A STUDY OF CLINICAL PROFILE, LABORATORY PROFILE AND COMPLICATIONS OF FEVER WITH THROMBOCYTOPENIA

Dissertation submitted in partial fulfilment of regulation

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M.D. Degree in General Medicine

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A STUDY OF CLINICAL PROFILE, LAB0RATORY PROFILE AND COMPLICATION OF FEVER WITH THROMBOCYTOPENIA

by

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Dissertation submitted to the Dr.M.G.R Medical University, Chennai in partial

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MD IN GENERAL MEDICINE

Under guidance of

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DECLARATION

I here by declare that this dissertation entitled **"A STUDY OF CLINICAL PROFILE, LABORATORY PROFILE AND COMPLICATIONS OF FEVER WITH THROMBOCYTOPENIA"** is a bonafide and genuine research work carried out by me under the guidance of Prof. DR.M.RAVEENDRAN MD., Associate professor, Coimbatore medical college, Coimbatore.

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Above all I would to thank the almighty for all this guidance and his blessing, to be in this noble profession rendering comfort to the suffering.

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Introduction

ABSTRACT

Background: Infection is a common cause of thrombocytopenia. Detection of thrombocytopenia associated with fever helps to narrow differential diagnosis and management of fever. It also helps to know the various complications of thrombocytopenia and its management and outcome of the patient.

Methods: 100 patients aged > 12 years with fever and thrombocytopenia seen between December 2010 to November 2011 were included for this study.

Results: Infection was the commonest cause of thrombocytopenia and dengue was the commonest infection. Bleeding manifestations were seen in 12 % of patients. PetechIae/purpura as the commonest bleeding manifestation followed by gum bleeding. Good recovery was noted in 92% while 8% had mortality. 50% of patients with platelet count below 20,000 does not needed platelet transfusion.

Conclusions: Infections, particularly dengue was the commonest cause of fever with thrombocytopenia. In majority of patients, thrombocytopenia was transient and asymptomatic but in significant number of cases there were bleeding manifestations. Spontaneous bleeding was noted in platelet count of < 20,000/mm3 in majority of patients, petechiae /purpura was seen in platelet count in range of 20,000-40,000/mm3. On treating the specific cause drastic improvement in platelet count was noted during discharge and further follow-up.

INTRODUCTION

Pyrexia is from the Greek *pyretos* meaning *fire*. Febrile is from the Latin word *febris*, meaning *fever*, and archaically known as *ague*.

As early as the sixth century BC, fever was noted to be a cardinal sign of disease, as Akkadain cuneiform descriptions used a flaming brazier to symbolize both fever and the localization heat that occurs with inflammation (Atkins 1982).[1]

In the era of Hippocrates, fever was explained with the doctrine of the four humor: blood, phlegm, black bile, and yellow bile. Yellow bile was associated with the element of fire, and thus fever was considered to be caused by an excess of yellow bile. Fever was thought, at the time, to be beneficial sign during infection, because the increased heat or "fire" caused by the excess of yellow bile would cook the infection out of the patient (Atkins 1982, young & brown 1985).

According to Mackowiak *el al.* ' after over a millennium of clinical investigation, there is not even a generally accepted(clinical) definition of fever'.

The humoral concept of fever was the mainstay of clinical practice until the 17th century, when Harvey discovered that blood circulation through the body. Subsequently, the believe that fever was beneficial changed dramatically, and physicians thought that fever was produced in the body fluid as a result of

fermentation and putrefaction or because of friction from increased flow of blood (Atkins 1982).

Fever was then consider to be synonymous with infectious disease as, until the mid- 19 century, most of these disease were grouped under the generic term of " fever" . because patients with fevers were considered to be associated with disease and often death, they were frequently isolated in order to product the community, as a result, fever came to be feared, and thus warranted intervention. Fever is currently treated as 'the orgin of, rather than the response to, an illness'.

A growing body of research in immunology and neuro-physiology has led to the recent understanding that fever is generally an adaptive physiology response to some threat. This notion goes against the thinking of the last two centuries that fever was a sinister sign and required intervention to lower or control it.

Fever has many important functions in the healing process:

- Increased mobility of leukocytes.
- Enhanced leukocytes phagocytosis.
- Endotoxin effects decreased.
- Increased proliferation of T cells.

Daniel G. Fahrenheit used human body temperature as a reference point for his temperature scale, defining it to be 96°F in the early 18th century. Later

redefinition of his scale to use the boiling point of water as a reference point caused the numerical value for normal body temperature to drift.

Carl Reinhold August Wunderlich released his summary of the armpit, or axillary, temperatures of twenty five thousand people in 1861, and reported the mean to be 37.0 °C (98.6 °F), with a range of 36.25 °C (97.25 °F) to 37.5 °C (99.5 °F). .

Fever is defined as an elevation of the normal body temperature that above the normal circadian variation as a result of the changes in the thermoregulatory center, located in the hypothalamus and occurs in conjunction with an increase in hypothalamic set point e.g 37°c to 39°c .

Thrombocytopenia is defined as platelet count <1,50,000/microliter. This is due to decreased production, increased destruction, increased sequestration in spleen. Of this infection is the most common cause. Fever with thrombocytopenia narrows the differential diagnosis of the clinical entity.

Infection like malaria , dengue, leptospirosis, typhoid, HIV, and miliary tuberculosis are some of the common causes of fever with thrombocytopenia.

Therefore a well organised systemic approach that is carried out with an awareness of cause of fever with thrombocytopenia can shorten the duration of investigation and bring out diagnosis.

Hence, need for study to know the causes and complications of fever with thrombocytopenia.

Aims of the Study

AIM OF THE STUDY

1. To study of incidence of various etiological agents for patients getting admitted for fever with thrombocytopenia.

2. To analyse relevant epidemiological data like seasonal variation

3. To analyse the clinical feature among various positive cases.

4. To analyse the investigation done on positive cases.

5. To study the incidence of complication.

6. To analyse about outcome of the patient.

Review of Literature

REVIEW OF LITERATURE

HISTORY OF FEVER:

Fever is perhaps the most ancient hallmark of disease. It dates back as far as civilization itself. For most of the history, fever was feared by ordinary people as a manifestation of punishment, induced by evil spirits or a marker of death.

The oldest civilizations (Egyptian, Mesopotamian, Chinese, Indian, and Greek) demonstrated extensive knowledge of fever, but tended to view it as being induced by evil spirits. Hence exorcism was used in many ancient cultures (to a lesser extent in Greek medicine) for the treatment of fever.[1]

Hippocratic writings, for example, contain evidence that fever was thought to be beneficial to the infected host. Rufus of Ephesus in the second century AD strongly advocated the beneficial role of fever.

Fever therapy was the principal form of treatment, not only for syphilis and gonorrhoea, but also for patients with rheumatoid arthritis and asthma. This belief, held for about 2000 years, should not be ignored.

Virtually all cultures use some form of "fever therapy" in the form of "saunas," or "sweat lodges," or "steam baths," or other ways to raise body temperature artificially. This probably dates back to the Hippocratic era and is based on the "humoral" theory of disease, where one of the forms of therapy was to "cook" the bad "humour."

Celsius, of the early Roman empire first suggested the possible relationship between fever and the cardinal manifestation of inflammation- heat, swelling, redness and pain.

The thermometer was not a single invention, however, but a development.

Galileo is often said to be the inventor of the thermometer, what he produced were thermoscopes. He also discovered that objects (glass spheres filled with aqueous alcohol) of slightly different densities would rise and fall, which is now the principle of the Galileo thermometer.(2)

The first person to put a scale on a thermoscope is variously said to be Francesco Sagredo or Santorio Santorio in about 1611 to 1613. The word thermometer (in its French form) first appeared in 1624 in *La Récréation Mathématique* by J. Leurechon, who describes one with a scale of 8 degrees.

In 1694 Carlo Renaldini proposed using them as fixed points on a universal scale. He described the normal diurnal variation of the body temperature. He described the normal diurnal variation of the body, established 100.4F as the upper limit of the normal range and gave the first quantitative definition of fever. Wunderlich is generally regarded as the father of clinical thermometer.(3)

Finally in 1724 Daniel Gabriel Fahrenheit used mercury as the thermometric liquid, produced a temperature scale which now (slightly adjusted) bears his name. Mercury's thermal expansion is large and fairly

uniform, it does not adhere to the glass, and it remains a liquid over a wide range of temperatures. Its silvery appearance makes it easy to read.

In 1780, J. A. C. Charles, a French physician, showed that it is possible to establish a temperature scale based on a single fixed point rather than the two fixed- point scales, such as the Fahrenheit and Celsius scales. This brings us back to a thermometer that uses a gas as the thermometric medium.

Sir William Siemens, in 1871, proposed a thermometer whose thermometric medium is a metallic conductor whose resistance changes with temperature. The element platinum does not oxidize at high temperatures and has a relatively uniform change in resistance with temperature over a large range. The *Platinum Resistance Thermometer* is now widely used as a thermoelectric thermometer and covers the temperature range from about -260° C to 1235° C.

The world's first temporal artery thermometer, a non-invasive temperature sensor which scans the forehead in about 2 seconds and provides a medically accurate body temperature was introduced by Dr. Francesco Pompei in 1999.

Pattrick Murphy and late barry wood were the first to obtained a purified form of endogenous pyrogen from rabbit peritoneal exudate cells.

In 1972, Gery and Waksman described the chemical nature of " lymphocyte-activating factor" which showed striking similarity with endogenous pyrogens.

THERMOREGULATION

Pathophysiology of fever:.

The febrile response is a complex physiologic reaction to disease involving a numerous endocrinologic and immunologic systems. Understanding the basic mechanisms underlying this phenomenon helps to formulate rational approaches to treatment and interventions.

Fever is defined as an elevation of the normal body temperature that above the normal circadian variation as a result of the changes in the, thermoregulatory center, located in the hypothalamus and occurs in conjunction with an increase in hypothalamic set point e.g 37°c to 39°c .

Normally heat is being continuously produced in the body, and also it is being lost continuously to the surroundings. When the rate of heat produced is equal to the rate of heat loss, the person is said to be heat balanced. But when there is is disturbance of equilibrium between the two, then the body temperature may rise leading to fever, or may fall leading to hypothermia.

Once the hypothalamic set point is increased, neurons in vasomotor center are stimulated and vasoconstriction started. The individual first notices vasoconstriction in the hand and feet. Shunting of blood away from

the periphery to the internal organs essentially decrease heat loss from the skin , and the person feel cold. For most fever, body temperature increase by 1°c to 2°c . Shivering, which increase heat production from the muscles, may begin at this time; however, shivering is not required if heat conservation mechanism raises body temperature sufficiently.

The process of heat conservation and heat production (shivering) continue until the temperature of body bathing the hypothalamic neurons matches the new thermostat setting. Once the point is reached , the hypothalamus maintain the temperature at the febrile level by the same mechanism of heat balance that function in the aferile state.

When the hypothalamic set point is again reset downward in response to either a decrease in concentration of pyrogens or use of drugs, the processes of heat loss through vasodilation and sweating are initiated. Loss of heat by sweating and vasodilation continue until the body temperature at the hypothalamic level matches the lower setting.

The hypothalamus is central to this process, functioning as a thermostat, controlling thermoregulatory mechanisms that balance heat production with heat loss. Integral to the process are the heat-sensitive receptors located in the preoptic area of the anterior hypothalamus. These receptors, which are sensitive to elevations in blood temperature, increase signal output as the temperature rises above a fixed thermal set point (37.1°C average) and decrease output when the temperature drops below the set point.

Similar receptors are in the skin, spinal cord, and abdomen, sending impulses to the hypothalamus via the spinal cord.

With core body temperature elevations, the sympathetic system is inhibited, leading to vasodilation of skin vessels and stimulation of the sweat glands to facilitate evaporative loss. This process prevails until the body temperature matches the thermal set point, when heat production matches heat loss.

Similarly, when body temperature is below the thermal set point, a variety of responses are initiated to conserve and increase production of heat. They include activation of the sympathetic nervous system to induce vasoconstriction of skin blood vessels; inhibition of sweating; activation of the shivering center in the posterior hypothalamus, thereby increasing muscle heat production; and secretion of neurotransmitters, which increase cell metabolism and, consequently, heat production.[9]

NEURAL MECHANISM OF THERMOREGULATORY RESPONSE:

No single center within the nervous system controls body temperature. Rather, thermoregulation is a process that involves a continum of neural structures and connections extending from the hypothalamus and limbic system through the lower brainstem and reticular formation to the spinal cord and sympathetic ganglia. Nevertheless, an area of the brain located in and near the rostral hypothalamus

seems to have a pivotal role in the process of thermoregulation. Although generally referred to as the *preoptic region,* it actually includes the medial and lateral aspects of the preoptic area, anterior hypothalamus, and septum. Numerous studies extending more than 60 years have established that neurons located in this region are thermo sensitive and exert at least partial control over physiologic and behavioural Thermoregulatory responses.[8]

NEURONAL MODEL EXPLAINING FEVER:

Endogenous agents and various drugs affect temperature by altering the activities of Hypothalamic neurons. The best examples are pyrogens that cause fever by elevating the set point temperature. Pyrogen inhibition of warm sensitive neurons will raise the regulated set point temperature to a higher level such as 39^0c. In response to the new set point temperature, thermoregulatory mechanism are activated to increase the pre optic temperature to 39^0c, thus leading to development of fever.

The whole body metabolic rate increase relative to the febrile state because, as result of the Q10 effect, increase in temperature induces increase in all metabolic reaction.

PYROGENS:

Pyrogens traditionally have been divided into 2 general categories: those that originate outside the body (exogenous pyrogens) and those derived from host cells (endogenous pyrogens). Exogenous pyrogens are, for the most part, microbes, toxins, or other products of microbial origin, whereas

endogenous pyrogens are host cell–derived (pyrogenic) cytokines that are the principal central mediators of the febrile response.[5].

EXOGENOUS PYROGENS:

Infection can be local or systemic in which Gram-positive or Gram-negative organisms release cell wall products such as endotoxins, peptidoglycans, teichoic acids and others. Also, enterotoxins from Staphylococcus aureus and other products from Gram-positive organisms are released. Gaining access to the circulation, for example in pneumonia entering the pulmonary vein and exiting via the carotid artery, microbial products reach the organum vasculosum laminae terminalis (OVLT) of the hypothalamus. The endothelium of the OVLT expresses LR receptors and microbial products such as endotoxins bind to their respective TLR on the OVLT and activate the endothelium. Microbial products also bind to TLR on phagocytic cells (macrophages, neutrophils, Kupffer cells) or to TLR on systemic endothelial cells. Pyrogenic cytokines IL-1, TNF-α, IL-6 and other cytokines are synthesized, processed, released and gain access to the circulation. In the case of enterotoxins, binding occurs on the T-cell receptor (in which case enterotoxins are called superantigens) resulting in production of IL-1 and TNF-α.. These pyrogenic cytokines enter the circulation and bind to their respective cytokine receptors on the OVLT. Activation of TLR and cytokine receptors induce COX-2, which results in synthesis of PGE2

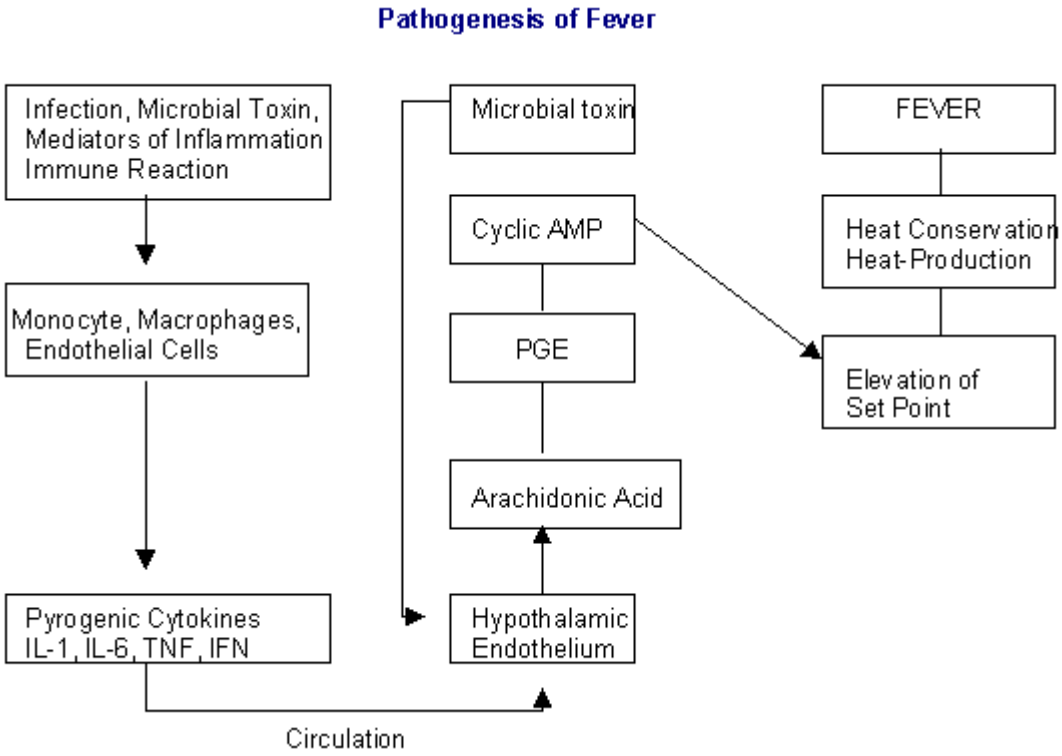
on the brain side of the OVLT. Increases in brain PGE 2 stimulate the release of cAMP and other neurotransmitters triggering thermo sensitive neurons in the thermoregulatory center to raise the hypothalamic thermostatic set point. Neuronal signals to the cortex initiate behavioural changes in humans to conserve body heat (posturing, clothing and others). Hypothalamic signals activate peripheral efferent nerves to blood vessels via the sympathetic pathways. Vasoconstriction of the blood vessels supplying the peripheral circulation decrease heat loss and core temperature increases (measured as fever). The resulting increase in blood temperature is detected by the hypothalamic center, which maintains these mechanisms of heat conservation (and behavioral changes) until hypothalamic PGE 2 levels fall (as a result of inhibitors of COX-2 and/or COX-3).

PGE2 RELEASE

PGE2 release comes from the arachidonic acid pathway. This pathway is mediated by the enzymes phospholipase A2 (PLA2), cyclooxygenase-2 (COX-2), and prostaglandin E2 synthase(6). These enzymes mediate the synthesis and release of PGE2. PGE2 is the ultimate mediator of the febrile response. During fever, levels of PGE_2 are elevated in hypothalamic tissue and the third ventricules. The circumventricular vascular organ surrounding the hypothalamic regulatory centers have the highest concentration of PGE2. Destruction of theses organs reduces the ability of pyrogens to produce the fever. Circulating cytokines by their systemic effect induces the synthesis of PGE2 which causes the fever.

The increase in PGE2 in the periphery account for the nonspecific myalgias and arthralgias that often accompany fever. It is thought that some systemic PGE2 escapes destruction by the lung and gain access to the hypothalamus via the internal carotid. However, it is the elevation in the brain that starts the process of raising the hypothalamic set point for core temperature.

There are four receptors for PGE2, and each signals the cell in different ways. Of the four receptors, the third(EP-3) is essential for fever: when the gene for this receptor is deleted in mice, no fever follows the injection of IL-1 or endotoxin. Deletion of the other PGE2 receptor genes leaves the fever mechanism intact. Although PGE2 is essential for fever, it is not a neurotransmitter. Rather, the release of PGE2 from the brain side of the hypothalamic endothelium triggers the PGE2 receptor on glial cells, this stimulation results in the rapid release of cyclic AMP, Which is a neurotransmitter. The release of cyclic AMP from glial cells activates neuronal endings from the thermoregulatory center extend in to the area. The elevation of cyclic AMP is thought to account for changes in the hypothalamic set point either directly or indirectly. Distinct receptors for microbial product are located on the hypothalamic endothelium. These receptor are called toll- like receptors and are similar in many ways to IL-1 receptors. The direct activation of toll-like receptor also result in PGE2 production and fever.[9]



CYTOKINES AS ENDOGENOUS PYROGENS:

In essence, all endogenous pyrogens are cytokines, molecules that are a part of the innate immune system. They are produced by phagocytic cells and cause the increase in the thermoregulatory set-point in the hypothalamus. Major endogenous pyrogens are interleukin 1 (α and β), interleukin 6 (IL-6) and tumour necrosis factor-alpha. Minor endogenous pyrogens include interleukin-8, tumour necrosis factor-α, tumour necrosis factor-β, macrophage inflammatory protein-α and macrophage inflammatory protein-β as well as interferon-α, interferon-β, and interferon-γ.

These cytokine factors are released into general circulation, where they migrate to the circumventricular organs of the brain due to easier absorption caused by the blood-brain barrier's reduced filtration action there. The cytokine factors then bind with endothelial receptors on vessel walls, or interact with

local microglial cells. When these cytokine factors bind, the arachidonic acid pathway is then activated.

Some endogenous molecules can also induce endogenous pyrogens not requiring exogenous stimuli, for e.g. antigen-antibody complexes, certain androgenic steroid metabolites, inflammatory bile acids, and some lymphocytes product.

INTERLEUKIN (IL) -1

IL-1α is produced mainly by activated macrophages, as well as neutrophils, epithelial cells, and endothelial cells. In general, Interleukin 1 is responsible for the production of inflammation, as well as the promotion of fever and sepsis.

Interleukin 1 was discovered by Gery in 1972. He named it lymphocyte-activating factor (LAF) because it was a lymphocyte mitogen. It was not until 1985 that interleukin 1 was discovered to consist of two distinct proteins, now called interleukin-1 alpha and interleukin-1 beta.

IL-1α is also known as fibroblast-activating factor (FAF), lymphocyte-activating factor (LAF), endogenous pyrogen (EP), osteoclast-activating factor (OAF).

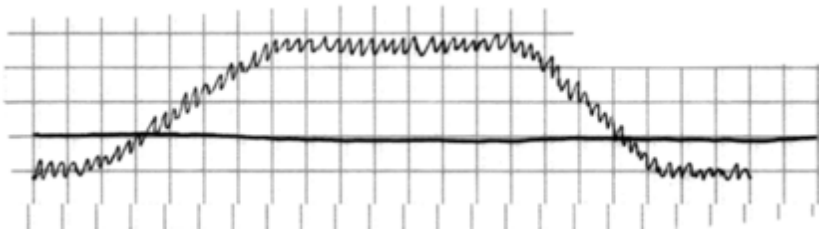
In Vivo Shortly after an onset of an infection into organism, IL-1α activates a set of immune system response processes.

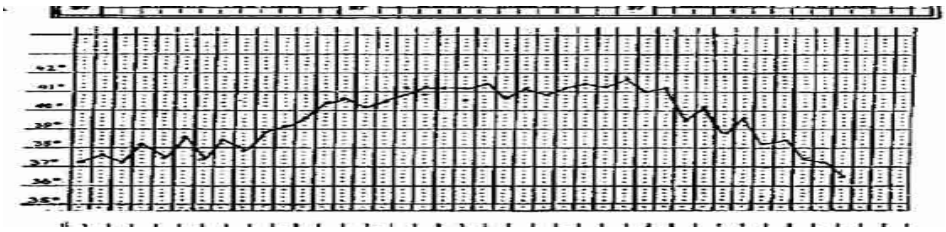
- induces synthesis of proteases, subsequent muscle proteolysis, release of all types of amino acids in blood and stimulates acute phase proteins synthesis
- stimulates fibroblasts proliferation
- increases blood neutrophils
- induces cycloxygenase synthesis and prostaglandin PGE2 release
- activates lymphocyte proliferation and induces fever
- changes the metallic ion content of blood plasma by increasing copper and decreasing zinc and iron concentration in blood

FEBRILE PATTERN:

Continuous fever: Temperature remains elevated above normal without touching the baseline and the fluctuation does not exceed 0.6c in 24 hours, *e.g.* lobar pneumonia, typhoid,urinary tract infection, brucellosis, or typhus. Typhoid fever may show a specific fever pattern, with a slow stepwise increase and a high plateau.

Continuous fever

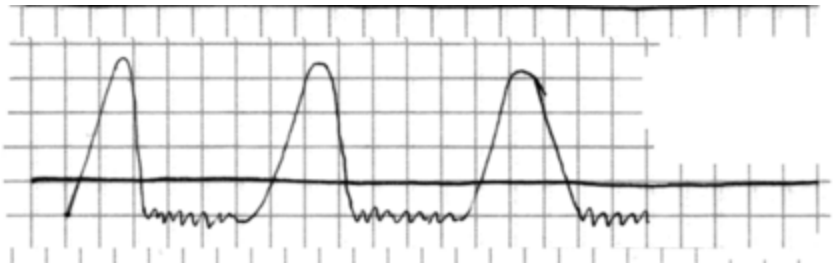




Intermittent fever: The elevated temperature touch the baseline in between. In hectic or septic type of intermittent fever, the diurnal variation elevation is extremely large, as occurs in septicaemia. Quotidian fever is a hectic fever occurring daily. *e.g.* malaria, kala-azar, or septicemia. Following are its types

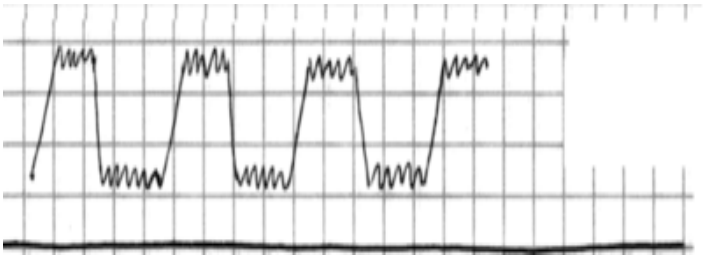
- Quotidian fever, with a periodicity of 24 hours, typical of Malaria
- Tertian fever occurs on the first and third day, e.g P. Vivax and ovale, falciparum.
- Quartan fever occurs on first and fourth day e.g *Plasmodium malariae*

Intermittent fever pattern



Remittent fever: the temperature fluctuation exceeds 0.6^0c, but without touching the baseline *e.g.*, infective endocarditis.

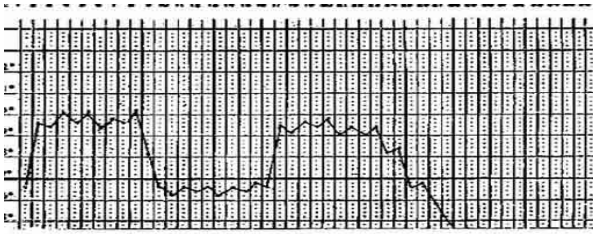
Remittent fever pattern



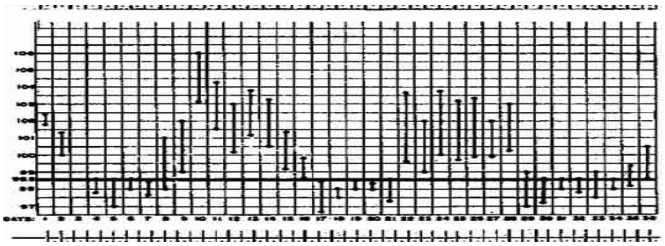
Relapsing fever pattern:

Febrile episode are separated by normal temperature for more than several days, e.g borrelia infection, rat bite fever.

Borrelia(relapsing pattern)

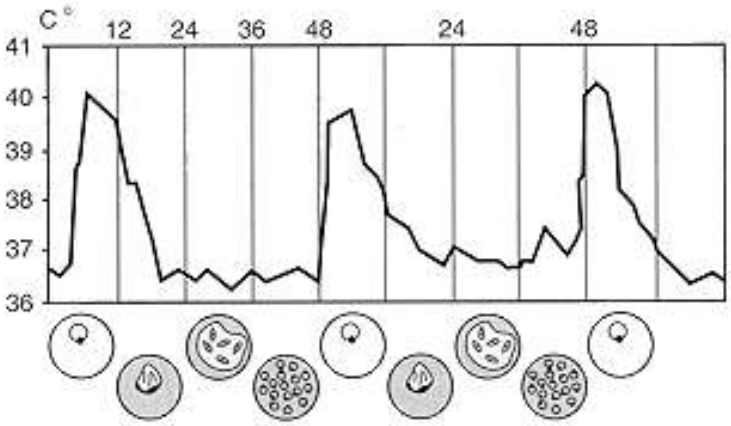


Pel-Ebstein fever: It is a type of fever lasting for 3 to 10 days followed by an afebrile period of 3 to 10 days e.g hodgkins lymphomas

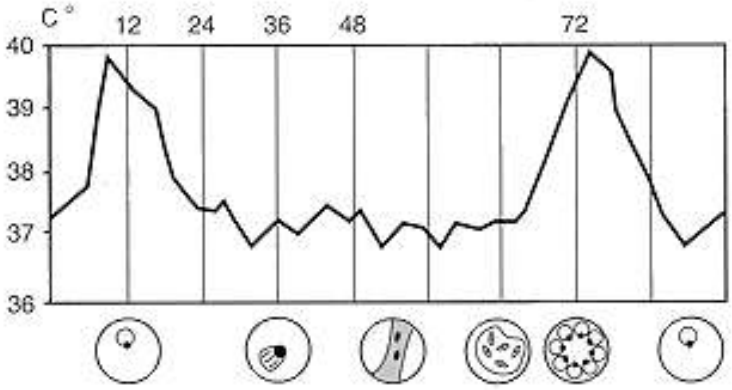


Malaria fever chart

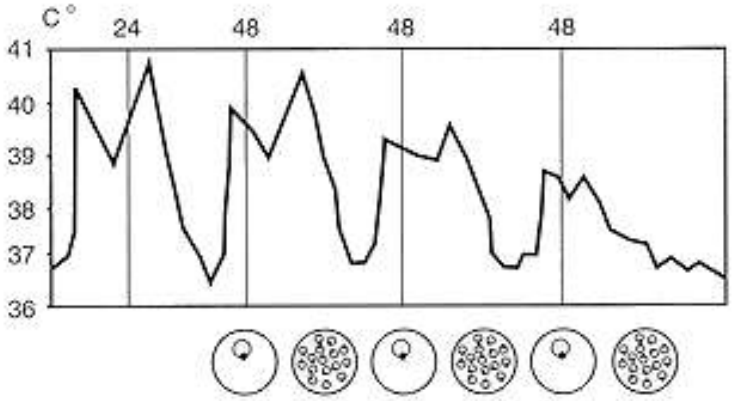
Typical temperature chart of *P vivax* infection showing tertian periodicity related to the maturation and rupture of erythrocytic schizonts



Typical temperature chart of *P malariae* infection showing quartan periodicity



Typical temperature chart of *P falciparum* infection showing irregular tertian periodicity and the influence of successful treatment



SADDLE BACK (BIPHASIC FEVER):

With several days of fever, gap of reduced fever of about 1 day, and several additional days of fever, this type characterises dengue and yellow fever, rift valley fever, Colorado tick fever and viral fever like influenza, polio.

Fever magnitude:

Most infectious disease produce temperature between 99^0F to 106°F . However some patient with infectious disease remain afebrile. These includes immunocompromised host, those with CRF, elders, alcoholic.

Hyperpyrexia is a elevation of body core temperature greater than or equal to 41 °C (106.7 °F) due to inadequate dissipation of heat. It is a medical emergency, since they are prone for sudden cardio respiratory arrest. Such a high temperature is considered a medical emergency as it may indicate a serious underlying condition or lead to significant side effects. The most common cause is an intracranial haemorrhage.[15] Other possible causes include sepsis, Kawasaki syndrome,[17] neuroleptic malignant syndrome, drug effects, serotonin syndrome, and thyroid storm.[16] Infections are the most common cause of fever, however as the temperature rises other causes become more common. Infections commonly associated with hyperpyrexia include: roseola, rubeola and enteroviral infections.

WHY ARE FEVER TEMPERATURE 0VER 106°F RARE?

Hippocrates maintained that " heat is the immoratal substance of life endowed with intelligence...." hence, heat must also be refrigerated by respiration and kept within bounds if source or principle of life is to persist; for if refrigeration is not provided the heat will consume itself'.

THROMBOCYTOPENIA

Platelets, or **thrombocytes** (from Greek , "clot" "), are small, irregularly shaped clear cell fragments (i.e. cells that do not have nucleus containing DNA), 2–3 μm in diameter, which are derived from fragmentation of precursor megakaryocytes. The average lifespan of a platelet is normally just 5 to 9 days.

- The physiological range for platelets is $(150–400)\times10^9$ per liter.
- Around 10^{11} platelets are produced each day by an average healthy adult.

Megakaryocyte and platelet production is regulated by thrombopoietin, a hormone usually produced by the liver and kidneys.

PRODUCTION OF PLATELET

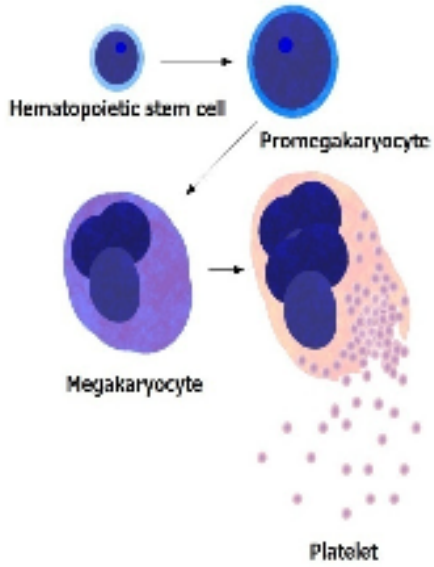


TABLE: 1

Causes of thrombocytopenia:

Decreased marrow production	**Marrow infiltration with tumours, aplastic, hypoplastic anemia, , B12 or folic acid deficiency, viral infection, thrombopoietin deficiency.**
Spleenic sequestration of circulating platelet	hypersplenism Hypothermic anaesthesia
increased destruction of circulating platelet	Non immune mechanism like DIC, TTP, HUS, massive blood transfusion,
	immune mechanism like ITP, SLE, HIV, drug induced, sarcoidosis autoimmune thyroiditis.

THROMBOCYTOPENIA ASSOCIATED WITH INFECTION:

Purpura was recognised as a manifestation of **peltissutial f**ever 2000 year ago. Several factor known to cause bleeding in associated with infection of which thrombocytopenia is the common cause.

Viral causes:

Dengue, HIV, HSV, CMV, Hanta virus.

Mechanism:

Many virus causes thrombocytopenia, out of this dengue is the most common cause.[9]

Most patients who develop dengue hemorrhagic fever or dengue hemorrhagic shock syndrome have had prior infection with one or more dengue serotypes. When an individual is infected with another serotype (ie, secondary infection), these non neutralizing antibodies recognize the dengue virus but do not neutralize or inhibit virus replication.

Instead, the virus and antibody form an antigen-antibody complex. This complex is recognized by receptors on macrophages, which then internalize the immune complex and allow the virus to replicate unchecked. This phenomenon is called antibody-dependent enhancement.

The affected macrophages release vasoactive mediators that increase vascular permeability, leading to vascular leakage, hypovolemia, and shock. This

mechanism, along with individual host and viral genome variations, plays an active role in pathogenesis.

Some researchers suggest that T-cell immunopathology may play a role, with increased T-cell activation and apoptosis. Increased concentrations of interferon have been recorded 1-2 days following fever onset during symptomatic secondary dengue infections. The activation of cytokines, including TNF-alpha, TNF receptors, soluble CD8, and soluble IL-2 receptors, has been correlated with disease severity.[20]

Mechanism of thrombocytopenia in HIV:

Thrombocytopenia is found in 3-40% of individuals with HIV infection. It can be the first presentation, and may occur at any stage of HIV infection.

HIV-related thrombocytopenia can be caused by the following:

Immune thrombocytopenic purpura

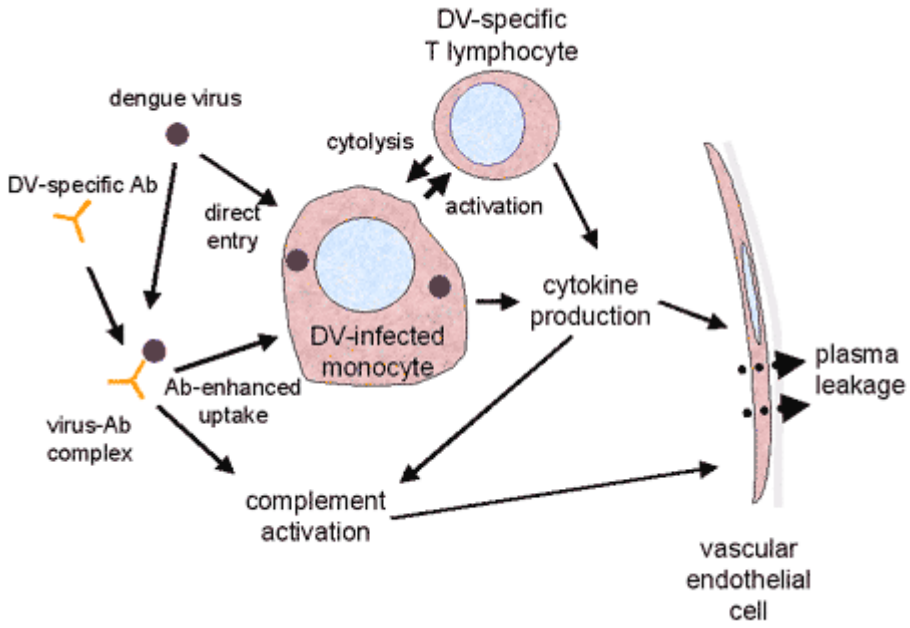
Immune thrombocytopenic purpura (ITP) is very common in HIV infection. This condition, characterised by very low platelet counts with an otherwise normal haematocrit and white blood cell count, is caused by an immune-mediated destruction of platelets. Patients with ITP may bruise easily or have petechiae, and bleeding from the gums or other sites. Serious haemorrhage is uncommon.[27,29]

Thrombotic thrombocytopenic purpura

Thrombotic thrombocytopenic purpura (TTP) is very rare, in comparison to ITP.

TTP presents with fever, haemolytic anaemia, thrombocytopenia, renal and neurological problems, caused by thrombi forming in small blood vessels affecting multiple organs. The diagnosis is confirmed by fragmented red blood cells (schistocytes) found on peripheral blood smear. Pregnancy and disseminated intravascular coagulation should be excluded. TTP is a medical emergency requiring plasmapheresis.[16][34,35]

PATHOGENSIS



BACTERIAL CAUSE:

Gram +ve and –ve septicaemia, military tuberculosis, leptospirosis, typhoid, mycoplasma pneumonia.

Tuberculosis is one of the world's most challenging communicable diseases. Bone marrow changes include changes in marrow cellularity

manifesting as myeloid hyperplasia that occurs more commonly with pulmonary tuberculosis, plasmacytosis, megaloblastic changes in cell lineages, and marrow aplasia. Miliary tuberculosis is associated with histiophagocytosis of all cell lineages and caseating granuloma formation in 60–70% cases. Other marrow changes in tuberculosis include marrow necrosis and myelofibrosis.[18] Thrombocytopenia in tuberculosis can occur due to marrow suppression as a side effect of histiophagocytosis as described above, tuberculosis complicated by thrombotic thrombocytopenic purpura (TTP), or disseminated intravascular coagulopathy (DIC), or due to immune-mediated platelet destruction. The immune basis of tuberculosis-induced thrombocytopenia is supported by the presence of either the platelet antigen-specific antibodies or platelet surface membrane IgG, or by response to immunomodulatory therapy .[19]

Protozoal causes:

Thrombocytopenia seen in complicated falciparum malaria is due to disseminated intravascular coagulation along with platelet endothelial activation, but the one seen in uncomplicated malaria like *P. vivax* has multifactorial etiology. Few postulated mechanisms are macrophage activation leading to platelet destruction, increased levels of cytokines, immunological destruction due to antiplatelet IgG6, oxidative stress, shortened platelet life span in peripheral blood and sequestration in nonsplenic areas and partly due to pseudo thrombocytopenia due to clumping of platelets.[12, 13]

Other cause:

Certain haematological condition also caused thrombocytopenia by marrow infiltration.

General view of burden of common infectious causes in india:

Dengue:

Dengue fever (DF) is the most common arthropod-borne viral disease and is one of the most important emerging infectious diseases of urban and peri-urban areas of tropical regions.

Since the 18th century, dengue has caused repeated epidemics worldwide. H. Graham in 1903 implicated *Aedes aegypti* as the vector for the disease and the virus was isolated in 1944 by Albert Sabin *et al*1. Dengue haemorrhagic fever gained nosologic status in 1954 and subsequently became endemic in many areas of tropical Asia. India belongs to category B, where dengue is an emerging disease with cyclical epidemics becoming more frequent. There have been repeated epidemics with the latest outbreak in the capital city of Delhi and various other states of India.[30]

AETIOLOGY

(a) Causative agent:

Four dengue viruses (types 1-4) within the genus *flavivirus* and family *flaviviridae*, are the causative agents. All four subtypes are found in India. Dengue virions are small particles with lipoprotein envelope and nucleocapsid of single stranded RNA genome with positive polarity. There is a close

antigenic similarity between the four serotypes but the cross protection in humans is at best partial and transient.

(b) Transmission:

Reservoir of infection is both man and mosquito. The transmission cycle is "Man-Mosquito-Man". *Aedes aegypti* is the principal vector, the other less important vectors being *Aedes albopictus, Aedes polynesiensis,* and several species of *Aedes scutellaris* complex1, 2. The extrinsic incubation period is 8-11 days, with mosquitoes remaining infected for life. *Aedes aegypti* – also called the "tiger mosquito" – is a daybiter with limited flight range, and breeds in fresh water collected indoors and peri-domestically in tropical areas. The outbreaks coincide with the monsoon. The epidemiologic significance of transovarian mosquito cycle and jungle transmission cycle is unknown.[31,32]

EPIDEMIOLOGY

Dengue affects more than 100 countries all over the world except Europe. In India, the first recorded outbreak of dengue fever was in 1812 and serological survey was conducted in 1952. In 1996, an outbreak involving 10,000 cases occurred in New Delhi; 400 deaths were reported in the outbreak.([33]

MALARIA:

Malaria is a protozoan disease transmitted by the bite of infected anopheles mosquitoes. It is the most important of the parasite disease of human, with transmission in 107 countries containing 3 billion people and causing 1-3 million death each year. Approximately 5% of the world

population is infected. Malaria has infected humans for over 50,000 years, and *Plasmodium* may have been a human pathogen for the entire history of the species. Close relatives of the human malaria parasites remain common in chimpanzees. Some new evidence suggests that the most virulent strain of human malaria may have originated in gorillas.

References to the unique periodic fevers of malaria are found throughout recorded history, beginning in 2700 BC in China. Malaria may have contributed to the decline of the Roman Empire, and was so pervasive in Rome that it was known as the "Roman fever". A number of regions in ancient Rome were considered at-risk for the disease because of the favourable conditions present for malaria vectors. This included areas such as: southern Italy, the island of Sardinia, the Pontine Marshes, the lower regions of coastal Etruria and the city of Rome along the Tiber River. The presences of stagnant water in these places were preferred by mosquitoes for breeding grounds. Irrigated gardens, swamp-like grounds, runoff from agriculture, and drainage problems from road construction led to the increase of standing water.

The term malaria originates from Medieval Italian: *mala aria* — "bad air"; the disease was formerly called *ague* or *marsh fever* . [22]

DISCOVERY OF THE PARASITE:

On 20 october 1880 charles Louis alphonse was examining the fresh blood of patient with ague, and observed moving bodies which he surmised correctly were parasites of red blood cells. The transmissibility of the infection in blood was proved 4 years later, by Gerhardt, but the route of natural

infection was not discovered until next decade. He, therefore, proposed that malaria is caused by this organism. For this and later discoveries, he was awarded the 1907 Nobel Prize for Physiology of Medicine. The malarial parasite was called *Plasmodium* by the Italian scientists Ettore Marchiafava and Angelo Celli.

Malaria is a mosquito-borne infectious disease of humans and other animals caused by eukaryotic protists of the genus *Plasmodium*. The disease results from the multiplication of Plasmodium parasites within red blood cells, causing symptoms that typically include fever and headache, in severe cases progressing to coma or death. It is widespread in tropical and subtropical regions, including much of Sub-Saharan Africa, Asia, and the Americas.

Five species of *Plasmodium* can infect and be transmitted by humans. Severe disease is largely caused by *Plasmodium falciparum* while the disease caused by *Plasmodium vivax*, *Plasmodium ovale*, and *Plasmodium malariae* is generally a milder disease that is rarely fatal. *Plasmodium knowlesi* is a zoonosis that causes malaria in macaques but can also infect humans.[23]

There were an estimated 225 million cases of malaria worldwide in 2009.[8]. An estimated 781,000 people died from malaria in 2009 according to the World Health Organization's 2010 World Malaria Report, accounting for 2.23% of deaths worldwide. Ninety percent of malaria-related deaths occur in sub-Saharan Africa, with the majority of deaths being young children. *Plasmodium falciparum*, the most severe form of malaria, is responsible for the vast majority of deaths associated with the

disease.[10] Malaria is commonly associated with poverty, and can indeed be a cause of poverty[11] and a major hindrance to economic development.(24,25)

LEPTOSPIROSIS:

Leptospirosis (also known as Weil's syndrome, canicola fever, canefield fever, nanukayami fever, 7-day fever, Rat Catcher's Yellows, Fort Bragg fever, black jaundice and Pretibial fever) is caused by infection with bacteria of the genus *Leptospira*, and affects humans as well as other mammals, birds, amphibians, and reptiles.

The role of the rat as a source of human infection was discovered in 1917. while the potential for leptospiral disease in dogs was recognized, but clear distinction between canine infection with *L. interrogans* serovars icterohaemorrhagiae and canicola took several years. Leptospirosis in livestock was recognized some years later. The infection is commonly transmitted to humans by allowing water that has been contaminated by animal urine to come in contact with unhealed breaks in the skin, the eyes, or with the mucous membranes. Outside of tropical areas, leptospirosis cases have a relatively distinct seasonality with most of them occurring in spring and autumn.(15)

COMPLICATION:

Platelet count>1 lakh, are usually asymptomatic and bleeding time remains normal. Platelet count of 50,000 and 1,00,000 cause mild prolongation of the bleeding time only after sever trauma.

Platelet count of<50,000 have easy bruising , manifested by skin purpura after minor trauma.

Platelet count <20,000 have spontaneous bleeding, they usually have petechiae and may have intracranial or spontaneous internal bleeding.

INVESTIGATION:

Many investigation should be included to work up of patient with fever and thrombocytopenia which including biochemical test, peripheral smear, haemogram etc.

a) Complete haemogram:
    1. ESR: >30mm/hr suggest – TB; malignancy. It is a non specific test, it is raised in most conditions.
    2. Leucopenia: early dengue
    3. Leucocytosis: predominately neutrophils indicates septicaemia.
    4. Peripheral smear: malarial parasites, malignant cells.
b) Rapid spot test:

    For plasmodium vivax and plasmodium palciparum

    It is very sensitivity test.
c) WIDAL – tube method for identification of enteric fever
d) IgM ELISA dengue:
e) IgM ELISA leptospirosis antibody;
f) Blood culture: at least 3 blood culture sample should be taken.
g) Bone marrow aspiration/ biopsy: for leukemia, lymphoma etc.

So in patient with fever and thrombocytopenia with renal and liver parameter being abnormal, then consider following

1. Leptospirosis
2. Malaria
3. Dengue
4. Septicaemia with multi organ failure.

Platelet count should also be repeated and observed for bleeding manifestation.

Platelet transfusion are indicated when platelet count is <20,000. Treating the underlying condition will result in drastic improvement of platelet count and its complication.

Materials and Methods

MATERIALS AND METHODS

The present study was done in patients admitted to Coimbatore medical college ,Coimbatore, tamilnadu over a period of one year.

SELECTION CRITERIA:

All patients more than 12years of age with fever (temperature >99.9^{0}F) and platelet count less than 1,50,000 cells/cu.mm.

No. Of patients selected: 100

EXCLUSION CRITERIA:

All patients less than 12 yrs of age.

All patients with thrombocytopenia without fever.

Diagnosed cases of platelet disorders and dysfunction

Patients on treatment with antiplatelet drugs and other drugs causing thrombocytopenia.

PEROID OF STUDY:

All patients were age of ≥ 12 years who were admitted as

in-patients between dec 2010 to nov 2011 in the Medicine Department in Coimbatore Medical College Hospital for fever with thrombocytopenia.

STUDY DESIGN: Prospective study

METHODS

All patients admitted in Coimbatore Medical College Hospital with fever and thrombocytopenia were evaluated. History was taken regarding duration of fever, occupation and history of travel. Symptoms other than fever, headache, nausea, vomiting, abdominal pain, diarrhea, cough, , anorexia, myalgia, gum bleeding, hemetemesis , conjunctival suffusion, oliguria, hematuria, loss of weight, etc., were noted.

Signs like rashes, signs of dehydration, petechiae, jaundice, lymphadenopathy, hepatomegaly, splenomegaly, anaemia, abdominal tenderness, added sounds in lungs , altered sensorium, etc., were also noted.

Investigations like complete hemogram, ESR, Liver function tests, routine urinary examination, urine for bile salts and bile pigments, Renal parameters like blood urea, serum creatinine, serum electrolytes, peripheral smear , xray chest, USG abdomen were done on admission.

Other special investigations like peripheral smear for MP, dengue serology, widal study, IgM antibody for leptospirosis, sputum AFB, ELISA for HIV1 and 2, blood culture and urine culture, bone marrow aspiration.

During the hospital stay, all the patients were subjected repeat CBC once in 2 days. The renal function tests were repeated every third day unless the patient developed ARF for whom the tests were done daily. Follow up of all patients regarding treatment and outcome were done during the hospital stay.

The causes of fever with thrombocytopenia are so numerous, a simple workable classification is presented in-

1. Viral causes:

   CMV, HSV, HIV, hantana, dengue, parvo-B19

2. Bacteria:

   Gram +ve and gram-ve septicaemia, military tuberculosis, leptospirosis, typhoid.

3. Protozoal causes:

   Malaria

4. Others:

   Leukemia, lymphoma etc

Observations and Results

RESULTS AND ANALYSIS

Analysis of clinical symptoms, laboratory profile and complication of 100 patients presented with fever with thrombocytopenia admitted at Coimbatore medical college and hospital, between December 2010 to November 2011, who met the inclusion criteria was done.

Total number of cases admitted with fever with thrombocytopenia is 100. Out of this 100 cases, 70 cases were male and 30 cases were female with male female ratio is 2.3:1. Most of the cases admitted between age 26 to 35. The mean age for male and female cases was 33.76 and 33.1 respectively.

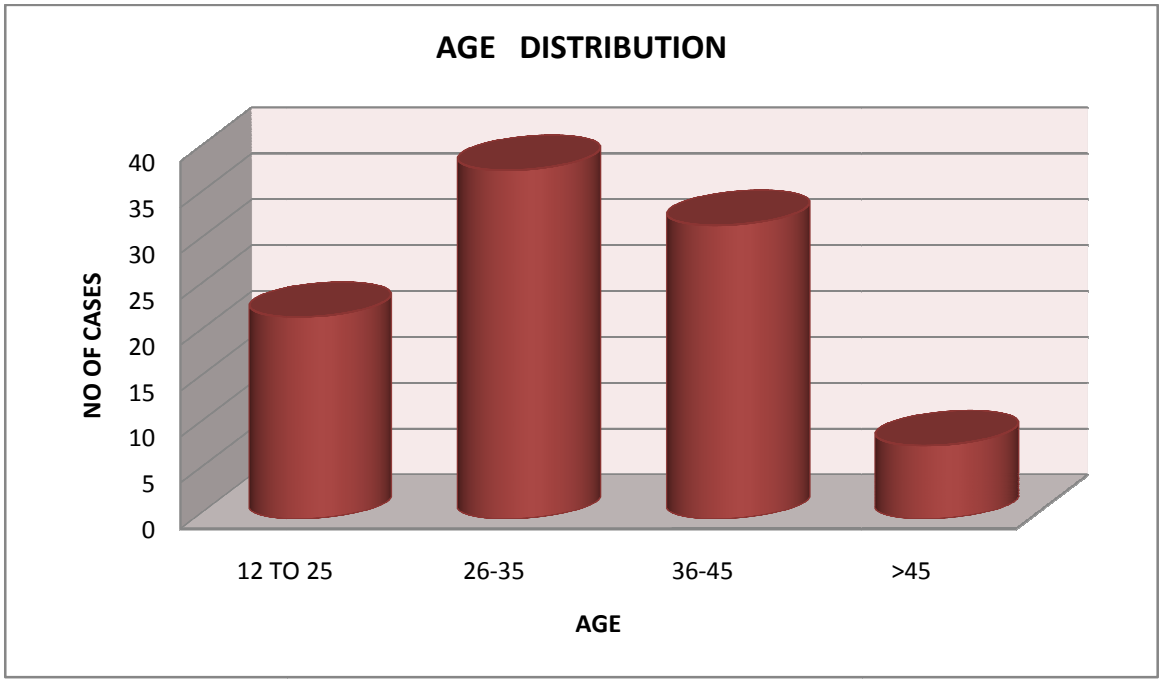
The age and sex distribution is given below fig 1 , 2, and 3.

AGE WISE DISTRIBUTION OF CASES

Table 2

AGE	NO. OF CASES	PERCENTAGE
12 -25	22	22%
26-35	38	38%
35-45	32	32%
>45	8	8%

GRAPH 1

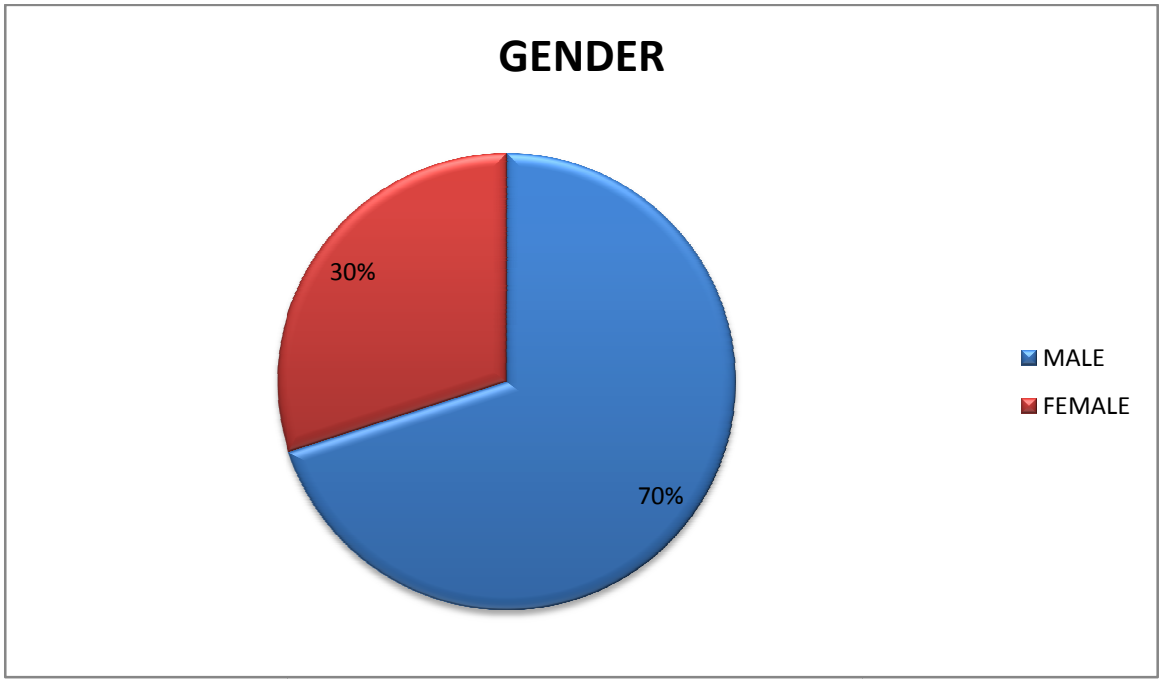


SEX WISE DISTRIBUTION

Table 3

SEX	NO OF CASES	PERCENTAGE
MALE	70	70%
FEMALE	30	30%

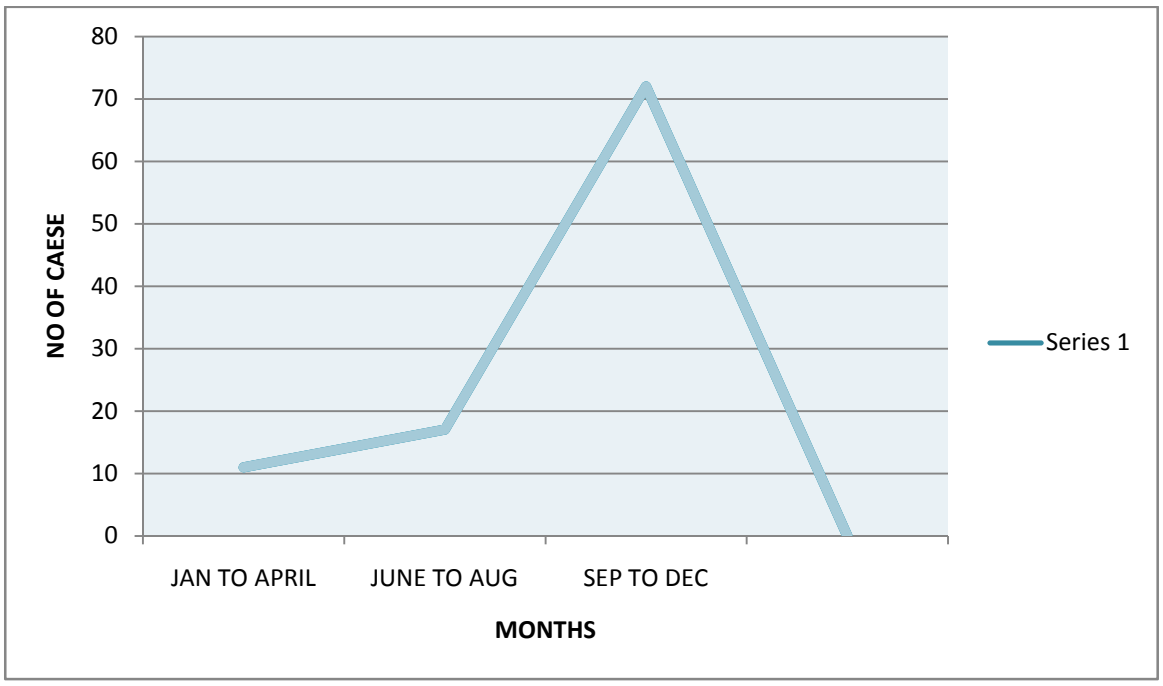
GRAPH 2



SEASONAL VARIATION

Out of 100 cases, 50% of fever with thrombocytopenia admitted between September and November month. The seasonal variation distribution is given below.

GRAPH 3



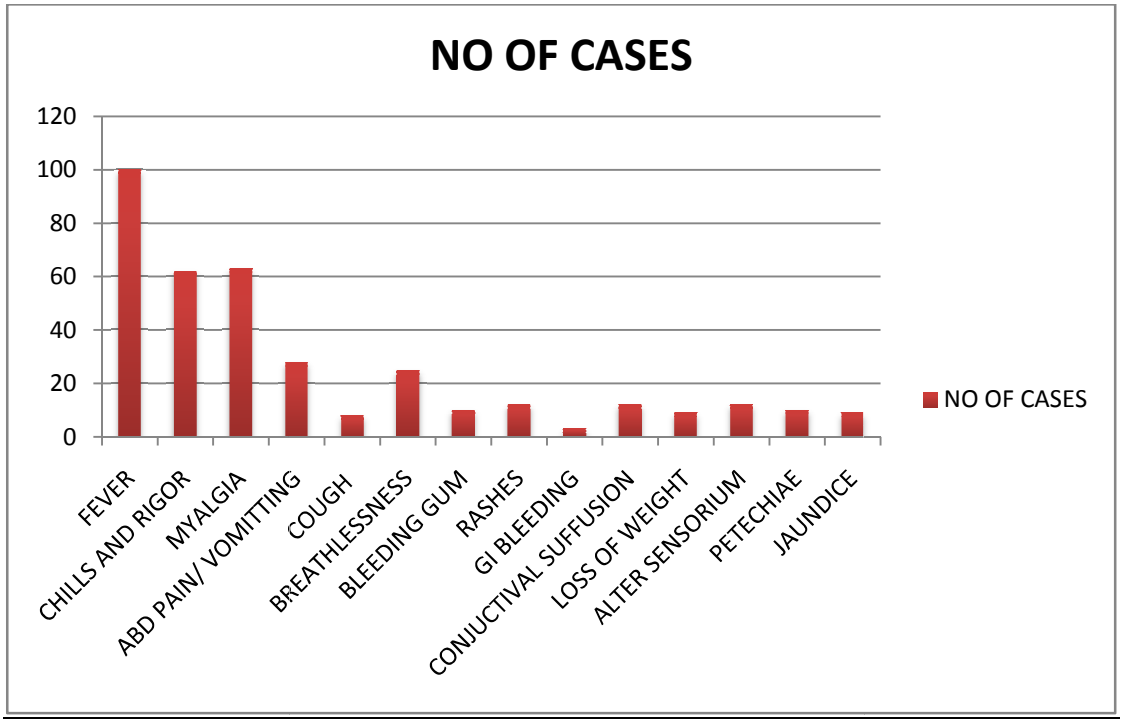
DISTRIBUTION OF SYMPTOMS

Most common presenting symptoms is fever with chills and rigor, myalgia followed by abdominal pain and vomiting.

TABLE 4

SI NO	SYMPTOMS	NO OF CASES	PERCENTAGE
1	FEVER	100	100%
2	CHILLS AND RIGOR	62	62%
3	MYALGIA	63	63%
4	ABD PAIN/ VOMITTING	28	28%
5	COUGH	8	8%
6	BREATHLESSNESS	25	25%
7	BLEEDING GUM	10	10%
8	RASHES	12	12%
9	GI BLEEDING	3	3%
10	CONJUCTIVAL SUFFUSION	12	12%
11	LOSS OF WEIGHT	9	9%
12	ALTERED SENSORIUM	12	12%

GRAPH 4



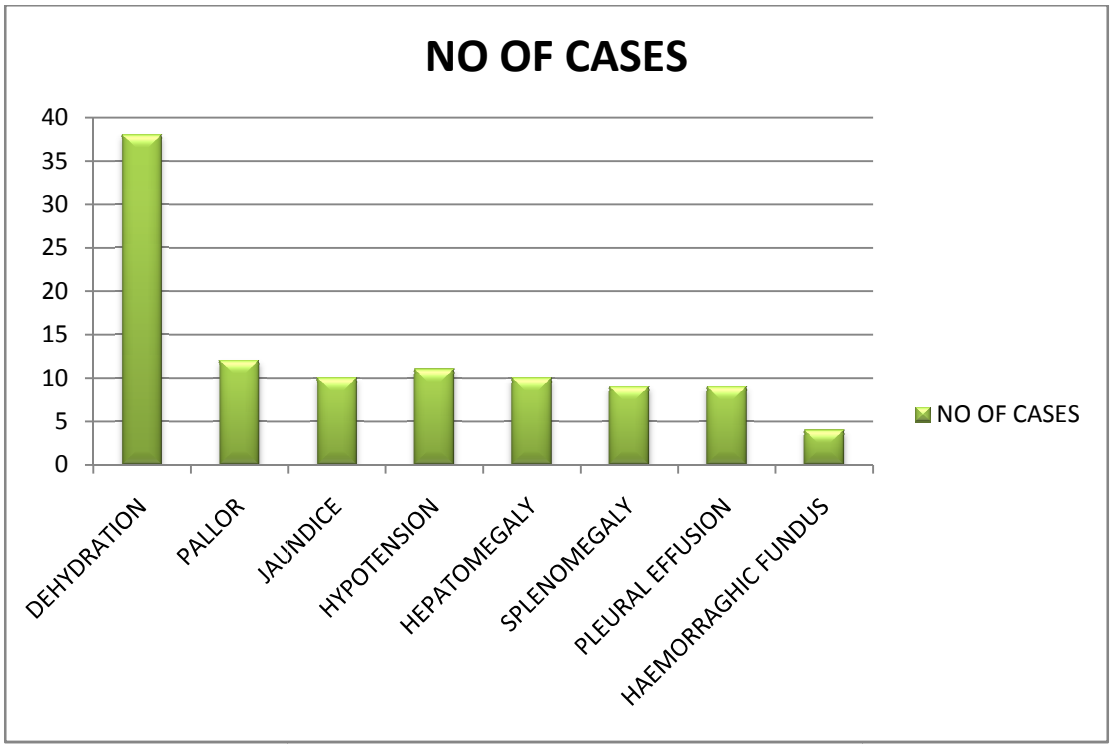
DISTRIBUTION OF SIGNS:

TABLE 5

SI NO	SIGN	NO OF CASES	PERCENTAGE
1	DEHYDRATION	38	38%
2	PALLOR	12	12%
3	JAUNDICE	10	10%
4	HYPOTENSION	11	11%
5	HEPATOMEGALY	10	10%
6	SPLENOMEGALY	9	9%
7	PLEURAL EFFUSION	9	9%
8	HAEMORRAGHIC FUNDUS	4	4%

SIGNS DISTRIBUTION

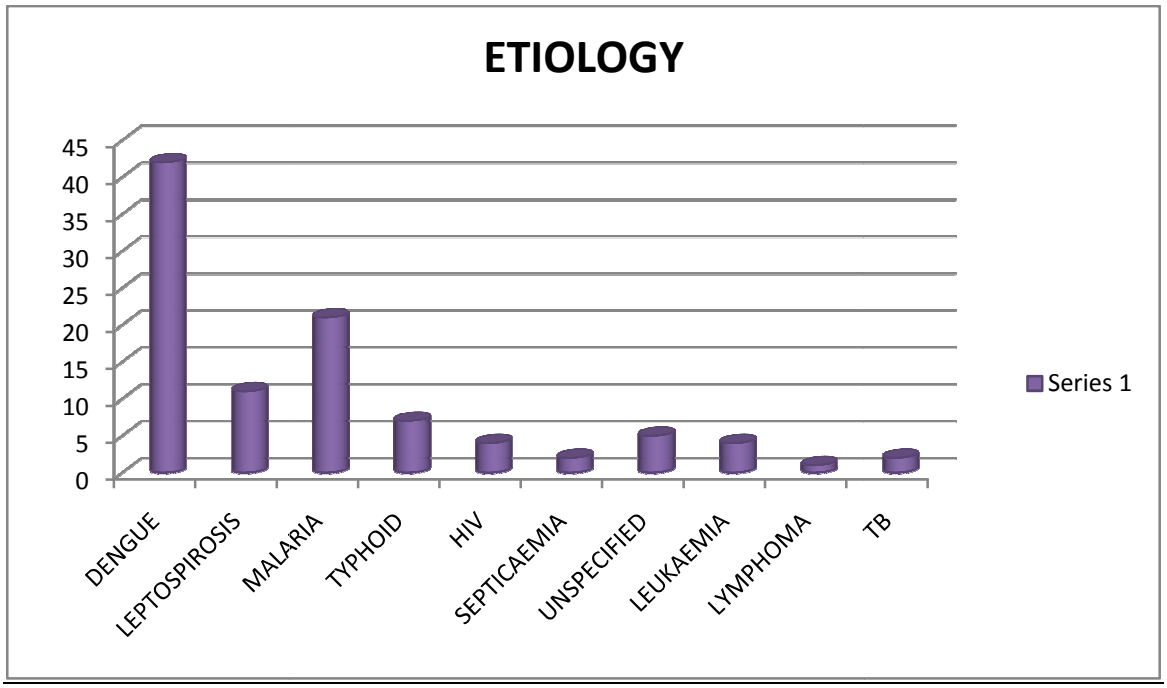
GRAPH -5



DISTRIBUTION OF ETIOLOGICAL AGENTS

Out of 100 cases, most common causes for fever with thrombocytopenia is dengue fever(42) , followed by malaria (21) and leptospirosis(11). Etiological distribution is given below

GRAPH 5



ANALYSIS OF LAB INVESTIGATION:

COMPLETE HEMOGRAM:

- Mean hemogloblin value was 11.8gm%
- Range 5.5- 14.4gm%

PLATELET COUNT:

- Mean platelet count was 52,175.00 / μ L

- Ranges from 5,500/ μ L to 1,20,000/ μ L
- Out of 100 cases with thrombocytopenia (<1,50,000), 60 cases were above 40,000. 17 cases were between 20,000 to 40,000. But 23 cases were below 20,000.

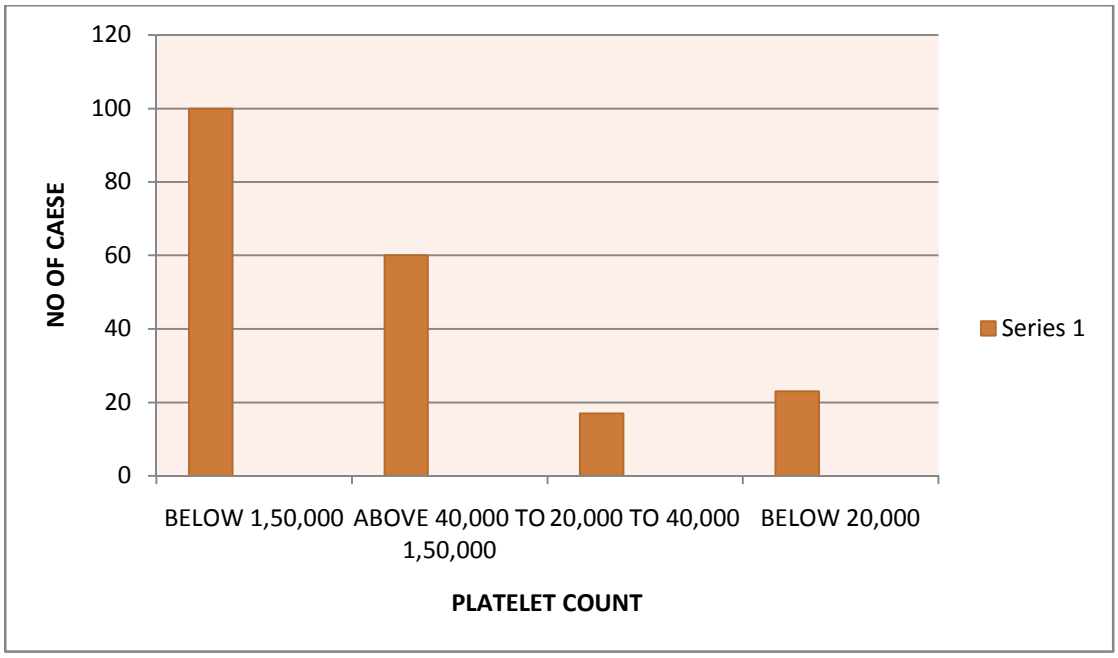
TABLE 6

PLATELET COUNT

SI.NO	PLATELET COUNT	NO OF CASES	PERCENTAGE
1	<1,50,000	100	100%
2	>40,000	60	60%
3	20,000 TO 40,000	17	17%
4	<20,000	23	23%

PLATELET COUNT

GRAPH 6



HAEMATOCRIT:

- Mean haemotocrit value was 32.05
- The value ranges from 21 to 55.

RENAL FUNCTION TEST:

Out of 100 cases, 12 cases had elevated renal parameter. Out of this 12 cases, 8 patients renal parameter improved with rehydration alone. Only 4 cases who needed nephrologist's intervention and dialysis.

TABLE 7

SI NO	BLOOD UREA	NO OF CASES
1	<40	88
2	40-60	5
3	60-100	2
4	>100	5

LIVER FUNCTION TEST:

Out of 100 cases, 13 cases had elevated liver function test. Out of this 13 cases , 6 patient had bilirubin level > 5.0mg/dl. Out of this 6 cases, 3 patients were died.

Distribution of bilirubin level, SGOT, SGPT, ALP is given below.

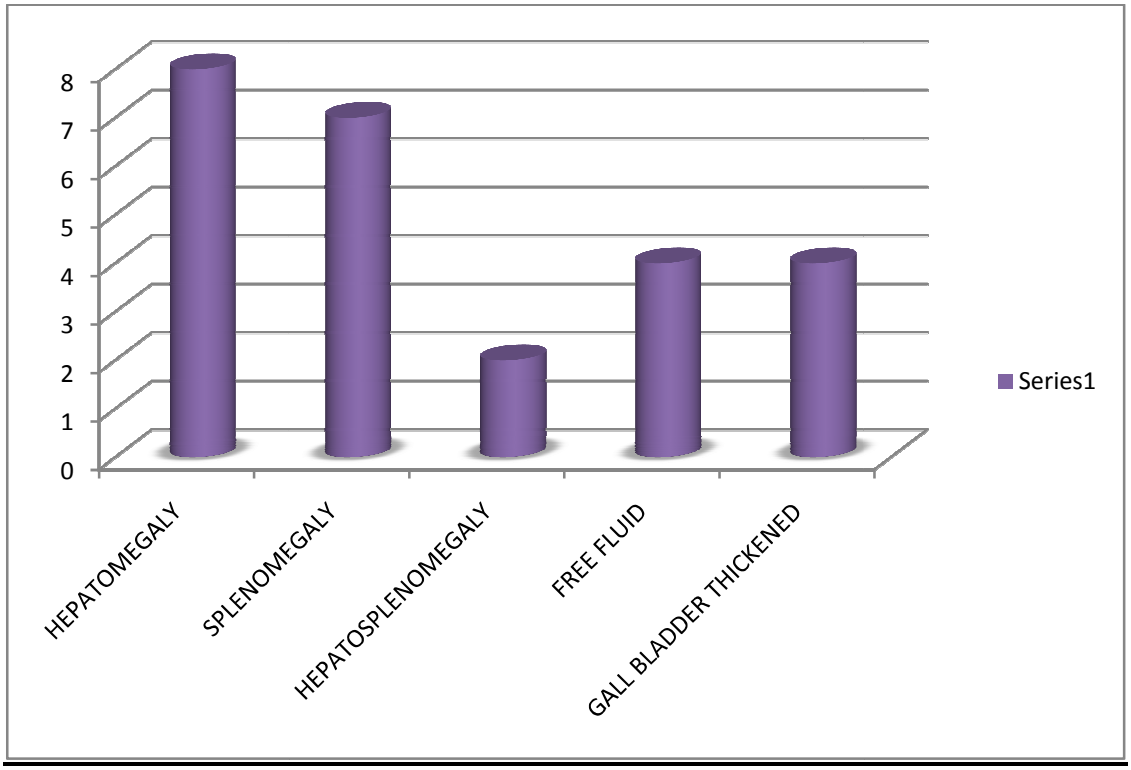
TABLE 8

LIVER FUNCTION TEST

LFT	MEAN	RANGE
Sr. bilirubin	2.14mg/dl	0.8- 18.0mg/dl
SGOT(U/L)	53.10	21-360
SGPT(U/L)	57.54	23-336
ALP(IU/L)	82.53	54-179

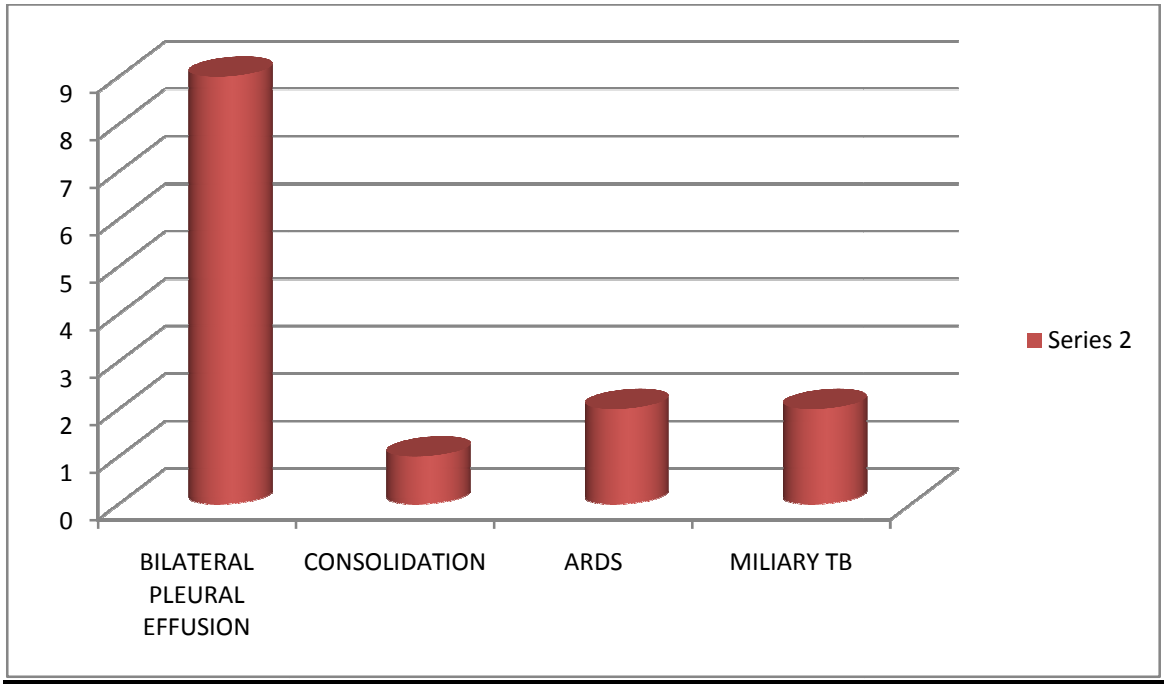
USG ABDOMEN:

GRAPH :7



GRAPH 8

X RAY CHEST PA VIEW

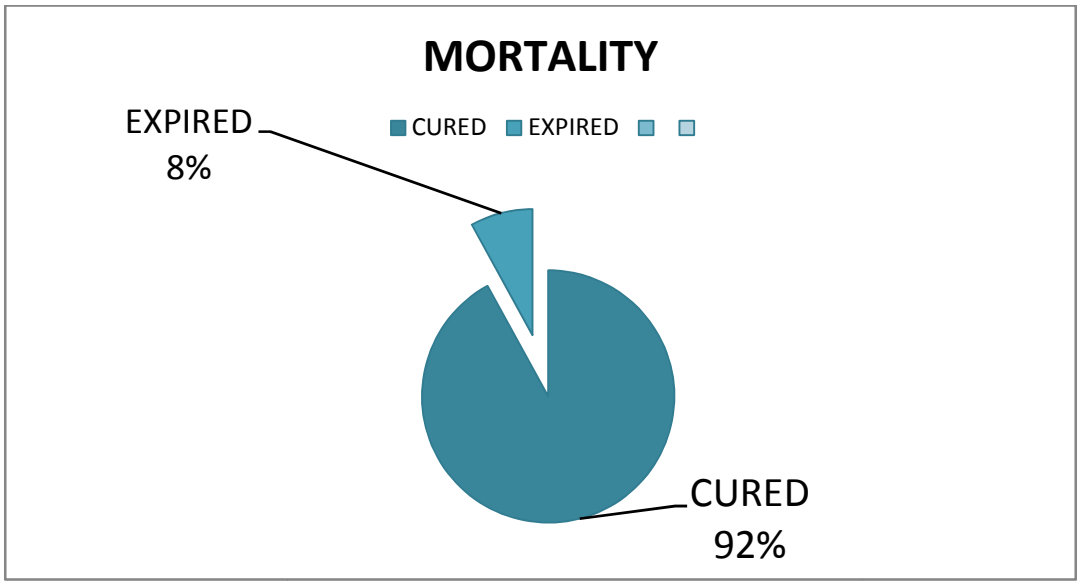


MORTALITY:

No of patients expired is -8.

Mortality rate is -8%.

GRAPH 9



Discussion

DISCUSSION

For a study of fever with thrombocytopenia, patient must satisfy above mentioned criteria's, prospective case collection is necessary and careful follow up is warranted. The three conditions allow the delineation of standard study population.

Out of 100 cases admitted with history of fever with thrombocytopenia, most common causes is infectious.

Out of this infectious condition, dengue is the most common cause (42%), second most common cause is malaria 21%.

Most of the patients were in the working group aged between 25 and 35 years. Further most of them were males (2.3:1).

Further as far as the Seasonal Distribution of cases were concerned, most of the cases were admitted during the months of September, October and November during which the Northwest monsoon is active in Tamil Nadu though sporadic cases were also seen during other months of the year.

Among symptoms other than fever, myalgia is most common 63%, followed by chills and rigor 62%.

Among bleeding manifestation, purpura 12% is the most common followed by bleeding gum 10% and GI bleeding is least 3%.

Out of 100 cases, 38% patients had dehydration at the time of admission. Among 38% , 11% patients had hypotension during admission. Most of the patients improved with treatment except few.

Among 100 cases with reduced platelet count, 60% cases had platelet above 40,000. 17% case had platelet between 20,000 to 40,000. Remaining 23% cases had platelet below 20,000.

Among 17 patient with platelet count between 20,000 and 40,000, 15 patients cured without blood transfusion. Only 2 patients needed blood transfusion.

Among 23 patient with <20000 platelet count, 12 patients improved without platelet transfusion. So only 11 patients actually need blood transfusion. Out of this 11 patients, 5 patients platelet count improved with FFB alone. So 6 out of 23 patients actually needed platelet transfusion.

Platelet count	**No of cases.**	**No of cases improved without bl. Transfusion**	**No of cases improved after bl. Transfusion**
40,000 -1,50,000	60	60	0
20,000-40,000	17	15	2
<20,000	23	12	11

A similar type **of study conducted in India by Nair Ps, Jain A at st.Stephen's hospital, New delhi, for period of one and half year**. A total study of 109 cases were studied with same criteria as in our study.[17]

Septicaemia 29% was the leading cause of fever associated with thrombocytopenia. Second common cause was enteric fever followed by dengue, malaria.

Comparison of nair study and our study

Disease category	Nair study		Our study	
Septicaemia	29	26.6%	2	2%
Dengue/ VHF	15	14.7%	42	42%
Malaria	10	9.2%	21	21%
Haematological	17	15.6%	5	5%
others	20	18.3%	30	30%

Distribution of platelet count	Nair study		Our study	
0-20,000	19	17.5%	23	23%
20,000-40,000	28	25%	17	17%
40-1,50,000	62	56.8%	60	60%

In conclusion our study of fever with thrombocytopenia reveals that

Infection is the most commonest cause, among infections , dengue is the common cause because of seasonal and regional variation. Second most common cause is malaria, in that p.vivax is more common. Petechiae is common bleeding manifestation. Blood transfusion is not needed for all the cases even when platelet is below 20,000. Acute renal failure is the common complication.

SUMMARY

A prospective study of 100 patients, who had fever and thrombocytopenia was done in our hospital. The inclusion and exclusion criteria were followed according to the criteria' mentioned in the material and methods of the study.

1. The age range of the patient was 18-62 years, with male and female ratio being 2.3:1.
2. Most of the cases admitted during September to December.
3. Among symptoms other than fever, myalgia is most common 63%, followed by chills and rigor 62%.
4. Among bleeding manifestation, purpura 12% is the most common followed by bleeding gum 10% and GI bleeding is least 3%.
5. A definitive diagnosis was made in 95% of the case.
6. Among the diagnosed case, dengue is most common cause of fever with thrombocytopenia (42%). Out of this 42 cases, 37 cases were dengue haemorrhagic fever. 5 cases were dengue haemorrhagic shock.
7. Other cases diagnosed were malaria, leptospirosis, typhoid, septicaemia, HIV, miliary tuberculosis, haematological malignancy.
8. Out of 21 cases, 19 cases were p.vivax and 2 cases were p.falciparum.
9. Among 100 cases, most of the patients with platelet count above 40,000, 17 cases between 20,000 to 40,000., 23 cases had platelet below 20,000.

10. Clinical manifestation of the thrombocytopenia are present only in 12 cases out of 100 cases.
11. Among 23 patients with <20000 platelet count, 12 patients improved without platelet transfusion. So, only 11 patients actually need blood transfusion.
12. Out of 100 cases, 12 cases had elevated renal parameter. Out of this 12 cases, 8 patients renal parameter improved with rehydration alone. Only 4 cases who needed nephrologist's intervention and dialysis.
13. In general, 92 cases had recovered and 8 cases had expired.
14. In 92 cases who had good recovery 45 cases followed up and platelet count reached normal at the time of discharge.

Conclusion

CONCLUSION

1. Fever with thrombocytopenia is one of the most challenging problems in the field of medicine.
2. Fever with thrombocytopenia consists of occult presentation of common disease rather than rare disease.
3. Infection is the most common cause of fever with thrombocytopenia.
4. Dengue, malaria, leptospirosis still present clinically in atypical and occult form, making diagnosis more difficult. So high index of clinical suspicion is needed.
5. So other than routine investigation they should do specific test like rapid spot test, IgM ELISA for dengue, IgM ELISA leptospirosis antibodies, widal test etc for correct diagnosis.
6. In majority of the patient, thrombocytopenia without bleeding manifestation.
7. Generally , spontaneous bleeding was noted in platelet count <20,000 , even some patients not have any bleeding manifestation with this platelet count. But due to qualitative defect it was seen in platelet count in the range of 40,000 cell cu/mm also.
8. Even with platelet count less than 20,000, platelet transfusion is not needed for all the cases.
9. Overall mortality for fever with thrombocytopenia is 8%.

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Appendix

PROFORMA

" A STUDY OF CLINICAL PROFILE, LABORATORY PROFILE AND COMPLICATION OF FEVER WITH THROMBOCYTOPENIA"

NAME : AGE: SEX: IP NO:

DOA: OCCUPATION: PLACE:

SYMPTOMS AND SIGN:

- FEVER
- CHILLS AND RIGOR
- MYALGIA
- HEADACHE
- ABDOMINAL PAIN
- VOMITING
- DIARRHOEA/ CONSTIPATION
- COUGH
- BREATHLESSNESS
- RASHES
- BLEEDING GUM
- PETECHIAE
- GI BLEED

- HEMATURIA
- OLIGURIA
- ALTERED SENSORIUM
- CONJUCTIVAL SUFFUSION
- LOSS OF WEIGHT
- DEHYDRATION
- ANAEMIA
- LYMPHADENOPATHY
- VITAL SIGNS
- HEPATOMEGALY
- SPLENOMEGALY
- ADDED SOUNDS IN LUNGS

INVESTIGATIONS:

BLOOD SUGAR :
UREA :
Sr. CREATININE :
ELECTROLYTES :

COMPLETE BLOOD COUNT:

Hb	:	g %
TC	:	cells/µL
DC	:	P: , L: , E: %
Platelets	:	cells/micro Ltr
haematocrit:		
RBC	:	cells/µL
ESR	:	½ hr: mm
	:	1 hr: mm

URINE ROUTINE

Albumin :

Sugar :

Deposits :

Bile Salts :

Bile Pigments:

LIVER FUNCTION TEST

Date: Day 1 After 1 week

Total Bilirubin ***mg/dl***
Direct Bilirubin ***mg/dl***
Indirect Bilirubin ***mg/d***l
SGOT ***U/L***
SGPT ***U/L***
Sr.Alkaline Phosphatase: ***IU***

Peripheral smear: Peripheral smear for MP:

Dengue serology: IgM Antibody for leptospirosis:

Widal test: Sputum AFB

HIV 1 AND 2: BLOOD CULTURE:

URINE CULTURE: USG ABDOMEN:

X RAY CHEST AND ECG:

BONE MARROW ASPIRATION:

Fundus:

Blood transfusion:

Diagnosis:

Prognosis:

ABBREVATIONS:

DHF- DENGUE HAEMORRAGHIC FEVER

DHS	-	DENGUE HAEMORRAGHIC SHOCK
MAL	-	MALARIA
LEPTO	-	LEPTOSPIROSIS
TYP	-	TYPHOID
SEPT	-	SEPTICAEMIA
CML	-	CHRONIC MYELOID LEUKAEMIA.
CLL	-	CHRONIC LYMPHOCYTIC LEUKAEMIA.
UNS	-	UNSPECIFIED (VIRAL FEVER)
M TB	-	MILIARY TUBERCULOSIS
PANCYT	-	PANCYTOPENIA.
CYTOP	-	CYTOPENIA.
P.VIVAX	-	PLASMODIUM VIVAX.
P.FALCI	-	PLASMODIUM FALCIPARUM.
GB	-	GALL BLADDER THICKENED.
FF	-	FREE FLUID
B/L PE	-	BILATERAL PLEURAL EFFUSION
HM	-	HEPATOMEGALY
SM	-	SPLENOMEGALY.
HSM	-	HEPATOSPLENOMEGALY.
C. PYLEO	-	CHRONIC PYELONEPHRITIS.
PLT	-	PLATELET COUNT.
PC	-	PACKED CELL / FFP FRESH FROEN PLASMA
GI	–	GASTRO INTESTINAL BLEED

MASTER CHART

SI NO	NAME	AGE	SEX	DOA	IP NO	FEVER	DURATION	CHILLS/ RIGOR	MYALGIA	ABD PAIN	VOMITING	COUGH/ EXP	GUM BLEED	PETECHIAE	GIT BLEED	LOSS WT	CONJ SUFFUSION	ALTERED SENSORIUM	DEHYDRATION	OLIGURIA	DYSPNOEA	PALLOR	JAUNDICE	HYPOTENSION	HEPATOMEGALY	SPLEENOMEGALY	PLEURAL EFFUSION	ADD SOUND	FUNDUS
1	JAYAMANI	34	F	14/1/11	1337	P	7	-	-	-	-	-	P	P	-	-	-	-	p	-	-	-	-	-	-	-	-	-	N
2	MURUGAN	30	M	15/1/11	1478	P	5	P	-	P	-	-	-	-	-	-	-	-	--	-	-	-	-	-	-	-	-	-	N
3	DURAI	45	M	15/1/11	1596	P	5	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
4	SAMPATH	32	M	17/1/11	2775	p	5	-	P	-	-	-	-	P	P		P	-	P	-	-	-	P	P	-	-	-	-	H
5	RAGUPATHI	27	M	19/1/11	2856	P	4	P	-	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
6	KUMAR	35	M	24/1/11	4070	P	3	P	-	-	P	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
7	ISFMAIL	44	M	15/2/11	6112	P	4	-	P	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
8	KACHANA	33	F	28/2/11	8462	P	7	-	P	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
9	ANTONY	30	M	13/3/11	12352	P	7	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
10	MANIKANDAN	39	M	8/4/11	19391	P	8	P	-	P	-	-	-	P	-	-	-	-	P	-	P	-	P	-	-	-	P	P	N
11	SARAVANAN	28	F	15/4/11	21384	P	40	P	P	-	-	P	P	-	-	P	-	P	P	-	P	P	-	-	P	-	-	P	N
12	SIVARAJ	34	M	5/5/11	24362	P	30	P	-	-	-	P	-	-	-	P	-	-	-	-	P	P	-	-	-	-	P	P	N
13	SHAJAHAN	37	M	9/6/11	26742	P	10	P	-	P	-	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
14	PONNUSAMY	62	M	15/6/11	36014	P	30		P	P	-	-	P	P	P	P	-	P	P	P	P	P	-	P	P	-	-	-	H
15	MANI	38	M	18/6/11	36456	P	6	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	--	-	-	-	N
16	KALIYAPPAN	60	M	30/6/11	37114	P	45	-	P	P	-	-	P	-	-	P	-	-	-	P	-	P	-	-	P	-	-	-	N
17	SATIYAMURTY	22	M	10/7/11	38985	P	7	P	-	-	P	-	-	-	-	-	-	-	P	-	-	P	-	-	-	-	-	-	N
18	GURUSAMY	42	M	13/7/11	39124	P	8	-	P	-	P	P	P	P	-	-	-	P	P	P	P	-	-	P	P	-	P	P	H
19	ANIFA	36	F	20/7/11	43432	P	4	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
20	KARTHICK	19	M	24/7/11	45760	P	2	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
21	SHANMUGAM	29	M	27/7/11	46436	P	35	P	-	P	P	P	-	-	-	P	-	-	P	-	-	P	-	-	P	-	-	P	N
22	SAYETH ABAS	14	M	30/7/11	43074	P	3	-	P	P	P	-	-	-	-	---	-	-	-	P	-	-	-	-	-	-	-	-	N
23	PADHMINI	44	F	2/8/11	47126	P	4	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
24	DEVIKA	26	F	8/8/11	47764	P	15	-	P	P	P	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
25	KUPUSAMY	36	M	13/8/11	47926	P	5	P	--	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
26	NAGALINGAM	40	M	18/8/11	48412	P	3	-	-	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
27	SIVAKUMAR	29	M	28/8/11	49136	P	4	P	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
28	MUNUSAMY	40	M	5/9/11	49360	P	7	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
29	RAMESH	34	M	13/9/11	51915	P	5	P	-	-	-	-	-	-		-	-	-	-	-	P	-	-	-	-	P	P	P	N
30	KARIYAN	35	M	17/9/11	52659	P	7	P	P	P	-	-	-	P	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
31	MARIYAPPAN	40	M	6/9/11	50299	P	8	P	P	P	-	-	P	P	-	-	P	-	P	-	P	-	-	-	-	P	P	P	N
32	BASHEER	60	M	3/9/11	49486	P	10	P	-	-	-	-	-	-	-	-	-	-	P	P	-	-	-	P	-	-	-	-	N
33	KAVITHA	24	F	16/9/11	52441	P	14	P	-	P	P	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	P	P	N
34	ERAMMA	36	F	22/9/11	53732	P	7	P	-	P	P	P	-	P	-	-	-	P	P	-	P	P	-	-	-	P	P	P	H
35	NISHA	22	F	24/9/11	54130	P	8	P	P	-	-	-	-	-	-	-	-	-	-	--	-	-	-	-	--	-	-	-	N
36	MUTHURAJ	41	M	26/9/11	54789	P	15	P	P	-	P	-	P	P	-	-	P	P	P	P	P	-	P	P	P	P	-	P	H
37	SIVASHANKAR	28	M	28/9/11	55134	P	14	-	-	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
38	RABIK	22	M	1/10/11	55781	P	7	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
39	TAMIL ARASU	30	M	3/10/11	51678	P	6	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
40	AISHA	43	F	5/10/11	50225	P	35	-	P	P	-	-	P	-	-	P	-	-	-	-	-	P	-	-	P	P	-	-	N
41	LAKSHMI	52	F	5/10/11	50289	P	14	P	P	P	P	-	-	-	-	-	-	P	P	P	-	--	-	-	-	-	-	-	N
42	RAMYA	25	F	6/10/11	51657	P	4	P	P	-	-	-	-	-	-	-	-	--	-	-	-	-	-	-	-	-	-	-	N
43	SELVARAJ	36	M	6/10/11	53278	P	4	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
44	CHINNU	45	M	7/10/11	52456	P	7	P	-	-	-	-	-	-	-	-	--	-	P	-	-	-	-	-	-	-	-	-	N
45	SHAJAHAN	40	M	7/10/11	51786	P	35	P	P	-	-	-	-	-	-	P	-	P	-	-	P	P	-	-	-	-	-	P	N
46	RAJAMMAL	36	M	8/10/11	52145	P	5	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
47	SUNDARAM	32	M	8/10/11	53789	P	7	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N

48	MANOHARAN	30	M	9/10/11	53419	P	4	P	P	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
49	MADHESH	31	M	10/10/11	55661	P	15	-	P	-	-	-	-	-	-	-	P	-	P	-	-	-	P	-	-	-	-	-	N
50	GOMATHY	60	F	15/11/11	64857	P	3	-	-	P	P	-	-	-	--	-	-	-	P	P	-	P	-	P	P	-	-	-	N
51	RAMATHAL	63	F	17/11/11	57767	P	45	-	P	P	-	-	-	-	-	P	-	P	P	-	-	P	-	-	-	-	-	-	N
52	ARUMUGAM	25	M	19/11/11	65805	P	10	P	-	P	P	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
53	RAJA	34	M	12/11/11	64200	P	14	P	P	P	P	-	-	-	-	-	P	-	-	-	--	-	P	-	P	-	-	-	N
54	PALANI	41	M	13/11/11	64231	P	8	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
55	KRISHNAN	29	M	13/11/11	65879	P	6	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
56	RAJESH	26	M	13/11/11	65467	P	10	-	P			-	-	-	-		P	-	--	-	-	-	P	-	-	-	-	-	N
57	RANI	38	F	14/11/11	63278	P	3	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
58	UNNI	28	M	14/11/11	64534	P	6	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
59	SAKTHIVEL	30	M	15/11/11	65231	P	3	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
60	SANKAR	26	M	15/11/11	65347	P	5	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
61	SURYA	18	M	15/11/11	65785	P	7	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
62	VIJI	29	M	15/11/11	65245	P	5	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	--	-	-	N
63	ANITHA	24	F	15/11/11	65765	P	8	P	P	-	P	-	-	P	-	-	P	-	P	-	P	-	-	P	P	-	P	P	N
64	SRIDHAR	27	M	16/11/11	65197	P	15	-	-	P	P	-	-	-	-	-	-	-	P	-	-	-	-	-	-	P	-	-	N
65	BOOPATHI	21	M	16/11/11	66129	P	4	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
66	SURESH	19	M	16/11/11	66321	P	8	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
67	KARUPAN	38	M	16/11/11	66078	P	7	P	-	P	P	-	-	-	-	-	-	P	P	P	-	-	-	P	-	-	-	-	N
68	RAMALAKSHMI	21	F	16/11/11	66749	P	5	P	P	-	P	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
69	SANGEETHA	18	F	16/11/11	66758	P	5	P	P	-	P	P	-	P	P	-	-	P	P	-	P	-	-	P	-	-	-	P	N
70	JAYA	18	F	16/11/11	66850	P	7	P	P	-	-	-	-	-	-	--	-	-	-	-	-	-	-	-	-	-	-	-	N
71	SIVA SHANKAR	22	M	17/11/11	67123	P	3	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
72	RAJA	26	M	17/11/11	67345	P	6	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
73	RANGAN	26	M	17/11/11	67231	P	14	-	P			-	P				P		P	-	-	-	P	-	-	-	-	-	N
74	JAFFER	29	M	17/11/11	67432	P	10	-	P	-	-	P	P	-	-	-	P	-	P	P	P	-	P	P	-	-	P	P	H
75	PANNEER	40	M	17/11/11	67489	P	8	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
76	MADHAN	31	M	17/11/11	68145	P	9	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
77	ARAVINDH	27	M	18/11/11	68254	P	6	-	-	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
78	SELVI	26	F	18/11/11	68367	P	6	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
79	SENTHIL	29	M	18/11/11	68167	P	7	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
80	KANDASAMY	40	M	18/11/11	68289	P	14	-	P	-	-	-	-	-	-	-	P	-	-	-	-	-	P	-	-	-	-	-	N
81	KANNIYAPAN	45	M	18/11/11	68356	P	40	P	-	-	-	-	-	-	-	P	-	P	P	-	-	-	-	-	-	-	-	-	N
82	MARY	36	F	18/11/11	68412	P	2	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
83	SENTHIL	37	M	18/11/11	67356	P	12	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	P	-	-	N
84	RAVI	44	M	19/11/11	61239	P	34	P	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	-	-	-	N
85	GOWRI	23	F	19/11/11	61849	P	3		P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
86	PONNUSAMY	62	M	19/11/11	61987	P	5	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
87	ANGALASWARI	19	F	20/11/11	66200	P	10	P	P	P	P	P	-	P	-	-	-	P	P	-	P	-	-	P	-	-	-	P	N
88	HARI	24	M	21/11/11	66128	P	6	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
89	SUJA	30	F	22/11/11	63778	P	7	P	-	-	-	-	-	-	-	-	-	-	-		-	-	-	-	-	-	-	-	N
90	THANGAM	18	F	27/11/11	66236	P	8	P	P	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
91	SUDHA	18	F	27/11/11	66246	P	5	P	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
92	MAHESWARI	19	F	27/11/11	63246	P	6	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
93	RAMALAKSHMI	20	F	27/11/11	66208	P	5	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	--	-	-	N
94	VELDURAI	45	M	27/11/11	67552	P	30	P	P	P	-	-	-	-	-	P	-	-	-	-	-	P	-	-	-	P	-	-	N
95	RAVI	46	M	27/11/11	68918	P	14	P	P	-	-	-	-	-	-	-	P	-	-	-	-	-	P	-	-	-	-	-	N
96	SANTHIYA	21	F	27/11/11	67418	P	3	P	P	-	-	-	-	-	-	-	-	-	P	-	-	-	-	-	-	-	-	-	N
97	RANGITHAM	40	F	28/11/11	62567	P	10	-	-	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
98	GANESH	37	M	28/11/11	64346	P	6	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
99	PANDISHWARI	18	F	28/11/11	67325	P	3	P	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N
100	MANIKAM	38	M	28/11/11	67456	P	4	P	-	P	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	N

Sl	NAME	HAEMOGLOBIN	TC (Cells/ml/µl	Platelet Count	HAEMATOCRIT	ESR (1Hr)	T.BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALP	BL Urea	Sr. Creatinine	Smear for MP	Dengue serology	IgM LEPTO	Widal test	Sputum AFB	HIV 1 & 2	BLOOD CULTURE	URINE CULTURE	PERIPHERAL SMEAR	USG ABDOMEN	XRAY CHEST	BONE MARROW ASPIRATION	TRANFUSION	OUTCOME	DIAGNOSIS
1	JAYAMANI	12	5.8	20,000	34	22	1.2	34	23	67	23	1.1	-	P	-	-	-	-	-	-	N	N	N		3 FFP	C	DHF
2	MURUGAN	11	7.9	67,000	45	21	1.4	33	45	56	34	1.3	P	-	-	-	-	-	-	-	P.VIVAX	MILD HM	N	-	-	C	MAL
3	DURAI	13.4	13.3	44,000	40	34	1.3	21	26	66	21	1.0	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
4	SAMPATH	15	6.3	19,000	38	34	18	360	250	140	24	0.8	-	-	P	-	-	-	-	-	N	N	N		1PLT,2FFP	C	LEPT
5	RAGUPATHI	11.9	5.8	77,000	36	25	1.6	23	34	88	34	0.9	-	-	-	P	-	-	-	-	N	N	N	-	-	C	TYP
6	KUMAR	15.8	5.7	40000	45	30	1.4	34	27	56	29	0.7	-	P	-	-	-	-	-	-	N	GB/ FF+	N	-	1PLT,2FFP	C	DHF
7	ISHMAIL	12.8	6.7	120000	37	24	1.2	35	44	89	21	1.5	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
8	KACHANA	13.8	14.2	45,000	36	22	1.1	44	28	92	25	1.1	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
9	ANTONY	10.8	11.5	65,000	32	34	1.5	56	45	90	24	1.6	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
10	MANIKANDAN	12.2	9.2	7500	40	15	2.3	83	97	113	32	1.6	P	-	-	-	-	-	-	-	P.VIVAX	HSM	B/L PE	-	4 PLT	C	MAL
11	SARAVANAN	8.7	14.6	28,000	55	78	1.8	45	34	78	27	1.1	-	-	-	-	-	P	-	-	CYTOPENIA	HM	N	-	1PC	C	HIV
12	SIVARAJ	9.1	15.1	55,000	34	88	1.2	34	46	77	34	1.3	-	-	-	-	P	-	-	-	CYTOPENIA	N	M. TB	-	-	C	M.TB
13	SHAJAHAN	10.2	7.2	5,500	30	24	1.0	40	33	55	36	1.3	P	-	-	-	-	-	-	-	P.VIVAX	N	N	-	-	C	MAL
14	PONNUSAMY	6.4	2.7	6,500	55	20	1.2	45	28	60	39	1.0	-	-	-	-	-	-	-	-	PANCYTO	MILD HM	N	AML	2PC/ 4PLT	E	AML
15	MANI	12.4	6.7	45,000	38	23	1.3	56	34	67	24	1.1	-	P	-	-	-	-	-	-	N	GB / FF+	N	-	-	C	DHF
16	KALIYAPPAN	7.4	3.3	39,000	35	15	1.4	55	45	58	24	0.8	-	-	-	-	-	-	-	-	PANCYTO	MILD HM	N	AML	2PC/1PLT	C	AML
17	SATIYAMURTY	9.7	7.5	12,000	24	25	1.8	44	36	70	30	0.9	-	P	-	-	-	-	-	-	N	N	N	-	2PLT	C	DHF
18	GURUSAMY	12.1	14.5	14,000	55	24	1.2	45	48	78	88	2.8	-	P	-	-	-	-	-	-	N	GB/ FF+	B/L PE	PE	2PLT/2FFP	C	DHS
19	ANIFA	12.8	12.1	56,000	34	12	1.1	23	34	98	24	1.4	P	-	-	-	-	-	-	-	P.VIVAX	N	N	-	-	C	MAL
20	KARTHICK	11.8	97	75,000	30	24	1.2	34	49	67	34	1.1	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
21	SHANMUGAM	8.8	2.4	36,000	35	60	1.5	45	65	77	25	0.8	-	-	-	-	-	P	-	-	PANCYTO	MILD HM	N	-	2PC	C	HIV
22	SAYETH ABAS	12.7	4.1	85,000	34	20	1.0	40	45	78	36	1.4	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
23	PADHMINI	12.2	6.6	66,000	24	22	1.1	45	34	89	34	1.1	P	-	-	-	-	-	-	-	P. VIVAX	N	N	-	-	C	MAL
24	DEVIKA	12.4	5.6	58,000	34	12	1.1	34	45	78	36	0.9	-	-	-	P	-	-	-	-	N	MILD SM	N	N	-	C	TYP
25	KARUPUSAMY	11.9	6.7	52,000	43	20	1.7	45	23	88	34	0.8	-	P	-	-	-	-	-	-	N	N	N	N	-	C	DHF
26	NAGALINGAM	12.9	11.4	66,000	34	18	1.4	33	24	78	23	0.9	-	P	-	-	-	-	-	-	N	N	N	N	-	C	DHF
27	SIVAKUMAR	13.1	13.5	75,000	40	23	1.5	34	45	79	34	1.4	-	P	-	-	-	-	-	-	N	N	N	N	-	C	DHF
28	MUNUSAMY	12.4	11.3	110000	44	24	1.3	55	34	99	28	1.5	-	P	-	-	-	-	-	-	N	N	N	N	-	C	DHF
29	RAMESH	10.6	5.5	78,000	55	23	1.1	55	34	67	28	1.3	P	-	-	-	-	-	-	-	P.VIVAX	SM/ FF+	B/L PE	-	-	C	MAL
30	KARIYAN	11.5	7.7	12,000	40	21	1.6	41	43	71	29	1.0	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
31	MARIYAPPAN	12.5	6.1	40,000	41	34	1.2	35	45	65	36	0.8	-	-	P	-	-	-	-	-	N	SM	B/L PE	-	-	C	LEPT
32	BASHEER	12.7	7.1	35,000	23	12	1.1	45	56	77	55	2.0	P	-	-	-	-	-	-	-	P.VIVAX	N	N	-	-	C	MAL
33	KAVITHA	10.8	3.6	28,000	55	23	1.3	34	41	65	46	1.8		-	-	P	-	-	-	-	N	FF+	B/L PE	-	-	C	TYP
34	ERAMMA	9.3	11	10,000	50	18	5.8	214	148	179	114	4.9	P	-	-	-	-	-	--	-	P.FALCI	SM	ARDS	-	2PLT/2FFP	E	MAL
35	NISHA	12.1	5.5	99,000	24	12	1.1	35	51	66	27	0.8	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
36	MUTHURAJ	11.4	7.9	15,000	50	40	7.5	188	110	143	180	4.6	-	-	P	-	-	-	-	-	N	HSM	N	-	2PLT/2FFP	E	LEPT
37	SIVASHANKAR	13.4	14.6	77,000	24	21	1.3	34	45	79	34	1.1	-	-	-	P	-	-	-	-	N	N	N	N	-	C	TYP
38	RABIK	12.4	12	55,000	34	22	1.1	22	45	56	34	0.9	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
39	TAMIL ARASU	10.6	18	10,000	35	20	1.7	45	35	89	35	1.2	P	-	-	-	-	-	-	-	P.FALCI	N	N	N	-	C	MAL
40	AISHA	6.5	1.00	65,000	35	15	1.5	34	46	89	30	1.1	-	-	-	-	-	-	-	-	CML -AP	MOD HSM	N	CML- AP	2PC	C	CML
41	LAKSHMI	11.3	15.6	54,000	45	56	1.3	35	45	78	170	3.5	-	-	-	-	-	-	-	P	N	CH.PYLEO	N	-	-	E	SEPT
42	RAMYA	12.3	7.8	65,000	34	12	1.2	45	34	76	34	1.1	-	-	-	-	-	-	-	-	N	N	N	N	-	C	UNS
43	SELVARAJ	13.4	7.3	78,000	24	10	0.9	55	23	89	29	0.8	-	-	-	-	-	-	-	-	N	N	N	N	-	C	UNS

44	CHINNU	14.5	13.4	40,000	25	12	0.8	34	45	67	34	1.4	-	P	-	-	-	-	-	-	N	N	N		-	C	DHF
45	SHAJAHAN	8.9	13.2	55,000	34	87	3.1	55	66	119	28	1.5	-	-	-	-	P	-	-	-	CYTOPENIA	N	M. TB	-	-	E	M.TB
46	RAJAMMAL	12.4	5.6	78,000	28	13	1.4	54	28	78	34	1.1	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
47	SUNDARM	12.5	7.4	120000	35	20	1.7	22	38	90	21	1.3	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
48	MANOHARAN	11.6	8.9	34,000	24	12	1.3	45	56	77	27	1.4	-	P	-	-	-	-	-	-	N	THICK GB	N	-	-	C	DHF
49	MADESH	13.3	3.5	22,000	42	13	3.1	98	112	134	34	1.6	-	-	P	-	-	-	-	-	N	N	N	-	-	C	LEP
50	GOMATHY	11.3	9.7	11,000	30	10	1.5	48	150	54	134	3.2	-	-		P	-	-	-	-	N	MILD HM	N	N	2FFP	C	TYP
51	RAMATHAL	4.8	1,53	36,000	34	56	1.6	45	34	88	20	0.7	-	-	-	-	-	-	-	-	CLL- SMUDGE	SM	N	CLL	2PC	E	CLL
52	ARUMUGAM	13.7	4.1	100000	35	15	1.4	50	55	89	34	1.3	-	-		P	-	-	--	-	N	MILD HM	N	-	-	C	TYP
53	RAJA	11.1	9.4	89,000	38	10	8.9	293	336	114	20	0.9	-	-	P	-	-	-	-	-	N	MILD HM	N	-	-	C	LEPT
54	PALANI	13.5	5.6	77,000	23	21	1.3	34	56	87	24	0.8	P	-	-	-	-	-	-	-	P.VIVAX		N	-	-	C	MAL
55	KRISHNANA	12.9	7.7	55,000	34	23	1.1	45	67	89	34	0.7	P	-	-	-	-	-	-	-	P.VIVAX	N	N	-	-	C	MAL
56	RAJESH	11.4	14.1	22,000	35	21	3.1	112	134	105	24	1.5	-	-	P	-	-	-	-	-	N	N	N	-	-	C	LEPT
57	RANI	12.5	11.5	18,000	36	16	1.1	45	28	78	26	1.2	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
58	UNNI	13.8	12.4	23,000	40	23	1.2	45	56	78	24	1.5	-	-	-	-	-	-	-	-	N	N	N	-	-	C	UNS
59	SAKTHIVEL	12.6	12.5	45,000	34	25	1.4	34	37	76	27	0.9	P	-	-	-	-	-	-	-	P. VIVAX	N	N	-	-	C	MAL
60	SANKAR	13.4	14.5	18,000	36	13	1.4	45	34	89	29	1.4	P	-	-	-	-	-	-	-	P.VIVAX	N	N	-	-	C	MAL
61	SURIYA	12.4	5.5	10,000	34	11	1.5	56	67	98	34	1.2	P	-	-	-	-	-	-	-	P.FALCI	N	N	-	-	C	MAL
62	VIJI	13.9	6.7	56,000	24	23	1.4	45	36	78	35	1.5	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
63	ANITHA	10.4	13.6	12,000	56	34	1.3	56	66	89	38	1.6	-	P	-	-	-	-	-	-	N	MILD HM	B/L PE	-	2 FFP	C	DHS
64	SRIDHAR	12.3	3.2	45,000	34	32	1.6	45	44	78	24	1.2	-	-	-	P	-	-	-	-	N	SM	N	-	-	C	TYP
65	BOOPATHI	13.2	4.6	18,000	28	21	1.4	45	56	90	28	1.5	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
66	SURESH	11.8	5.8	33,000	32	12	1.2	34	56	67	34	1.2	P	-	-	-	-	-	-	-	P.VIVAX	N	N	N	-	C	MAL
67	KARUPAN	10.3	15.6	55,000	56	55	1.8	56	77	90	88	3.2	-	-	-	-	-	-	-	P	-	C. PYLEO	N	N	-	C	SEPT
68	RAMALAKSMI	12.4	6.7	120200	28	15	1.7	45	33	80	34	1.2	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
69	SANGEETHA	13	13.7	14,000	55	25	1.6	56	60	90	24	1.5	-	P	-	-	-	-	-	--	N	N	B/L PE	-	6PLT/3FFP	C	DHS
70	JAYA	13.9	5.1	100000	34	33	1.1	44	45	78	33	0.8	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
71	SIVASHANKAR	12.4	12.4	44,000	23	21	1.4	34	56	67	34	0.7	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
72	RAJA	13.4	5.8	78,000	21	19	1.1	36	59	81	50	2.4	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
73	RANGAN	10.4	3.5	13,000	32	17	3.8	101	89	76	23	1.3	-	-	P	-	-	-	-	-	N	N	N	-	2FFP	C	LEPT
74	JAFFER	12.4	6.7	11,000	57	34	6.7	123	142	90	142	3.7	-	-	P	-	-	-	-	-	N	N	CONSO	-	2PLT/FFP	E	LEPT
75	PANNEER	13.2	4.8	44,000	23	12	1.2	34	56	78	34	1.2	P	-	-	-	-	-	-	-	N	N	N	-	-	C	MAL
76	MADHAN	11.2	5.6	51,000	27	19	1.4	25	58	72	38	1.2	P	-	-	-	-	-	-	-	N	N	N	-	-	C	MAL
77	ARAVIND	13.4	9.9	19,000	25	15	1.8	43	56	87	25	1.1		P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
78	SELVI	11.9	12.3	43,000	32	14	1.2	34	45	88	43	1.8	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
79	SENTHIL	13.6	5.6	77,000	23	19	1.3	33	76	90	34	1.3	P	-	-	-	-	-	-	-	N	N	N	-	-	C	MAL
80	KANDASAMY	12.4	3.4	47,000	34	10	3.5	43	56	87	36	1.6	-	-	P	-	-	-	-	-	N	N	N	-	-	C	LEPT
81	KANNIYAPPAN	8.9	2.7	35,000	35	55	1.6	56	67	98	27	1.6	-	-	-	-	-	P	-	-	PANCYTO	N	N	N	2PC	E	HIV
82	MARY	12.4	6.8	89,000	23	12	1.7	34	65	90	34	1.3	-	-	-	-	-	-	-	-	N	N	N	N	-	C	UNS
83	SANTHI	12.4	3.5	77,000	26	13	3.2	45	56	89	26	1.4	-	-	P	-	-	-	-	-	N	SM	N	-	-	C	LEPT
84	RAVI	8.9	2.7	45,000	26	14	1.4	34	25	79	34	1.5	-	-	-	-	-	P	-	-	PANCYTO	N	N	N	2PC	C	HIV
85	GOWRI	13.4	6.8	55,000	28	19	1.6	39	56	90	59	2.2	P	-	-	-	-	-	-	-	P.VIVAX	N	N	N	-	C	MAL
86	PONNUSAMY	12.4	8.9	79,000	28	24	1.3	45	67	89	34	1.6	-	P	-	-	-	-	-	-	N	N	N	N	-	C	DHF
87	ANGALESWARI	13.1	7.4	10,000	57	30	1.4	54	55	88	60	2.4	-	P	-	-	-	-	-	-	N	N	ARDS	-	4PPT/2FFP	C	DHS
88	HARI	11.1	8.8	57,000	30	21	1.3	45	59	79	31	1.1	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
89	SUJA	12.5	7.9	88.000	31	23	1.5	47	61	91	33	1.3	P	-	-	-	-	-	-	-	P.VIVAX	N	N	N	-	C	MAL
90	THANGAM	11.9	8.9	19,000	24	23	1.2	34	47	100	38	0.9	-	P	-	-	-	-	-	-	N	N	N	-	3PLT/3FFP	C	DHS
91	SUDHA	12.5	5.5	80,000	25	22	1.3	35	59	78	24	0.8	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
92	MAHESWARI	13	7.1	50,000	34	12	1.4	45	26	69	34	1.3	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
93	RAMALAKSMI	14	9.4	22,000	44	18	1.2	54	45	79	28	0.9	-	P	-	-	-	-	-	-	N	N	N	-	3FFP	C	DHF
94	VELDURAI	8.9	33.4	45,000	36	15	1.5	45	55	98	36	1.2	-	-	-	-	-	-	-	-	NHL	MOD SM	N	NHL	2PC	C	NHL
95	RAVI	11.1	4.4	50,000	45	30	5.3	36	45	90	34	1.6	-	-	P	-	-	-	-	-	N	N	N	-	-	C	LEPT
96	SANTHIYA	12.8	5.7	61,000	47	39	1.7	55	56	89	24	1.1	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
97	RANGITHAM	11.4	3.1	34,000	43	33	1.3	51	57	90	36	1.4	-	-	-	P	-	-	-	-	N	N	N	-	-	C	TYP
98	GANESH	12.4	7.8	55,000	39	23	1.2	45	67	89	23	1.2	P	-	-	-	-	-	-	-	N	N	N	-	-	C	MAL
99	PANDISHWARI	13.4	7.9	90,000	24	23	1.1	45	49	88	38	1.3	-	P	-	-	-	-	-	-	N	N	N	-	-	C	DHF
100	MANIKAM	12.8	6.9	45,000	25	21	1.6	34	67	78	34	1.4	-	-	-	-	-	-	-	-	N	N	N	N	-	C	UNS