But to confirm this finding another larger sample size study required

Key words : Dengue Fever; ocular manifestation; platelet count.

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INTRODUCTION

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.[1]

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.[2] Among children of South East Asian Region Countries, these infections including dengue fever (DF), dengue haemorrhagic fever are self limiting. This mosquito born viral diseases caused by the bite of female anopheles Aedes aegypti leading to Dengue haemmohragic fever (DHF) and dengue shock syndrome (DSS) are the leading causes of hospitalization and death. It preceded by diarrheal 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.[3]

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 manifestations of dengue infection varies from asymptomatic to severe life threatening illness in the form of DHF/DSS. Dengue

hemorrhagic 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 (< 20^0C) 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.[5] 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.[5] 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.

Ocular manifestation of DF have received little attention in published literature. There are isolated reports of DF who primarily presented with visual impairment due to posterior segment involvement. Other manifestation are in form of retinal haemorrhages and retinopathy. DF and DHF can cause ophthalmic symptoms that were not previously well-described in the medical literature. Blurring of vision typically coincides with the nadir of thrombocytopenia and occurs ≈1 week after onset of fever.[6] Clinical features include retinal edema, blot haemorrhages, and vasculitis. Less common features include exudative retinal detachment, cotton wool spots, and anterior uveitis. Prognosis is generally good as the disease is often selflimiting, resolving spontaneously even without treatment. However, patients may experience mild relative central scotoma that may persist for months. The use of steroids in treating this inflammatory eye condition is controversial. A randomized controlled trial is under way to evaluate the effect of systemic steroids on dengue retinopathy; results will be reported in due course.[6]

Dengue is now widely prevalent in the areas in and around Belgaum and no studies have been documented ophthalmic complications in dengue patients in the present settings. Most studies on the prevalence of ocular manifestation of dengue fever have been carried out in industrialized countries; nevertheless, more than 90% of all the patients infected with dengue fever live in poor and middle income countries. Dengue fever is considered to be rarely associated with ocular manifestations$_6$. The published literature includes reports of patients presenting primarily with moderate to marked visual impairment due to various posterior segment alteration. This study need to be undertaken for documenting the spectrum of ocular manifestation in dengue fever.

OBJECTIVES

To estimate the prevalence of ocular manifestations in a group of patients with dengue fever.

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 arthraligia. 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 Queens land, 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 land 2) and 1950's (type 3 and 4).

In 1954, Filipino pediatricians 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 infections[5]

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 Queens land (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 India[6]

Dengue fever is endemic in many parts of India except the Himalayan and other mountainous regions where conditions are not conductive 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 pea 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

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 heterologus 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]

There 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 homologus 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 coagulopahty, 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.

Ophthalmologic manifestations [6]

Ocular manifestations in DF have received little attention in published literature . There are isolated reports of DF who primarily presented with visual impairment due to posterior segment involvement.

In the published literature significant number of patients presented with subconjunctival haemorrhage. The majority of patients had multiple, dot-like petechial haemorrhages. This pattern of subconjunctival haemorrhage has been reported to be occasionally associated with systemic conditions like meningococcal septicaemia, subacute bacterial endocarditis, measles, and sandfly fever, but not yet with dengue fever.[6]

This characteristic type of subconjunctival haemorrhage in dengue infection should be an important diagnostic consideration when conjunctival petechial haemorrhages are seen in association with fever, especially in patients living in endemic areas or in travellers who have recently visited a tropical country.

Other manifestation are in form of dilatation and tortusity of vessels, cotton wool spots, superficial retinal haemorrhages, hard exudates. The macula, however was spared in patients of published literature. This is in contrast to other reports, where patients presented with sever visual impairment due to choroidal effusion, optic neuritis, exudative maculopathy, or retinal thickening in the macula.

DF and DHF can cause ophthalmic symptoms that were not previously well-described in the medical literature.[7]

The difference in severity and type presentation may be due to variation in virulence and serotype of the infecting virus.

The pathophysiologic mechanisms involved in dengue infection are complex and not completely understood. The various manifestations of the disease are believed to be a result of either direct viral invasion or a complex immune-mediated process. It has been speculated that viral invasion of endothelial cells, dendritic cells, monocytes, and hepatocytes causes apoptosis and cellular dysfunction. This may be followed by the transient aberrant immune response, resulting in CD4/CD8 ratio inversion and cytokine overproduction, that has deleterious effects on these cells. In addition, overproduction of interlukin-6 triggers the formation of autoantibodies against platelets and endothelial cells, and this results in further immune mediated damages.

Subconjunctival haemorrhages, retinal haemorrhages, and exudates, which could be due to generalized increased capillary permeability, plasma leakage, and haemorrhagic diathesis associated with endothelial dysfunction ,platelet destruction, and consumptive coagulopathy.[6]

The pathogenesis of cotton wool spots may be related to occlusion of precapillary arterioles in the retinal nerve fiber layer by immune complex deposition.

Lim et al in their study suggested the possibility of specific autoantibodies being produced against retina, retinal pigment epithelium, or choroid, but precise mechanisms responsible for the various ocular alterations in dengue still remain unknown.

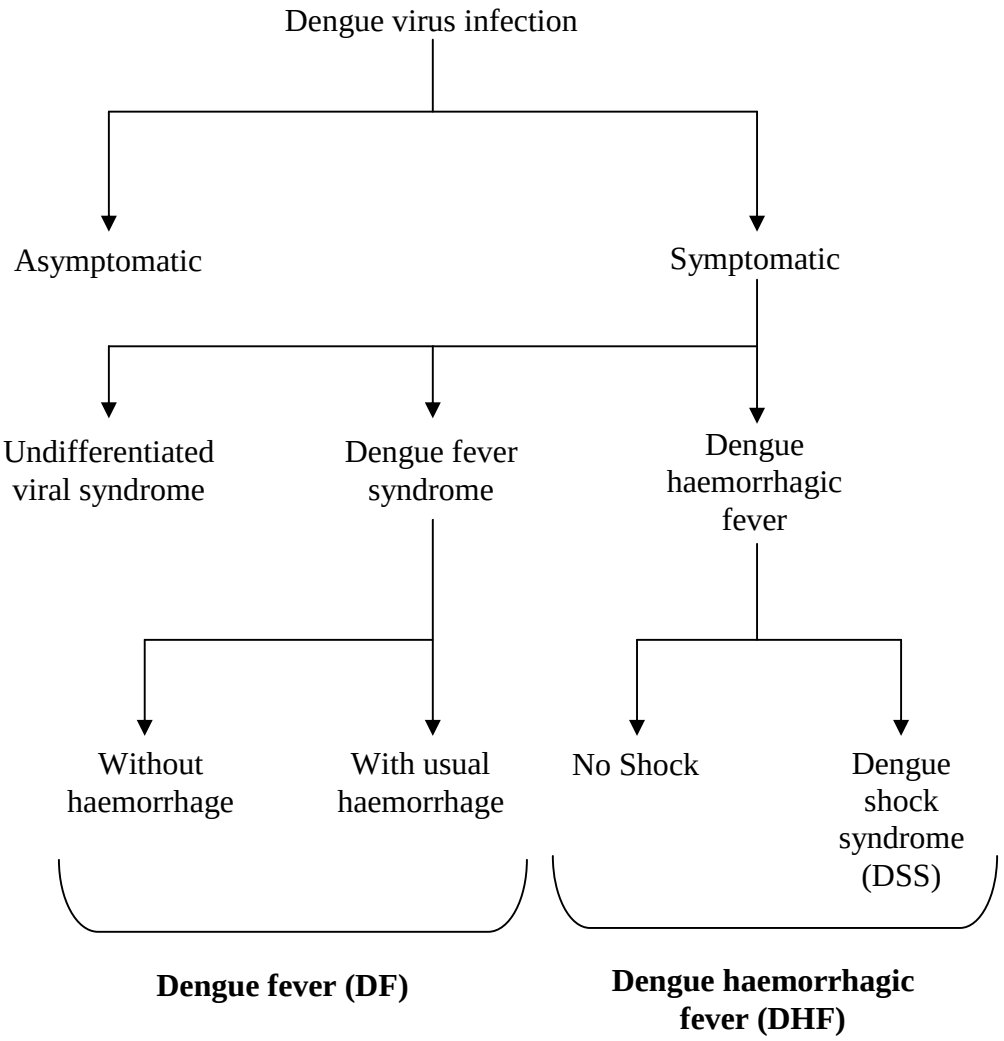
Haemorhagic manifestations of dengue fever are believed to be multifocal in origin. Gomber et al, however, reported that there was no significant association between thrombocytopenia and haemorrhagic manifestations, signifying that there may be other factors like platelet dysfunction and disseminated intravascular coagulopathy, responsible for bleeding.

Blurring of vision typically coincides with the nadir of thrombocytopenia and occurs ≈1 week after onset of fever.[5.] Less common features include exudative retinal detachment, cotton wool spots, and anterior uveitis. Prognosis is generally good as the disease is often selflimiting, According to Harutoglou et al, most of findings resolve spontaneously even without treatment. However, patients may experience mild relative central scotoma that may persist for months. The use of steroids in treating this inflammatory eye condition is controversial. A randomized controlled trial is under way to evaluate the effect of systemic steroids on dengue retinopathy.[7]

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 infection[8]



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 spleenomegaly. All patients have some degree of haemorrhagic phenomenon like positive tourniquet test, petechial spots, bruising at venepuncture site, bleeding from gums, epistaxis, hemetemesis 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 glomerulopnephritis.[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 tires 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 over hydration.

Indications of hospitalization[17]

- 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/mm^3
- Acute abdominal pain
- Evidence of plasma leakage eg. Pleural effusion, ascites.

Dengue fever

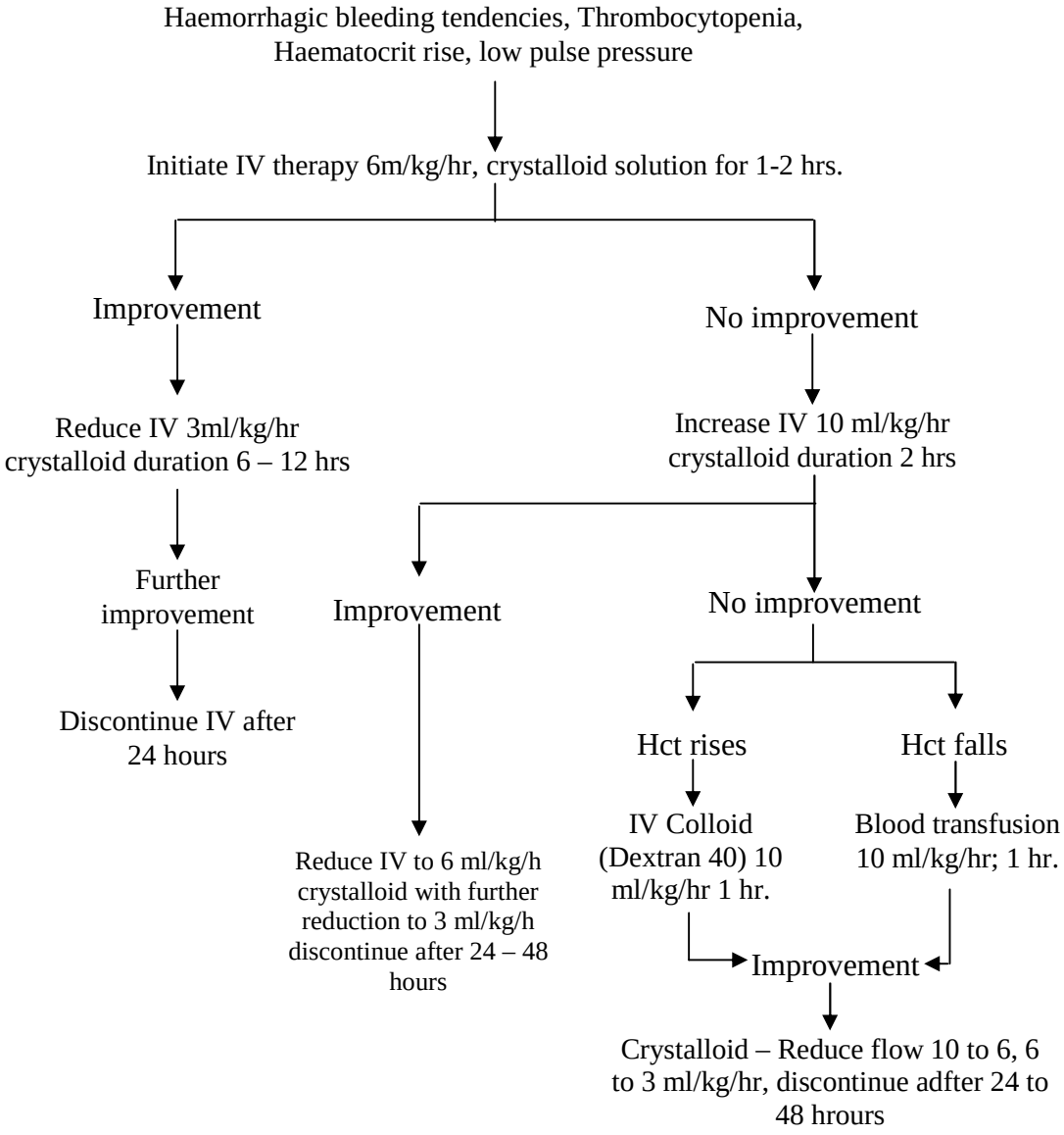
Patient with dengue fever require rest, oral fluids to compensate for losses via diarrhea or vomiting, analgesics and antipyretics preferably paracetamol. Antibiotics are not indicated in uncomplicated patients.[18]

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.

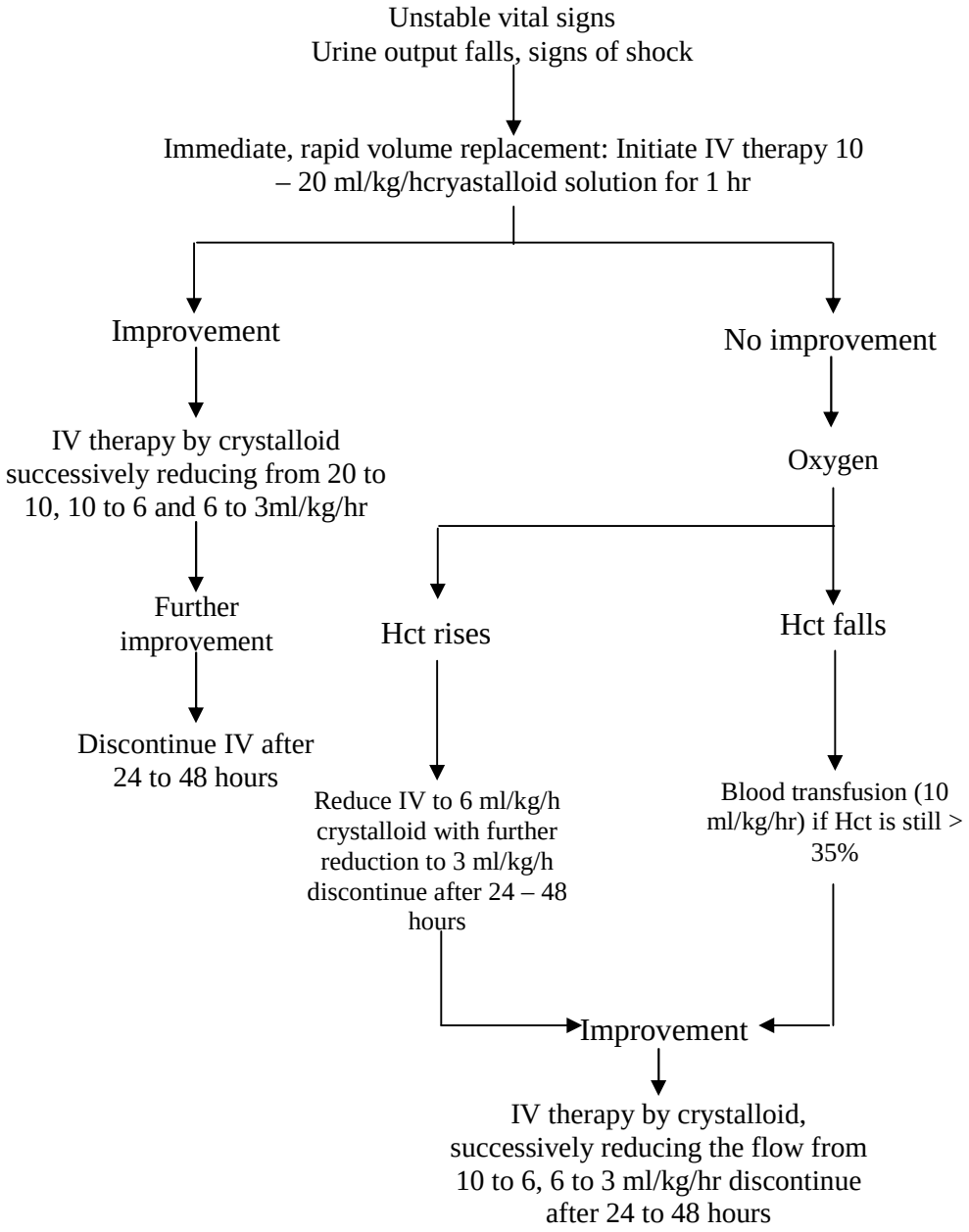
Figure 2: Flow chart of the treatment for DHF Grade – I and Grade – II[9]



DHF Grade – III and Grade – IV[19]

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 DSS[9]



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 haemorrhage. 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/mm^3 and have severe haemorrhage.[1] In children, prophylactic platelet transfusion indicated when platelet count is less than 20,000/mm^3 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 haemorrhage and outcome in these patients were not improved compared to control.

Use of steroids

There are two clinical trails in pediatric age group namely Sumarmo et al[17] which used hydrocortisone and Sampson Tassniyom et al[20] 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 immunoglobin in DSS and efficacy of heparin in DIC have not yet been documented.

Prognosis[2]

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 ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum on a patients with dengue fever during the period of October 2011 to September 2012.

Study design

One year cross-sectional study.

Study period

The present study was conducted during October 2011 to October 2012.

Method of collection of data

Source of Data

Patients admitted with diagnosis of dengue fever ,confirmed by serological tests in inpatient wards at Medical, Pediatric and Ophthalmic departments belonging to age> 6 years and of either gender at KLE'S Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum, were enrolled in this study. **.**

Sample size and sampling procedure

All the patients with dengue fever admitted KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during the study period that is 100 admissions were recorded during the study and same considered as sample size.

Selection criteria

Inclusion Criteria

- One hundred consecutive patient admitted in Medical and Paediatric wards with diagnosis of serologically positive dengue fever.

Exclusion Criteria

- Participants not willing to give consent.
- Participants with history of diabetes mellitus, hypertension , anemia or any other systemic or ophthalmic disorder

Methodology

- Diagnosis of dengue fever was based on clinical features and laboratory parameters. Serological evaluation was done by using the dengue IgM capture ELISA TEST KIT. The ophthalmic evaluation was done in Ophthalmic OPD in all the patients except for those who were too ill to be moved to the OPD in whom the evaluation was done at bed side.
- Detailed ocular examination in all patients was carried out, including visual acuity, anterior segment examination with torch light while examining bed side, Slit lamp bio microscopy in ambulatory patients examined in OPD, and fundus examination with direct and/or indirect ophthalmoscope. Visual acuity was recorded by Snellen's test chart. In few patients where visual acuity by Snellen's test chart could not be recorded due to poor general condition and hence clinically recorded bed side by finger counting. Ability to count fingers at 6 meters was considered clinically good. Visual acuity in unconscious or comatose patients, and with dementia or disoriented could not be tested.

  Anterior segment examination was performed in all conscious and cooperative patients with diffuse illumination on the Slit lamp. The pupils were dilated with Phenylephrine 5% and Tropicamide 0.8% combination eye drops. Direct and indirect ophthalmoscopy was performed in all patients. Documentation of

relevant findings of the ocular adnexa and eye were made in the form of external photography, fundus diagrams and fundus photography.

The relevant treatment for ocular complaints was instituted along with the consultation with the physician for systemic condition when required.

STATISTICAL ANALYSIS:

- Prevalence rate of ocular manifestations of DF as the percentage of the total patients examined having ocular manifestation of DF.
- P value is calculated to study association of platelate count with the ocular complication.

RESULTS

The present study was conducted in the Department of ophthalmology, KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum. on patients with dengue fever during the period of October 2011 to September 2012. One hundred patients hospitalized with a diagnosis of dengue fever or DHF were included in this study.

Table 1 Age Distribution of the dengue patients

Age group	Frequency	Percent
6-10	24	24.0
11-20	29	29.0
21-30	25	25.0
31-40	8	8.0
41-50	9	9.0
Above 50	5	5.0
Total	100	100.0

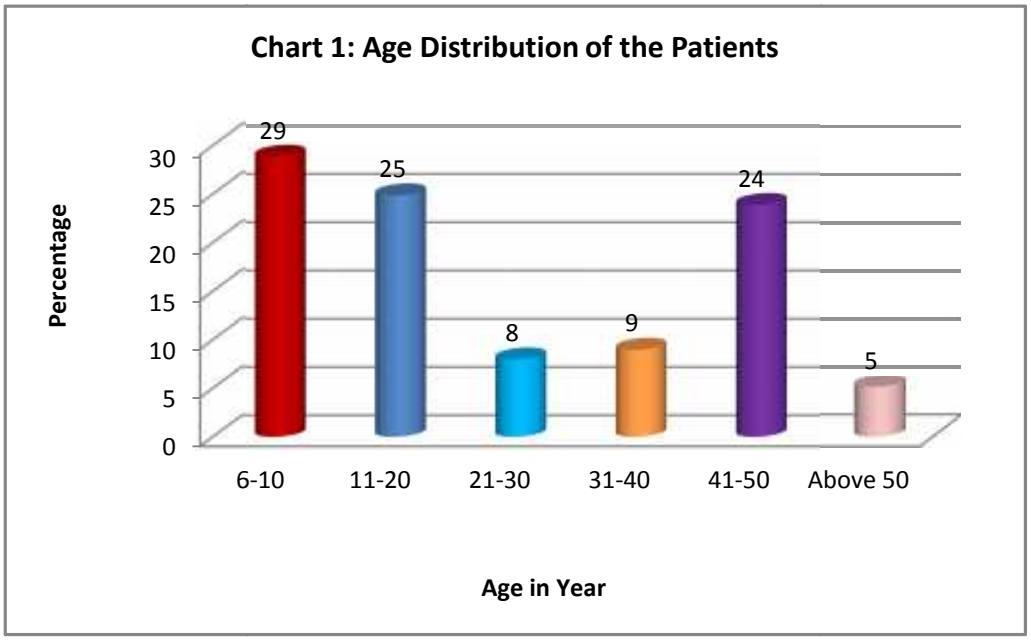
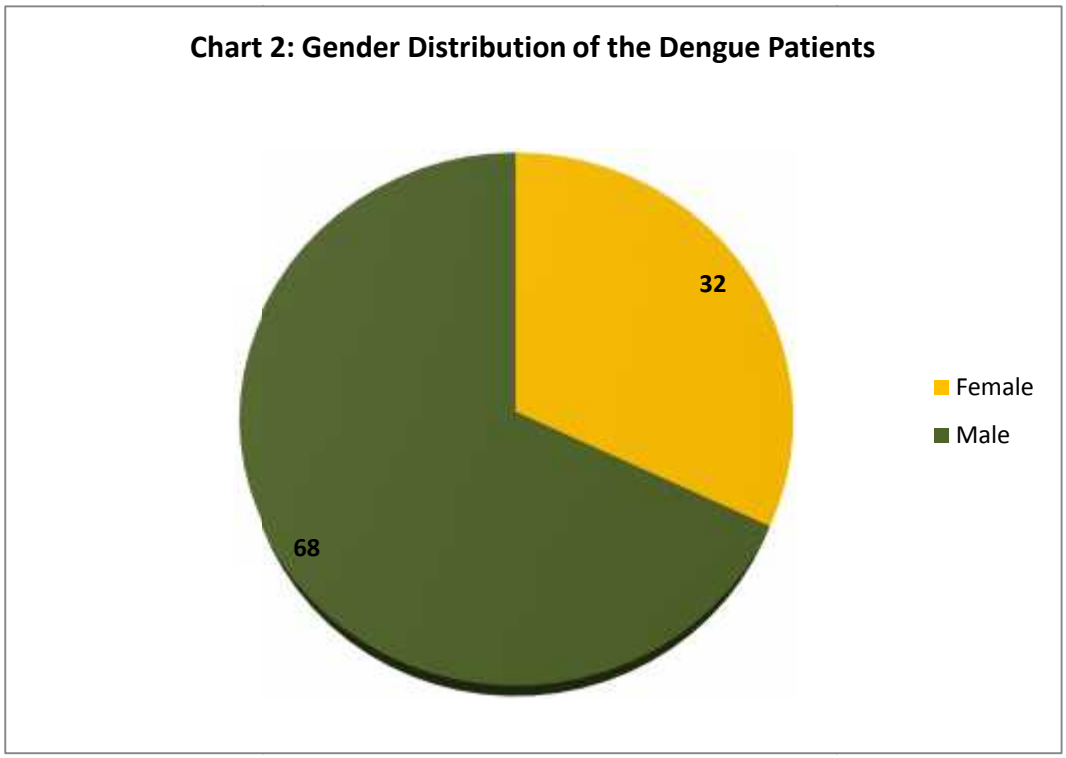


Table 2: Gender Distribution of the dengue patients

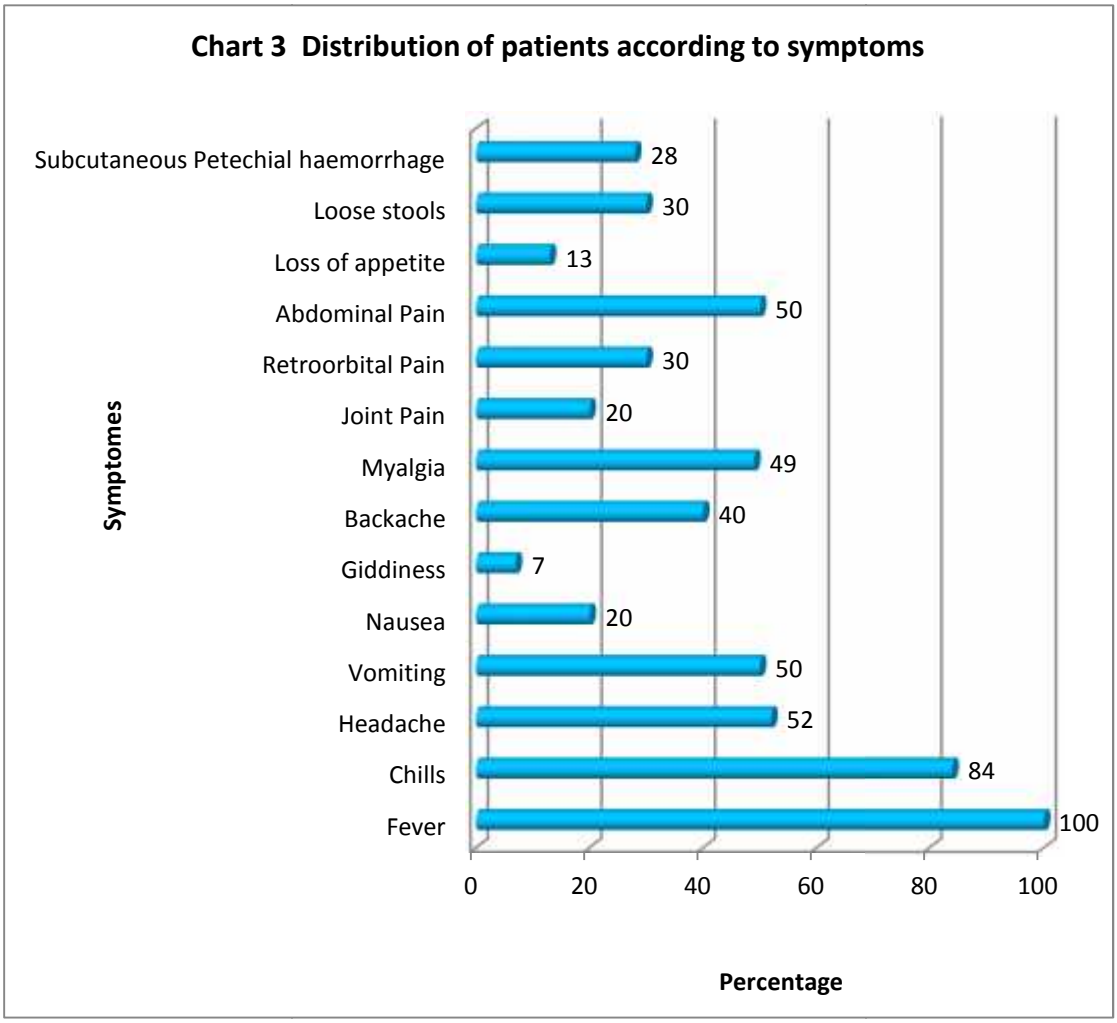
Sex	Frequency	Percent
F	32	32.0
M	68	68.0
Total	100	100.0



The patients ranged in age from 6 to 59 years with a mean of 22.32 years; 68 % were males and 32 % were females. 54 % patients were the under age of 30 rest all were above the age of 30(Table 1& 2). 70 patients lived in urban areas, 45(45%) patients were employed, & the rest were either students or housewives. The median duration of hospital stay was 5 days

Table No 3 Distribution of patients according to symptoms

Symptoms	Number	Percentage
Fever	100	100%
Chills	84	84%
Headache	52	52%
Vomiting	50	50%
Nausea	20	20%
Giddiness	7	7%
Backache	40	40%
Myalgia	49	49%
Joint Pain	20	20%
Retroorbital Pain	30	30%
Abdominal Pain	50	50%
Loss of appetite	13	13%
Loose stools	30	30%
Subcutaneous Petechial haemorrhage	28	28%



All patients presented with a history of fever ranging from 37.8^0 C to 40.3^0 C. Fever was present in all 100 (100%) patients. Other common presenting symptoms were chills (84%), headache (52%), vomiting (50%), nausea (20%), giddiness (7%), backache (40%), mayalgia (49%), joint pain (20%), retroorbital pain (30%), abdominal pain (50%), loss of appetite (13%), loose stools (30%), subcutaneous petechial haemorrhage (28%). (Table 3)

Table 4 Classic dengue fever and dengue haemorrhagic fever, grades I - IV

	No of patients					
	CDF	**DHF-I**	**DHF-II**	**DHF-III**	**DHF-IV**	**Total**
With ocular findings	1	2	35	1	1	40
Without ocular findings	36	15	9	0	0	60
Total	37	17	44	1	1	100

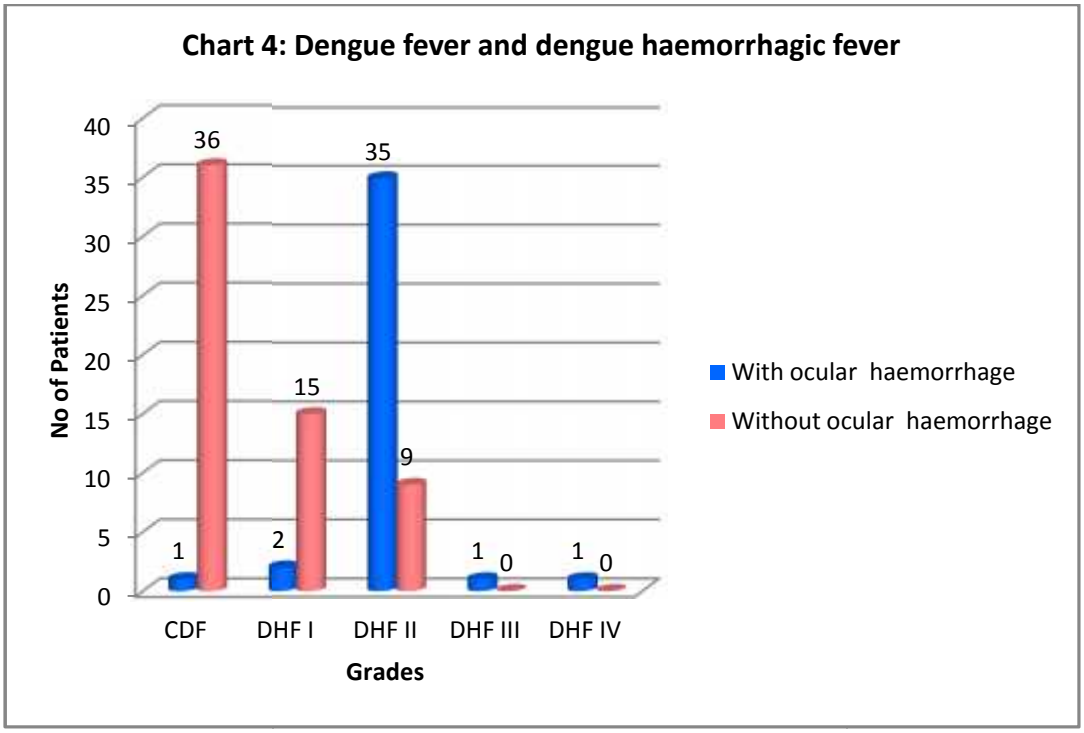


Table 4 sums up the symptoms observed at the time of admission. The 60% of the patients were not having any ocular manifestation. Only 40% patient had ocular symptoms and sign .20% patient complained of redness and watering of the eyes and only one had diminution of vision which was not subsided till the time of discharge from hospital.37 patients had classic dengue fever and remaining 63 patients had dengue hemorrhagic fever ,out of which 44 were in DHF grade ll and remaining were in other category. Out of 40 patients having ocular findings 35 (90 %) had DHF grade II.

Table 5 – Ocular findings in patients with dengue fever

Ocular finding	No of patients	% of total, n = 100
Subconjunctival haemorrhage	37	37.0
Petechial-type haemorrhage	31	31
Diffuse-type haemorrhage	6	6
Posterior segment alterations	8	8
Superficial retinal haemorrhages	2	2.0
Cotton-wool spots	0	0.0
Dilatation and tortuosity of vessels	5	5.0
Macular haemorrhage	1	1.0

Ocular bleeding manifestations were present in 40(40%) patients. Subconjunctival haemorrhage was the commonest haemorrhagic manifestation & was found in 37(37%) patients, followed by superficial retinal haemmohrage in 2 (2%) patients and macular haemmorhage in 1(1%) patients. Petechial type of subconjuctival haemmorhage was seen in 31 patients and only 6 patients out of 40 were having diffuse type of subconjuctival haemmorhage .cutaneous haemorrhage in the form of petechiae and ecchymosis seen in 14% of patients. 5(5%) patient had posterior segment alteration in form of dilatation and tortuosity of retinal vessels. Both anterior and Posterior segment manifestation is seen in only patients who had platelet counts less than 50 000/µL (table 5).

Table 6 – Laboratory findings and association with ocular haemorrhage

Laboratory Parameter	No (%) of patients		P - value
	With ocular haemorrhage	Without ocular haemorrhage	
>100 000/µL	1(2.5)	7 (11.7)	0.026
50 – 100 000/µL	4 (10.0)	15 (25.0)	
<50 000/µL	35 (87.5)	38 (63.3)	
Total	40 (100)	60 (100)	
Prothrombin time*			0.430
Normal	15 (38.5)	16 (29.7)	
Prolonged	24 (61.5)	40 (70.3)	
Total	39 (100)	56 (100)	
APTT*			0.463
Normal	4 (10.3)	10 (17.9)	
Prolonged	35 (89.7)	46 (82.1)	
Total	39 (100)	56 (100)	

* Prothrombin time and APTT were carried out in 95 patients only.

Chart 5: Laboratory findings and association with ocular haemorrhage

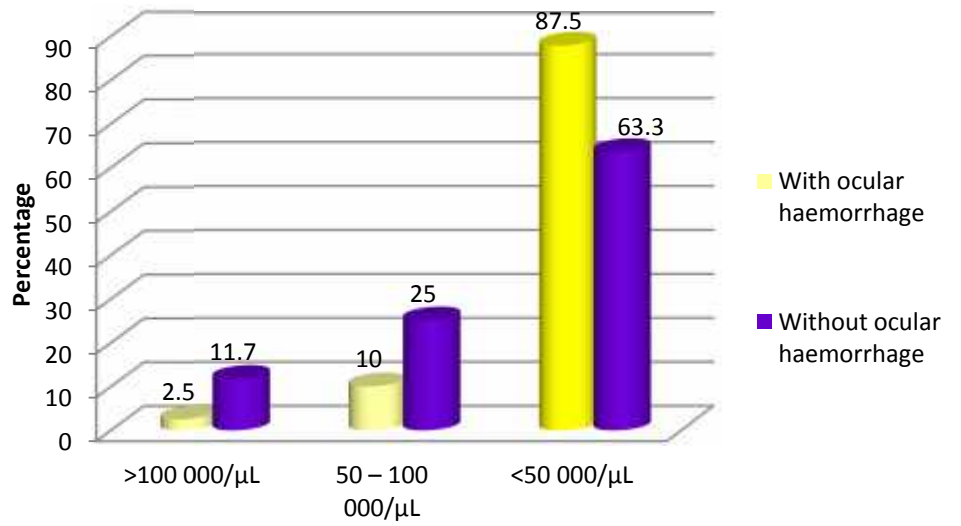
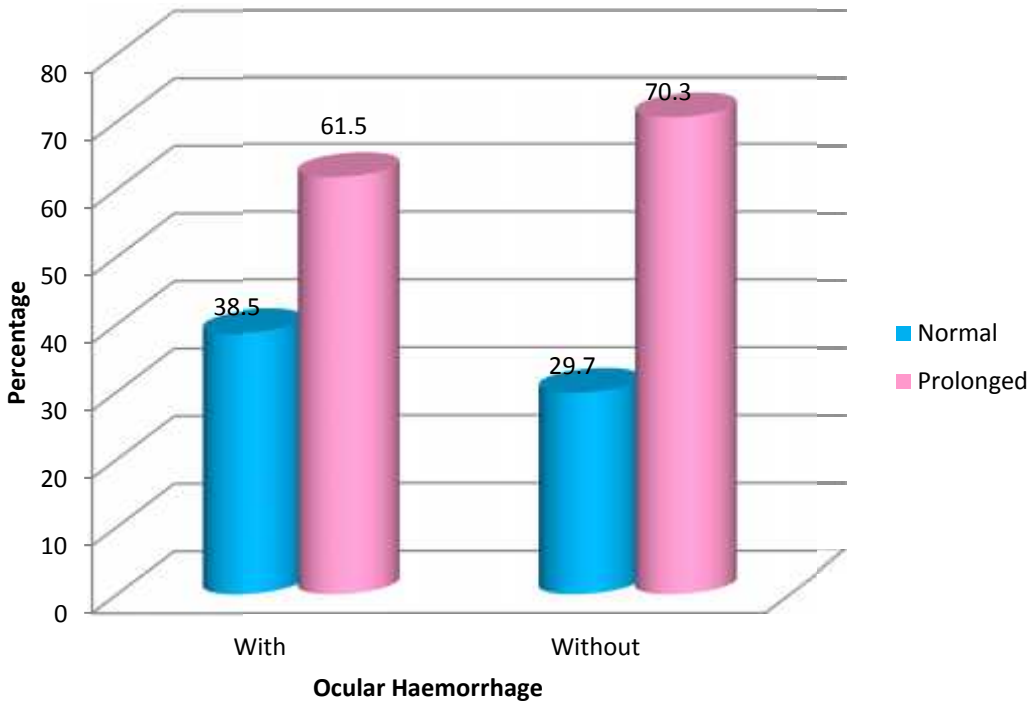
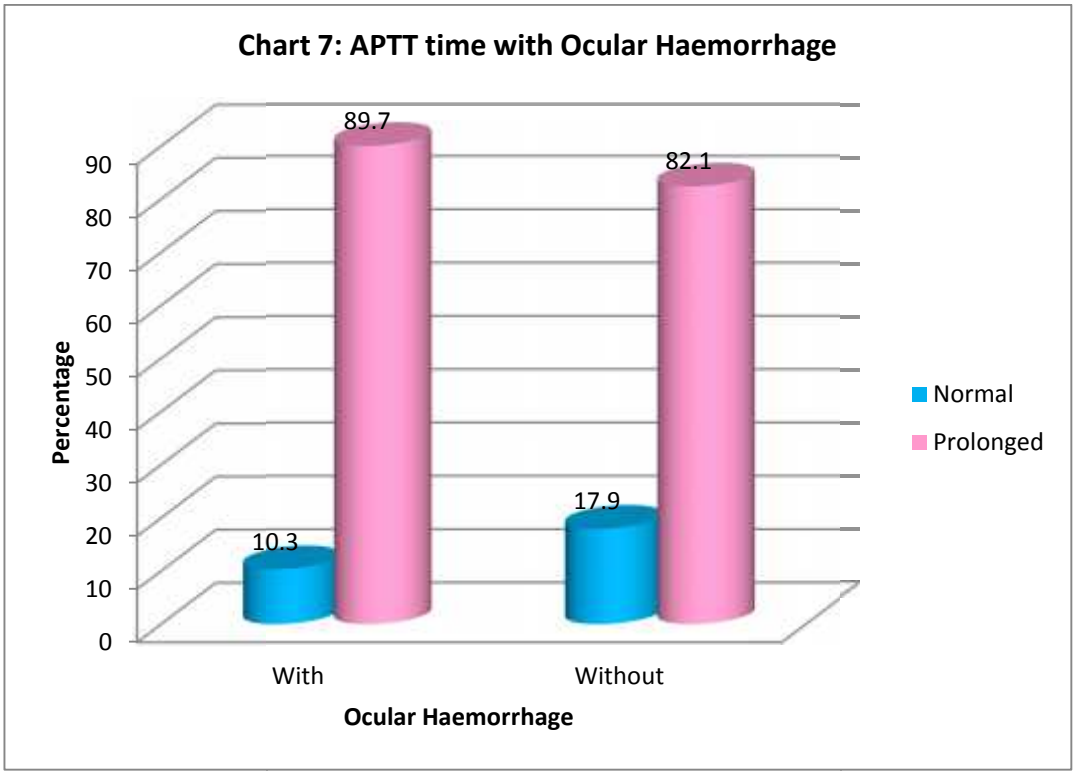


Chart 6: Prothrombin time with Ocular Haemorrhage





Of all the laboratory parameters evaluated, marked thrombocytopenia (platelet count less than 50,000/µL) was present in 73% of patients.35 out of 73 patients who had thrombocytopenia had ocular haemorrhage, and the association was statistically significant (*p<0.026*). 38 patients out of 73 who had thrombocytopenia did not had ocular findings which was also significant. Prothrombin time (PT) and partial thromboplastin time (PTT) were carried out in 95 patients. Although 89.7% of patients with ocular haemorrhage had prolonged APTT values, the association was not statistically significant (*p>0.463*). PT was prolonged in 61.5% of patients with ocular haemorrhage(*p>0.430)(Table 6).*

DISCUSSION

The incidence**,** geographic distribution, and clinical severity of epidemic and endemic dengue have increased in a great extent in the past few decades. The expansion of this flavivirus infection is linked to many reasons like explosion of population ,unhygienic conditions, resurgence of the mosquito vector Aedes aegypti, population growth in the tropics, uncontrolled urbanization and overcrowding without appropriate water management, and global spread of dengue via travel and trade.[13]

The pathophysiologic mechanisms involved in dengue infection are complex and not completely understood. The various manifestations of the diseases are believed to be a result of either direct viral invasion or a complex immune- mediated process. It has been speculated that viral invasion of endothelial cell, dendritic cells, monocytes, and hepatocytes causes apoptosis and cellular dysfunction. This may be followed by a transient aberrant immune response, resulting in CD4/CD8 ratio inversion and cytokine overproduction that has deleterious effects on these cells, in addition, overproduction of interleukin-6 trigger the formation of autoantibodies against platelets and endothelial cells, and this result in further immune mediated damages. Ocular findings in our patients primarily included subconjunctival haemorrhages, retinal haemorrhages, and exudates, which could be due to generalized increased capillary permeability, plasma leakage, and haemorrhagic diathesis associated with endothelial dysfunction, platelet destruction, and consumptive coagulopathy. The pathogenesis of cotton-wool spots may be related to occlusion of precapillary arterioles in the retinal nerve fibre layer by immune complex deposition. Lim et al in their study suggested the possibility of specific autoantibodies being produced against retina, retinal pigment epithelium, or choroid, but precise

mechanisms responsible for the various ocular alterations in dengue still remain unknown.[6]

Haemorrhagic manifestations of dengue fever are believed to be multifactorial in origin. Thrombocytopenia and abnormal coagulation profiles (PT, PTT) have been reported to have a predictive value for spontaneous systemic bleeds in dengue infection. Gomber et al, however, reported that there was no significant association between thrombocytopenia and haemorrhagic manifestations, signifying that there may be other factors, like platelet dysfunction and disseminated intravascular coagulopathy, responsible for bleeding.[24.] There are no reports in the literature of an association of ocular haemorrhage with abnormal laboratory parameters in dengue infection.

All 4 distinct serotypes of dengue virus (DEN 1-4) can cause dengue fever. Moreover, variations in the virus strains within and between the 4 serotypes influence the disease severity. Apparently, infection by serotype 2 is more severe.[25] Epidemological evidence also suggests that the risk of severe disease is most likely when infection with one serotype is followed by a secondary infection with another serotype, particularly if the secondary infection involves serotype 2. The study conducted by our microbiology department on dengue patients, showed that majority have serotype 1 and 3 as causative virus by PCR method. Kapoor et al study serotype 3 was reported to be causative agent of the epidemic when their study was conducted.[6] Since none of the earlier published reports have mentioned the serotype of causative virus, the possibility of variations in clinical presentation due to difference in the serotype of the dengue virus cannot be ruled out. Further prospective studies

with a large sample are required to document the spectrum of ocular manifestations of dengue fever, correlating these with the serotype of the causative virus.

The clinical presentation of the patients in our series was characteristic of classic dengue infection. Fever was a consistent symptom present in all patients with chills, vomiting, myalgia, headache, backache, and abdominal pain being among the other common presenting symptoms. Ocular manifestations of dengue fever have received little attention in the published literature.[27] Ocular manifestations of dengue fever have been described ranging from 10-47 % in various literature.

In our study 40 % patients had ocular manifestations while 60 % were asymptomatic. Among 40 % patients with ocular findings, the subconjunctival haemorrhage was the commonest finding seen in 37 (37%) patients, out of which majority of patients i,e 31(31%) had multiple, dot-like petechial haemorrhages in one or both eyes ,remaining 6(6%) patients had diffuse type of haemorrhage .This pattern of subconjunctival haemorrhage has been reported to be occasionally associated with systemic conditions like meningococcal septicaemia, subacute bacterial endocarditis, measles, and sandfly fever, but not yet with dengue fever. Since the majority of our patients had this characteristic type of subconjunctival haemorrhage, dengue infection should be an important diagnostic consideration when conjunctival petechial haemorrhages are seen in association with fever, especially in patients living in endemic areas or in travellers who have recently visited a tropical country.

There are isolated reports of dengue fever patients who primarily presented with visual impairment due to posterior segment involvement.

8 patients in our series had posterior segment findings, including dilatation and tortuosity of vessels as only finding in 5(5%) patients, macular haemmohrage in1(1%) patient and, isolated superficial retinal haemorrhages in 2 (2%)patients

In our series, 37(37%) patients had classic dengue fever and remaining 63 (63%) patients had dengue haemmorrhagic fever, out of which 44(44%) patients were categorised into DHF grade II and remaining were in other category. Ocular findings were present in 35 patients that means 90 % patient with DHF grade II. As compared to other studies, the difference in the severity and type of presentation in our series may be attributed to variations in the virulence and serotype of the infecting virus.[28.]

Our study revealed that 87.5 % of patients with ocular haemorrhage (subconjunctival or retinal) had marked thrombocytopenia (platelet counts less than 50,000/µL) and the association was statistically significant *($p<0.026$*).The correlation with other parameters, like prolonged PT, PTT tests were not significant.(Table 5)which is consistent with other several studies .

Ocular alterations in dengue are usually self-limiting. According to Haritoglou et al, most of the findings resolve without specific treatment, but occasionally visual recovery may be prolonged or vision may remain permanently impaired in patients with a severe maculopathy. In our study, one patient had visual impairment till the time of discharge. We are not able to comment on the recovery in this case, as we lost follow up of this patient.

Lim et al in a recent report suggested the use of periocular steroids for treatment of vision threatening maculopathy in dengue patients.[29] Their study, however, included 6 patients only, thus inferences regarding definitive management

of such patients cannot be conclusively drawn from their observations. Further studies with a larger sample size are required to evaluate the treatment options in patients presenting with visual impairment in dengue infection.

Dengue fever, and in particular life-threatening DHF, continues to be a global challenge: the pathogenesis of the disease is not completely understood, there is neither specific treatment available nor any immediate prospect of a vaccine, and the mosquito control measures in most of the hyperendemic areas are inadequate. The incidence and geographic distribution of dengue has increased dramatically in the past several years and their ocular manifestations are likely to follow this trend. Ophthalmologists should thus be aware of the various ocular manifestations of dengue, as early diagnosis and referral for appropriate supportive therapy can considerably reduce the mortality of this potentially fatal disease. Furthermore, patients of dengue fever with marked thrombocytopenia (platelet count <50,000/µL) are predisposed to spontaneous ocular haemorrhages, though majority are with no consequences, if present in macula can lead to impairment of vision.

Table 7 - Summary of literature on ocular manifestation of dengue fever

AUTHOR	YEAR	NO.&SEXOF PATIENTS	OCULAR FINDINGS
Spitznas(in German)	1978	1F	Bilateral intraretinal haemorrhages in macula.
Wen et al(in chinese)	1989	14F 10M	Macular haemorrhages,10 maculopathy,7 Macular and retinal haemorrhages, 5 Peripapillary haemorrhages, 3 Roth's spot, 2 vitreous/aqueous cells,2 Diffuse retinal oedema,1 Optic neuritis,1Ischemic optic neuropathy,1.
Haritoglou et al (in German)	2000	2	Intraretinal haemorrhages, retinal pigment epithelial lesions in the fovea, cotton-wool spots, maculopathy.
Haritoglou et al	2002	1F	Bilateral exudative maculopathy, haemorrhages in the nerve fibre layer.
Cruz Villegas et al	2003		Bilateral choroidal effusion.
Lim et al	2004	1M 5F	Intraretinal whitish lesions with localized retinal & RPE disturbance, small dot haemorrhages vascular sheathing around macula & papillomacular bundle.
Squeira et al	2004	1M	Bilateral vascular sheathing , retinal haemorrhages at the equator, and cotton-wool spots in the macula.

Kapoor et al (India)	2006	134	Subconjunctival haemorrhage, superficial retinal haemorrhages, dilatation and tortuosity of vessels, cotton wool spots, retinopathy.
Stephen C.B. Teoh et al (Singapore)	2006	50	Subconjunctival haemorrhage, Anterior uveitis, intermediate uveitis Macular haemorrhages, maculopathy, retinal haemorrhages, disc swelling, optic neuritis.

NOTE: RPE, retinal pigment epithelium

CONCLUSION

The present study is a hospital based cross sectional study representing the patients with dengue infection for ocular manifestations.

The study concluded that dengue is a significant cause of ocular disease with 40(40%) patients having ocular manifestations. The commonest ocular lesions observed were the anterior segment manifestations such as, subconjunctival petechial haemorrhage and diffuse haemorrhage. The less common ocular manifestations seen were the posterior segment manifestations such as superficial retinal haemorrhage and macular haemorrhage.

The study shows that the spectrum of the ocular lesions associated with dengue infection in this region is similar to Indian study Kapoor et al. The prevalence of ocular manifestations correlated significantly with Dengue WHO clinical stage 2. (DHF II) .The association between the prevalence of the ocular manifestations and the WHO clinical stages suggest that ocular manifestations are related to disease progression. We found close correlation with ocular manifestations of dengue and thrombocytopenia(platelet count <50,000cells/µL which was statically significant.. In microbiology department of our hospital it was found that serotype 1 and 3 are causative virus in most of dengue patients of Belgaum territory. But to confirm this finding another larger sample size study is required .

While further studies are still under way to elucidate the cause and incidence of this increasing trend of ophthalmic manifestations in this region. Including randomized controlled trials are being taken to determine the best form of treatment and management in these patients, it may be prudent for ophthalmologists and

physicians to be aware of, and to take a re-look at this lesser known but re-emerging complication of dengue fever.

SUMMARY

The present cross sectional study was conducted in Department of ophthalmology at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum during October 2011 to September 2012 on 100 patients with dengue fever proved by micro ELISA test. The objective of present study was to estimate the prevalence of ocular manifestations in a patients with dengue fever.

- Ocular examination in all patients was carried out, including visual acuity, anterior segment examination with torch light while examining bed side, Slit lamp bio microscopy in ambulatory patients examined in OPD, and fundus examination with direct and/or indirect ophthalmoscope. Percentage of the total patients having ocular manifestation of DF was calculated and P value is calculated to study association of platelet count with the ocular complications.

One hundred patients hospitalized with a diagnosis of dengue fever or DHF were included in the study. The patients ranged in age from 6 to 59years with a mean of 22.32 years. All patients presented with a history of fever ranging from 37.8^0 C to 40.3^0 C. Fever was present in all 100 (100%) patients. Other common presenting symptoms were chills (84%), headache (52%), vomiting (50%), nausea (20%), giddiness (7%), backache (40%), myalgia (49%). The 60 % of the patients were not having any ocular manifestation. Only 40% patient had ocular symptoms and signs. Ocular bleeding manifestations were present in 40(40%) patients Subconjunctival haemorrhage was the commonest haemorrhagic manifestation & was found in 37(37%) patients, followed by superficial retinal haemmohrage in 2 (2%) patients and macular haehmorrhage in 1(1%) patients. Marked thrombocytopenia (platelet count less than 50,000/µL) was present in 87.5 % of patients.35 out of 73 patients

who had thrombocytopenia had ocular haemorrhage, and the association was statistically significant ($p<0.026$).

The study concluded that dengue is a significant cause of ocular disease with 40(40%) patients having ocular manifestations. The commonest ocular lesions observed were the anterior segment manifestations such as, subconjunctival petechial haehmorrhage and diffuse haemorrhage. The less common ocular manifestations seen were the posterior segment manifestations such as superficial retinal haemorrhage and macular haemorrhage. The prevalence of ocular manifestations correlated significantly with Dengue WHO clinical stage 2. We found close correlation with ocular manifestations of dengue and thrombocytopenia (platelet count <50,000cells/µl) which was statistically significant. The study conducted by our microbiology department on dengue patients, showed that majority have serotype 1 and 3 as causative virus by PCR method. But to confirm this finding another larger sample size study is required.

BIBLIOGRAPHY

1. Pande JN, Kabra SK. Dengue hemorrhagic fever and shock syndrome. Natl Med J India. 1996; 9(6): 256-8.
2. Singh M. Medical emergencies in children. 4th Ed. New Delhi: Sagar Publications; 2004.
3. Halstead SB. Dengue hemorrhagic fever. A pubic health problem and a field for research. Bull World Health Organ 1980; 58(1):1-21.
4. Cohen SN, Halstead SB. Shock associated with dengue infection. J Paediatr 1966; 68(3): 448-55.
5. Halstead SB. The XXth century dengue pandemic: need for surveillance and res. World Health Stat Q 1992; 45 (2-3): 292-8.
6. Harpreet K Kapoor;Saloni Bhai et al.Ocular manifestations of dengue fever in East Indian epidemic.Can J Opthalmol 2006;41:741-6.
7. Stephen C.B. Teoh et al.A re-look at ocular complications in dengue fever and dengue haemorrhagic fever. Dengue Bulletin-volume 30,2006.
8. Dengue hemorrhagic fever and the Dengue shock syndrome in India. Review Article. Nat Med J Ind 1996; 9(1): 20-3.
9. World Health Organization. Key issues in dengue vector control towards the operationalisation of a global strategy: report of consultation. (CTD/FIL(Den)/IC 96.1). Geneva: The Organization; 1995.
10. Gubler DJ. Epidemic dengue/dengue hemorrhagic fever as a public health, social and economic problem in the 21st century. Trends Microbiol. 2002;10:100–3.

11. Lim WK, Mathur R, Koh A, Yeoh R, Chee SP. Ocular manifestations of dengue fever. Ophthalmology. 2004;111:2057–64.

12. Haritoglou C, Scholz F, Bialasiewicz A, Klauss V. Ocular manifestation in dengue fever. Ophthalmologe. 2000;97:433–6.

13. Wen KH, Sheu MM, Chung CB, Wang HZ, Chen CW. The ocular fundus findings in dengue fever. Gaoxiong Yi Xue Ke Xue Za Zhi. 1989;5:24–30.

14. Deutman AF, Bos PJ. Macular bleeding in dengue fever. Klin Monatsbl Augenheilkd. 1979;175:429.

15. Spitznas M. Macular haemorrhage in dengue fever. Klin Monatsbl Augenheilkd. 1978;172:105–7.

16. Nainiwal S, Garg SP, Prakash G, Nainiwal N. Bilateral vitreous haemorrhage associated with dengue fever. Eye. 2005; 19:1012–3.

17. Haritoglou C, Dotse SD, Rudolph G, Stephan CM, Thurau SR, Klauss V. A tourist with dengue fever and visual loss. Lancet. 2002;360:1070.

18. Siqueira RC, Vitral NP, Campos WR, Orefice F, de Moraes Figueiredo LT. Ocular manifestations in dengue fever. Ocul Immunol Inflamm. 2004;12:323–7.

19. Cruz-Villegas V, Berrocal AM, Davis JL. Bilateral choroidal effusions associated with dengue fever. Retina. 2003:23:576–8.

20. Gubler DJ. Dengue and dengue hemorrhagic fever: its history and resurgence as a global public health problem. Epidemiology of dengue and dengue hemorrhagic fever. In: Gubler DJ, Kuno G, editors. Dengue and dengue hemorrhagic fever. Wallingford, United Kingdom: CAB International Press; 1997. pp. 1–22.

21. Gubler DJ. Dengue and dengue hemorrhagic fever. Clin Microbiol Rev. 1998;11:480–96.

22. Halstead SB. Epidemiology of dengue and dengue hemorrhagic fever. In: Gubler DJ and Kuno G, editors. Dengue and dengue hemorrhagic fever. Wallingford, United Kingdom: CAB International Press; 1997. pp. 23–44.

23. Kurane I, Ennis FA. Immunity and immunopatholgy in dengue virus infections. Semin Immunol. 1992;4:121.

24. Gomber S, Ramchandran VG et al. Hematological observations and diagnostic markers in dengue hemorrhagic fever-a reappraisal. Indian pediatr 2001;38: 477-81.

25. Gibbons RV, Vaughn DW. Dengue: an escalating problem.BMJ 2002;324:1563-6.

26. Spitznas M. [Macular haemorrhage in dengue fever (author's transl)]. Klin Monatsbl Augenheilkd 1979 Sep;175(3):429.

27. Wen KH, Sheu MM, Chung CB, Wang HZ, Chen CW. [The ocular fundus findings indengue fever]. Gaoxiong Yi Xue Ke Xue Za Zhi 1989 Jan;5(1):24-30.

28. Haritoglou C, Scholz F, Bialasiewicz A, Klauss V. [Ocular manifestation in dengue fever]. Ophthalmologe 2000 Jun;97(6):433-6.

29. Lim WK, Mathur R et al. Ocular manifestation of dengue fever. Opthalmology 2004:111:2057-64.

ANNEXURE – I: INFORMED CONSENT FORM

ID NO ☐ ☐ ☐

TITLE: "ONE YEAR CROSS SECTIONAL STUDY OF OCULAR MANIFESTATIONS IN DENGUE FEVER PATIENTS ADMITTED IN TERTIARY CARE HOSPITAL BELGAUM"

INVESTIGATORS: Dr. _____, Post-Graduate in M.S. Ophthalmology under the guidance of Dr. _____ Associate Professor, Department of Ophthalmology, J N Medical College, Belgaum.

Mr. /Mrs./Ms_____

you are invited to participate in our above research study.

Respected sir/ madam,

We request you to enroll yourself to participate in our study as, you are eligible for this study. Your participation in research is voluntary, your decision whether or not to participate in the study will not affect your relationship with J N Medical College.

Purpose of the Study:

The main purpose of research is

- To estimate the prevalence of ocular manifestations in a group of patients with dengue fever
- To study pattern of ocular involvement in a group of patients with dengue fever

Procedure Involved:

In this study, you will be asked to give detailed history of the disease you have and undergo necessary investigations. You will then be examined and findings will documented, and if necessary photographed.

Risks and Benefits:

As such there are no major risks involved, however some discomfort may occur during the process of examination and investigation for which all precautions will be taken. The investigator does not promise or guarantee that you will receive direct benefit being in the study.

Your participation may benefit you and others suffering from same ailment in future, by helping us learn more about the disease process and better treatment modalities. No financial incentives are promised to you for being a part of study.

Alternatives:

Your decision whether or not to participate in this study will not affect the quality of treatment you receive and if you are not willing to participate, further you may withdraw from the study at any time.

Privacy and Confidentiality:

The only people to know that you are a research subject are members of the research team. No information about you or information provided by you during the research will be disclosed to others without your written permission, except:

1. In emergency to protect your rights and welfare.
2. If required by law

Compensation:

In the event of injury related to the study, treatment will be made available through KLE's Dr Prabhakar Kore Hospital and M R C, Belgaum. There is no compensation or payment for such medical treatment by law. The doctors and the staff will provide facilities and medical attention to you.

Costs for participating in this research:

There will not be any extra cost incurred by you except for the investigations which are the part of the existing management protocol for this ailment. There is no commitment for any reimbursement or any other compensation for the participant.

Authorization to Publish Results:

The results of the research may be published or discussed in a conference, or used for teaching purpose. However the participant's identity will be kept confidential.

Questions:

If you have any questions about the research you may please contact:

1) Chief investigator, Dr. _____ P.G. Department of Ophthalmology, J N Medical College, Belgaum . Contact No: _____.
2) Guide, Dr. _____, Professor, Department of Ophthalmology, J N Medical College, Belgaum . Ph: _____.
3) Dr. _____, Principal, J N Medical College Belgaum and Chairman of Institutional Ethics Committee. Ph: _____.

Consent Statement:

I voluntarily agree for the participation as a subject of study. By signing this consent form I am not giving up any of my legal rights. I am signing the consent form after having read or been read for me in vernacular language, including the risks and the benefits and having all my questions answered.

Subject name: _____

Signature or the Left Thumb Print of Subject: _____

Witness Name: _____

Signature or the Left Thumb Print of Witness: _____

Investigators Name: _____

Signature of Investigator: _____

Date: _____

Place: _____

ANNEXURE – II: SCREENING FORM

Sl.No:

ID No ☐ ☐ ☐

IP/OP No: ☐

Name: ☐ ☐ ☐

(First Name) (Middle Name) (Surname)

Age: ☐ Years

Sex: ☐ 1 – Male; 2 – Female

Address: _____

Phone No if any;

Occupation: ☐

Religion: ☐ 1- Hindu 2 – Muslim 3 – Christian 4 – Sikh 5 Others (Specify)

Date of Examination: ☐☐ ☐☐ ☐☐☐☐

DENGUE STATUS: ☐ 1.Positive 2. Negative

Diagnosis: Ocular Diagnosis _____

Systemic Diagnosis _____

Is the patient eligible for Study? ☐ 1 – Yes 2 – No

Has informed consent been taken? ☐ 1 – Yes 2 – No

Final Result Information :

1. Ineligible ☐
2. Eligible, Refusal
3. Eligible, Participating

Doctor's Name: _____

Doctor's Signature: _____

☐☐ ☐☐ ☐☐☐☐

ANNEXURE – III: PROFORMA

Patient Name: I.D. No:-

Age: Sex:

Address:

Date of admission: Date of discharge:

Chief complaints:

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

15. Jaundice

Ocular:

1. Diminution of vision	☐	Gradual	☐	Sudden
	☐	Progressive	☐	Static
	☐	Painless	☐	Painful
	☐	(1-Distance; 2 Near; 3 Both)		

2. History of Redness/ watering / Discharge	☐	1 - Yes 2 - No
3. H/O Pain	☐	1 - Yes 2 - No
4. History of Coloured halos	☐	1 - Yes 2 - No
5. H/o wearing spectacles:	☐	1 - Yes 2 - No
Any other complaints (if present, specify):	☐	1 - Yes 2 - No

Past history

Diabetes:	☐	1-Present	2-Absent	Duration: _____months / years
Hypertension :	☐	1-Present	2-Absent	Duration: _____months / Years
Asthma	☐	1-Present	2-Absent	
Tuberculosis:	☐	1-Present	2-Absent	

Any other medical disorders: _____

Personal history:

H/o Bowel and Bladder habits: ☐ 1-Regular 2-Irregular

H/o Blood transfusion: ☐ 1-Present 2-Absent

Family History:

☐ 1- Married 2-Unmarried

☐ No of Childrens:

Health status of childrens: ☐ 1- Healthy 2- Diseased 3-Dead

If dead specify the cause: _____

General physical examination

Pallor: ☐ 1-Present 2-Absent

Icterus ☐ 1-Present 2-Absent

Edema: ☐ 1-Present 2- Absent

Lymphadenopathy ☐ 1-Present 2-Absent

Cyanosis: ☐ 1-Present 2-Absent

Clubbing: ☐ 1-Present 2-Absent

Cardiovascular system: ☐ 1 normal; 2-Abnormal; if abnormal specify: _____

Vital signs:

Pulse rate (Per minute):

Blood Pressure: ☐☐☐ / ☐☐☐ (mm of Hg)

Temperature: ☐ ^{0}C

Respiratory system: ☐ 1-Normal; 2-Abnormal; if abnormal specify: _____

Nervous System: ☐ 1-Normal; 2-Abnormal; if abnormal specify: _____

Per abdomen/Genital: ☐ 1-Normal; 2-Abnormal; if abnormal specify: _____

Examination

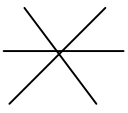
Ocular Examination:

Head Posture: ☐ (1- Errect; 2 Tilted)

Facial Symmetry: ☐ (1-Symmetrical; 2-Asymmetrical)

Visual Axes: ☐ (1-Parallel; 2-Deviated)

Visual Acuity:	Right Eye	Left Eye
Distant:		
Pinhole:		
Near:		
With spectacles:		

Extra Ocular Movements:	Right Eye	Left Eye	Binocular
			

ANTERIOR SEGMENT EXAMINATION

	(RIGHT EYE)	(LEFT EYE)
Adnexa		
Conjunctiva		
Cornea		
Sclera		
Anterior chamber		
Iris		
Pupil		
lens		

	DIRECT		**INDIRECT**	
FUNDUS:	**Right Eye**	**Left Eye**	**Right Eye**	**Left Eye**

Glow:

Media

Optic disc:

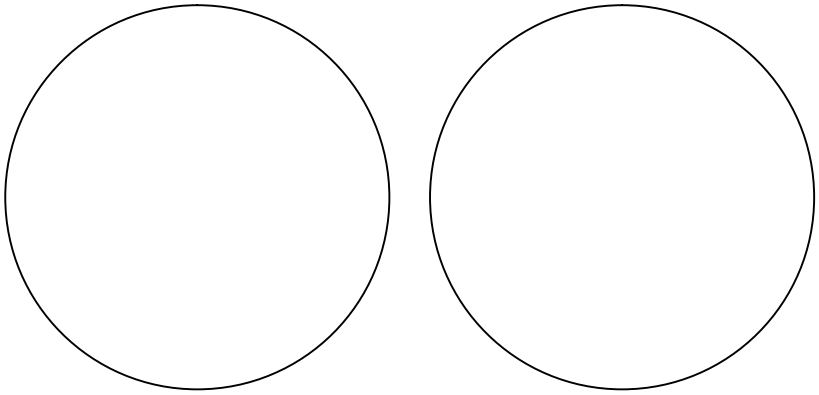
Cup: Disc ratio:

Vessels:

Background:

Foveal reflex:

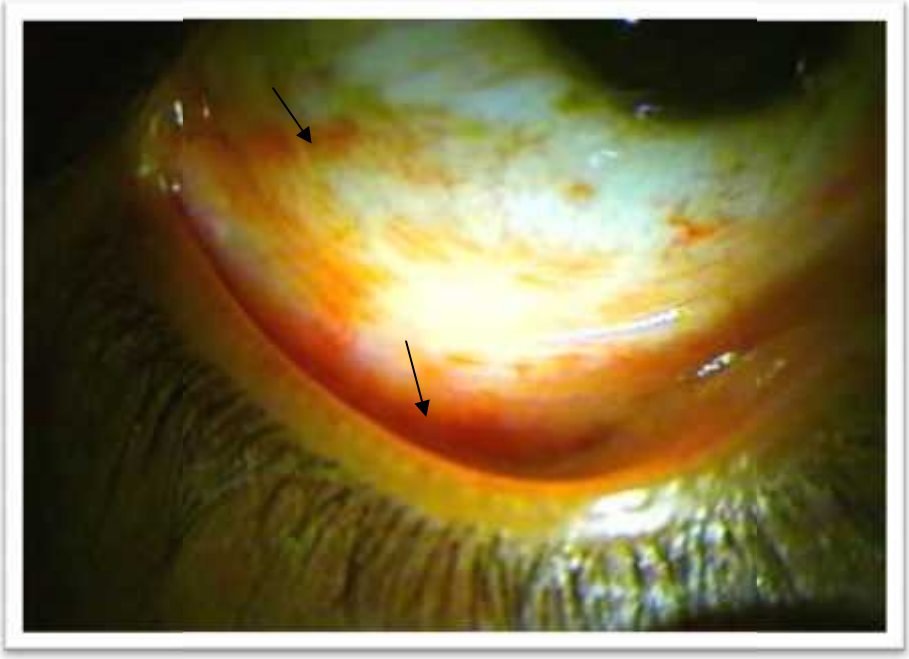
FUNDUS: **Right Eye** **Left Eye**



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
- Chest X-ray
- USG Abdomen
- Lipid profile

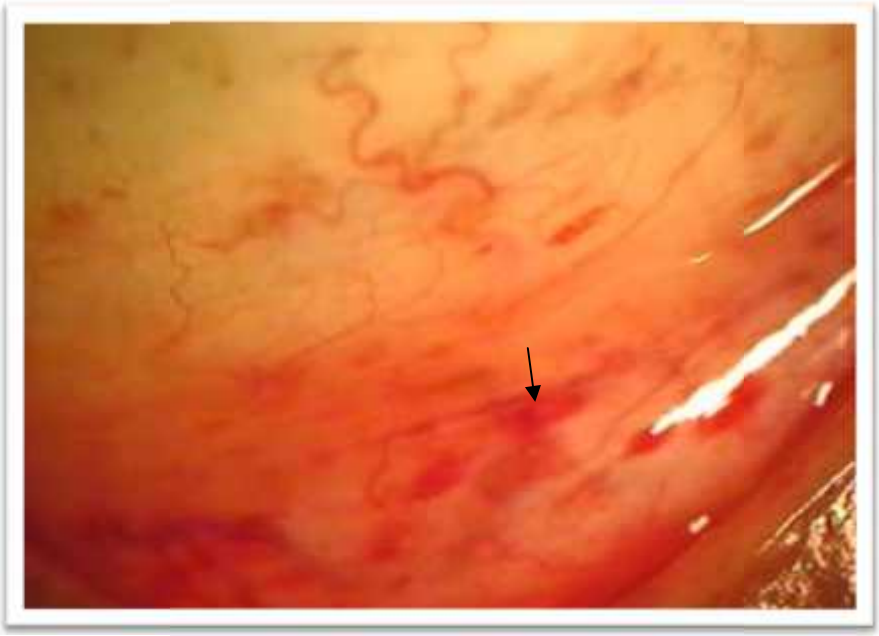
ANNEXURE – IV: PHOTOGRAPHS



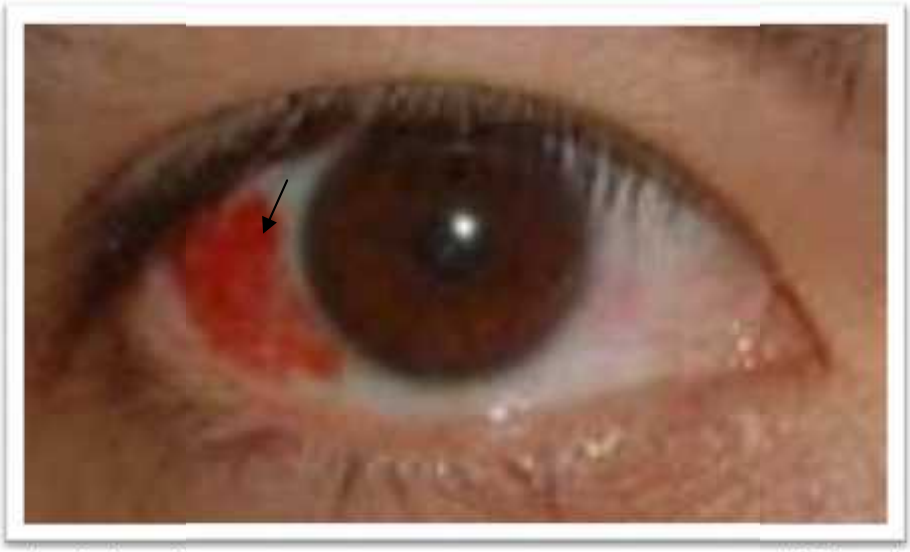
Photograph 1 : Case 2. Clinical photograph showing petechial type of Subconjunctival haemorrhage in both palpebral and bulbar conjunctiva (arrow pointing Subconjunctival haemorrhage) in a 25years male patient seropositive for dengue admitted with the diagnosis of DHF II with platelet cell count 42000cells

Photograph 2 : Case 2. Clinical photograph showing petechial type of Subconjunctival haemorrhage only in palpebral conjunctiva (arrow pointing Subconjunctival haemorrhage) in a 7 years female patient, seropositive for dengue admitted with the diagnosis of DHF II with platelet cell count 21000cells/µL

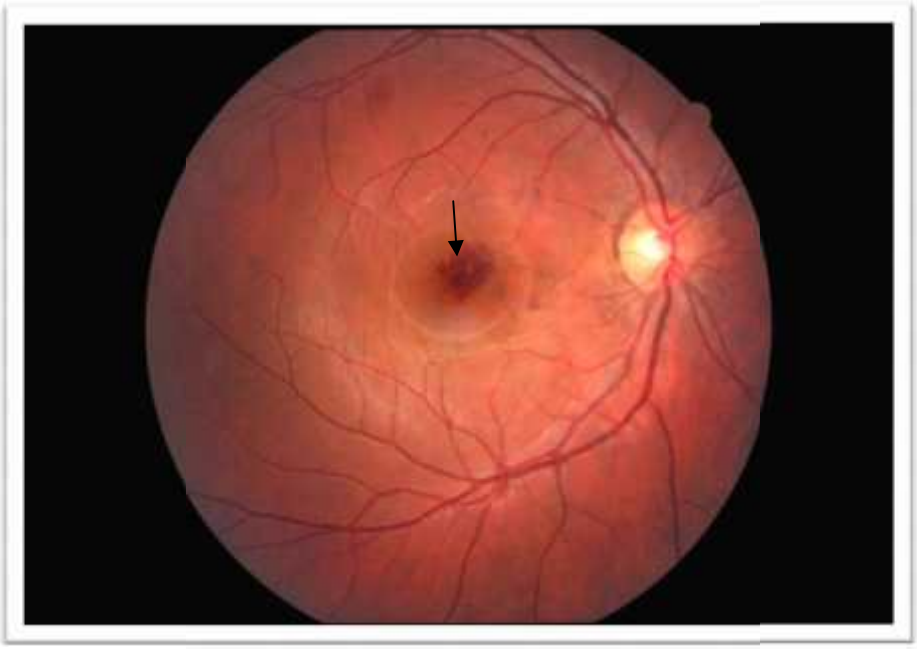
Anterior segment manifestations



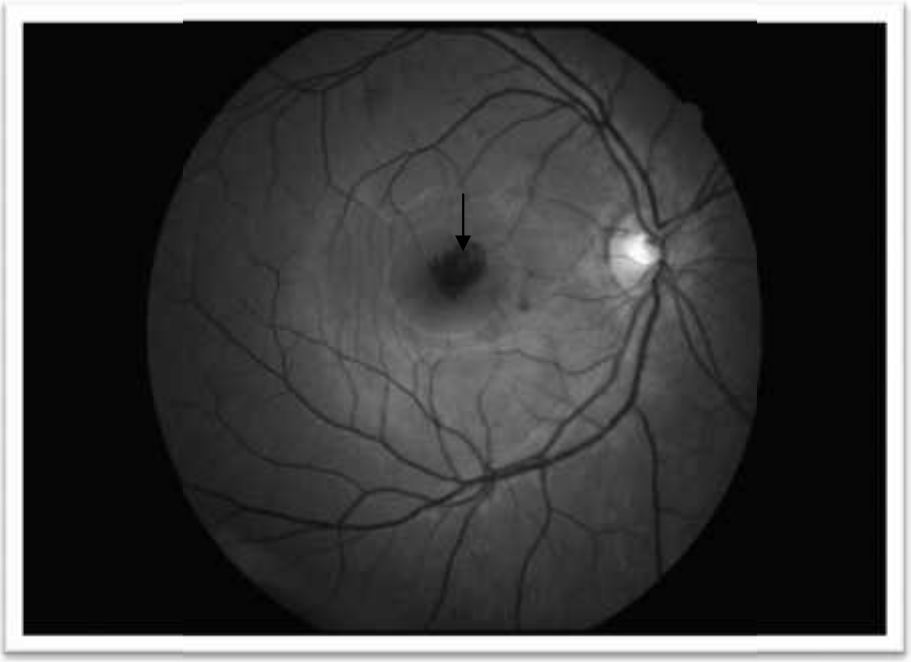
Photograph 3 : Case 2. Clinical photograph showing petechial type of Subconjunctival haemorrhage (arrow pointing Subconjunctival haemorrhage) in a 30years male patient seropositive for dengue admitted with the diagnosis of DHF I with platelet cell count 30000 cells/µL



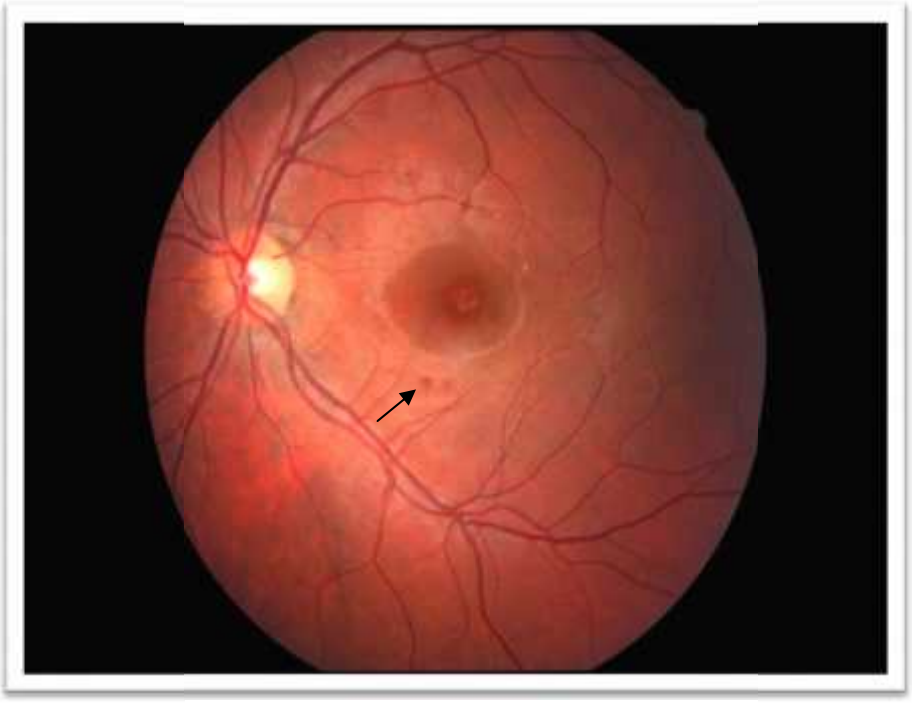
Photograph 4: Case 10. Clinical photograph showing Diffuse type of Subconjunctival haemorrhage in right eye (arrow pointing diffuse Subconjunctival haemorrhage) in a 6years male patient seropositive for dengue admitted with the diagnosis of DHF II with platelet cell count 29000 cells/µL



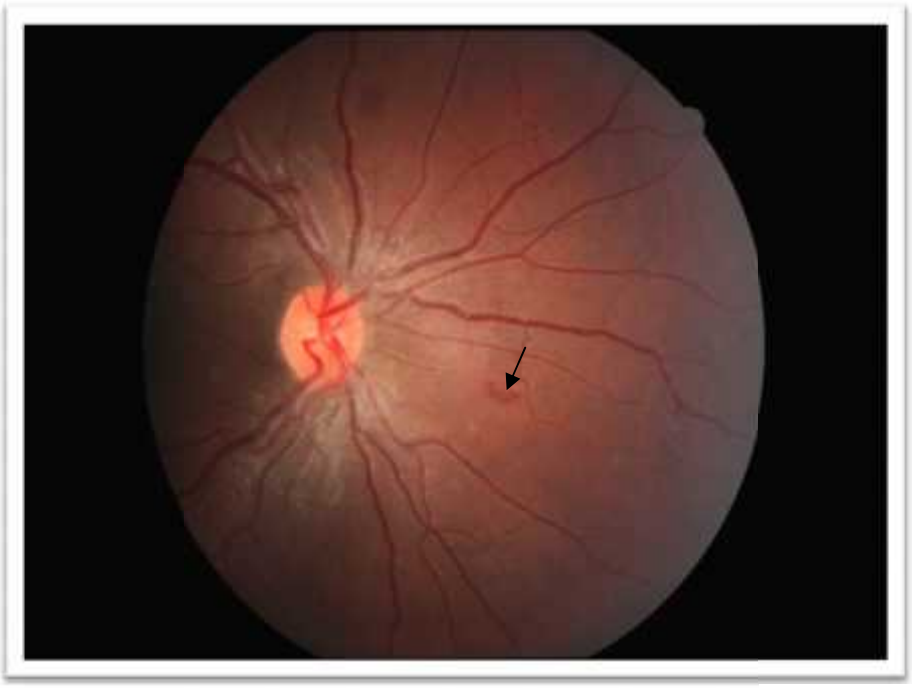
Photograph 5: Case 76.Fundus photograph showing foveal haemorrhage and superficial retinal haemorrhage in macular region in right eye in a 20 year old male seropositive for dengue admitted with the diagnosis of DHF II with platelet cell count 15000cells/µL. BCVA in RE was 6/24 and NV-N8.



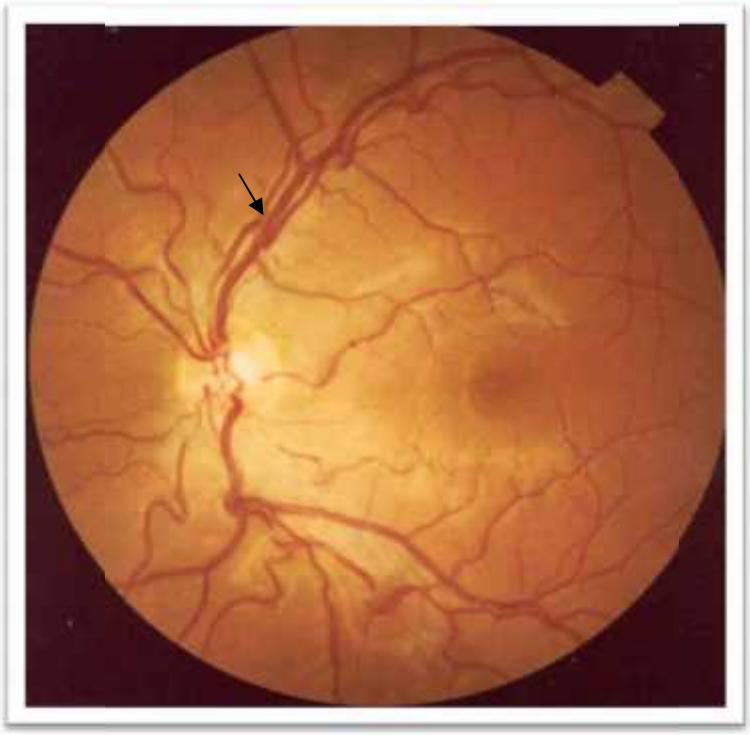
Photograph 6 : Case 76 Red free.Fundus photograph showing foveal haemorrhage and superficial retinal haemorrhage in macular region right eye in a 20 year old male seropositive for dengue admitted with the diagnosis of DHF II with platelet cell count 15000cells/µL. BCVA in RE was 6/24 and NV-N8



Photograph 7: Case 76.Fundus photograph showing superficial retinal haemorrhage at macula in left eye in a same 20 year old male seropositive for dengue admitted with the diagnosis of DHF II with platelet cell count 15000cells/µL. BCVA in LE was 6/6 and NV-N6.



Photograph 8 : Case 5.Fundus photograph showing superficial retinal haemorrhage at superior nasal quadrant of right eye in a 21 year old male seropositive for dengue admitted with the diagnosis of DHF II with platelet cell count 44000 cells/µL therapy. VA in BE was 6/6.



Photograph 9 : Case 76.Fundus photograph showing dilatation and tortusity of retinal vessels in both eyes in a 36year old male seropositive for dengue admitted with the diagnosis of DHF II with platelet cell count 44000 cells/µL therapy. VA in BE was 6/6.

ANNEXURE – V: MASTER CHART

KEY TO MASTER CHART

-	-	Absent
+	-	Present
ABN	-	Abnormal
^{0}F	-	Degree Fahrenheit
aPTT	-	Activated partial thromboplastin time
BCVA	-	Best corrected visual acuity
C	-	Control
CDF	-	Classic dengue fever
cmm	-	Cubic millimeter
c/o	-	Complaints of
DBS	-	Decreased breath sounds
DHF	-	Dengue haemorrhagic fever
DTOV	-	Dilatation and tortusity of vesseles
F	-	Female
Hb	-	Haemoglobin
h/o	-	History of
IgG	-	Immunoglobulin G
IgM	-	Immunoglobulin M
IP. No.	-	In patient number
L	-	Left eye
M	-	Male
MH	-	Macular haemorrhage

N	-	Normal
PCV	-	Packed cell volume
PL	-	Perception of light
PT	-	Prothombin time
R	-	Right eye
SCPH	-	Subconjuctival petechial haemorrhage
SCDH	-	Subconjuctival diffuse haemorrhage
SFRH	-	Superficial retinal haemorrhage
Sr. No.	-	Serial number
T	-	Test
THM	-	Tender hepatomegaly
TC	-	Total count

				GENERAL SYMPTOMS															OCULAR SYMPTOMS					
Sr. No.	IP No.	Age (Years)	Sex	Fever	Chills	Headache	Neck rigidity	Vomiting	Nausea	Giddiness	Backache	Mayalgia	Joint pain	Retrobulbar pain	Abdominal pain	Loss of appeptite	Loose stools	Jaundice	Diminution of vision	H/O redness,watering	H/O pain	Coloured halos	H/O wearing spectacles	Any other c/o
1	471234	49	M	+	+	+	-	-	-	-	+	+	-	+	+	-	+	-	-	-	-	-	-	-
2	471354	25	M	+	+	+	-	+	-	-	+	+	-	-	+	-	-	-	-	-	-	-	-	-
3	471864	25	F	+	+	+	-	+	-	-	-	+	+	+	+	-	+	-	-	+	-	-	-	-
4	471584	9	F	+	+	-	-	+	-	-	-	-	-	-	-	+	+	-	-	+	-	-	-	-
5	472675	21	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	-	-	-
6	474077	15	F	+	+	-	-	+	-	-	+	+	-	-	+	-	-	-	-	+	-	-	-	-
7	473935	16	F	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
8	475205	25	M	+	+	+	-	-	-	-	-	+	+	+	+	-	+	-	-	+	-	-	-	-
9	474978	17	M	+	+	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-
10	475166	6	M	+	+	+	-	+	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-
11	475945	12	F	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
12	475926	26	M	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
13	475107	7	F	+	+	-	-	+	-	-	-	+	+	+	+	-	+	-	-	-	-	-	-	-
14	476849	13	M	+	+	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-
15	477401	30	M	+	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	+	-	-	-
16	477130	45	F	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	+
17	477481	6	M	+	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
18	477473	20	M	+	+	+	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
19	477300	11	M	+	+	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-
20	477877	13	F	+	+	+	-	+	-	-	-	-	-	-	-	+	+	-	-	+	-	-	-	-
21	478013	7	M	+	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
22	478456	59	F	+	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
23	479101	8	F	+	+	-	-	+	-	-	+	-	-	+	+	-	-	-	-	-	-	-	-	-
24	479390	11	M	+	+	-	-	-	-	-	-	+	+	+	+	+	+	-	-	-	-	-	-	-
25	479445	13	M	+	+	+	-	+	-	-	-	+	+	+	+	-	+	-	-	+	-	-	-	-
26	479444	15	M	+	+	-	-	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	-	-
27	479084	12	M	+	+	-	-	+	-	-	-	-	+	+	-	-	-	-	-	+	-	-	-	
28	479978	44	F	+	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
29	480637	22	F	+	+	+	-	-	-	-	+	-	-	-	+	-	-	-	-	+	-	-	-	-
30	480353	44	M	+	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-
31	481538	45	M	+	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
32	481428	6	F	+	+	+	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-
33	482229	58	M	+	+	-	-	+	-	-	-	+	+	+	+	+	+	-	-	-	-	-	-	-
34	481872	11	F	+	+	+	-	+	-	-	+	+	-	-	+	-	-	-	-	-	-	-	-	-
35	481878	10	M	+	+	+	-	+	-	-	-	+	+	+	+	-	+	-	-	-	-	-	-	-
36	481967	7	M	+	-	-	-	+	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-
37	482408	9	F	+	+	-	-	+	-	-	+	-	-	+	+	-	+	-	-	-	-	-	-	-
38	482506	32	F	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
39	482774	18	M	+	+	+	-	+	-	-	+	+	+	+	-	-	-	-	-	-	-	-	-	-
40	483486	11	M	+	+	+	-	+	-	+	-	+	-	-	-	+	+	-	-	-	-	-	-	-
41	483740	7	F	+	+	-	-	+	-	+	-	-	-	-	+	-	-	-	-	-	-	-	-	
42	483905	28	F	+	+	+	-	+	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-
43	483711	38	M	+	+	+	-	-	-	-	+	-	+	+	+	-	+	-	-	-	-	-	-	-
44	483843	8	M	+	+	-	-	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-
45	483986	21	M	+	+	+	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-
46	484082	22	M	+	+	+	-	+	+	-	-	-	-	-	+	-	+	-	-	-	-	-	-	-
47	484379	55	M	+	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
48	484289	23	M	+	+	+	-	+	+	-	+	+	-	-	+	-	-	-	-	-	-	-	-	-
49	484669	26	M	+	+	-	-	+	+	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-
50	484801	29	M	+	+	-	-	+	+	-	+	-	-	-	+	-	+	-	-	-	-	-	-	-
51	484459	26	M	+	+	-	-	+	+	-	+	+	+	+	-	+	-	-	-	-	-	-	-	-
52	484513	13	F	+	+	-	-	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
53	484642	25	F	+	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
54	484820	43	M	+	+	-	-	+	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-
55	485379	40	M	+	+	-	-	+	+	+	+	+	+	+	+	-	+	-	-	-	-	-	-	-
56	485612	47	M	+	+	+	-	-	-	-	+	-	+	+	-	-	-	-	-	-	-	-	-	-
57	485779	8	F	+	+	+	-	-	-	-	-	-	+	+	-	-	-	-	-	-	-	-	-	-
58	485623	11	M	+	+	-	-	-	-	-	+	-	-	-	+	+	-	-	-	+	-	-	-	-
59	485823	27	F	+	+	+	-	+	+	-	-	+	-	-	+	-	-	-	-	-	-	-	-	-
60	485989	30	M	+	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-
61	485980	22	M	+	+	-	-	+	+	-	+	+	-	-	-	+	-	-	-	-	-	-	-	-
62	486090	55	F	+	+	-	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-
63	486745	15	M	+	-	+	-	-	-	-	+	+	-	-	-	-	-	-	-	+	-	-	-	-
64	486853	13	M	+	+	-	-	+	+	-	-	-	-	-	+	-	-	-	-	+	-	-	-	-
65	486740	28	M	+	+	+	-	+	+	-	+	-	-	+	+	-	+	-	-	-	-	-	-	-
66	486738	17	M	+	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
67	486440	6	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
68	487356	7	M	+	+	-	-	+	+	-	+	-	+	+	+	-	+	-	-	-	-	-	-	-
69	487059	34	M	+	+	-	-	-	-	-	+	+	-	+	+	-	+	-	-	-	-	-	-	-
70	487169	35	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
71	488381	28	M	+	+	+	-	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-
72	486078	20	M	+	+	+	-	-	-	+	+	+	-	-	+	-	+	-	-	-	-	-	-	-
73	488926	18	M	+	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-
74	489528	6	F	+	+	-	-	-	-	-	-	+	+	+	-	-	-	-	-	-	-	-	-	-
75	489767	21	M	+	+	-	-	-	-	-	+	+	-	+	+	-	+	-	-	-	-	-	-	-
76	489572	20	M	+	+	+	-	+	+	-	+	+	-	+	+	-	+	-	-	-	-	-	-	-
77	490534	74	M	+	+	+	-	-	-	-	+	-	-	+	+	-	+	-	-	-	-	-	-	-
78	490754	9	F	+	+	+	-	-	-	-	+	+	-	+	+	-	+	-	-	-	-	-	-	-

	GENERAL & SYSTEMIC EXAMINATION															OCULAR EXAMINATION		
Sr. No.	Pallor:	Icterus	Edema	Lymphadenopathy	Cyanosis	Clubbing	Pulse	BP	Temperature ^{0}F	CVS	RS	CNS	PA	Bowel and bladder habits	Blood transfusion	Head Posture	Facial Symmetry	Visual Axis
1	-	-	-	-	-	-	90	110/70	98.6	N	N	N	N	N	+	N	N	N
2	-	-	-	-	-	-	70	110/70	99	N	N	N	N	N	+	N	N	N
3	-	-	-	-	-	-	120	120/84	99.4	N	DBS	N	N	N	+	N	N	N
4	-	-	-	-	-	-	106	112/64	99	N	N	N	N	N	+	N	N	N
5	-	-	-	-	-	-	74	110/70	99.4	N	N	N	N	N	+	N	N	N
6	-	-	-	-	-	-	103	120/84	100	N	N	N	THM	N	-	N	N	N
7	-	-	-	-	-	-	96	104/66	100	N	N	N	N	N	+	N	N	N
8	-	-	-	-	-	-	66	100/70	104	N	N	N	N	N	+	N	N	N
9	-	-	-	-	-	-	70	100/70	99	N	N	N	N	N	+	N	N	N
10	-	-	-	-	-	-	79	102/70	99	N	N	N	THM	N	+	N	N	N
11	-	-	-	-	-	-	82	102/68	99	N	N	N	N	N	+	N	N	N
12	-	-	-	-	-	-	70	100/60	99	N	N	N	THM	N	+	N	N	N
13	-	-	-	-	-	-	112	108/60	101	N	N	N	THM	N	+	N	N	N
14	-	-	-	-	-	-	80	110/80	99	N	N	N	N	N	+	N	N	N
15	-	-	-	-	-	-	80	100/70	99	N	N	N	THM	N	+	N	N	N
16	+	-	-	-	-	-	86	130/100	99	N	N	N	N	N	+	N	N	N
17	-	-	-	-	-	-	90	102/66	99	N	N	N	N	N	+	N	N	N
18	-	-	-	-	-	-	80	110/72	99	N	N	N	THM	N	+	N	N	N
19	-	-	-	-	-	-	80	90/60	99	N	N	N	THM	N	+	N	N	N
20	-	-	-	-	-	-	82	106/72	99	N	DBS	N	THM	N	+	N	N	N
21	-	-	-	-	-	-	78	94/68	98.6	N	N	N	THM	N	+	N	N	N
22	-	-	-	-	-	-	70	102/70	99.2	N	N	N	N	N	-	N	N	N
23	-	-	-	-	-	-	82	108/68	99	N	N	N	N	N	+	N	N	N
24	-	-	-	-	-	-	90	112/60	99	N	N	N	THM	N	+	N	N	N
25	-	-	-	-	-	-	92	110/40	99	N	N	N	THM	N	+	N	N	N
26	-	-	-	-	-	-	78	120/40	99	N	N	N	N	N	+	N	N	N
27	-	-	-	-	-	-	88	88/50	99.2	N	N	N	N	N	+	N	N	N
28	-	-	-	-	-	-	90	160/100	98.6	N	N	N	N	N	+	N	N	N
29	-	-	-	-	-	-	74	110/70	99	N	N	N	N	N	+	N	N	N
30	-	-	-	-	-	-	100	110/80	99	N	N	N	THM	N	+	N	N	N
31	-	-	-	-	-	-	68	102/60	99.8	N	DBS	N	N	N	+	N	N	N
32	-	-	-	-	-	-	92	94/70	99	N	N	N	N	N	+	N	N	N
33	-	-	-	-	-	-	84	120/84	98.9	N	N	N	N	N	-	N	N	N
34	-	-	-	-	-	-	98	110/90	98	N	N	N	N	N	-	N	N	N
35	-	-	-	-	-	-	86	110/60	98	N	DBS	N	N	N	-	N	N	N
36	-	-	-	-	-	-	80	110/70	99	N	N	N	THM	N	+	N	N	N
37	-	-	-	-	-	-	92	100/70	99	N	N	N	N	N	+	N	N	N
38	-	-	-	-	-	-	90	120/90	99	N	N	N	N	N	+	N	N	N
39	-	-	-	-	-	-	80	110/70	99	N	N	N	N	N	+	N	N	N
40	-	-	-	-	-	-	90	108/70	99	N	N	N	N	N	-	N	N	N
41	-	-	-	-	-	-	82	100/66	98.6	N	N	N	N	N	-	N	N	N
42	-	-	-	-	-	-	80	110/70	99.8	N	N	N	N	N	-	N	N	N
43	-	-	-	-	-	-	80	120/70	99	N	N	N	N	N	-	N	N	N
44	-	-	-	-	-	-	80	110/70	99	N	N	N	N	N	+	N	N	N
45	-	-	-	-	-	-	80	110/70	99	N	N	N	THM	N	+	N	N	N
46	-	-	-	-	-	-	90	130/80	98.8	N	N	N	N	N	-	N	N	N
47	-	-	-	-	-	-	80	130/74	99	N	N	N	N	N	+	N	N	N
48	-	-	-	-	-	-	80	140/90	99	N	N	N	THM	N	-	N	N	N
49	+	-	-	-	-	-	74	110/70	99	N	N	N	THM	ABN	-	N	N	N
50	-	-	-	-	-	-	78	110/70	99	N	N	N	N	ABN	+	N	N	N
51	-	-	-	-	-	-	90	140/92	99	N	N	N	THM	N	-	N	N	N
52	-	-	-	-	-	-	100	110/70	99.8	N	N	N	N	N	-	N	N	N
53	-	-	-	-	-	-	78	110/70	99.8	N	N	N	N	N	+	N	N	N
54	-	-	-	-	-	-	90	110/70	99	N	N	N	N	N	-	N	N	N
55	-	-	-	-	-	-	77	110/70	98.8	N	N	N	N	N	-	N	N	N
56	-	-	-	-	-	-	70	110/88	99	N	N	N	N	N	+	N	N	N
57	-	-	-	-	-	-	76	110/70	98.6	N	N	N	N	N	-	N	N	N
58	-	-	-	-	-	-	84	110/70	99	N	N	N	N	N	+	N	N	N
59	-	-	-	-	-	-	98	110/80	99	N	N	N	N	N	+	N	N	N
60	-	-	-	-	-	-	70	110/70	98.8	N	N	N	THM	N	-	N	N	N
61	-	-	-	-	-	-	77	110/70	98.8	N	N	N	THM	N	+	N	N	N
62	-	-	-	-	-	-	64	130/80	98.8	N	N	N	N	N	-	N	N	N
63	-	-	-	-	-	-	86	110/64	99	N	N	N	N	ABN	+	N	N	N
64	-	-	-	-	-	-	92	110/70	100	N	N	N	N	N	+	N	N	N
65	-	-	-	-	-	-	70	110/70	99	N	N	N	THM	N	-	N	N	N
66	-	-	-	-	-	-	94	110/70	99	N	N	N	N	N	-	N	N	N
67	-	-	-	-	-	-	96	106/70	98.6	N	N	N	N	N	+	N	N	N
68	-	-	-	-	-	-	106	110/66	99	N	DBS	N	N	N	+	N	N	N
69	-	-	-	-	-	-	74	124/70	99.2	N	N	N	N	N	-	N	N	N
70	-	-	-	-	-	-	80	110/80	99	N	N	N	THM	N	-	N	N	N
71	-	-	-	-	-	-	80	110/70	98	N	N	N	N	N	-	N	N	N
72	-	-	-	-	-	-	80	120/80	98	N	N	N	N	N	+	N	N	N
73	-	-	-	-	-	-	90	100/60	99	N	N	N	THM	N	+	N	N	N
74	-	-	-	-	-	-	80	130/70	99	N	N	N	N	N	-	N	N	N
75	-	-	-	-	-	-	80	120/90	98	N	N	N	N	ABN	-	N	N	N
76	-	-	-	-	-	-	80	120/70	98.6	N	N	N	N	N	+	N	N	N
77	-	-	-	-	-	-	74	110/70	99	N	N	N	N	N	+	N	N	N
78	-	-	-	-	-	-	77	100/60	98	N	N	N	THM	N	+	N	N	N

	OCULAR EXAMINATION																						INVESTIGATIONS										
	Visual acuity						Extraocular movements				Anterior segment							Posterior segment										PT			APTT		
	Distant		Near		BCVA													Direct fundoscopy		Indirect fundoscopy													
Sr. No.	RE	LE	RE	LE	RE	LE	RE	LE	Binocular	Adnexa	Conjunctiva	Cornea	Sclera	Anterior chamber	Iris	Pupil	Lens	RE	LE	RE	LE	Diagnosis	Hbgm%	PCV	Platelet cell/μL	IgM DENGUE	IgG DENGUE	C	T	INR	C	T	Chest X-ray
1	6/9	6/9	N36	N36	N	N	N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	CDF	15.3	45.0	16000	+	+	15.8	15.7	1.51	31.5	51.4	N
2	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	16.19	40.0	42000	+	+	15.8	16.2	1.52	31.5	54.7	N
3	6/6	6/6	N6	N6			N	N	N	N	SCDH	N	N	N	N	N	N	N	N	N	N	DHF II	7.4	19.1	21000	+	+	15.8	16.0	1.48	31.5	31.9	PE
4	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF II	12.1	34.2	21000	+	+	10.8	22.4	1.05	27.4	28.3	N
5	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	SFRH	N	SFRH	N	DHF II	14.6	42	44000	+	+	15.8	28.9	1.54	31.5	49.9	N
6	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	10.1	30.4	25000	+	+	15.8	15.6	1.02	31.5	32.7	N
7	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF II	10.5	34.5	17000	+	+	15.8	30.1	1.52	31.5	49.5	N
8	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	14.2	41.6	18000	+	+	15.8	31.2	1.48	31.5	49.9	N
9	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.8	37,1	43000	+	+	15.8	37.3	1.46	31.5	50.1	N
10	6/6	6/6	N6	N6			N	N	N	N	SCDH	N	N	N	N	N	N	N	N	N	N	DHF II	12.8	41.6	29000	+	+	15.8	41.1	1.51	31.5	51.6	N
11	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	12.4	36.5	29000	+	+	15.8	15.9	1.56	31.5	55.2	N
12	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	14.8	47.2	17000	+	+	15.8	37.5	1.38	30.4	48.6	N
13	6/6	6/6	N6	N6			N	N	N	LE	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	15.4	45.4	45000	+	+	15.8	29.8	1.13	31.5	58.8	N
14	6/6	6/6	N6	N6			N	N	N	N	SCDH	N	N	N	N	N	N	N	N	N	N	DHF II	12.9	38.5	12000	+	+	15.8	16.2	1.06	31.5	31.6	N
15	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF I	10.0	38.9	30000	+	+	15.8	40.3	1.62	31.5	50.6	N
16	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	9.8	29.1	21000	+	+	15.8	38.8	1.58	31.5	48.9	N
17	6/18	6/12	N6	N6	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.9	35.3	48000	+	+	15.8	43.6	1.46	31.5	53.1	N
18	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	14.5	44.1	13000	+	+	15.8	44.2	1.08	31.5	32.1	N
19	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF II	13.5	41.5	25000	+	+	15.8	36.7	1.54	31.5	52.3	N
20	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF II	11.7	36.5	7000	+	+	15.8	39.8	1.56	31.5	56.1	PE
21	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.6	35.7	40000	+	+	15.8	48.7	1.57	31.5	49.7	N
22	6/9	6/9	N36	N36	6/6 N6	6/6 N6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.9	35.3	48000	+	+	15.8	18.0	1.56	31.5	55.4	N
23	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF I	12.5	42.5	49000	+	+	15.8	18.5	1.59	31.5	52.9	N
24	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.6	40.7	55000	+	+	15.8	41.9	1.54	31.5	49.4	N
25	6/6	6/6	N6	N6			N	N	N	N	SCDH	N	N	N	N	N	N	N	N	N	N	DHF II	11.7	36.5	6000	+	+	15.8	39.6	1.62	31.5	57.1	N
26	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	11	44	214000	+	+	15.8	37.8	1.32	31.5	52.9	N
27	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.8	38.6	24000	+	+	10.8	11.6	1.42	27.4	30.8	N
28	6/18	6/18	N8	N8	6/6 N6	6/6 N6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	13.1	40	7000	+	+	15.8	29.9	1.36	31.5	49.5	N
29	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF I	12.6	38.1	85000	+	+	15.8	42.0	1.44	31.5	38.9	N
30	6/6	6/6	N10	N10	N6	N6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF I	15.7	45.4	42000	+	+	15.8	16.1	1.13	31.5	31.7	N
31	6/6	6/6	N12	N12	N6	N6	N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	15.5	47.4	9000	+	+	15.8	38.9	1.58	31.5	57.6	N
32	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF I	14.8	46.0	15000	+	+	15.0	17.0	1.56	31.5	53.5	PE
33	6/12	6/12	N10	N10	6/6 N6	6/6 N6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.9	38.6	95000	+	+	15.8	16.1	1.02	31.5	31.2	N
34	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	13.3	40	130000	+	+	15.8	39.9	1.03	31.5	55.9	N
35	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF IV	12.5	37	160000	+	+	15.8	40.4	1.05	31.5	50.6	N
36	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	13.8	40.0	18000	+	+	14.1	14	1.54	28	54.8	N
37	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	13.2	40.3	130000	+	+	15.8	14.9	1.01	31.5	59.7	N
38	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	11.7	37.8	82000	+	+	15.8	39.4	1.02	31.5	57.8	N
39	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF I	15.1	45.0	18000	+	+	15.8	41.7	1.54	31.5	58.4	N
40	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.9	37.1	49900	+	+	15.8	38.7	1.58	31.5	55.9	N
41	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	13	39.4	119000	+	+	15.8	47.3	1.02	31.5	56.8	N
42	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12	37	41000	+	+	15.8	38.6	1.58	31.5	54.9	N
43	6/6	6/6	N6	N6			N	N	N	N	SCDH	N	N	N	N	N	N	N	N	N	N	DHF II	15.9	45.0	11000	+	+	15.8	37.9	1.54	31.5	49.9	N
44	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	13.4	40.4	45000	+	+	15.8	16.1	1.05	31.5	51.7	N
45	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.3	37	21000	+	+	15.8	16.3	1.58	31.5	53.2	N
46	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	15.4	45.3	11000	+	+	15.8	38.6	1.16	31.5	58.3	N
47	6/18	6/12	N36	N36			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	13.4	41.6	41000	+	+	15.8	15.9	1.07	31.5	55.4	N
48	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	14.9	42.5	46000	+	+	15.8	16.3	1.16	31.5	32.7	N
49	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	14.1	41.6	41000	+	+	15.8	27.7	1.58	31.5	56.2	N
50	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	16.2	47.4	18000	+	+	15.8	39.9	1.54	31.5	57.4	N
51	6/9	6/12	N6	N6	6/6	6/6	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	13.7	39.7	79000	+	+	15.8	36.8	1.21	31.5	56.8	N
52	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	14.6	41.6	60000	+	+	15.8	16.3	1.03	31.5	51.1	N
53	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	12.7	37.5	45000	+	+	15.8	39.0	1.02	31.5	57.3	N
54	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	10.8	30.6	188000	+	+	15.8	36.8	1.02	31.5	55.9	N
55	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	14	32.6	44000	+	+	15.8	35.5	1.29	31.5	49.4	N
56	6/6	6/6	N36	N36			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF I	17.0	51.3	10000	+	+	15.8	15.9	1.54	31.5	58.4	N
57	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	11.6	34.1	98000	+	+	15.8	37.7	0.89	31.5	57.3	N
58	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	13.4	39.7	90000	+	+	15.8	20.0	1.29	31.5	60	N
59	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	DHF I	11.6	35.7	43000	+	+	15.8	38.1	1.05	31.5	58.4	N
60	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	16.3	47.4	12000	+	+	15.8	34.7	1.03	37.5	55.3	N
61	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	DTOV	N	DTOV	N	DHF II	15.6	45.3	71000	+	+	15.8	16.2	1.10	31.5	57.2	N
62	6/9	6/6	N8	N8			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	13.3	39.7	27000	+	+	15.8	34.9	1.02	31.5	58.1	N
63	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	DTOV	N	DTOV	N	DHF II	13.8	41.2	33000	+	+	15.8	33.7	1.05	31.5	59.2	N
64	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	DTOV	N	DTOV	N	DHF II	16	45.3	3000	+	+	15.8	16.4	1.54	31.5	31.8	N
65	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	15.3	45.3	81000	+	+	15.8	38.4	1.05	31.5	58.7	N
66	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	13.4	39.7	1030000	+	+	15.8	16.5	1.03	31.5	57.9	N
67	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.3	36.3	21000	+	+	15.8	16.3	1.54	31.5	60.1	N
68	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	14.2	30.6	16000	+	+						PE
69	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	14.6	45.3	93000	+	+	15.8	36.4	1.03	31.5	49.2	N
70	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	13.5	39.7	25000	+	+	15.8	16.1	1.54	31.5	53.8	N
71	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	16.4	47.4	45000	+	+	15.8	37.5	1.03	31.5	49.3	N
72	6/6	6/6	N6	N6			N	N	N	N	SCDH	N	N	N	N	N	N	N	N	N	N	DHF II	12.1	36.3	7000	+	+	15.8	33.9	1.05	31.5	60.2	N
73	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N	DHF II	10.7	30.6	28000	+	+	15.8	37.4	1.02	31.5	52.6	N
74	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	13.5	37.5	78000	+	+	15.8	39.3	1.05	31.5	55.8	N
75	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	CDF	12.4	36.3	80000	+	+	15.8	14.2	1.56	31.5	56.5	N
76	6/24	6/6	N8	N6	6/18	6/6	N	N	N	N	SCDH	N	N	N	N	N	N	SFRH	N	SFRH	N	DHF II	15.6	45.3	15000	+	+	15.8	40.0	1.62	31.5	58.2	N
77	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	DTOV	N	DTOV	N	DHF II	12.	32.6	65000	+	+	15.8	39.7	1.05	31.5	33.8	N
78	6/6	6/6	N6	N6			N	N	N	N	SCDH	N	N	N	N	N	N	N	N	N	N	DHF II	11.6	35.7	5000	+	+	15.8	33.4	1.44	31.5	59.2	N

Sr. No.	IP No.	Age (Years)	Sex	GENERAL SYMPTOMS: Fever	Chills	Headache	Neck rigidity	Vomiting	Nausea	Giddiness	Backache	Mayalgia	Joint pain	Retrobulbar pain	Abdominal pain	Loss of appeptite	Loose stools	Jaundice	OCULAR SYMPTOMS: Diminution of vision	H/O redness,watering	H/O pain	Coloured halos	H/O wearing spectacles	Any other c/o
79	490923	30	M	+	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	-	-	-
80	491323	7	F	+	+	-	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
81	491091	24	M	+	-	+	-	-	-	-	+	+	-	-	+	+	-	-	-	-	-	-	-	-
82	491148	45	F	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-
83	491602	37	F	+	-	-	-	+	+	-	-	+	-	-	-	-	-	-	-	-	-	-	-	-
84	491483	6	F	+	+	+	-	-	-	-	-	-	-	-	-	+	-	-	-	+	-	-	-	-
85	491972	20	M	+	-	+	-	+	+	-	+	+	-	-	+	-	+	-	-	+	-	-	-	-
86	492089	10	F	+	+	+	-	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	-	-
87	491988	36	M	+	-	+	-	-	-	-	+	-	-	-	+	-	-	-	-	+	-	-	-	-
88	492365	12	M	+	+	-	-	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-
89	492381	7	M	+	-	-	-	-	-	+	-	-	+	+	+	-	+	-	-	+	-	-	-	-
90	492300	22	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-
91	492383	7	F	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-
92	492382	6	F	+	-	+	-	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
93	492619	35	M	+	+	-	-	-	-	-	-	+	+	+	+	-	+	-	-	-	-	-	-	-
94	492634	20	M	+	-	+	-	-	-	-	+	+	-	-	+	-	+	-	-	-	-	-	-	-
95	492821	13	M	+	-	-	-	+	+	-	+	+	-	+	+	-	+	-	-	-	-	-	-	-
96	492404	10	M	+	+	-	-	-	-	-	-	+	+	+	+	-	+	-	-	-	-	-	-	-
97	492967	47	M	+	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-
98	493004	17	M	+	+	-	-	+	+	-	-	-	-	-	+	-	-	-	-	-	-	-	-	-
99	493305	21	M	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	-	-	-	-
100	493373	12	M	+	-	-	-	-	-	-	+	+	-	-	+	-	-	-	-	+	-	-	-	-

Sr. No.	GENERAL & SYSTEMIC EXAMINATION: Pallor:	Icterus	Edema	Lymphadenopathy	Cyanosis	Clubbing	Pulse	BP	Temperature ^{0}F	CVS	RS	CNS	PA	Bowel and bladder habits	Blood transfusion
79	-	-	-	-	-	-	60	94/70	100	N	DBS	N	THM	N	+
80	-	-	-	-	-	-	80	130/70	98	N	N	N	N	N	-
81	-	-	-	-	-	-	78	120/70	99	N	N	N	N	N	-
82	-	-	-	-	-	-	100	86/58	101	N	N	N	N	N	+
83	-	-	-	-	-	-	84	112/74	98.4	N	N	N	N	N	+
84	-	-	-	-	-	-	76	110/78	98.8	N	N	N	N	N	-
85	-	-	-	-	-	-	82	130/60	99	N	N	N	N	N	+
86	-	-	-	-	-	-	78	120/70	99	N	N	N	N	N	-
87	-	-	-	-	-	-	80	110/80	98.6	N	N	N	N	N	-
88	-	-	-	-	-	-	60	90/60	98.8	N	N	N	N	N	+
89	-	-	-	-	-	-	108	86/58	98.6	N	DBS	N	N	N	+
90	-	-	-	-	-	-	100	110/70	101	N	N	N	N	N	-
91	-	-	-	-	-	-	98	104/62	99	N	DBS	N	N	N	+
92	-	-	-	-	-	-	120	100/70	99	N	DBS	N	N	N	+
93	+	-	-	-	-	-	60	120/70	99	N	N	N	N	N	+
94	-	-	-	-	-	-	82	110/80	100	N	N	N	N	N	+
95	-	-	-	-	-	-	110	110/60	99	N	DBS	N	THM	N	-
96	-	-	-	-	-	-	96	92/68	99	N	N	N	THM	N	+
97	-	-	-	-	-	-	78	130/90	99	N	N	N	THM	N	+
98	-	-	-	-	-	-	76	90/60	99	N	N	N	THM	N	-
99	-	-	-	-	-	-	60	140/70	98.8	N	N	N	N	N	+
100	-	-	-	-	-	-	82	108/70	99	N	N	N	N	N	-

Sr. No.	OCULAR EXAMINATION: Head Posture	Facial Symmetry	Visual Axis	Visual acuity: Distant RE	Distant LE	Near RE	Near LE	BCVA RE	BCVA LE	Extraocular movements RE	LE	Binocular	Anterior segment: Adnexa	Conjunctiva	Cornea	Sclera	Anterior chamber	Iris	Pupil	Lens	Posterior segment: Direct fundoscopy RE	LE	Indirect fundoscopy RE	LE
79	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
80	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
81	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
82	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
83	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
84	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
85	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
86	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
87	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	DTOV	DTOV	DTOV	N
88	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
89	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
90	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
91	N	N	N	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N
92	N	N	N	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N
93	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
94	N	N	N	6/6	6/6	N6	N6			N	N	N	N	SCDH	N	N	N	N	N	N	N	N	N	N
95	N	N	N	6/6	6/6	N6	N6			N	N	N	N	SCPH	N	N	N	N	N	N	N	N	N	N
96	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
97	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
98	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N
99	N	N	N	6/6	6/6	N6	N6			N	N	N	N	SCDH	N	N	N	N	N	N	MH	N	MH	N
100	N	N	N	6/6	6/6	N6	N6			N	N	N	N	N	N	N	N	N	N	N	N	N	N	N

Sr. No.	Diagnosis	INVESTIGATIONS: Hbgm%	PCV	Platelet cell/µL	IgM DENGUE	IgG DENGUE	PT C	PT T	INR	APTT C	APTT T	Chest X-ray
79	DHF I	10.7	30.6	9000	+	+	15.8	16.2	1.02	31.5	33.1	N
80	CDF	13.5	39.7	81000	+	+	15.8	15.8	1.52	31.5	57.9	N
81	CDF	15.6	45.3	44000	+	+	15.8	37.5	1.26	31.5	58.4	N
82	DHF I	12.9	35.7	12000	+	+	15.8	38.8	1.46	31.5	59.4	N
83	CDF	13.1	39.6	99000	+	+	15.8	39.4	1.28	31.5	35.0	N
84	DHF I	12.6	36	8000	+	+	15.8	29.9	1.32	31.5	59.2	N
85	DHF I	10	28	14000	+	+						N
86	DHF I	11.8	36	43000	+	+	15.8	38.2	1.56	31.5	60.1	N
87	DHF II	13	34.8	44000	+	+	15.8	37.5	1.04	31.5	31.8	N
88	CDF	12.9	36.4	42000	+	+	15.8	39.1	1.54	31.5	55.2	N
89	CDF	14.7	43.5	28000	+	+						N
90	CDF	13.1	40.2	183000	+	+	15.8	32.2	1.58	31.5	55.4	N
91	DHF III	11	32.2	98000	+	+	11.4	19.7	1.64	26.8	59.4	N
92	DHF II	12.4	37.1	21000	+	+	15	17.5	1.52	31.5	57.8	N
93	CDF	15.5	32.4	5000	+	+	14.4	10.7	1.62	31.5	37.0	N
94	DHF II	16.5	47.1	8000	+	+	15.8	16.8	1.58	31.5	60.3	N
95	DHF II	14.6	42.8	10000	+	+	15.8	32.2	1.50	31.5	59.6	N
96	DHF I	13.4	41.7	20000	+	+	15.8	16.8	1.58	31.5	67.0	N
97	DHF I	15	44.6	82000	+	+	15.8	16.3	1.52	31.5	62.3	N
98	DHF I	17.2	48.9	19000	+	+						N
99	DHF II	15.2	31.4	14000	+	+	15.6	15.4	1.58	31.5	57.8	
100	DHF I	12.5	36.8	74000	+	+						N

	Symptoms											Signs								
Sl. No.	Fever	Myalgia	Arthralgia	Vomiting	Headache	Abdominal pain	Backache	Diarrhoea	malena	Altered sensorium	Seizure	Petechiae / Ecchymosis	Pleural effusion	Hepatomegaly	Bradycardia	Ascites	Subconjunctival haemorrhage	Icterus	Spleenomegaly	HCT