“CLINICAL AND ETIOLOGICAL PROFILE OF ACUTE FEBRILE ILLNESSES WITH THROMBOCYTOPENIA - A ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY”

By

Dr. P. RAHUL

Dissertation submitted to the
KLE University, Belgaum, Karnataka

In Partial Fulfillment
of the requirements for the degree of

M. D. (GENERAL MEDICINE)

Under the Guidance of

Dr. ANNASAHEB J. DHUMALE MD
ASSOCIATE PROFESSOR

DEPARTMENT OF MEDICINE,
JAWAHARLAL NEHRU MEDICAL COLLEGE,
BELGAUM, KARNATAKA

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KLE UNIVERSITY, BELGAUM,

KARNATAKA

DECLARATION

I hereby declare that this dissertation entitled **"CLINICAL AND ETIOLOGICAL PROFILE OF ACUTE FEBRILE ILLNESSES WITH THROMBOCYTOPENIA - A ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY"** is a bonafide and genuine research work carried out by me under the guidance of **Dr. ANNASAHEB J. DHUMALE** $_{MD}$, Associate Professor, Department of Medicine, Jawaharlal Nehru Medical College, Nehru Nagar, Belgaum – 10.

Date:

Place: Belgaum (Dr. P. RAHUL)

KLE UNIVERSITY, BELGAUM,

KARNATAKA

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This is to certify that the dissertation entitled **"CLINICAL AND ETIOLOGICAL PROFILE OF ACUTE FEBRILE ILLNESSES WITH THROMBOCYTOPENIA - A ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY"** is a bonafide research work done by **Dr. P. RAHUL** in partial fulfillment of the requirement for the degree of **M.D. (GENERAL MEDICINE).**

Date:

Place: Belgaum

Dr. ANNASAHEB J. DHUMALE MD
Associate Professor,
Department of Medicine,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

KLE UNIVERSITY, BELGAUM,

KARNATAKA

ENDORSEMENT

This is to certify that the dissertation entitled **"CLINICAL AND ETIOLOGICAL PROFILE OF ACUTE FEBRILE ILLNESSES WITH THROMBOCYTOPENIA - A ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY"** is a bonafide research work done **Dr. P. RAHUL** under the guidance of **Dr. ANNASAHEB J. DHUMALE** MD Associate Professor, Department of Medicine, J. N. Medical College, Nehru Nagar, Belgaum – 590 010.

Dr. V. A. Kothiwale MD, Ph.D.
Professor and Head,
Department of Medicine,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

Dr. V. D. Patil MD, DCH
Principal,
J. N. Medical College,
Nehru Nagar, Belgaum – 10

Date:
Place: Belgaum

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KARNATAKA

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Date :

(Dr. P. RAHUL)

Place : Belgaum

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Date:
Place: Belgaum **Dr. P. RAHUL**

LIST OF ABBREVIATIONS USED

APP	Acute phase protein
APR	Acute phase response
CMV	Cytomegalovirus
CRP	C-Reactive protein
DIC	Disseminated Intravascular coagulation
DOA	Date of Admission
DOD	Date of Discharge
EP	Endogenous Pyrogen
HIV	Human Immunodeficiency Virus
HSV	Herpes simplex virus
IL-1	Interleukin-1
LAF	Lymphocyte Activating Factor
OVLT	Organum vasculosum laminae terminalis.
POAH	Pre optic/Anterior hypothalamus
SCN	Supra Chiasmatic Nucleus
TNF	Tumor Necrosis Factor

ABSTRACT

Background and Objectives

Infection is a commonest cause of Thrombocytopenia. Thrombocytopenia associated with fever helps to narrow the differential diagnosis and management of fever. It also helps to know the various complications of thrombocytopenia and its management.

Methods

200 patients aged more than 12 years with Fever and Thrombocytopenia, between January 2008 to December 2008 were included in this study.

Results

Infection was the commonest cause of thrombocytopenia and Dengue was the commonest of the infections. Bleeding manifestations were seen in 45% of patients. 85% of patients with Bleeding tendencies had Petechiae/Purpura as the commonest bleeding manifestation, followed by spontaneous bleeding in 51%. Good recovery was noted in 97%, while 3% had mortality. Septicemia accounted for 50% of deaths followed by Dengue with 33% and Leptospirosis with 17%.

Interpretation and Conclusion

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 counts of less than 20,000 in majority of patients. Petechiae/Purpura were seen more commonly in

platelet count in range of less than or equal to 50,000. On treating the specific cause drastic improvement in platelet count was noted during discharge and further follow-up.

Key words

Dengue; Infection; Mortality; Petechiae/purpura; Spontaneous bleeding.

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INTRODUCTION

Fever is a pervasive and ubiquitous theme in human myth, art and science. Fever is such a common manifestation of illness that it is not surprising to find accurate descriptions of the febrile patients in early recorded history.[1]

Hippocrates and, later during the Roman Empire, physicians gave detailed descriptions of fever and their varied patterns of presentations.[1]

With the construction, in the early eighteenth century, of an effective thermometer by the Dutch instrument maker Gabriel Daniel Fahrenheit, new interest surfaced in the relationship between body temperature and disease.

Modern research had its beginning in 1948, when Dr. Paul Beeson determined that fever is caused by a product of host inflammatory cells. Initially thought to be a product of polymorphonuclear leukocyte, this endogenous pyrogen is generated by mononuclear phagocytes. It is identical or very similar in composition to substances previously identified as lymphocyte activating factor (LAF), mononuclear cell factor and leukocyte endogenous mediator collectively known as Interleukin-1 (IL-1). Interleukin-1 has now been shown to have a major role in thermoregulation and fever.

Normal body temperature displays a diurnal pattern with lower values in the early morning hours and higher values in the afternoon. Normal ranges are between 35.8°C (96.5°F) and 37.2°C (99°F). Fever is superimposed on this pattern and thus temperatures are usually greatest in the afternoon and evening.

Fever is defined as an elevation of the body temperature above the normal circadian range, as the result of a change in the thermoregulatory center located in the anterior hypothalamus.

A morning temperature of more than 37.2°C (98.9 °F) or evening temperature of more than 37.7°C (99.9°F) would define fever.[1]

Thrombocytopenia is defined as platelet count less than 150,000 /µl. This is due to decreased production, increased destruction (immunogenic and non-immunogenic) and increased sequestration in spleen. Of these, infections being the commonest cause of thrombocytopenia.[2,13]

At times the fever course is prolonged and fever with thrombocytopenia narrows the differential diagnosis of the clinical entity.

Septicemia, infections like Malaria, Dengue, Leptospirosis, Typhoid, Human Immunodeficiency Virus (HIV) and Miliary tuberculosis (TB) are some of the common causes of fever with thrombocytopenia.

Therefore, a well-organized systemic approach, carried out with an awareness of causes of fever with thrombocytopenia can shorten the duration of investigations and bring out the diagnosis.

In view of the above context the present study was carried out to assess causes and complications of fever with thrombocytopenia.

OBJECTIVES

The objectives of the present study were to;

- To evaluate clinical profile of fever with thrombocytopenia.
- To identify the cause of fever with thrombocytopenia.
- To assess the clinical complications associated with fever and thrombocytopenia.

REVIEW OF LITERATURE

History of fever

Depicted in the Sumerian pictographs as flaming brazier, fever was recognized as a cardinal feature of disease.[3]

Sir William Osler stated "Humanity has but three great enemies: Fever, famine and war; of these by far the greatest, by far the most terrible, is fever". Like Osler, physicians since antiquity have viewed fever as an entity worthy of unremitting attention.

Hippocrates mentioned that "Heat is the immortal substance of life endowed with intelligence….., hence, heat must also be refrigerated by respiration and kept within bounds if the source or principle of life is to persist; for if refrigeration is not provided, the heat will consume itself" The writing of Hippocrates provided the detailed description of febrile disease.[4]

Celsius, of the early Roman Empire, first suggested the possible relationship between fever and the cardinal manifestations of inflammation - heat, swelling, redness and pain. Carl Reinhold August Wunderlich (1815-1877) in his book, Das Verhalten der Eigenwarme in Krankenheiten (The course of Temperature in Diseases) gave 98.6^0 F (37^0 C) its special significance, vis-à-vis, the normal temperature. He described the normal diurnal variation of the body temperature, established 100.4°F (38°C) 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 thermometry.[5]

He also wrote that "(fever) can give more certainly than anything else information as to the grade of disease". Because of his work, fever, which has previously been viewed as a disease, came to be recognized more appropriately as a clinical sign.[5]

The mercury thermometer had been perfected in Holland in the early 18th century by Gabriel Daniel Fahrenheit. The word "thermometer" surfaced in the literature of Leurechon's "Recreation Mathematique" (1624), which mentioned the use instrument "to test the intensity of fever".[5]

The concept of central set-point temperature was introduced by H. T. Hammel, who proposed an original neuronal model to explain regulation of a set-point temperature by preoptic. Heat production responses were shown to regulate near a set-point of 37°C by the respective effector neurons.

In 1961, Pittendrigh enumerated all of the characteristics required to explain temporal organization in living organism. He proposed that internal time keeping is achieved by a self-sustained oscillator(s) with a temperature-compensated period that can be entrained by the external environment. These characteristics of circadian temporal organization were described in detail by a group of scientists' meeting at the Cold Spring Harbor symposium (1960) on biological clocks.

The term “Circadian” (derived from circa or “about” and dies or “day”) was proposed by Dr. Franz Harberg in the late 1950s to denote these daily cycles.[6]

In 1948, Kleitmann and Ramsaroop, provided some of the first detailed information concerning endogenous and exogenous influences on the diurnal rhythm of core (oral) temperature. In most of their subjects, there was a 12 hour difference between the maximum and minimum observed temperatures.

The current concept of fever physiology is that, host cell-derived molecules induce fever, which usually occurs in the context of an overall inflammatory response directed against pathogenic microbes. The host derived molecules responsible for fever used to be known as endogenous pyrogens, as first demonstrated by Paul Beeson in 1948. He described temperature-elevating effect of a substance obtained from polymorphonuclear leucocytes.

The relative roles of exogenous pyrogens and cytokines in fever can be summarized by a quote from the cartoon character Pogo, who said “We have met the enemy and he is us”.

Pattrick Murphy and late Barry Wood were the first to obtain a purified form of endogenous pyrogen from rabbit peritoneal exudate cells.[7]

The late Phyllis Bodel described an intracellular form of Endogenous pyrogen (EP) and reported production of EP by both murine macrophages and human lymphoma cells.[7]

In 1972, Gery and Waksman described the chemical nature of "Lymphocyte-activating factor" which showed striking similarity with endogenous pryrogens.[7]

Kluger and co-workers provided proof that endotoxin-induced fever is mediated by IL-1 B induction of IL-6, suggesting that IL-6 might be the final common pathway for such fever.[7]

Milton and Wendlandt originally proposed that E-series prostaglandins (PGE) might mediate the febrile response to pyrogens. This consensus of opinion still favors the proposition that PGE2, the endogenous isoform of PGE, plays an essential role in production.[8]

Rotondo et all proposed that the PGE2 involved in fever might be generated peripherally, transported to the Pre Optic/Anterior Hypothalamus (POAH) by the blood stream, and then, being Lipophilic, either cross the BBB at this site or diffuse to the POAH through the Organum Vasculosum Laminae Terminalis (OVLT) to cause the induction of fever.[8]

History of thrombocytes

In 1877, Osler coined the term thrombocytes or haematoblasts of Deetjen and Dekhuyzen (1901) and elucidated the role of these third corpuscles as fibrin formers in coagulation.

Thermoregulation

Pathophysiology of fever

Body temperature rarely varies more than 1°C throughout a person's lifetime. There are many interactions between thermoregulation and the homeostatic systems. Thermoregulation is closely related to various non thermal regulatory systems and thus, changes in the body temperature can affect these other systems, just as changes in non thermal parameters can affect these other systems, just as changes in non thermal parameters can affect thermoregulation.[9]

The physiologic mechanisms are controlled by the central nervous system, especially by the neurons in and near the rostral hypothalamus, including the anterior hypothalamus, preoptic area and adjacent septal regions. Such neurons sense changes in the deep body temperature and integrate this information with afferent sensory information from thermoreceptors in the skin and more central locations. In response to peripheral temperature changes, hypothalamic neurons initiate approximate thermoregulatory responses to maintain a constant core temperature.[9]

Neurons in the Supra Chiasmatic Nucleus (SCN) display a circadian rhythm in their firing rates and thermosensitivities, that form the basis of SCN's capacity to serve as a biological clock, that influences the activity of many homeostatic systems, including thermoregulation. Heat is derived from biochemical reactions in all living cells. The biological work ultimately produces

heat. All biochemical reactions display a "Q10 effect" in which warming increase heat producing reactions, leading to more warming.[9]

Shivering is the primary means of increasing heat production in response to cold environment, produced by contraction of the muscles.

Because most heat producing organs lie deep within the body core, the circulatory system plays a vital role in distributing heat throughout the body. Heat loss is often explained in terms of the physical mechanism for heat transfer: Conduction, Convection, Radiation and Evaporation.

Neural control of thermoregulatory response

Thermoregulation is controlled by a continuum of neural structures and connections extending from the hypothalamus and limbic system to the lower brainstem and reticular formation, to the spinal cord, and to sympathetic ganglia. Because the preoptic region is sensitive to its own temperature and controls virtually all thermoregulatory responses, it is often described in terms of a negative feed back loop in a control system that regulates around a set-point temperature. It integrates central and peripheral thermal information, apparently responding to such information by shifting the preoptic set-point temperature.

Neuronal model explaining fever

Various drugs and endogenous substances affect temperature regulation by altering the activity 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°C. In response to the new set -point temperature, thermoregulatory mechanisms are activated to increase the preoptic temperature to 39°C, thus leading to development of fever.

The whole body metabolic rate is increased relative to the febrile state because, as a result of the Q10 effect, increases in temperature induces increases in all metabolic reactions.

As the concentration of preoptic pyrogenic substances decrease, either naturally or as a result of an antipyretic drug, the body temperature begins to return to a normal afebrile level - i.e. “Breaks” or “Defervesence”.[9]

Exogenous pyrogens

The current concept of fever physiology is that host cell derived molecules induce fever, which usually occurs in context of an overall inflammatory response directed against pathogenic microbes.

Three different cytokines - interleukin-1 (IL-1), Tumor Necrosis Factor (TNF) and Interleukin-6 (IL-6) account for endogenous pyrogen activity, and it is clear that exogenous pyrogens by themselves do not cause fever unless they elicit cytokine release.

There are many different substances capable of causing fever in humans: microorganisms (primary cell wall components), microbial toxins, antigen

antibody complexes, activated compliment components (C3a, C5a), pyrogenic steroids (Etiocholanolone), Drugs, Polynucleic acids.

Gram negative bacteria possess two known pyrogens: Lipopolysaccharide (LPS), which is component of the bacterial outer membrane and peptidoglycan, which forms cross link lattice below the outer membrane.

LPS is the most potent stimulus known for TNF production and release. TNF causes fever by affecting brain prostaglandin production. LPS binds to Lipopolysaccharide Binding Protein (LBP) which is present in the normal human sera and its concentration rises 100-fold during acute phase response. LBP catalyses the binding of LPS to LPS receptor known as CD14, which is present on macrophages and granulocytes. This markedly enhances LPS induced inflammatory cytokine production by cells.

Gram positive bacteria lack LPS, but contain peptidoglycan, Lipotechoic acid and a group of rhamnose glucose polymers. The basic structure responsible for peptidoglycan's pyrogenicity is muramyl peptide (MDP-N-acetyl muramyl-L-alanine-d-isoglutamine).

Gram positive bacteria release exotoxins, which can also cause fever. Exotoxins act by binding to Major Histocompatibility Complex - Class I molecules on antigen-presenting cells, which then is able to bind to T-cell receptor, which then become activated and release TNF and IL-1. The ability of exotoxins to activate large numbers of T-cells has led to its designation as super antigen.

Cytokines as endogenous pyrogens

Patrick Murphy and the late Barry Wood were the first to obtain a purified form of Endogenous Pyrogens (EP) from rabbit peritoneal exudate cells. In 1974 two distinct forms of EP were described, both of molecular weight 15,000 with one having pH of 5 and the other with a pH of 6.8 to 7.0, which introduced the concept of multiple, chemically induced distinct EPs.

Like interferon (IFN), TNF and IL-6, IL-1 is produced by many different (non leucocytic) cells and acts on many non leucocytic targets, hence these polypeptides are regarded as a special class of substances called "Cytokines".[10]

The following cytokines are known to be intrinsically pyrogenic, in that they produce rapid onset of fever by acting directly, on the hypothalamus (i.e. without a requirement for the formation of another cytokine): they are IL-1a, IL1b, TNF-α, TNF-β, IFN-α, IL-6. These now include a family of cytokines using the cell-signaling apparatus gp130.

Cells using this receptor are pyrogenic and currently include IL-6, IL-11, oncostatin-M, Ciliary Neurotrophic Factor (CNTF), Cardiotrophin-1 and Leukemic Inhibitory Factor (LIF). Some endogenous molecules can also induce EP not requiring an exogeneous stimuli, for e.g. antigen-antibody complexes, certain ancrogenic steroid metabolites, inflammatory bile acids, and some lymphocyte products.

Endotoxin is an example of a pyrogen, that can both act directly on the hypothalamus to cause fever and induce EP synthesis in various host cells, which then induce fever.[7]

EPs reaching the brain via the systemic circulation do not actually penetrate the Blood Brain Barrier (BBB). It seems more likely that Eps have their effect on the rich vascular network close to the cluster of neurons in the preoptic anterior hypothalamus. These sites called the circumventricular organs or Organum Vasculosum Laminae Terminalus (OVLT), possess little if any BBB. Thus it is likely that endothelial cells lining OVLT either offer no resistance to the movement of EPs into the brain or release arachidonic acid metabolites themselves when they encounter EPs in circulation. Alternatively PGE2α and other prostaglandins might be produced by endothelial cells, which in turn induce a neurotransmitter - like substance that acts to raise the thermal set - point.

PGE2 increases the levels of Cyclic AMP, which does have neurotransmitter properties in brain tissue and has been implicated in the pathogenesis of fever.

IL-1 and TNF induced effects on vascular tissue would make the endothelial surface in OVLT a prime site of action of EPs, in the initiation of fever.[7]

Interleukin-1 (IL -1)

The molecule IL-1 (either IL-1α or IL-1β) is a bonafide EP. At present there are two genes coding for two different IL-1 forms: IL-1α and IL-1β. The

pH 7 form has been designated IL-1β and the PH - 5 form has been designated IL-1α.

Both forms of IL - 1 are translated as 31 - KDa precursor peptides lacking the classic signal or cleavage sequence and hence is processed to its mature forms by enzymatic steps (by IL - 1 converting enzyme) leading to smaller peptides and the 4 K Da peptide causes fever.

The biological properties of the two forms is largely identical. Several studies have shown that IL-1β is readily secreted from activated cells, where as IL-1α remains cell associated and hence IL-1α is likely to be more relevant for local diseases than for systemic disorders.[7]

One of the most potent pro-inflammatory properties of IL-1 is its ability to induce gene expression for cyclooxygenase, resulting in the synthesis of large amounts of prostaglandins.

IL-1 activates cultured vascular endothelial cells in < 1 hour at relatively low concentrations by inducing the expression of Inter Cellular Adhesion Molecule-1 (ICAM-1), on the cell surface. This molecule interacts with the leucocyte-glycoprotein complex designated leucocyte function antigen.

Tumor Necrosis Factor (TNF)

TNF is a macrophage product that is directly cytotoxic for certain tumor cells. Human TNF has been cloned and has the same amino acid sequence as another macrophage product called “Cachectin”.

Recombinant IL-1 and recombinant TNF both stimulate synovial cell production of PGE and collagenases, endothelial cell procoagulant activity and release of Platelet Activating Factor (PAF). Both molecules are cytotoxic for certain tumor cells and both induce hepatic Acute Phase response (APR). In addition, lymphocyte activation, cytotoxicity for insulin producing-β cells are also shared properties of IL-1 and TNF. The receptors for TNF, however are distinct from those of IL-1. TNF also has the property of inducing the production of IL-1 in vivo. TNF increase PGE2 production within 30 minutes. This property is shared by IL-1. Recent studies indicate the TNF-β, a lymphokine also known as lymphotoxin, shares considerable (78%) homology with TNF.[7]

The biological activity of TNF most often measured is its cytotoxic effect on various susceptible cells.

In general systemic responses to LPS appear to be mediated by TNF. In many ways TNF can be viewed as a master fever producing cytokine because it induces both IL-1 and IL-6.

Interleukin-6 (IL-6) and other gp 130-transducing cytokines

IL-6 is a term given to a polypeptide cytokine that was initially isolated from fibroblasts and called interferon-β_2. IL-6 produces typical EP fever when injected into rabbits. 50-100 fold greater concentration of IL-6 are required to cause fever.

IL-1 and TNF are potent stimulators of IL-6 production. IL-6 belongs to a family of cytokines that triggers cells via the glycoprotein (gp) 130 signaling

apparatus. These receptors present on nearly all cells. Pyrogenic cytokines such as IL-6, IL-11, oncostatin-M, CNTF, cardiotrofin-1 all use these receptors.[10]

Kluger and co-workers provided proof that endotoxin-induced fever is mediated by IL-1 β induction of IL-6, suggesting that IL-6 might be the final common pathway for such fever.

The Interferons (IFN)

Interferons were the first cytokines administered to humans. Fever was recognized early as a prominent effect of interferon therapy.

IFN possess several biological activities in addition to antiviral activity. These include important effects such as their capacity for increasing natural killer activity and enhancing expression of class-I and II MHC antigens.

Recombinant human INF-β injected into humans at a dose of 10 to 100 U/kg causes chills and fever within 2 hours.

The IFN are species specific:

- IFN-β is an EP, induces fever via the same mechanism as that governing the activity of other EPs-synthesis of brain prostaglandins.
- IL-1 induces IFN-β. IFN-β is less pyrogenic for humans.
- IFN-β can also be pyrogenic in humans, it stimulates IL-1 production.
- IL-2 infusions induce fever in humans, induces TNF and IFN-β.

The concept of cytokines inducing other cytokines is vital to the understanding of how non-infectious disease produce fever. Vasculitis, rheumatoid arthritis, lupus, trauma, hemorrhage, thrombophlebitis, drug fever and cancer are examples of non-infectious diseases in which both fever and acute phase response are common. Fever is also a prominent side effect of cancer chemotherapy, and the treatment of transplant rejection with antibodies to CD3 is also associated with fever. It has been implicated that in all these diseases cytokines such as IL-1, TNF and IL-6 are involved in pathogenic process.

Prostaglandin E2: A putative Fever Mediator

Milton and Wendlandt originally proposed that E-series prostaglandins (PGE) might mediate the febrile response to pyrogens. PGE_2, the endogenous isoform of PGE plays an essential role in fever production. Induction of PGE_2, is triggered by peripheral signals evoked by early, non cytokine factors elicited in response to infectious challenge.[8]

In 1983, it was proposed that in lieu of entering the Preoptic-Anterior Hypothalamus (POAH) through OVLT, circulating cytokines might interact with specific receptors at this site to induce secondary signals that transducer and transmit the original pyrogenic message to brain and was thus suggested that transmission of blood pyrogenic messages to the brain might be mediated by PGE2.

Prostaglandin E2 synthesis involves the cleavage of Arachidonic Acid (AA) released from membrane phospholipids into the prostaglandin

endoperoxides, PGG_2 and PGH_1:PGH_2 is then quickly converted to PGE_2 by PGE_2 isomerase. The free AA concentration is thus rate limiting. In the context of fever production, phospholipase A_2 (PLA_2) has been considered the key enzyme. Its enhanced activation accounts for the release of AA. Prostaglandin H synthase (consisting of COX and hydroperoxidase together in a single protein) is believed to catalyze the transformation of AA into PGE_2.

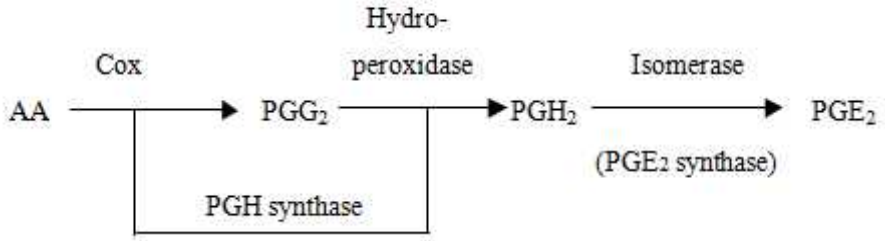


Figure 1: The pathway of PGE_2 synthesis from arachidonic acid.

Humoral mechanism

The anaphylatoxic components of the complement, C3a and C5a are rapidly produced in blood via both the classical and the alternative pathways as a result of the interaction of the lipid A moiety of LPS with C1 and C3. Within seconds of binding to its receptors on MP3, C3a triggers production of PGE_2.

Neural mechanism

It has been suggested that, neural mechanism involves activation of primary sensory nerves by cytokines released in their vicinity, presumably from principle sources such as hepatic macrophages (Kupffer cells). The message of the peripheral cytokines might be transmitted centrally via activated vagal

afferent nerves to the Nucleus Tractus Solitarious (NTS) and passes from NTS to POAH via ascending Noradrenergic (NA) projections originating in A1 and A2 region of the medulla oblongata, arriving in POAH via ventral NA bundle.

PGE_2 induced in the OVL-POAH region by these NA inputs might provide an early triggering signal for fever onset.

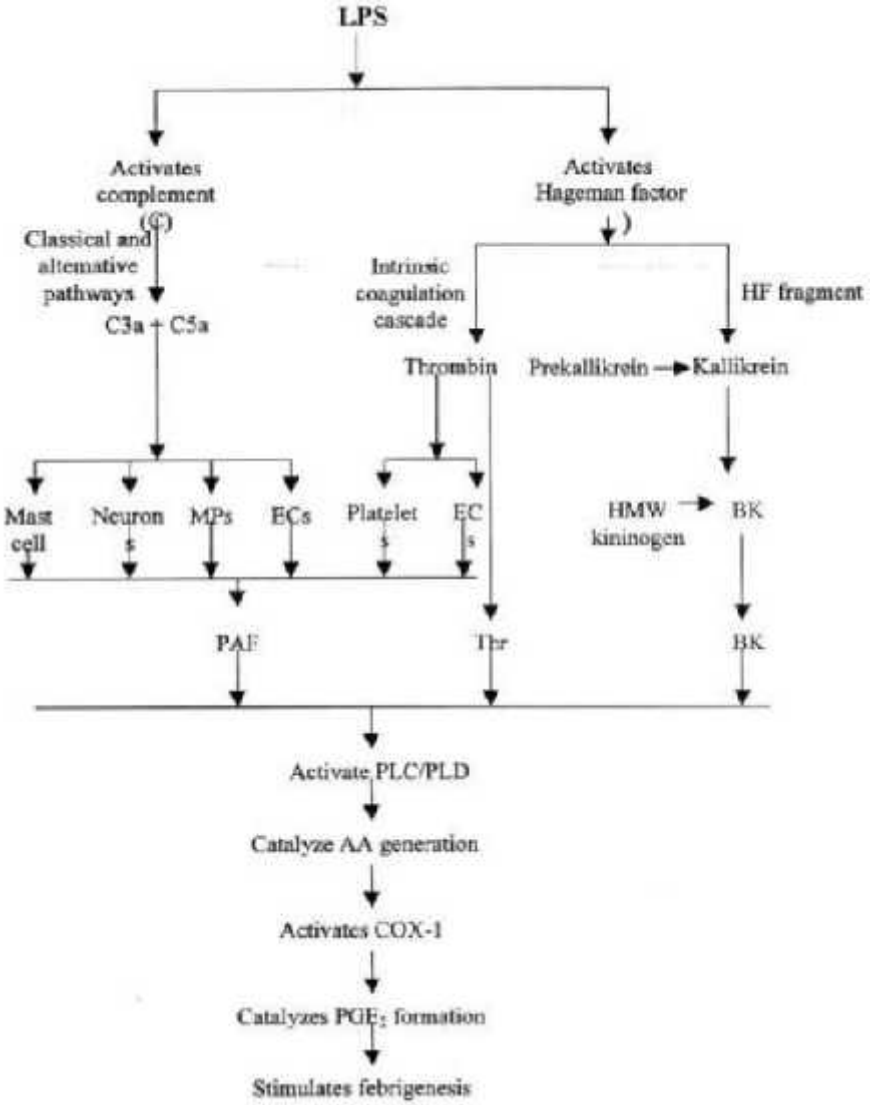


Figure 2: LPS Pathway - The hypothesized successive endogenous mediators evoked by intravenous LPS potentially contributing to the generation of PGE_2 in the OVLT. Ecs, endothelial cell; Thr. Thrombin; BK, bradykinin.

Why are fever temperatures over 106° F rare ?

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

Du Bois placed fever's upper limit between 41^{o}C and 42°C. The mechanism involved in such regulation might lie in the intrinsic properties of the neurons of the hypothalamus itself. Upper limit of febrile range might be determined simply by the maximum and minimum firing rates of the hypothalamus. There might be the release of endogenous antipyretic substances that antagonise the effects of pyrogens on the neurons. These are arginine vasopressin, melanocyte stimulating hormone, antipyretic neurochemicals etc.

In all likelihood, several different mechanisms are involved in the process of endogenous "Refrigeration" that prevents body heat from "Consuming itself" during the febrile response.

Acute phase response

Inflammation is the local response to inflammatory stimuli. If inflammation is severe enough, it is accompanied by a large number of systemic changes, referred to collectively as the Acute phase Response (APR). The APR consists of substitution of new "Set points" for the haemostatic mechanisms that normally maintain a constant internal environment during good health, and is presumed to play a major role in adaptation and defense.

The APR may be transient, dissipating with recovery, or can persist in chronic disease, resulting in chronic APR.

Fever is only one of the changes in homeostatic settings that occur during the APR. Other changes include Somnolence, Anorexia, changes in plasma protein synthesis and altered synthesis of many endocrine hormones. In addition negative nitrogen balance, gluconeogenesis, decreased levels of zinc and iron and increased level of copper occurs. There is leukocytosis and thrombocytosis, decreased erythropoeisis, resulting in what is commonly called “anaemia of chronic disease”.

Stimuli that commonly give rise to APR include bacterial, viral infections, surgical or other trauma, neoplasms, tissue infection, various immunologically mediate inflammatory states, strenuous exercise and child birth.

In a narrower sense APR refers to changes in concentrations of a large number of plasma proteins, the Acute phase Proteins (APP), which reflect reprogramming of the pattern of gene expression of secretary proteins in hepatocytes.

The two major human APP are C-reactive Protein (CRP) and Serum Amyloid A (SAA), Whose levels are increased by greater than 1000 fold in the plasma following stimulation in infected individuals. Positive APP (levels increased): Ceruloplasmin and the complement components C3, C4 and other proteins. Negative APP (levels decreased/synthesis is decreased): Albumin transthyretin, α_2-HS glycoprotein.

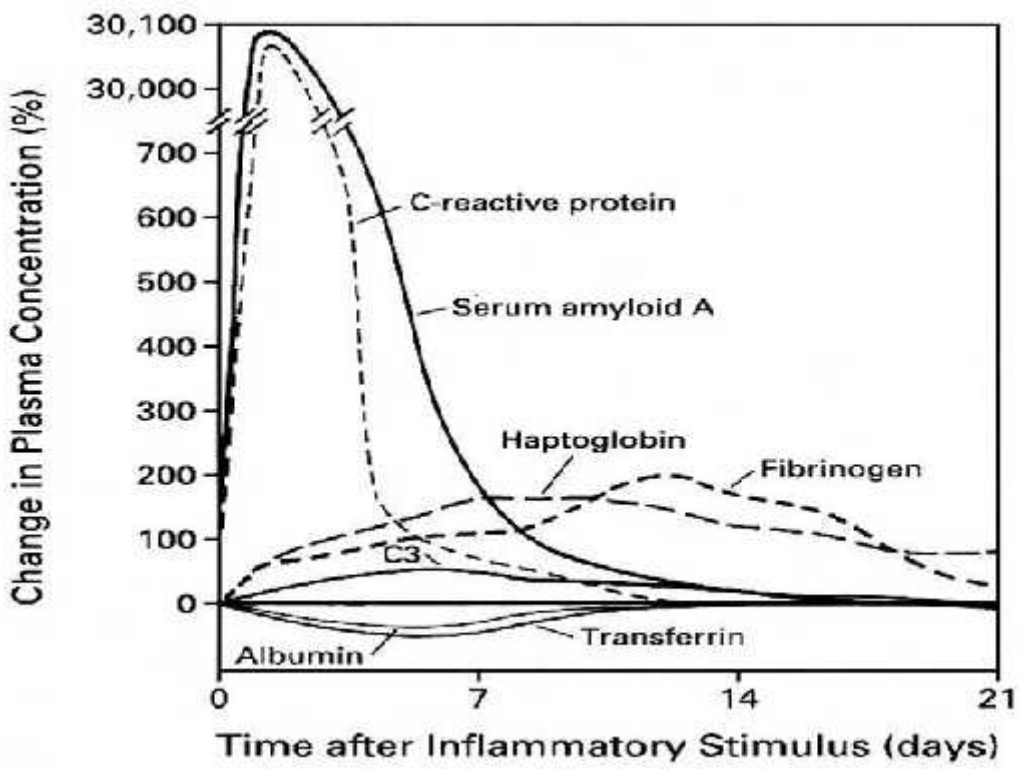


Figure 3: Acute phase reactants - Characteristic patterns of change in concentrations of some plasma proteins following a moderate inflammatory stimulus.

C-Reactive protein (CRP)

The major function of CRP has been presumed to be related to its ability to bind specifically to phosphocholine and, thus recognize some foreign pathogens as well as the phospholipid constituents of damaged or necrotic cells. The CRP then can activate the complement system, which suggest that it can initiate the elimination of targeted cells by interacting with humoral and cellular effect or systems of inflammation. Net induces adhesion and chemotaxis of phagocytic cells and lymphocytes.

The complement components, many of which are acute phase reactants can effect chemotaxis, opsonization and may lead to cytotoxity as well.

Fibrinogen can lead to endothelial cell adhesion, spreading and proliferation, all critical to tissue repair.

In the given individual, the APR represents the integrated sum of multiple, separately regulated, physiologic alterations.

Heat shock proteins (HSP)

One of the most interesting events in the febrile response is the heat shock stress or response. HSP participates in the development and maintenance of the thermotolerant state. Thermotolerance describes the phenomenon by which cells and animals previously expressed to a single intense but sublethal conditioning heat stress, become tolerant to a subsequent otherwise lethal heat stress.[11]

The inducers of Heat Shock Response are: Temperature (Hyperthermia and hypothermia), Ischemia, Hypoxia, Accident stress, endotoxin, cytokines (IL-1 and TNF), chemotherapeutic agents etc.

HSP confers protection against ischemic injury to the kidney, liver, and heart. HSP 70 gene has shown tolerance to both thermal and hypoxic stress.[11]

Alterations in the cellular energy status and ischemic stress are strong inducers of heat shock response. During these metabolic stresses, oxidative uncoupling might occur in mitochondria, leading to enhanced oxyradical

generation, leading to generation of HSP there by generating antioxidant enzymes.

The physiologic stress of fever involves temperature elevation, cytokine release, and increased metabolic activity. HSP plays important roles in the cellular response to these stresses. These include protein management, regulation of cytokine production and secretion, etc. HSP serves to stabilize epithelial and endothelial cell barriers.

It may be well that the febrile response, through the induction of cellular HSP, serve to generate a tolerant state that enables the organism to withstand further damage from endotoxin, cytokines and metabolic insults.[11]

Definitions of febrile patterns

The types of febrile patterns have been traditionally grouped according to the definitions listed below. Often, within these groups, specific infectious diseases may occur.[12,1]

1. ***Continuous (sustained):*** Fever does not fluctuate more than about (1.5°F) during 24 hours, but at no time touches the normal. Eg: Pneumonia, Rickettsial diseases, Typhoid fever, central nervous system disorders, Tularemia, and Falciparum (malignant tertian malaria).[1]

2. ***Intermittent fever:*** When fever is present only for several hours during the day, it is called intermittent fever. When a paroxysm of intermittent fever occurs daily, the fever is described as Quotidian, when on alternate

days, it is tertian, when two days intervene between consecutive attacks, it is quartan.[1]

Eg: Localized pyrogenic infections and bacterial endocarditis; Malaria (commonly with leukopenia) may present as quotidian (daily spike), tertian (spike every third day), or quartan (spike every fourth day) types.

A double quotidian pattern with two daily spikes occurs sufficiently often to be helpful in salmonellosis, Miliary tuberculosis, double malarial infections, and gonococcal and meningococcal endocarditis.

3. ***Remittent Fever:*** Fever with daily fluctuation exceeding 2°C in 24 hours.

4. ***Relapsing fever:*** Short febrile periods punctuating one or several days of normal temperature. Eg: Pel-Ebstein fever - Hodgkin's disease, Brucellosis of the Brucella melitensis type, Rat-bite fever, Dengue fever, Yellow fever, etc.

5. ***Saddleback (biphasic fever):*** With several days of fever, a gap of reduced fever of about 1 day, and then several additional days of fever. This type characterizes dengue and yellow fever, Colorado tick fever, Rift Valley fever and viral infections such as influenza, poliomyelitis, and lymphocytic chroiomeningitis.

Fever should be regarded as a reliable clinical sign and with the fever pattern mentioned above, it is possible to suggest a diagnosis within the group of disease and this can lead to specific therapy and ultimate cure.

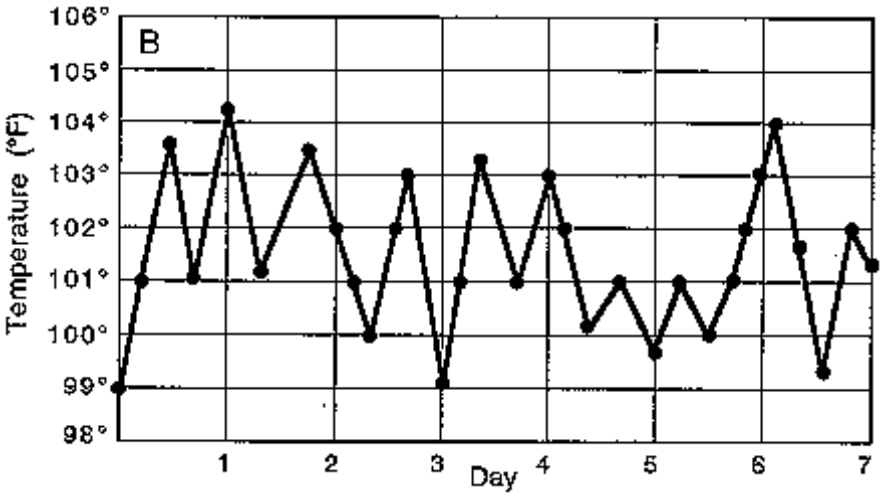


Figure 4: Remittent fever

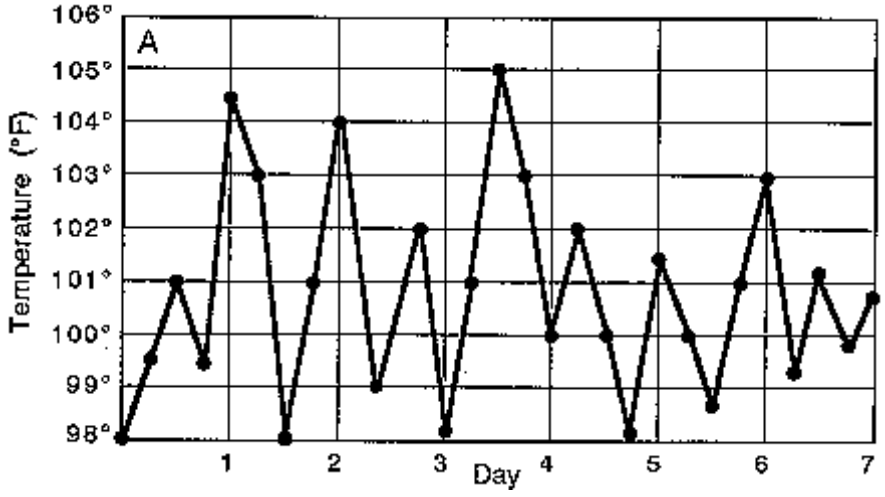


Figure 5: Intermittent fever

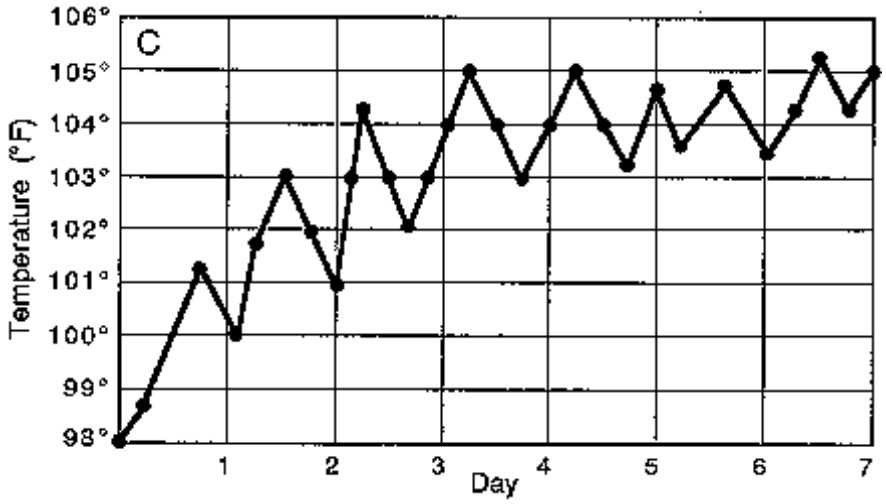


Figure 6: Continuous fever

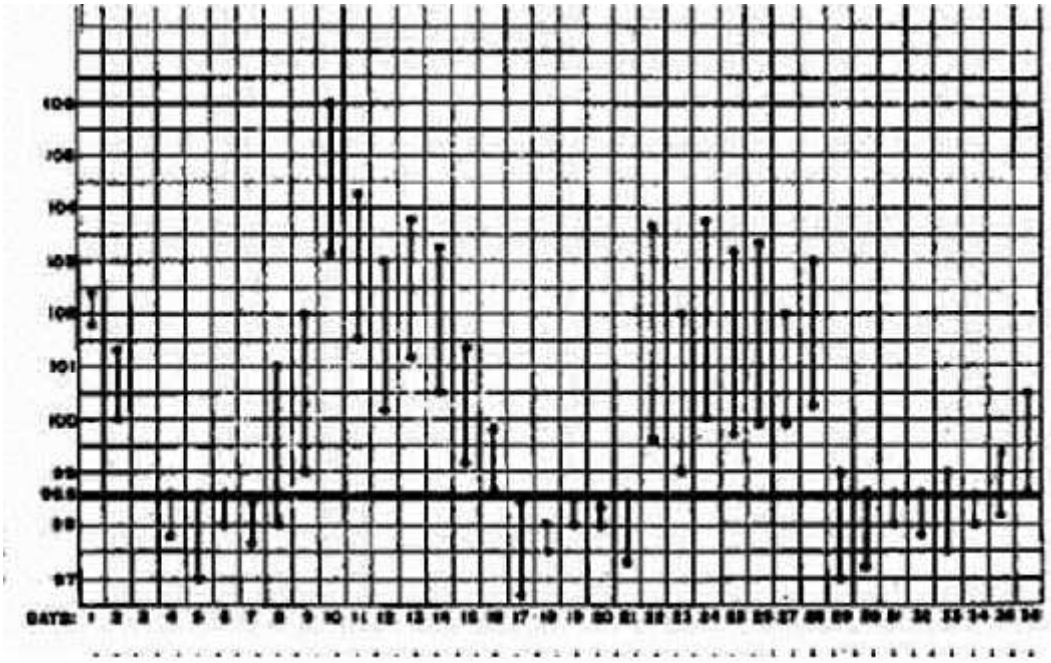


Figure 7: Pel-Ebstein fever

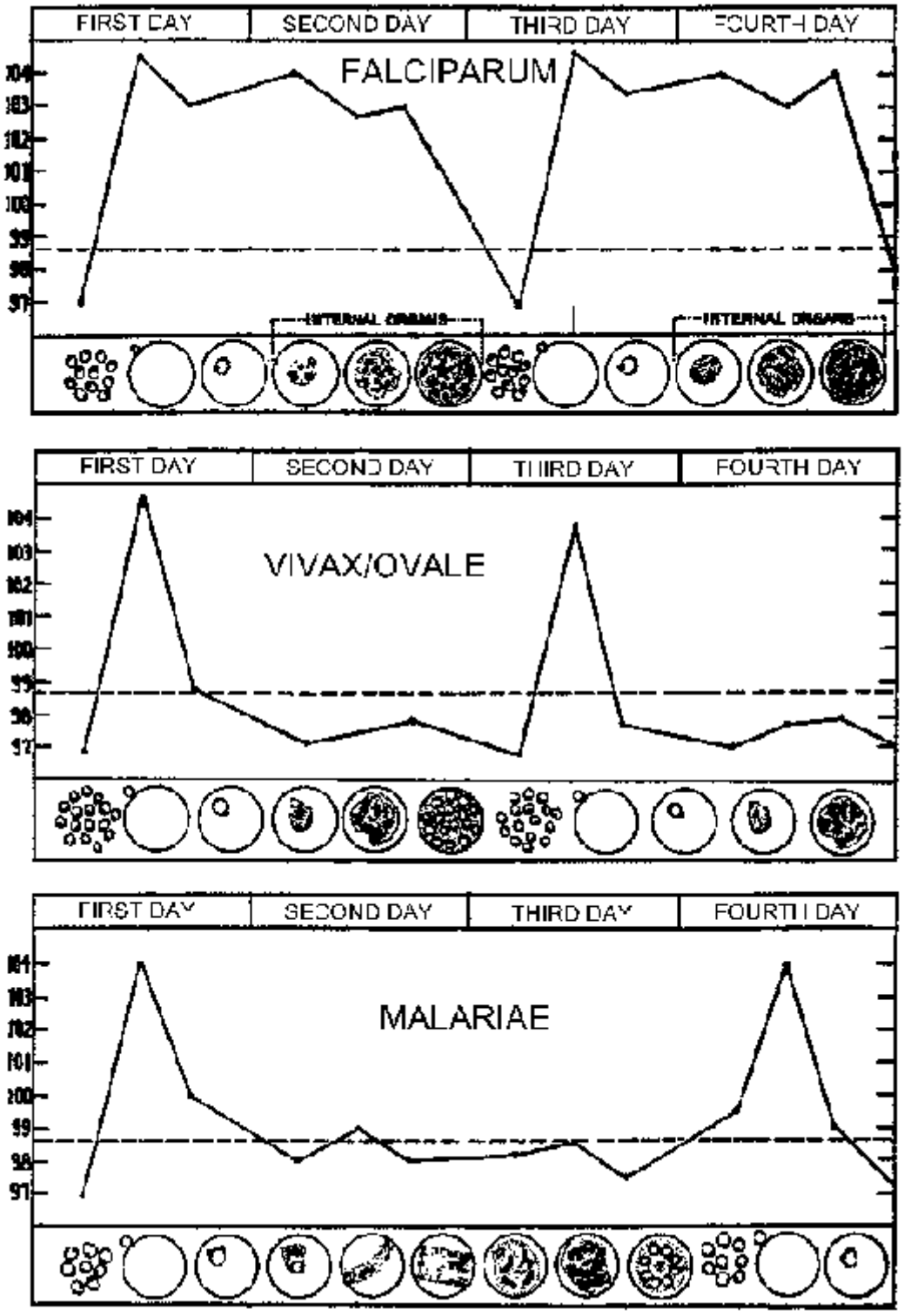


Figure 8: Malarial fever patterns

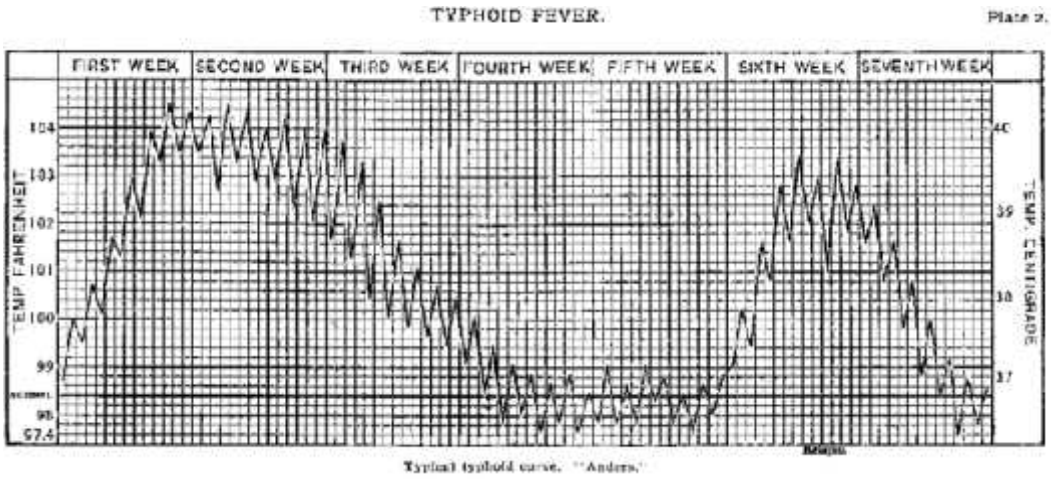


Figure 9: Classical typhoid fever pattern

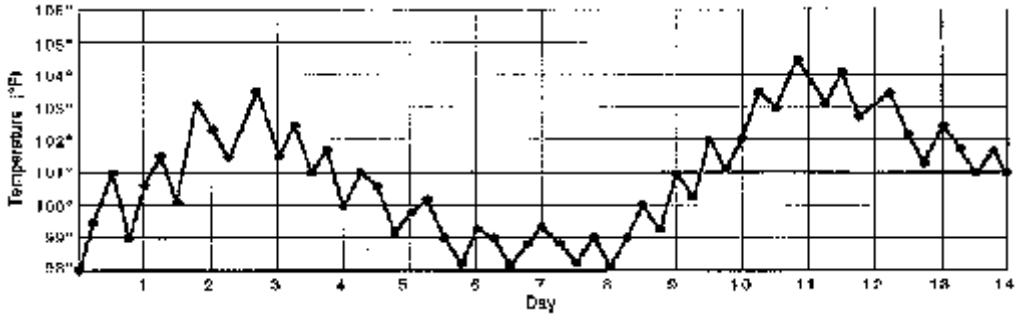


Figure 10: Relapsing fever

Fever Magnitude

Most infectious diseases produce temperatures between 37°C and 41°C (99°F and 106°F). However some patients with infectious diseases remain afebrile. These include immunocompromised hosts, those with chronic renal insufficiency, alcoholics and elderly persons.

Extreme pyrexia - Temperature exceeding 108° F- rarely, if ever accompanies an infectious disease. Conditions, in which extreme pyrexia is seen, include drug fevers, central nervous system fevers, malignant hyperthermia, heat stroke and human immunodeficiency virus infection.

Hypothermia is always a bad prognostic sign in the presence of infectious disease. This condition is seen with overwhelming sepsis, uremia, cold exposure and hypothyroidism.

It is well established that patients do better clinically when they have some degree of fever, which indicates their ability to respond to infection in general.

THROMBOCYTOPENIA

Thrombocytopenia is defined as a reduction in the peripheral blood platelet count below the lower normal limit of 150,000/µl. Because platelet counts are prone to error, a single platelet count that is lower than normal should be confirmed by a second count. It should also be confirmed by inspecting the blood film.[13,14]

Thrombocytes are involved in both thrombotic and bleeding disorders. Abnormalities of platelet production might lead to either dysfunction.

The life span of platelets once they enter the circulation is about 8-10 days. About 10% of the population is destroyed each day.[14]

Life span is measured by labeling the platelet with radioactive Chromium (51Cr) or Indium (111In).[14]

Thrombocytopenia may result from impaired platelet production, accelerated platelet destruction, or dilution/splenic sequestration.[13,14]

Table 1: Causes of thrombocytopenia

Decreased marrow production	• Marrow infiltration with tumor, fibrosis • Marrow failure- aplastic, hypoplastic anemias, drug effects
Splenic sequestration of circulating platelets	• Splenic enlargement due to tumor infiltration • Splenic congestion due to portal hypertension
Increased destruction of circulating platelets	**Nonimmune destruction** • Vascular prostheses, cardiac valves • Disseminated intravascular coagulation • Sepsis • Vasculitis.
	Immune destruction • Autoantibodies to platelet antigens • Drug-associated antibodies • Circulating immune complexes (Systemic lupus erythematosus, viral agents, bacterial sepsis).

Thrombocytopenia associated with infection

Purpura was recognized as a manifestation of peltisutial fever 2000 years ago. Several factors are known to cause bleeding in association with infections of which thrombocytopenia is the common cause.[14]

Viral causes

The viral causes are CMV, Dengue, Parvo-B19, HSV, HIV, Hantana virus etc.[14]

Mechanism

Viruses produce thrombocytopenia by impaired platelet production, as a result of invasion of megakaryocytes by the virus, toxic effects of viral protein on progenitor cells, virus induced haemophagocytosis, destruction of circulating platelets by viruses - by viral antigen-antibody complexes.[14]

Bacterial causes

Gram +ve and gram -ve septicemia, Miliary tuberculosis, Leptospirosis, Typhoid , Mycoplasma pneumonia, etc.[14,15]

Septicemia resulting from both gram negative and gram positive is the commonest cause of thrombocytopenia. May be caused by disseminated intravascular coagulation (DIC) and the diagnosis of DIC may be apparent when coagulation studies are performed.[15]

About 46% of these patients have elevated platelet associated Immunoglobulin G without evidence of DIC.[15]

Platelets adherence to damaged vascular surfaces also accounts for thrombocytopenia in certain bacterial infections, such as meningococcemia. Endotoxin, exotoxin, platelet activating factor may damage platelets, resulting in increased clearance.[14]

Patients with sepsis syndrome may develop hemophagocytic histiocytosis with phagocytosis of platelets, white cells and platelets in bone marrow histiocytes.[15]

Protozoal causes

Thrombocytopenia occurs in over 80% of patients with malaria and human platelets have been demonstrated to contain plasmodia species.[14]

Experimental evidence suggested it was immune mediated destruction with elevated platelet activated immunoglobulin. However in 1993, it was demonstrated that ultra structural changes in platelets, and the level of parasitemia was the cause for thrombocytopenia.

Other causes

Certain hematological conditions also cause thrombocytopenia by marrow infiltration (lymphoma, leukaemia).[14]

CLINICAL COMPLIATIONS OF THROMBOCYTOPENIA

- Platelet count of more than one lakh, are usually asymptomatic and bleeding time (BT) remains normal.[13]

- Platelet count of 50,000 - 1,00,000 cause mild prolongation of the BT, bleeding occurs only after severe trauma.[13]

- Platelet count of less than 50,000 have easy bruising, manifested by skin purpura after minor trauma.[13,14]

- Platelet count of less than 20,000/µl have spontaneous bleeding. They usually have Petechiae, and may have intracranial or spontaneous internal bleeding.[13,14]

P.S. Nair (2003) conducted a study of fever with thrombocytopenia and concluded that septicemia was the commonest cause.[16]

INVESTIGATIONS

The diagnostic work up of patients with fever and thrombocytopenia should include battery of investigations including biochemical tests; haemograms; peripheral smear etc.

Complete haemogram

1. ***ESR:*** More than 30 mm/hr suggests - TB; malignancy. It's a non specific test, it is raised in most conditions.

2. ***Leucopenia:*** In early dengue, before the IgM ELISA dengue is positive.

3. ***Leucocytosis:*** Predominantly neutrophils indicates septicemia.

4. ***Blood smear:*** Dohle bodies, toxic granules suggest septicemia also should be examined for malarial parasites.

Rapid spot test

This test is used for plasmodium vivax and plasmodium falciparum species. It is very sensitive for detection of malaria.

WIDAL

WIDAL tube method is used for identification of enteric fever.

IgM ELISA dengue

This test will be positive after fifth day of fever and rising titres are indicative of dengue.

IgM ELISA leptospiral antibodies

Used in very acute toxic presentation with conjunctival suffusion and renal and/or liver parameters are being abnormal.

Blood culture

At least 3 blood culture samples should be taken. Special techniques are required for fastidious organisms to grow, and incubation has to be continued for at least 2 weeks.

Bone marrow examination

Used in cases of leukemia, lymphoma etc.

So in patients with fever and thrombocytopenia with renal and liver parameters being abnormal, it is very important to consider:

a. Malarial infections

b. Leptospiral

c. Dengue infections

d. Septicemia with multiorgan dysfunction syndrome

Platelet counts should also be repeated and observed for bleeding manifestations.

Platelet transfusions are indicated when platelet count is <20,000.

Treating the underlying condition will result in drastic improvement of platelet count and its complications.

METHODOLOGY

The present study was conducted in the Department of Medicine, Jawaharlal Nehru Medical College, Belgaum on patients with fever and thrombocytopenia, who were admitted to KLES Dr. Prabhakar Kore Hospital and MRC, Belgaum during the period of January 2008 to December 2008.

Study design

One year cross-sectional study.

Study period

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

Method of collection of data

Source of Data

Patients with fever and thrombocytopenia admitted to the medical wards of KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belgaum.

Sample size

The present study comprised of 200 patients with fever and thrombocytopenia.

Sampling procedure

As no specific data was available, an estimated number of 200 cases, fulfilling the selection criteria and admitted to Medical wards of KLES Prabhakar

Kore Hospital and Medical Research Centre, Belgaum during the study period were included in the study.

Selection criteria

Inclusion Criteria

- Age more than 12 years.
- Fever less than 15 days duration.
- Fever more than 100^0 F atleast once a day.
- Platelet count less than 1,00,000/µl

Exclusion Criteria

- Known patients of Primary Thrombocytopenias
- Drug induced thrombocytopenia.

Procedure

The study was approved by the Ethical and Research Committee of Jawaharlal Nehru Medical College, Belgaum. During the study period, all patients presenting with fever and thrombocytopenia were screened for eligibility. The patients fulfilling the selection criterion were selected for the study and written informed consent (Annexure–I) was obtained. Further they were subjected to a detailed history and clinical examination according to predesigned and pretested proforma (Annexure II).

Once the patients are admitted with fever and thrombocytopenia, a careful history was recorded, general physical examination was done. Detailed

examination of various systems was done. Routine investigations were done. Specific and special investigations were done as and when indicated like,

- Complete blood count
- Erythrocyte sedimentation rate
- Platelet count
- Peripheral smear for morphology
- Peripheral smear for malarial parasite
- QBC for malarial parasite
- Bleeding time and clotting time
- Urine routine and microscopy
- Blood culture
- Urine culture
- Mini Renal Profile
- Liver function tests
- WIDAL
- Dengue IgG, IgM
- Brucella Standard Agglutnation Test
- Leptospira ELISA
- Bone marrow examination
- Other necessary special investigations

In whom a final definite diagnosis was reached, were treated for the disease, and in those who were affordable platelet count was repeated at the time of discharge and no effort was made to gather follow-up information, if the patient was not followed up in our institution.

Details of history, general physical examination and laboratory and technical investigation reports were noted down from time to time.

Once the specific diagnosis was reached, patients were treated for it specifically and symptomatically (Mechanical ventilators, haemodialysis etc.)

For bleeding complications platelet transfusions was done if platelet count was ≤ 10,000/µl. The causes of fever with thrombocytopenia are numerous. So a simple workable classification is presented in –

1. Viral causes: CMV; Dengue; Parvo-B19; HSV, HIV, Hantana etc.
2. Bacterial causes: Gram +ve and -ve septicemia, Miliary tuberculosis, Leptospirosis, typhoid etc.
3. Protozoal causes: Malaria.
4. Others: Leukemia, lymphoma, etc.

The patients were followed from the day of admission till their discharge from the hospital.

Statistical methods

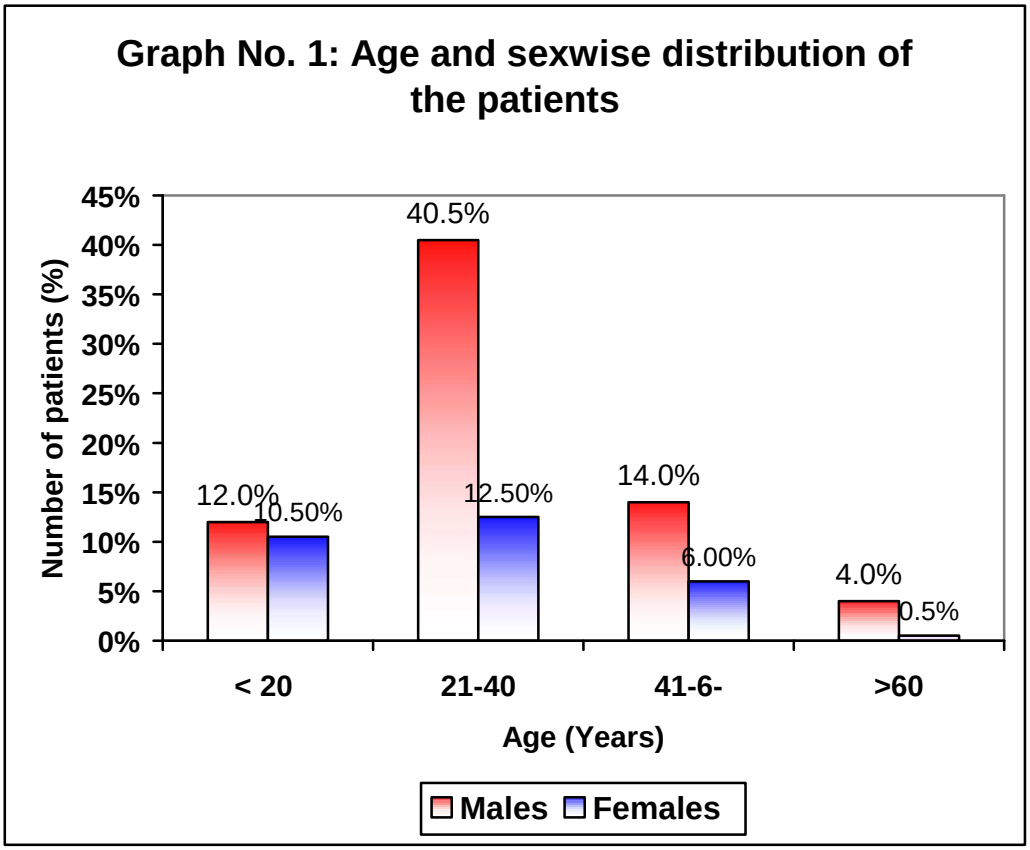
The data was tabulated and rates and ratios were computed. A p value of less than 0.05 was considered as significant.

RESULTS

The present study was conducted in the Department of Medicine, Jawaharlal Nehru Medical College, Belgaum on patients with fever and thrombocytopenia, who were admitted to KLES Dr. Prabhakar Kore Hospital and MRC, Belgaum during the period of January 2008 to December 2008. A total number of 200 patients admitted over a period of one year in our hospital were studied. The observations and findings were recorded as below.

Table 2: Age and sexwise distribution of the patients

Age in years	Males		Females	
	Number	Percentage	Number	Percentage
≤ 20	24	12.0%	21	10.5%
21 – 40	81	40.5%	25	12.5%
41 – 60	28	14.0%	12	6.0%
>60	08	4.0%	01	0.5%

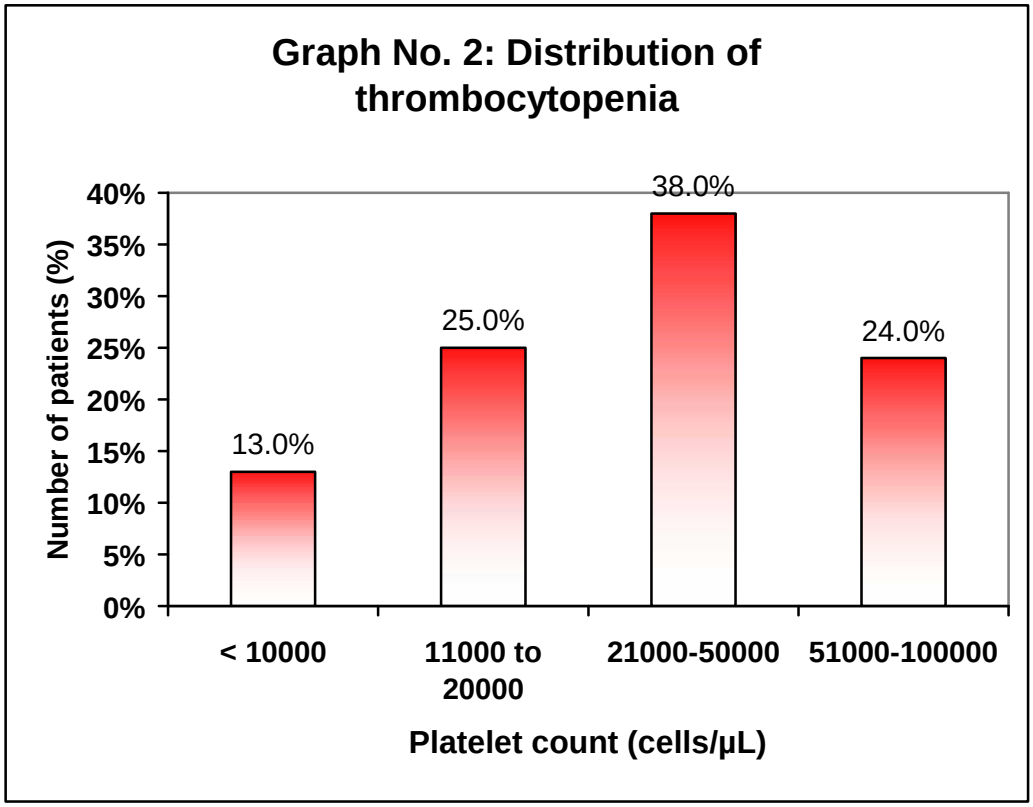


No particular age group was considered, but the study subjects were in the age group of 13-85 years.

The sex of the patient was not taken into consideration for the study. Out of 200 cases of fever with thrombocytopenia, 141 were males and 59 were females.

Table 3: Distribution of thrombocytopenia

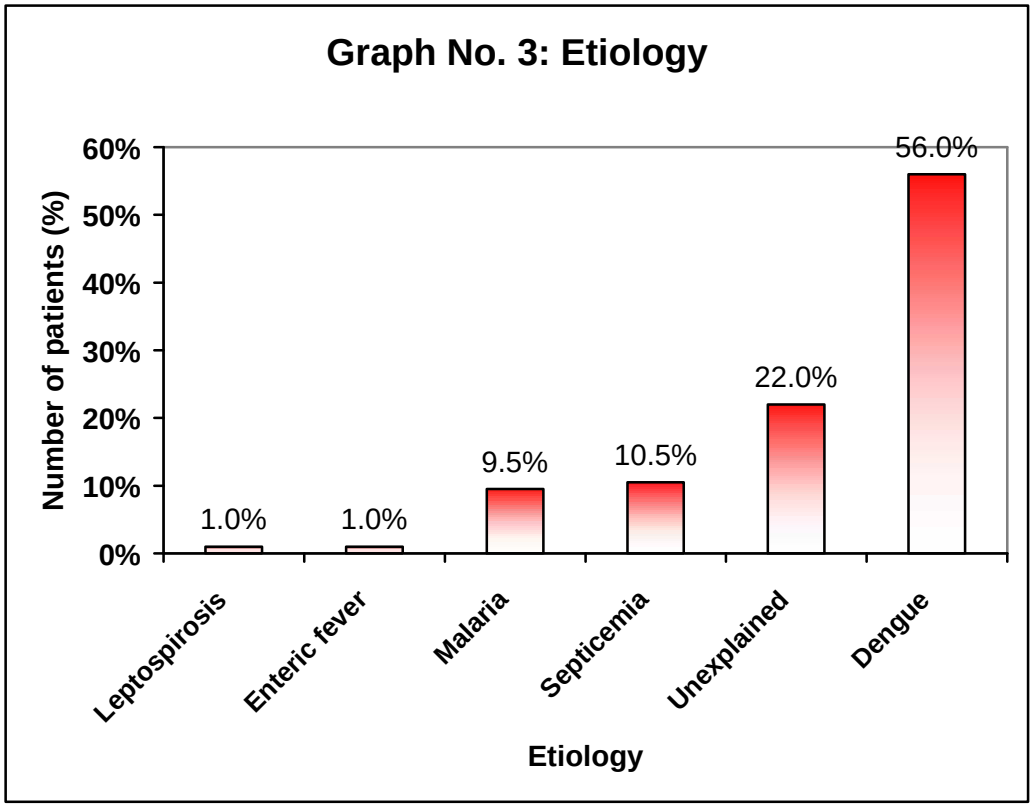
Platelet count (Cells/µl)	Thrombocytopenia	
	Number	Percentage
≤ 10000	26	13%
11 – 20000	50	25%
21 – 50000	76	38%
51 – 100000	48	24%



In our study 24% of the patients had platelet count in the range of 51,000 - 1,00,000, followed by 38%, 25% and 13% of the patients had platelet count in the range of 21,000 - 50,000; 11000-20,000 and ≤ 10000 respectively.

Table 4: Etiology

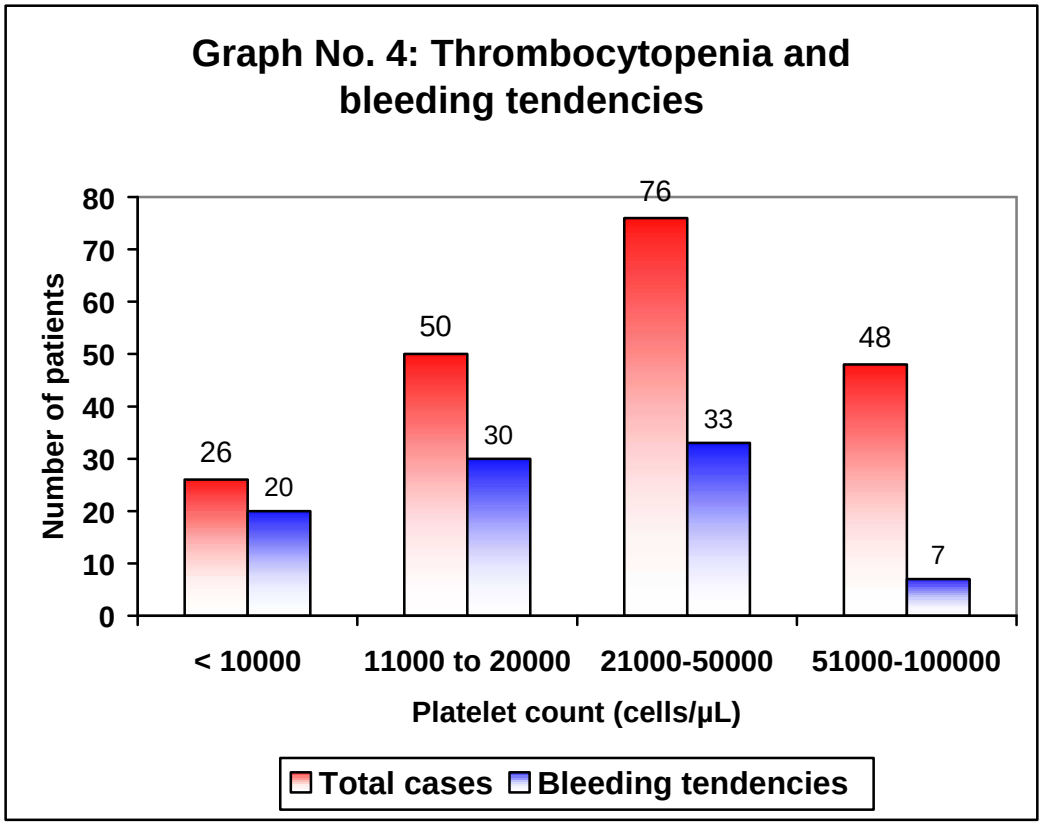
Etiology	Number of patients	
	Number	**Percentage**
Leptospirosis	2	1%
Enteric Fever	2	1%
Malaria	19	9.5%
Septicemia	21	10.5%
Unexplained	44	22%
Dengue	112	56%



Out of 200 cases, a definitive diagnosis could be made in 156 Cases. Among them Dengue was the major cause accounting for 112 cases and 56% of the total cases. Second major cause was Unexplained Causes in 44 cases (22%), followed by Septicemia in 21cases (10.5%), Malaria in 19 cases (9.5%), Enteric fever in 2 cases (1%) and Leptospirosis in 2 cases (1%). In the 19 cases of Malaria, 12 cases of mixed malaria (63%) was the commonest cause, followed by 6 cases of Vivax malaria (32%), and 1 case of Falciparum malaria (5%).

Table 5: Thrombocytopenia and bleeding tendencies

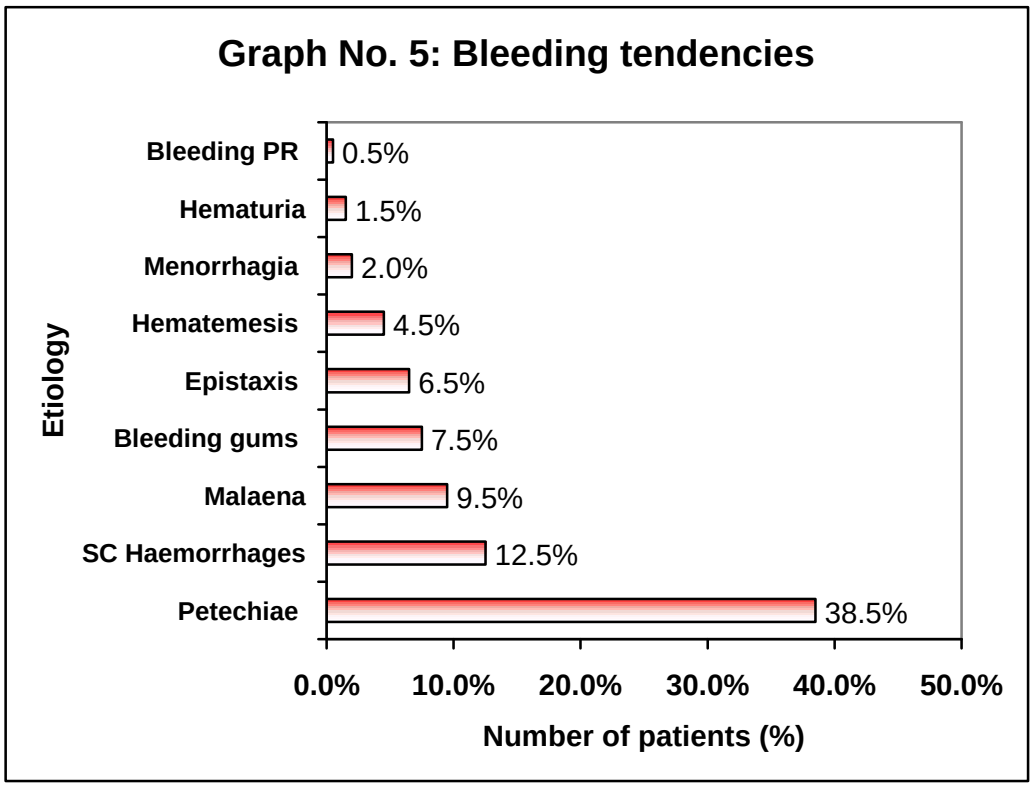
Platelet count (Cells/µl)	**Total number of patients**	**Bleeding tendencies**	
		Number	**Percentage**
≤ 10000	26	20	76.9%
11 – 20000	50	30	60.0%
21 – 50000	76	33	43.40%
51 - 100000	48	07	14.6%



Clinical manifestation of thrombocytopenia was there in 90 patients and there was no clinical manifestation of thrombocytopenia in 110 patients. Bleeding tendencies are more commonly seen in patients with platelet count less than 20,000.

Table 6: Bleeding tendencies

Bleeding Tendencies	Number of patients	
	Number	**Percentage**
Petechiae	77	38.5%
Sub conjunctival hemorrhages	25	12.5%
Malaena	19	9.5%
Bleeding gums	15	7.5%
Epistaxis	11	6.5%
Hematemesis	9	4.5%
Menorrhagia	4	2%
Hematuria	3	1.5%
Bleeding PR	1	0.5%

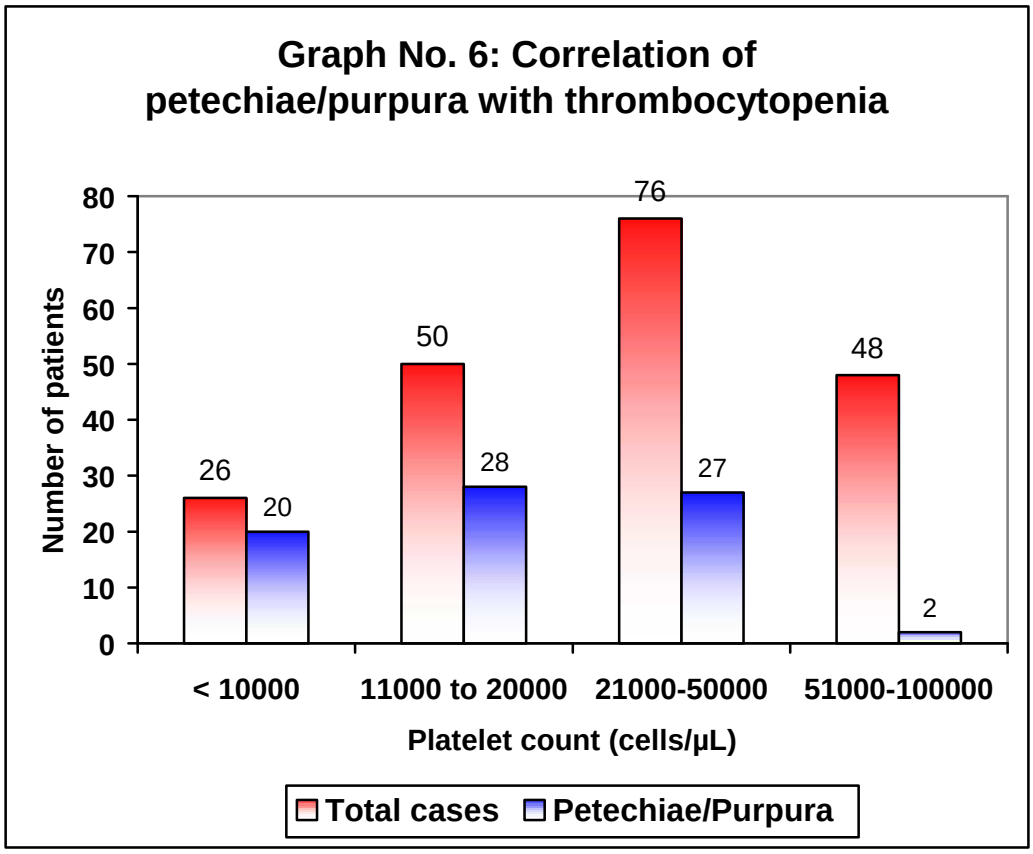


Out of 90 patients, 77 patients (85%) had petechiae/purpura and spontaneous bleeding was seen in 51 patients (57%).

Out of the 51 cases having spontaneous bleeding, 25 cases (49%) had Sub-conjunctival hemorrhages, 19 cases (37%) had Malaena, 15 cases (29%) had Bleeding gums, 11 cases (22%) had Epistaxis, 9 cases (18%) had Hematemesis, 4 cases (8%) had Menorrhagia, 3 cases (6%) had Hematuria and 1 case (2%) had Bleeding per rectum.

Table 7: Correlation of Petechiae/purpura with thrombocytopenia

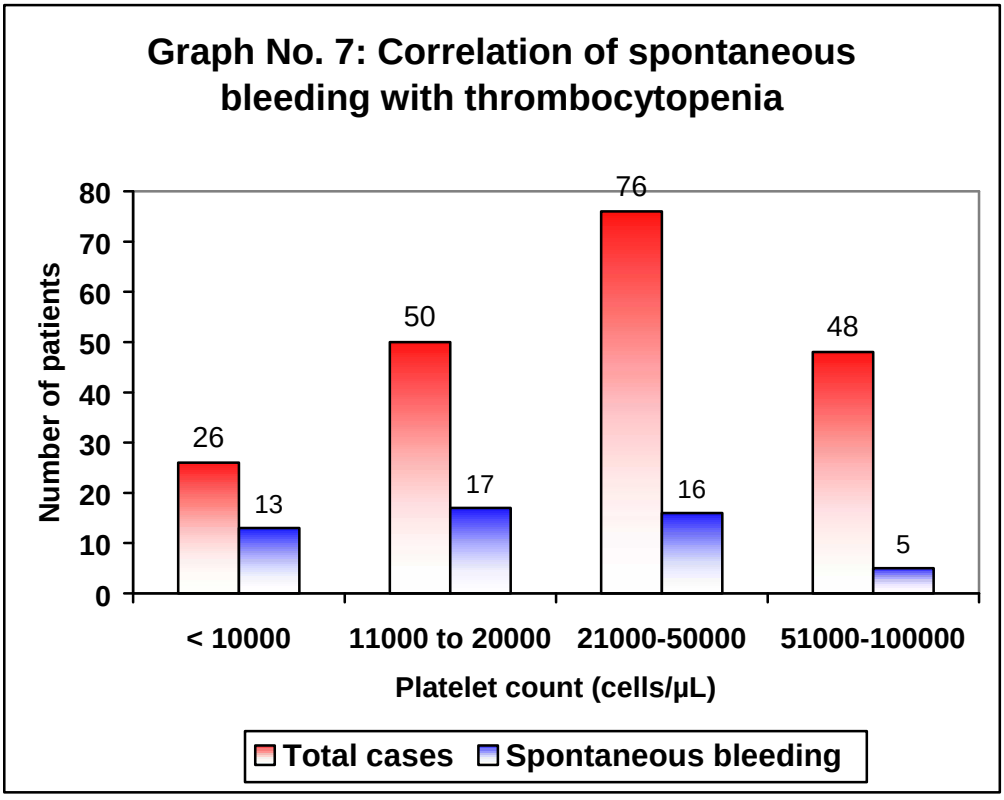
Platelet count (Cells/µl)	Total number of patients	Patients with Petechiae / purpura	
		Number	Percentage
≤ 10000	26	20	80%
11 – 20000	50	28	56%
21 – 50000	76	27	35%
51 - 100000	48	02	4%



Petechiae/purpura are more commonly seen in patients with platelet count less than 20,000 and usually seen upto platelet counts of 50,000.

Table 8: Correlation of spontaneous bleeding with thrombocytopenia

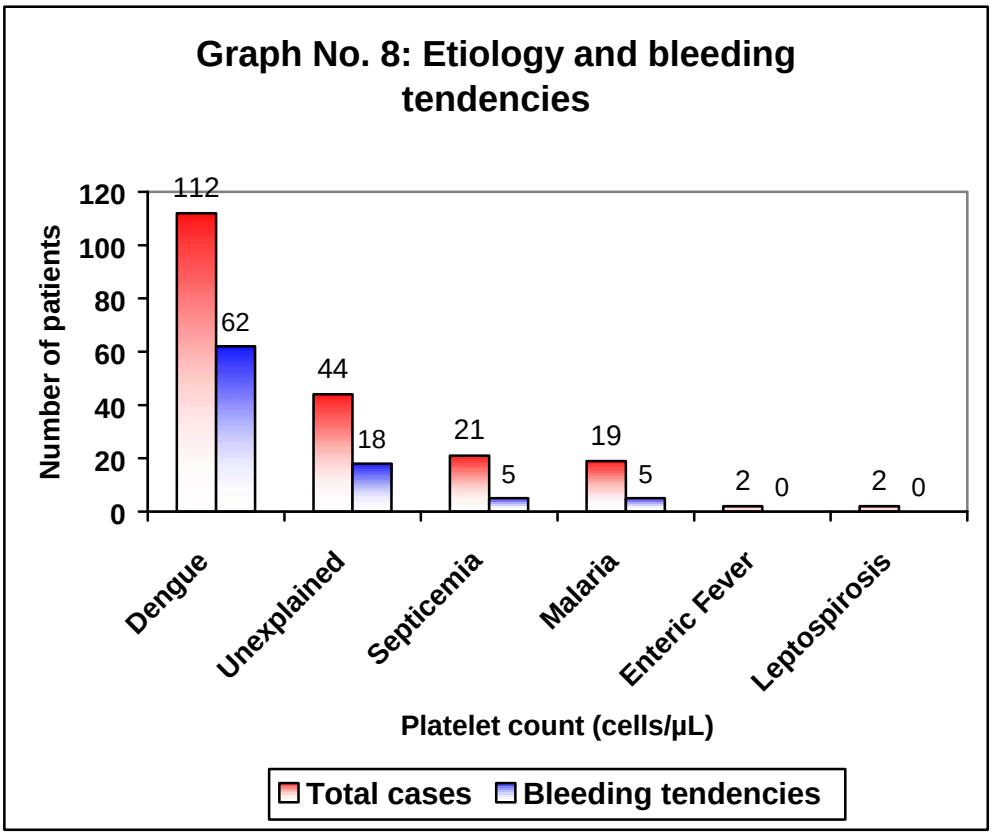
Platelet count (Cells/µl)	Total number of patients	Spontaneous bleeding	
		Number	Percentage
≤ 10000	26	13	50.00%
11 – 20000	50	17	34.00%
21 – 50000	76	16	21.00%
51 - 100000	48	05	10.00%



Generally, spontaneous bleeding was noted in platelet count < 20,000 but in some due to qualitative defects it was seen in platelet count in the range of > 50,000/µl also. Some patients with platelet count of 10,000 did not have spontaneous bleeding.

Table 9: Etiology and Bleeding tendencies

Platelet count (Cells/µl)	Total number of patients	Bleeding tendencies	
		Number	Percentage
Dengue	112	62	55.30%
Unexplained	44	18	40.90%
Septicemia	21	05	23.80%
Malaria	19	05	26.30%
Enteric Fever	2	00	0.0%
Leptospirosis	2	00	0.0%



Out of the 112 cases of Dengue, 62 cases (55.3%) had Bleeding tendencies. Out of the 44 cases of Unexplained causes, 18 cases (40.9%) had Bleeding tendencies.

Out of the 21 cases of Septicemia, 5 cases (23.8%) had Bleeding tendencies. Out of the 19 cases of Malaria, 5 cases (26.3%) had Bleeding tendencies.

Bleeding tendencies were more common in Mixed forms of Malaria.

Table 10: Thrombocytopenia and Bleeding tendencies in Dengue cases

Platelet count (Cells/µl)	Total number of patients	Bleeding Tendencies	
		Number	Percentage
≤ 10000	15	13	86.70%
11 – 20000	29	23	79.30%
21 – 50000	51	23	45.10%
51 – 100000	17	03	17.60%

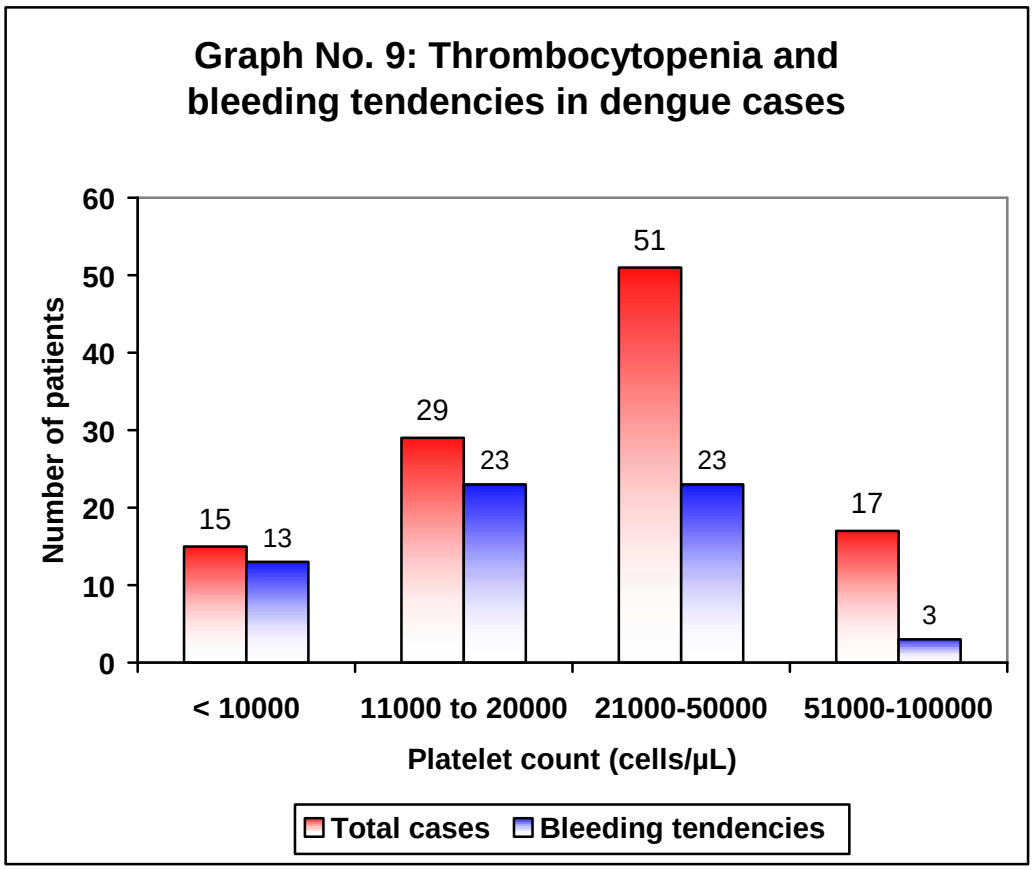


Table 11: Thrombocytopenia and Bleeding tendencies in Unexplained causes

Platelet count (Cells/µl)	Total number of patients	Bleeding Tendencies	
		Number	Percentage
≤ 10000	08	05	62.50%
11 – 20000	08	05	62.50%
21 – 50000	09	05	55.60%
51 – 100000	19	03	15.80%

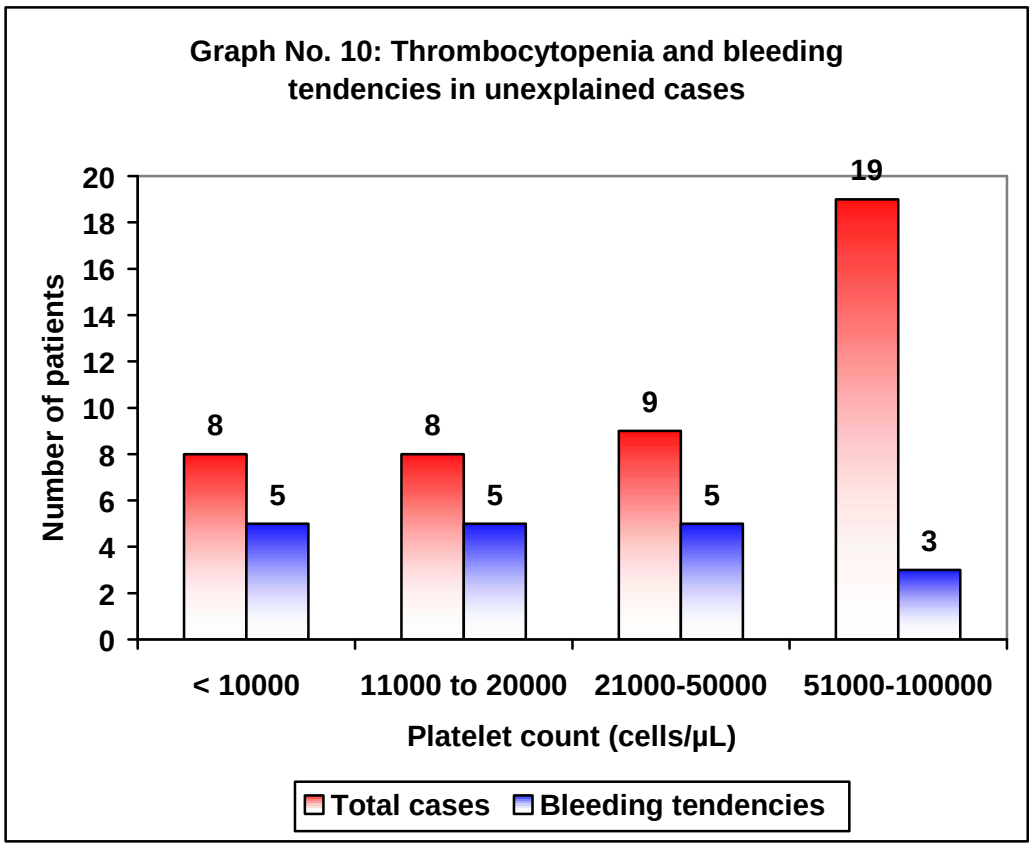


Table 12: Thrombocytopenia and Bleeding tendencies in Septicemia cases

Platelet count (Cells/µl)	Total number of patients	Bleeding Tendencies	
		Number	Percentage
≤ 10000	02	01	50.00%
11 – 20000	07	02	28.60%
21 – 50000	05	01	20.00%
51 – 100000	07	01	14.30%

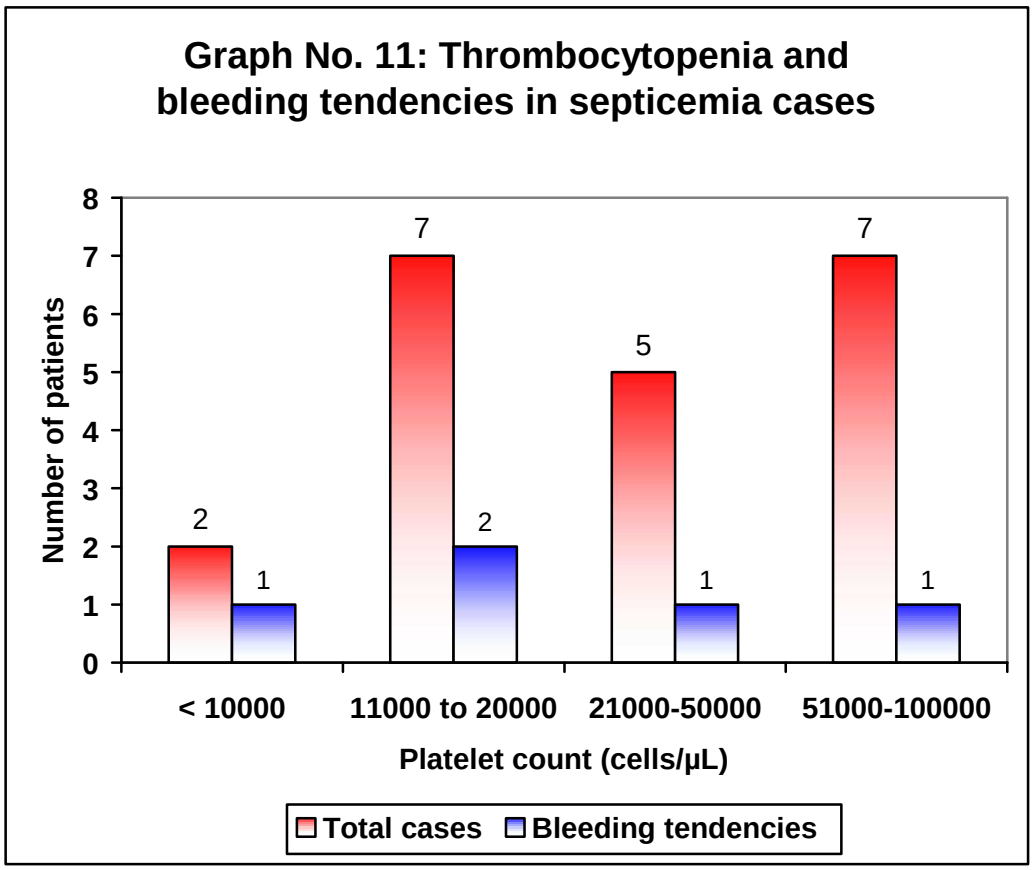


Table 13: Thrombocytopenia and Bleeding tendencies in Malaria cases

Platelet count (Cells/µl)	Patients with Malaria	Bleeding tendencies	
		Number	Percentage
≤ 10000	03	03	100%
11 – 20000	05	00	00%
21 – 50000	07	02	28.60%
51 – 100000	04	00	00%

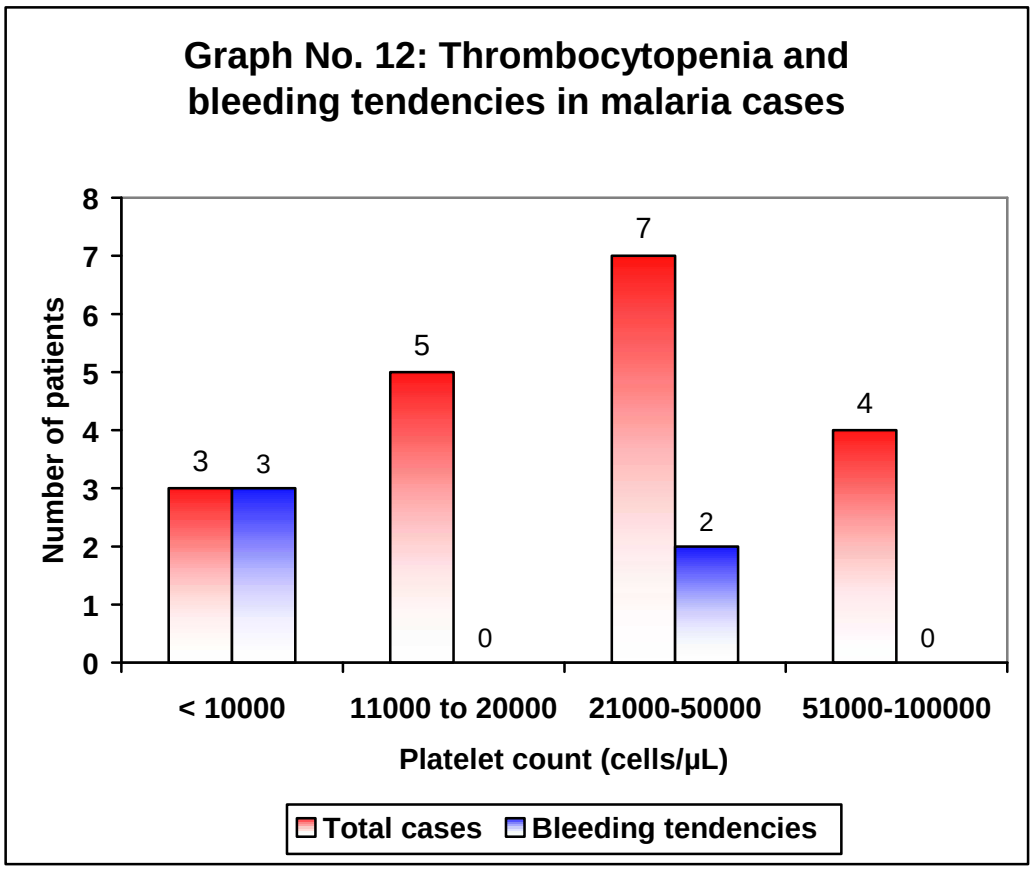
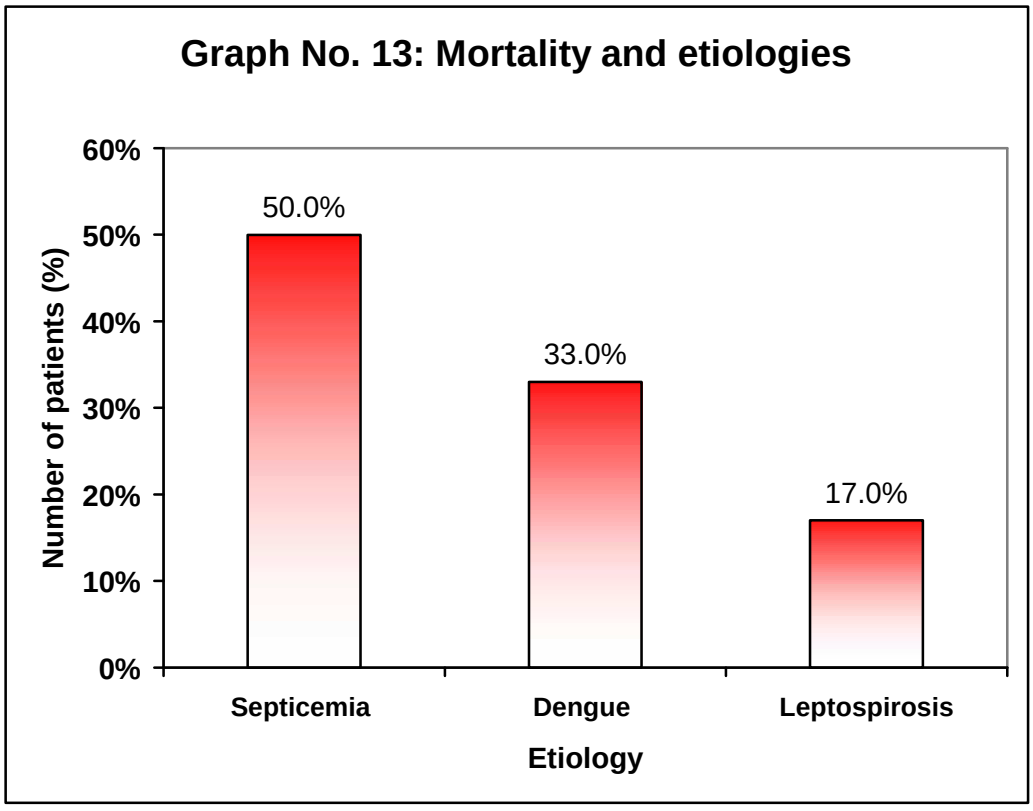


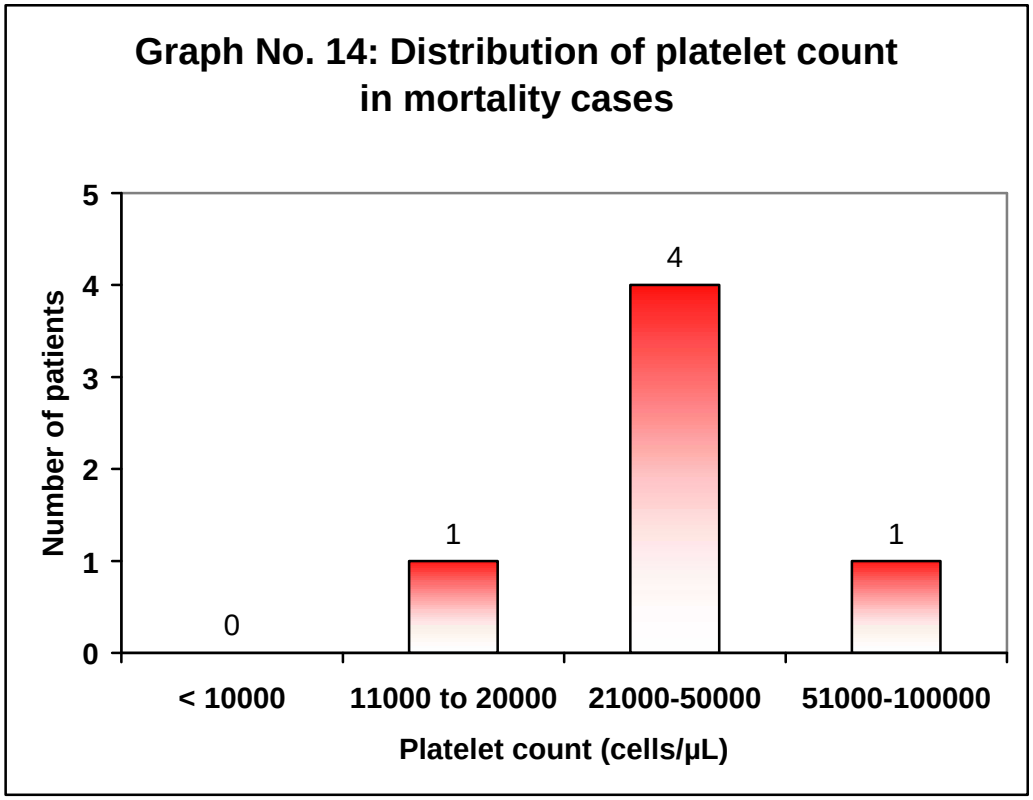
Table 14: Mortality and etiologies

Etilogy	Number of Mortalities	Percentage of Mortalities
Septicemia	3	50%
Dengue	2	33%
Leptospirosis	1	17%



Out of 200 cases, 194 cases had good recovery and 06 cases expired during stay in hospital. Out of 6 mortality cases, 3 cases due to Septicemia accounted for 50% of death, and was the common cause. 2 cases were due to Dengue accounting for 33% of death. 1 case died due to Leptospirosis (17%). In 6

mortality cases, majority of platelet count was in the range of 21000-50000/µl in 4 cases.



All 194 cases, who had good recovery were followed up and their platelet count was repeated at the time of discharge. It was noted that the platelet count in them were near normal.

DISCUSSION

For a study of fever with thrombocytopenia, patients must satisfy the above mentioned criteria, prospective case collection is necessary and careful follow up is warranted. These three conditions allow the delineation of a standard study population. The depth and means of exploration are also important but rather difficult to evaluate.

Indian study

This study was conducted by Nair PS, Jain A, Khanduri U, Kumar V. (2003) at St.Stephen's hospital, New Delhi, for period of one and half years. A total of 109 cases (76 male, 33 female patients) were studied with the same criteria as in our study[16].

Septicemia with 29 cases was the leading cause of fever associated with thrombocytopenia. Second common cause was Enteric fever followed by Dengue and Malaria with 16, 15 and 10 cases respectively.[16]

Out of 109 patients 62 patients (56.8%) had platelet count between 50,000-1,00,000, followed by 28 patients (25.7%) with count between 20,000 to 50,000.

Out of 109 patients 45 patients had thrombocytopenic signs accounting for 41.3%. Out of 45 patients spontaneous bleeding was seen in 31 patients accounting for 69% of the bleeding manifestations.[16]

During the course of follow up platelets showed increasing trends in 69 patients (63.3%) and continuously decreasing trends in 8 patients (7.3%).[16]

Totally infections represented the most important cause of fever with thrombocytopenia with a relative frequency ranging from 68% - 100%.

In our study infections was the established diagnosis in 78% of the cases as compared to other study in which infection accounted for 68% of the cases. Hence infection was the commonest cause of fever with thrombocytopenia in both the studies.

Among infections, Dengue (56%) was the commonest cause as compared to other study in which septicemia (27%) was the commonest cause. This was due to seasonal and regional variations.

In our study Septicemia was (10.5%) was the third most common cause of fever with thrombocytopenia, but Dengue (9.2%) was the third common cause in the other study.

In the other study Enteric fever was the second common cause and accounted for 14.7%, but in our study it accounted for only 1%.

Dengue / VHF infections constituted 56% in our study as compared to 13.8% in other study.

In our study 22% cases were undiagnosed, but in other study 18.3% cases remained undiagnosed.

In our study thrombocytopenic signs were present in 45% as compared to 41.3% in other study.

In our study Petechiae/ purpura (85%) was the commonest bleeding manifestations followed by spontaneous bleeding (57%). In other study spontaneous bleeding was the commonest bleeding manifestation (68%) followed by petechiae / purpura accounting for (22.22%), others (9.88%).[16]

In our study distribution of platelet count in the range of 50-100 thousands was seen in 24% as compared to 56.8% in other study. Platelet count in the range of 20-50000 was seen in 38% and 25.7% in our study and other study respectively.[16]

During the course of follow up platelet count showed increasing trends accounting for 63.3% and continuously falling counts in 7.3% in their study[16]. But in our setup 100% cases showed increasing trends in platelet count both at the time of discharge and in future follow up. There was no decreasing trends of platelet count observed.

In conclusion, our study of fever with thrombocytopenia reveals that infections was the commonest cause.

Among infections, Dengue was the commonest cause because of seasonal and regional variations.

Definitive increase in platelet count was noted after the underlying cause was treated.

Septicemia accounted for 50% of mortality in our study, followed by Dengue with 33% and Leptospirosis with 17% of the mortality cases.

Infectious diseases group formed the major portion of patients presenting with fever and thrombocytopenia lacking any specific signs. Dengue, Malaria, Leptospirosis and other viral infections formed the major chunk of this group.

Response to empirical therapy for locally prevalent disease may help the physician for better management of the patients.

In future various pathological and microbiological imaging modalities should be needed for research and diagnosis of many viral hemorrhagic fevers.

Thrombocytopenia is common finding in malaria and about 80% of malaria patients have the same.[17,18]

In a study conducted by UM Jadav, "Thrombocytopenia in malaria - Correlation with type and severity of malaria". Normal platelet count was noted in 21.6% cases (n=1565)[18].

It was observed that thrombocytopenia was rarely accompanied by clinical bleeding or biochemical evidence of DIC both in our and their study. Platelets count can fall to below 25,000/µl but this is uncommon[18].

Platelet count rise rapidly with recovery so no need of platelet transfusion in malaria cases.[18]

The prevalence of thrombocytopenia was 78.4% of cases in UM Jadav et al study of thrombocytopenia in malaria and it highlighted that persistent platelet count is unlikely in the lab findings of malaria. Thrombocytopenia was

seen in 40-90% of patients infected with plasmodium falciparum in India.[18]

The mechanism of thrombocytopenia in malaria could be due to peripheral destruction and consumption by DIC.[14,20]

Profound thrombocytopenia with platelet count as low as 5000/µl has been reported in Indian literature in a 43 year old female patient with vivax malaria[17].

CONCLUSION

1. Fever with thrombocytopenia is one of the most challenging problems in the field of medicine.
2. Fever with thrombocytopenia consists of occult presentations of common diseases rather than rare disease.
3. Infection is the commonest cause of fever with thrombocytopenia.
4. Among infection, Dengue was the commonest cause.
5. Malaria, Typhoid, Dengue still present clinically in atypical and occult forms, making diagnosis difficult and prolonged. So high index of clinical suspicion is needed.
6. So they should be investigated with some routine and specific test like rapid spot test; IgM ELISA for Dengue, IgM ELISA Leptospiral antibodies, etc. for correct diagnosis.
7. In majority of patients thrombocytopenia was transient and asymptomatic.
8. In significant number of cases thrombocytopenia lead to various bleeding manifestations and influenced the clinical profile of these febrile illness
9. Generally, spontaneous bleeding was noted in platelet count <20,000 but in some due to qualitative defects it was seen in platelet count in the range of > 50,000 cell cu/mm also.
10. Some patients with platelet count of 10,000 did not have spontaneous bleeding.

11. Spontaneous bleeding patients should be evaluated for Disseminated Intravascular Coagulation also.
12. Platelet count rise rapidly with treatment of malarial infection, so no need of platelet transfusion in malaria cases.

SUMMARY

A prospective study of 200 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 materials and methods of the study.

- The age range of the patient was 13-85 years, with male and female ratio being 70:30. These factors any way were not considered in our study.
- The duration of hospitalization ranged from 1-30 days, with an average period of hospitalization being 6.3 days.
- A definitive diagnosis was made in 156 cases.
- Among the diagnosed cases, Dengue formed the largest group with 56%.
- Other cases diagnosed were Septicemia 21 cases, Malaria 19 cases, Enteric fever 2 cases, Leptospirosis 2 cases, constituting 10.5%, 9.5%, 1%, and 1% respectively.
- Unexplained causes formed 22% of all the cases.
- Common range of platelet count at the time of admission was 21-50,000 in 76 cases, followed by 11-20 thousands in 50 cases, 51-100 thousands in 48 cases, and ≤ 10000 in 26 cases.
- Clinical manifestation of thrombocytopenia was present only in 90 cases and in 110 cases it was not present.
- Out of 90 cases which had thrombocytopenic manifestations petechiae/purpura was present in 77 cases accounting for 85% and spontaneous bleeding in 51 cases accounting for 57%.

- In general, 194 cases had good recovery and 6 cases had mortality.
- All 194 cases who had good recovery were followed up and platelet counts were near normal (1 - 1.5 lakhs) at the time of discharge.
- In 6 mortality cases, 3 were due to Septicemia - accounting for 50%, 2 were due to Dengue accounting for 33%, and 1 due to Leptospirosis accounting for 17% of the total mortalities.
- Common range of platelet count in mortality cases was in range of 21-50 thousands in 4 cases, followed by 15000 in 1 case and 80000 in 1 case.
- During discharge and follow up of all patients in our study platelet counts showed increasing trends and were near normal (around 1.5 lakhs/µl).

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ANNEXURE I

CONSENT FORM

INFORMED CONSENT FORM

"CLINICAL AND ETIOLOGICAL PROFILE OF ACUTE FEBRILE ILLNESSES WITH THROMBOCYTOPENIA-A ONE YEAR HOSPITAL BASED CROSS-SECTIONAL STUDY".

Objective and purpose of the study:

This research is intended to study clinical and etiological profile of acute febrile illnesses with thrombocytopenia. The principal investigator of the study is Dr. P. Rahul under the guidance of Dr. Annasaheb J. Dhumale My co-operation will be of great help to patients with acute febrile illnesses with thrombocytopenia in future.

Procedure:

If I agree to be part of the research study I will be asked the relevant history and will be subjected to relevant clinical examination and investigations. I will also have to give blood and urine samples for the necessary investigations

Risk and Benefits:

The only risk and possible discomfort I might get is while taking blood from my arm for the investigations. It may cause swelling, pain, redness, bruising or infection (rarely happens) at the site from where the blood is drawn.

Alternatives

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

Privacy and Confidentiality

All information collected about me during the course of this study will be kept confidential to the extent permitted by law. The code numbers will identify me in this research record. Information from this study may be published but my identity will be confidential in any publication.

Institution / Sponsor's policy

Does not apply to this research

Financial incentives for participation

I will not be paid / offered any gifts /incentives for participating in the study.

Authorization to publish the results

The results of the study would be forwarded to the KLE University, Belgaum as part of requirement towards the completion of MS degree, review and publishing.

If I have any questions about my rights as a participant I may call Dr. V. D. Patil, Principal and Chairman, J.N.M.C Ethical Committee for Human Research phone number 0831-2471350.

Consent Statement

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

Name of the Participant or legally authorised representative : _____

Signature / Thumb print _____

In case of the queries during study or in future you may contact following person

Principal investigator : Dr. P. Rahul

Guide : Dr. Annasaheb J. Dhumale

Name of the Witness _____

Signature _____

Investigator Name and Signature _____

Date: Place:

ANNEXURE II

PROFORMA

I.P number:

Patient Name:

Age: Sex:

Address:

Date of admission: Date of discharge:

SYMPTOMS AND SIGNS:

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

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

PAST HISTORY:

1. Any blood transfusions
2. Previous hospitalisation
3. Similar illnesses

TREATMENT HISTORY:

1. On drugs known to cause thrombocytopenia
2. Any other drugs

PERSONAL HISTORY:

(Menstrual history in females)

PHYSICAL EXAMINATION:

General condition:

Pallor:

Icterus:

Lymphadenopathy:

Conjunctiva:

Tongue:

Rashes:

Temperature:

Pulse:

Blood pressure:

SYSTEMIC EXAMINATION:

R.S:

C.V.S:

P/A

CNS

LABORATORY INVESTIGATIONS:

- CBC
- ESR
- Platelet count
- Peripheral smear for morphology
- Peripheral smear for MP
- QBC for MP
- BT, CT

- Urine routine and microscopy
- Blood culture
- Urine culture
- MR
- LFT
- WIDAL
- Dengue IgG,IgM
- Brucella Standard Agglutnation Test
- Leptospira ELISA
- Bone marrow examination
- Other necessary special investigations

DIAGNOSIS:

ANNEXURE IV - MASTER CHART

S. No	IP NO.	DATA			SYMPTOMS									P/H	GPE			SYST EXAM				INVESTIGATIONS																		DIAGNOSIS	OUTCOME
		AGE (in years)	SEX	HOSPITAL STAY (days)	FEVER	CHILLS & RIGORS	HEADACHE	JAUNDICE	COUGH & BREATHLESSNESS	ALTERED SENSORIUM	BLEEDING	OTHERS	TREATMENT HISTORY	TEMPERATURE	PALLOR, ICTERUS, EDEMA	BLEEDING MANIFESTATIONS	CVS	RS	PA	CNS	Hb (gm%)	TC (cells/cu mm)	PLATELET COUNT (cells/cu mm	ESR (mm/hr)	URINE ROUTINE	PS FOR MP	QBC FOR MP	BLOOD UREA (mg/dl)	SERUM CREATININE (mg/dl)	SERUM BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALKALINE PHOSPHATASE	COAGULATION PROFILE	WIDAL	DENGUE ELISA IgM/ IgG	LEPTOSPIRA IgM	HIV	BLOOD CULTURE	BONE MARROW		
1	320377	20	M	6								M/BP				SB/Pe	NS	PE	NS	NS	13	10900	21000	24	N			32	2	0.7	599	139	137	N							DENGUE	G
2	323343	25	M	11	+	+	–	–	–	–	+	NS	–	+	–		NS	NS	NS	NS	15.3	2400	57000	28	N	–	–	20	0.7	0.5	80	40	124	N	–	+	–	–	–	–	DENGUE	G
3	321474	45	M	7	+	+	–	–	–	–	–	NS	–	+	–	–	NS	NS	NS	NS	13.1	8200	90000	32	N	–	–	40	1.1	0.8	183	82	86	N	–	+	–	–	–	–	DENGUE	G
4	315058	30	M	2	+	–	+	–	–	–	–	PA/OG	–	+	–	–	NS	C	NS	AS	8.8	22000	35000	56	N	–	–	224	8	1.8	88	64	104	N	–	+	–	–	–	–	SEPTICEMIA	G
5	315294	21	M	4	+	–	–	–	–	+	–	M/BP	–	+	I	–	NS	NS	SPL	NS	10.8	3000	18000	30	N	PV/PF	–	45	0.8	1.6	40	64	103	N	–	–	–	–	–	–	MM	G
6	316388	27	M	4	+	+	–	+	–	–	–	PA	–	+		–	NS	NS	HEP	NS	15.1	2800	77000	20	N		+	27	1	1	56	40	90	N	–	–	–	–	–	–	UNEXPLAINED	G
7	322281	22	F	8	+	–	–	–	–	–	–		–	+	–	SB	NS	NS	NS	NS	12.3	8500	24000	32	N	–	–	37	0.7	0.9	212	48	112	N	–	–	–	–	–	–	DENGUE	G
8	315978	25	M	6	+	+	–	–	–	–	+	M/BP	–	+	–		NS	NS	NS	NS	13	4200	50000	11	N	–	–	20	0.6	1.8	346	123	122	N	–	+	–	–	–	–	DENGUE	G
9	317491	28	M	8	+	+	–	–	–	–	–	PA	–	+	–	Pe/Pu	NS	NS	NS	NS	17.6	8200	10000	10	N	–	–	22	0.7	1	346	400	140	N	–	+	–	–	–	–	DENGUE	G
10	323342	19	M	3	+	–	–	–	+	–	–	M/BP	–	+	–	SB	NS	NS	NS	NS	11	7300	49000	22	N	–	–	24	0.8	1.2	56	40	88	N	–	+	–	–	–	–	DENGUE	G
11	306500	20	M	4	+	–	+	–	–	–	–	M/BP	–	+	–	Pe/Pu	NS	NS	NS	NS	16	5600	30000	20	N	–	–	29	0.9	0.8	205	169	89	N	–	+	–	–	–	–	DENGUE	G
12	322186	24	M	2	+	–	–	–	–	–	–		–	+	–	Pe/Pu	NS	NS	NS	NS	13.2	2800	24000	10	N	–	–	10	0.7	0.9	890	300	90	N	–	+	–	–	–	–	DENGUE	G
13	320336	29	M	7	+	+	+	–	–	–	–	LS	–	+	P,I	SB/Pe	NS	NS	HEP	NS	19.2	10800	5000	10	N	–	–	38	1.3	5.7	3851	1970	186	N	–	+	–	–	–	–	DENGUE	G
14	321583	18	F	4	+	–	+	–	–	–	+	M/BP	–	+			NS	NS	NS	NS	12.4	8000	91000	38	N	–	–	20	1	1	50	38	80	N	–	+	–	–	–	–	DENGUE	G
15	320393	46	M	6	+	+	+	–	–	–	–	M/BP	–	+	–	–	NS	NS	NS	NS	13.7	10600	40000	28	N	–	–	40	1.5	2	366	152	174	N	–	+	–	–	–	–	DENGUE	G
16	319636	44	F	8	+	–	+	–	–	–	–	M/BP	–	+	I	SB/Pe	NS	NS	NS	NS	15.8	5400	17000	4	N	–	–	28	1.2	3.8	1889	691	308	N	–	+	–	–	–	–	DENGUE	G
17	312758	23	M	5	+	–	+	–	–	–	+		–	+		SB	NS	NS	NS	NS	16.5	5300	22000	8	N	–	–	10	0.7	0.8	182	82	78	N	–	+	–	–	–	–	DENGUE	G
18	305126	53	M	5								M/BP				Pe/Pu	NS	NS	NS	NS	15.1	5300	20000	5	N			24	0.8	0.3	105	56	106	N							DENGUE	G
19	318861	22	M	13	+	+	+	–	–	–	–	LS	–	+	–	SB/Pe	NS	NS	HEP	NS	14.6	4400	2000	28	N	–	–	19	0.8	1.8	234	88	96	N	–	+	–	–	–	–	DENGUE	G
20	321578	16	M	4	+	–	+	–	+	–	+	M/BP	–	+	–		NS	NS	NS	NS	12.8	9900	45000	18	N	–	–	18	0.7	0.3	379	140	130	N	–	+	–	–	–	–	DENGUE	G
21	322119	24	F	7	+	+	–	–	–	–	–		–	+	–	Pe/Pu	NS	PE	NS	NS	14.3	4700	14000	12	N	–	–	32	0.7	0.6	101	94	90	N	–	+	–	–	–	–	DENGUE	G
22	319874	13	F	6	+	+	–	–	–	–	–	M/BP	–	+	–	SB/Pe	NS	NS	NS	NS	13	14000	47000	24	N	–	–	56	1.2	0.8	154	80	237	N	–	+	–	–	–	–	DENGUE	G
23	319938	37	F	7	+	+	+	–	–	–	+	M/BP	–	+	I	SB/Pe	NS	NS	HEP	NS	17.5	22400	14000	32	N	–	–	58	2.3	2.2	3373	2083	191	N	–	+	–	–	–	–	DENGUE	G
24	323107	41	M	4	+		+	–	–	–	+		–	+			NS	NS	NS	NS	11.5	6150	22000	18	N	–	–	39	0.2	0.9	82	39	90	N	–	+	–	–	–	–	DENGUE	G
25	322044	65	M	9	+	+	–	–	–	–	–	–	–	+	–	SB	NS	NS	NS	NS	13	5800	88000	8	N	–	–	36	1.4	3.5	89	82	80	N	–	+	–	–	–	–	DENGUE	G
26	323497	25	M	7	+	+	–	–	+	–	+	M/BP	–	+	–	SB/Pe	NS	NS	NS	NS	16.6	2700	9000	8	N	–	–	14	0.9	0.9	109	64	85	N	–	+	–	–	–	–	DENGUE	G
27	323559	35	F	3	+	+	–	–	–	–	+	M/BP	–	+	–		NS	NS	NS	NS	13.2	10400	98000	30	N	–	–	27	0.9	0.8	68	94	96	N	–	+	–	–	–	–	DENGUE	G
28	320114	13	F	7	+	+	–	–	–	–	–	M/BP	–	+	–	–	NS	NS	NS	NS	12	7200	14000	28	N	–	–	27	0.7	0.2	139	99	88	N	–	+	–	–	–	–	DENGUE	G
29	321848	60	M	7	+	–	+	–	–	–	–	M/BP	–	+	–	–	NS	NS	NS	NS	17.9	6400	21000	2	N	–	–	26	1.4	1.9	994	298	140	N	–	+	–	–	–	–	DENGUE	G

S. No	IP NO.	DATA			SYMPTOMS									P/H	GPE			SYST EXAM				INVESTIGATIONS																										DIAGNOSIS	OUTCOME
		AGE (in years)	SEX	HOSPITAL STAY (days)	FEVER	CHILLS & RIGORS	HEADACHE	JAUNDICE	COUGH & BREATHLESSNESS	ALTERED SENSORIUM	BLEEDING	OTHERS	TREATMENT HISTORY	TEMPERATURE	PALLOR, ICTERUS, EDEMA	BLEEDING MANIFESTATIONS	CVS	RS	PA	CNS	Hb (gm%)	TC (cells/cu mm)	PLATELET COUNT (cells/cu mm	ESR (mm/hr)	URINE ROUTINE	PS FOR MP	QBC FOR MP	BLOOD UREA (mg/dl)	SERUM CREATININE (mg/dl)	SERUM BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALKALINE PHOSPHATASE	COAGULATION PROFILE	WIDAL	DENGUE ELISA IgM/ IgG	LEPTOSPIRA IgM	HIV	BLOOD CULTURE	BONE MARROW		
30	323472	24	M	7								M/BP				Pe/Pu	NS	NS	NS	NS	14.7	3600	8000	10	N			28	0.7	1.2	88	60	140	N							DENGUE	G
31	321409	29	M	4	+	–	+	–	–	–	–	M/BP	–	+	–	Pe/Pu	NS	NS	NS	NS	16.9	7400	30000	24	N	–	–	22	1.1	0.6	227	243	169	N	–	+	–	–	–	–	DENGUE	G
32	321505	28	M	4	+	–	+	–	–	–	–		–	+	–	SB/Pe	NS	PE	HEP	NS	14.6	6200	50000	12	N	–	–	10	0.7	0.8	253	189	123	N	–	+	–	–	–	–	DENGUE	G
33	321523	25	M	4	+	+	+	–	–	–	+	M/BP	–	+	I	Pe/Pu	NS	PE	HEP	NS	21	7100	20000	10	N	–	–	20	1.2	2	891	497	96	N	–	+	–	–	–	–	DENGUE	G
34	321651	26	M	1	+	–	+	–	–	–	–	M/BP	–	+		SB/Pe	NS	NS	NS	NS	9.7	8500	12000	24	N	–	–	38	1.2	0.5	197	103	73	N	–	+	–	–	–	–	UNEXPLAINED	G
35	309835	85	M	3	+	+	–	–	–	–	+		–	+	–		NS	NS	HEP	AS	13.3	12300	55000	20	N	–	–	111	3	1.1	63	90	144	N	–	–	–	–	–	–	SEPTICEMIA	G
36	313468	65	M	4	+	–	–	–	+	+	–	–	–	+	P_TE	Pe/Pu	NS	C	HEP	NS	6.9	7300	22000	88	UTI	–	–	60	1.2	2	58	44	110	N	–	–	–	–	–	–	SEPTICEMIA	G
37	304187	45	F	15	+	–	–	–	+	–	–	–	–	+	P	Pe/Pu	NS	NS	NS	NS	9.3	8000	9000	40	N	–	–	56	1	0.8	40	34	88	N	–	–	–	–	–	–	UNEXPLAINED	G
38	309445	18	F	5	+	+	–	–	–	–	–	PA	–	+			NS	NS	NS	NS	11	6200	90000	26	N	–	–	24	0.7	0.9	66	50	89	N	–	–	–	–	–	–	UNEXPLAINED	G
39	309549	24	F	10	+	+	+	–	–	–	–	M/BP	–	+	P	Pe/Pu	NS	NS	NS	NS	8	4900	12000	34	N	–	–	26	0.7	0.8	78	54	98	N	–	–	–	–	–	–	UNEXPLAINED	G
40	310461	42	M	7	+	–	–	–	–	–	–	M/BP	–	+		SB/Pe	NS	NS	NS	NS	16	12100	2000	3	N	–	–	16	0.7	0.6	58	36	94	N	–	–	–	–	–	–	UNEXPLAINED	G
41	310063	53	M	13	+	–	–	–	–	–	+	M/BP	–	+	–		NS	NS	SPL	NS	13.5	10500	88000	30	N	–	–	24	0.7	0.8	78	80	98	N	–	–	–	–	–	–	UNEXPLAINED	G
42	310809	30	M	5	+	+	+	–	+	–	–		–	+	P	–	NS	NS	NS	NS	5.7	4900	95000	85	N	–	–	11	0.9	0.8	23	39	70	N	–	–	–	–	–	–	UNEXPLAINED	G
43	311022	18	F	5	+	–	+	–	–	–	–	M/BP	–	+		Pe/Pu	NS	NS	NS	NS	13	1700	30000	22	N	–	–	20	0.5	1.3	66	54	89	N	–	–	–	–	–	–	UNEXPLAINED	G
44	311282	42	M	6	+	–	–	–	–	–	–	M/BP	–	+	I_TE	SB/Pe	NS	NS	HEP	NS	12.4	6500	15000	31	N	–	–	83	2.7	5.2	72	82	116	N	–	–	–	–	–	–	UNEXPLAINED	G
45	305899	20	F	5	+	–	–	–	–	–	+		–	+	P		NS	PE	HSM	NS	10.6	3500	83000	130	UTI	–	–	125	4	0.4	22	25	103	N	–	–	–	–	–	–	UNEXPLAINED	G
46	322878	21	M	6	+	+	–	–	+	–	–	M/BP	–	+		SB	NS	NS	NS	NS	13	3600	50000	4	N	–	–	52	0.7	0.9	182	32	78	N	–	–	–	–	–	–	DENGUE	G
47	321652	50	M	8								M/BP				SB/Pe	NS	C	HEP	NS	13.4	4500	10000	32	N			22	1.2	3.3	536	946	240	N							DENGUE	G
48	321428	30	M	6	+	–	+	–	–	–	+	LS	–	+	–	Pe/Pu	NS	NS	NS	NS	14.2	3800	12000	10	N	–	–	32	0.3	0.7	988	300	426	N	–	+	–	–	–	–	DENGUE	G
49	321733	20	F	8	+	–	–	–	–	–	–	M/BP	–	+	–	SB/Pe	NS	NS	NS	NS	11.3	5300	14000	12	N	–	–	14	0.7	0.8	275	98	96	N	–	+	–	–	–	–	DENGUE	G
50	323472	24	M	7	+	+	–	–	–	–	+	M/BP	–	+	–		NS	NS	NS	NS	14.7	3600	8000	32	N	–	–	26	0.8	0.9	184	94	102	N	–	+	–	–	–	–	DENGUE	G
51	316845	57	M	5	+	+	–	–	–	–	–		–	+	–	–	NS	NS	NS	NS	15	4600	30000	4	N	–	–	22	0.8	0.8	322	444	116	N	–	+	–	–	–	–	DENGUE	G
52	320393	46	M	4	+	+	–	–	–	–	–	–	–	+	–	–	NS	NS	HEP	NS	13.7	10600	40000	28	N	–	–	40	1.5	2	366	152	174	N	–	+	–	–	–	–	DENGUE	G
53	320955	19	M	7	+	–	+	–	–	–	–	PA	–	+	–	–	NS	NS	NS	NS	16.5	8200	20000	5	N	–	–	29	0.9	0.8	111	132	111	N	–	+	–	–	–	–	DENGUE	G
54	323239	30	F	1	+	+	–	–	–	–	–	PA	–	+	–	–	NS	NS	HEP	AS	12.6	55500	40000	78	N	–	–	72	3.5	6.2	22915	3590	368	N	–	+	–	–	–	–	DENGUE	E
55	316821	16	M	5	+	+	+	+	–	+	–	PA	–	+	–	Pe/Pu	NS	NS	NS	NS	14.7	5200	22000	8	N	–	–	19	0.7	0.8	229	241	156	N	–	+	–	–	–	–	DENGUE	G
56	321118	38	M	4	+	+	–	–	–	–	–	M/BP	–	+	–	SB/Pe	NS	NS	NS	NS	16.4	10700	10000	30	N	–	–	17	1.2	1.4	215	199	122	N	–	+	–	–	–	–	DENGUE	G
57	320342	18	M	4	+	–	+	–	–	–	+	M/BP	–	+	–		NS	NS	NS	NS	17.4	3500	21000	22	N	–	–	37	1	0.5	411	236	182	N	–	+	–	–	–	–	UNEXPLAINED	G
58	316734	25	M	6	+	+		–	–	–	–		–	+	–	Pe/Pu	NS	NS	HEP	NS	16.3	4100	25000	10	N	–	–	10	0.9	0.9	132	82	96	N	–	–	–	–	–	–	DENGUE	G

S. No	IP NO.	DATA			SYMPTOMS									P/H	GPE			SYST EXAM				INVESTIGATIONS																				DIAGNOSIS	OUTCOME
		AGE (in years)	SEX	HOSPITAL STAY (days)	FEVER	CHILLS & RIGORS	HEADACHE	JAUNDICE	COUGH & BREATHLESSNESS	ALTERED SENSORIUM	BLEEDING	OTHERS	TREATMENT HISTORY	TEMPERATURE	PALLOR, ICTERUS, EDEMA	BLEEDING MANIFESTATIONS	CVS	RS	PA	CNS	Hb (gm%)	TC (cells/cu mm)	PLATELET COUNT (cells/cu mm	ESR (mm/hr)	URINE ROUTINE	PS FOR MP	QBC FOR MP	BLOOD UREA (mg/dl)	SERUM CREATININE (mg/dl)	SERUM BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALKALINE PHOSPHATASE	COAGULATION PROFILE	WIDAL	DENGUE ELISA IgM/ IgG	LEPTOSPIRA IgM	HIV	BLOOD CULTURE	BONE MARROW		
59	316085	40	F	6								M/BP					NS	NS	NS	NS	13	5800	25000	8	N			20	0.6	0.7	346	432	188	N							DENGUE	G
60	320242	26	F	3	+	+	–	–	–	–	–	M/BP	–	+	–	SB/Pe	NS	NS	NS	NS	14.4	6100	20000	34	N	–	–	16	0.9	1.1	88	96	100	N	–	+	–	–	–	–	DENGUE	G
61	318730	24	M	8	+	+	–	–	–	–	+	M/BP	–	+	–	SB/Pe	NS	NS	NS	NS	10.6	12500	5000	68	N	–	–	48	1.1	0.5	260	112	97	N	–	+	–	–	–	–	UNEXPLAINED	G
62	317350	30	F	7	+	–	+	–	–	+	M/BP	–	+	–	Pe/Pu	NS	NS	HEP	NS	15.4	4100	11000	22	N	–	–	19	0.8	0.5	248	120	63		–	–	–	–	–	–	DENGUE	G
63	313692	53	M	3	+	–	–	–	–	–	–	M/BP	–	+	–		NS	NS	NS	NS	14.6	8900	59000	10	N	–	–	10	0.6	0.7	75	54	77	N	–	+	–	–	–	–	DENGUE	G
64	304984	20	F	6	+	+	–	–	–	–	–	M/BP	–	+	–	Pe/Pu	NS	NS	NS	NS	11	5000	26000	22	N	–	–	14	0.6	0.9	55	64	89	N	–	+	–	–	–	–	UNEXPLAINED	G
65	322618	31	M	4	+	–	+	–	–	–	–	M/BP	–	+	–	SB/Pe	NS	NS	NS	NS	15.1	6500	11000	22	N	–	–	19	1	0.6	167	93	97	N	–	–	–	–	–	–	DENGUE	G
66	322337	19	F	6	+	+	–	–	–	–	+	M/BP	–	+	–		NS	NS	NS	NS	14.4	1600	70000	12	N	–	–	13	0.8	0.4	30	26	91	N	–	+	–	–	–	–	UNEXPLAINED	G
67	322323	21	M	5	+	–	+	–	–	–	–	M/BP	–	+	–	–	NS	NS	NS	NS	15.2	6700	25000	17	N	–	–	30	0.8	0.7	56	82	100	N	–	–	–	–	–	–	DENGUE	G
68	319537	58	F	5	+	–	+	–	+	–	–		–	+	–	–	NS	NS	NS	NS	16.2	3800	62000	10	N	–	–	10	0.5	0.9	132	82	106	N	–	+	–	–	–	–	DENGUE	G
69	316820	28	M	3	+	–	–	–	–	–	–	–	–	+	–	–	NS	NS	NS	NS	9.4	7900	25000	10	N	–	–	13	0.7	0.9	445	198	131	N	–	+	–	–	–	–	DENGUE	G
70	320772	23	M	3	+	–	+	–	–	–	–	–	–	+	–	–	NS	NS	NS	NS	16.8	3900	83000	12	N	–	–	10	0.7	0.9	112	72	116	N	–	+	–	–	–	–	DENGUE	G
71	320365	27	M	3	+	+	–	–	–	–	–	M/BP	–	+	–	–	NS	NS	NS	NS	14	6800	50000	10	N	–	–	26	0.8	1.2	132	112	96	N	–	+	–	–	–	–	DENGUE	G
72	320635	48	M	14	+	–	+	–	–	–	–	M/BP	–	+	–	–	NS	NS	NS	NS	12.7	8200	55000	14	N	–	–	50	1.8	2.3	75	72	131	N	–	+	–	–	–	–	UNEXPLAINED	G
73	322875	24	M	4	+	–	–	–	–	–	–		–	+	–	–	NS	NS	NS	NS	11.4	4600	11000	36	N	PF/PV	–	22	1	5.7	72	96	203	N	–	–	–	–	–	–	MM	G
74	322650	34	F	5	+	+	+	–	–	–	–	M/BP	–	+	–	–	NS	NS	NS	NS	11.9	5200	44000	28	N		+	11	0.6	0.3	63	48	80	N	–	–	–	–	–	–	UNEXPLAINED	G
75	322878	21	M	5	+	–	+	–	–	–	–	M/BP	–	+	–	SB/Pe	NS	NS	NS	NS	12.3	6400	9000	38	N	–	–	18	0.8	0.6	96	88	122	N	–	–	–	–	–	–	DENGUE	G
76	317831	19	M	5								M/BP				SB/Pe	NS	NS	NS	NS	18.1	2800	13000	7	N			12	0.8	0.6	223	107	111	N							DENGUE	G
77	321275	55	M	8	+		+	–	–	–	+	M/BP	–	+	–	Pe/Pu	NS	NS	HEP	NS	13.1	7800	20000	25	N	–	–	13	0.7	1	1662	582	101	N	–	+	–	–	–	–	DENGUE	G
78	316586	80	M	2	+	+	–	–	–	–	–	PA	–	+	–		NS	NS	NS	AS	10	76000	13000	78	UTI	–	–	102	5	0.7	21	27	300	N	–	+	–	–	–	–	SEPTICEMIA	G
79	316979	80	M	4	+	+	–	–	–	+	–		–	+	–	–	NS	C	NS	AS	13.6	24000	5000	28	N	–	–	69	1.5	0.6	31	31	125	N	–	–	–	–	–	–	SEPTICEMIA	G
80	319657	34	M	10	+	–	–	–	–	–	–	M/BP	–	+	P	Pe/Pu	NS	NS	NS	NS	6.8	3400	9000	98	N	–	–	34	0.7	1.3	66	26	88	N	–	–	–	–	–	–	DENGUE	G
81	319750	17	F	9	+	+	–	–	–	–	–		–	+		Pe/Pu	NS	NS	NS	NS	13	3400	13000	112	N	–	–	37	0.7	0.6	1338	459	137	N	–	+	–	–	–	–	DENGUE	G
82	322969	26	M	6	+	–	–	–	–	–	–	M/BP	–	+	–	Pe/Pu	NS	NS	HEP	NS	14.4	8500	23000	18	N	–	–	12	0.7	0.8	255	119	80	N	–	+	–	–	–	–	DENGUE	G
83	323323	26	M	4	+	+	+	–	+	–	–	M/BP	–	+	–	Pe/Pu	NS	NS	NS	NS	16.4	6000	48000	44	N	–	–	19	0.6	1.2	88	126	120	N	–	+	–	–	–	–	DENGUE	G
84	323092	19	F	4	+	–	+	–	+	–	–		–	+	–	SB/Pe	NS	NS	NS	NS	14	8600	22000	22	N	–	–	28	1.2	0.8	90	60	112	N	–	+	–	–	–	–	DENGUE	G
85	323044	22	F	4	+	+	–	–	–	–	+	M/BP	–	+	P		NS	NS	NS	NS	7.2	8100	32000	45	N	–	–	31	0.7	0.9	66	54	90	N	–	+	–	–	–	–	DENGUE	G
86	323026	36	F	7	+	+	–	–	–	–	–		–	+	I	SB/Pe	NS	NS	HEP	NS	13.6	20500	22000	22	N	–	–	37	1.1	3.4	802	363	277	N	–	+	–	–	–	–	DENGUE	G
87	323014	32	M	7	+	–	+	+	–	–	+	–	–	+		SB/Pe	NS	NS	NS	NS	12.8	6700	10000	60	N	–	–	22	0.7	0.8	88	60	110	N	–	+	–	–	–	–	DENGUE	G

S. No	IP NO.	DATA			SYMPTOMS									P/H	GPE			SYST EXAM				INVESTIGATIONS																								OUTCOME
		AGE (in years)	SEX	HOSPITAL STAY (days)	FEVER	CHILLS & RIGORS	HEADACHE	JAUNDICE	COUGH & BREATHLESSNESS	ALTERED SENSORIUM	BLEEDING	OTHERS		TREATMENT HISTORY	TEMPERATURE	PALLOR, ICTERUS, EDEMA	BLEEDING MANIFESTATIONS	CVS	RS	PA	CNS	Hb (gm%)	TC (cells/cu mm)	PLATELET COUNT (cells/cu mm	ESR (mm/hr)	URINE ROUTINE	PS FOR MP	QBC FOR MP	BLOOD UREA (mg/dl)	SERUM CREATININE (mg/dl)	SERUM BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALKALINE PHOSPHATASE	COAGULATION PROFILE	WIDAL	DENGUE ELISA IgM/ IgG	LEPTOSPIRA IgM	HIV	BLOOD CULTURE	BONE MARROW	DIAGNOSIS	
88	323006	33	M	8								M/BP						NS	NS	NS	NS	17.4	5400	45000	20	N			14	0.8	0.8	184	90	134	N							DENGUE	G
89	316094	55	M	30	+	+	+	–	–	–	–			–	+	–	–	NS	NS	HEP	NS	14.9	13700	78000	15	N	–	–	76	1.8	4.8	67	42	113	N	–	+	–	–	–	–	SEPTICEMIA	G
90	322821	27	M	5	+	–	–	–	+	–	M/BP			–	+	–	–	NS	NS	NS	NS	16.8	3000	39000	18	N	–	–	13	0.6	1.8	69	48	107	N	–	–	–	–	–	–	DENGUE	G
91	322843	27	M	8	+	–	+	–	–	–	+	M/BP		–	+	–	Pe/Pu	NS	NS	NS	NS	15.2	5200	38000	12	N	–	–	16	0.9	0.5	98	58	71	N	–	+	–	–	–	–	DENGUE	G
92	322283	30	M	6	+	+	+	–	–	–	–	M/BP		–	+	–	Pe/Pu	NS	NS	NS	NS	12.4	6600	26000	10	N	–	–	38	1.6	1.6	110	88	140	N	–	+	–	–	–	–	UNEXPLAINED	G
93	322222	18	M	5	+	+	+	–	–	–	–	M/BP		–	+	–		NS	NS	NS	NS	12	7400	94000	22	N	–	–	27	1	0.9	66	60	82	N	–	–	–	–	–	–	DENGUE	G
94	322210	24	M	4	+	+	–	–	–	–	–	M/BP		–	+	–	SB/Pe	NS	NS	NS	NS	12.4	5600	13000	20	N	–	–	28	1	1.3	96	88	110	N	–	+	–	–	–	–	DENGUE	G
95	322147	45	M	3	+	+	–	–	–	–	+	M/BP		–	+	–		NS	NS	NS	NS	12	8600	96000	28	N	–	–	20	0.6	0.8	99	48	110	N	–	+	–	–	–	–	DENGUE	G
96	321915	40	F	6	+	–	–	–	–	–	–	M/BP		–	+	–	SB/Pe	NS	NS	NS	NS	12.2	7200	10000	26	N	–	–	11	1	1	366	220	199	N	–	+	–	–	–	–	DENGUE	G
97	321694	42	F	4	+	–	+	–	–	–	+	M/BP		–	+	–		NS	NS	NS	NS	13.3	5900	20000	8	N	–	–	11	0.8	0.6	119	79	79	N	–	+	–	–	–	–	DENGUE	G
98	321693	21	F	4	+	–	+	–	–	–	–	M/BP		–	+	–	Pe/Pu	NS	NS	NS	NS	12	7900	31000	20	N	–	–	23	0.7	0.9	98	69	101	N	–	+	–	–	–	–	DENGUE	G
99	320645	34	M	6	+	–	+	–	–	–	–	M/BP		–	+	–	SB/Pe	NS	NS	NS	NS	14.4	8200	17000	36	N	–	–	18	0.8	1.1	66	72	60	N	–	+	–	–	–	–	DENGUE	G
100	320371	50	M	6	+	–	+	–	–	–	+	M/BP		–	+	–	SB/Pe	NS	NS	NS	NS	18.1	8500	16000	24	N	–	–	67	1.9	2.8	407	183	229	N	–	+	–	–	–	–	DENGUE	G
101	319374	20	M	5	+	+	–	–	–	–	+			–	+	–		NS	NS	NS	NS	16	3600	61000	2	N	–	–	22	1.2	0.9	87	36	114	N	–	+	–	–	–	–	UNEXPLAINED	G
102	319298	26	M	6	+	+	–	–	–	–	–	–		–	+	–	SB/Pe	NS	NS	NS	NS	16	6100	10000	4	N	–	–	22	0.7	2.7	63	34	98	N	–	–	–	–	–	–	UNEXPLAINED	G
103	318551	53	M	4	+	+	–	–	–	–	+	–		–	+	–		NS	NS	SPL	NS	14	3200	52000	8	N	–	–	29	0.8	0.5	96	95	109	N	–	–	–	–	–	–	UNEXPLAINED	G
104	319949	17	M	6	+	–	–	–	–	–	–	M/BP		–	+	–	SB	NS	C	NS	AS	11.6	5100	83000	25	N	–	–	302	5.2	8.1	269	238	264	N	–	–	–	–	–	–	UNEXPLAINED	G
105	282363	60	F	3														NS	NS	NS	AS	14.5	10000	5000	60	N			208	3.3	8.1	176	103	295	N							UNEXPLAINED	G
106	267786	22	M	8	+	–	–	–	+	–	–	–		–	+	P	–	NS	NS	HEP	NS	4	800	36000	110	N	PF/PV	–	96	2	2.4	100	56	140	N	–	–	–	–	–	–	MM	G
107	290141	25	M	6	+	+	+	–	–	–	–	–		–	+	P	–	NS	NS	HSM	NS	8.6	1600	11000	44	N		+	30	0.8	3.4	107	57	98	N	–	–	–	–	–	–	UNEXPLAINED	G
108	276898	45	F	10	+	+	+	–	+	–	–	PA/LS		–	+	P	–	NS	NS	HSM	NS	9.4	2300	16000	75	N	PF	–	43	0.8	1.5	85	92	118	N	–	–	–	–	–	–	FM	G
109	254014	36	M	10	+	–	–	–	–	–	–	M/BP		–	+	P	–	NS	NS	HSM	NS	3.1	1800	4000	38	N		+	30	0.7	1.1	80	62	99	N	–	–	–	–	–	–	UNEXPLAINED	G
110	295683	28	F	8	+	+	–	–	+	–	–	LS		–	+	P	–	NS	NS	HEP	NS	9.4	1800	33000	68	N	PF/PV	–	54	0.9	1.8	110	68	122	N	–	–	–	–	–	–	MM	G
111	312937	20	F	20	+	–	–	–	–	–	–			–	+		–	NS	NS	NS	AS	12.7	9400	27000	20	N		+	53	2.1	0.7	48	33	74	N	–	–	–	–	–	–	UNEXPLAINED	G
112	303575	55	M	6	+	–	+	–	–	+	–	OG		–	+	I,E	–	NS	NS	NS	NS	10.9	2900	20000	22	UTI	–	–	264	2.6	4.6	32	43	140	AB	–	–	–	–	–	–	SEPTICEMIA	G
113	304161	64	M	3	+	+	–	–	–	–	–	LS		–	+	P	–	NS	NS	NS	NS	7.1	22000	89000	20	UTI	–	–	36	2.3	1	45	40	95	AB	–	–	–	–	–	–	SEPTICEMIA	G
114	302585	25	F	2	+	+	–	–	–	–	–	PA		–	+		–	NS	NS	NS	AS	16.9	30400	15000	40	N	–	–	68	2	1.7	56	66	107	N	–	–	–	–	–	–	SEPTICEMIA	E
115	282405	63	M	4	+	+	–	–	+	+	–			–	+	–	–	NS	NS	NS	AS	12.5	9400	41000	20	UTI	–	–	17	0.9	0.7	51	31	1835	N	–	–	–	–	–	–	DENGUE	E
116	293929	55	F	8	+	–	+	–	–	+	–	M/BP		–	+	–	–	NS	NS	NS	NS	10.3	16400	44000	56	N	–	–	24	0.8	6.9	132	107	132	N	–	+	–	–	–	–	LEPTOSPIROSIS	G

S. No	IP NO.	DATA			SYMPTOMS									P/H	GPE			SYST EXAM				INVESTIGATIONS																						DIAGNOSIS	OUTCOME
		AGE (in years)	SEX	HOSPITAL STAY (days)	FEVER	CHILLS & RIGORS	HEADACHE	JAUNDICE	COUGH & BREATHLESSNESS	ALTERED SENSORIUM	BLEEDING	OTHERS	TREATMENT HISTORY	TEMPERATURE	PALLOR, ICTERUS, EDEMA	BLEEDING MANIFESTATIONS	CVS	RS	PA	CNS	Hb (gm%)	TC (cells/cu mm)	PLATELET COUNT (cells/cu mm	ESR (mm/hr)	URINE ROUTINE	PS FOR MP	QBC FOR MP	BLOOD UREA (mg/dl)	SERUM CREATININE (mg/dl)	SERUM BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALKALINE PHOSPHATASE	COAGULATION PROFILE	WIDAL	DENGUE ELISA IgM/ IgG	LEPTOSPIRA IgM	HIV	BLOOD CULTURE	BONE MARROW		
117	318227	40	M	5								LS			P	Pe/Pu	NS	NS	NS	NS	13	6650	75000	18	N			22	0.6	1.2	68	54	97	N							UNEXPLAINED	G
118	300923	26	F	18	+	+	–	–	+	–	–	M/BP	–	+	E		NS	NS	NS	NS	9.8	5900	21000	100	UTI	–	–	29	0.5	1.3	120	88	210	N	–	–	–	–	–	–	SEPTICEMIA	G
119	302088	25	F	12	+	+	–	–	–	–	–		–	+	P	Pe/Pu	NS	NS	NS	NS	3.5	14400	4000	65	UTI	–	–	42	0.8	0.6	29	25	87	N	–	–	–	–	–	–	UNEXPLAINED	G
120	305016	46	M	4	+	+	–	–	–	–	–	LS	–	+			NS	C	NS	AS	13.6	8800	42000	20	N	–	–	150	4.5	3.3	47	36	148	N	–	–	–	–	–	–	SEPTICEMIA	G
121	304126	20	F	3	+	+	–	–	–	+	–	HOA	–	+	P_1I	–	NS	NS	NS	AS	10.5	16100	26000	68	UTI	–	–	34	3.3	14	149	28	569	AB	–	–	–	–	–	–	SEPTICEMIA	E
122	302764	20	M	3	+	–	–	+	–	+	–		–	+	P_1I	SB/Pe	NS	NS	SPL	NS	13.8	6600	14000	22	UTI	–	–	220	7.2	13	96	56	100	N	–	–	–	–	–	–	UNEXPLAINED	G
123	302768	60	M	9	+	+	–	+	–		+	–	–	+	P	SB/Pe	NS	NS	NS	AS	8	17400	18000	32	N	–	–	88	2.2	1.2	88	76	140	N	–	–	–	–	–	–	SEPTICEMIA	G
124	302474	22	M	9	+	–	–	–	–	+	+	–	–	+			NS	NS	NS	NS	9.7	5100	21000	22	N	PF/PV	–	43	1.2	1	56	44	87	N	–	–	–	–	–	–	MM	G
125	304235	50	M	6	+	+	+	–	–		–	M/BP	–	+	–	Pe/Pu	NS	NS	NS	AS	13.4	14400	35000	34	N		–	173	1.8	0.4	66	54	78	N	–	–	–	–	–	–	DENGUE	G
126	304579	22	M	4	+	+	+	–	–	+	–		–	+	–		NS	NS	NS	NS	14.4	2600	98000	20	N	–	–	34	0.6	0.8	44	32	68	N	–	+	–	–	–	–	ENTERIC FEVER	G
127	305310	30	M	6	+	–	–	–	+	–	–	PA	–	+	–	–	NS	NS	SPL	NS	11.9	2000	23000	50	N	PV	–	34	0.6	1	31	44	89	N	+	–	–	–	–	–	VM	G
128	306023	18	F	4	+	+	+	–	+	–	–	M/BP	–	+	–	–	NS	NS	NS	NS	9.9	2100	19000	58	N		+	12	0.6	0.2	575	269	77	N	–	–	–	–	–	–	DENGUE	G
129	306045	26	M	7	+	–	–	–	–	–	–	M/BP	–	+	–	–	NS	NS	HSM	NS	10.3	3200	45000	24	N	PV	–	24	0.8	0.8	44	56	97	N	–	+	–	–	–	–	VM	G
130	308363	19	F	11	+	+	–	–	–	–	–		–	+	–	–	NS	NS	HEP	NS	9.8	3400	73000	55	N		+	40	1	0.9	66	58	122	N	–	–	–	–	–	–	UNEXPLAINED	G
131	308768	32	M	7	+	–	–	–	+	–	–	PA	–	+	P_1I	–	NS	C	HEP	AS	10.3	19300	52000	36	UTI	–	–	93	5.4	2.8	88	63	93	AB	–	–	–	–	–	–	SEPTICEMIA	G
132	308661	24	M	6	+	–	–	–	–	+	–		–	+		–	NS	NS	NS	AS	11	9600	79000	22	N	PF/PV	–	25	1.6	1.1	160	101	112	N	–	–	–	–	–	–	MM	G
133	308113	25	F	4	+	+	–	–	–	+	–	PA	–	+	P_1E	Pe/Pu	NS	NS	HSM	AS	5.9	7900	4000	105	N	PF/PV		89	1.2	0.8	73	59	206	N	–	–	–	–	–	–	MM	G
134	322175	25	M	6								M/BP				Pe/Pu	NS	NS	NS	NS	11	5600	34000	22	N			47	1.5	0.8	44	32	101	N							UNEXPLAINED	G
135	321622	30	M	3	+	–	–	–	–	–	–		–	+	–	Pe/Pu	NS	NS	NS	NS	15.6	5600	27000	20	N	–	–	19	1	0.7	304	140	84	N	–	–	–	–	–	–	DENGUE	G
136	311041	24	M	4	+	–	–	–	–	–	–	PA	–	+	–		NS	NS	NS	NS	13.4	5800	16000	36	N	–	–	179	1.4	0.5	124	58	98	N	–	+	–	–	–	–	DENGUE	G
137	311624	35	M	13	+	–	–	–	–	–	–		–	+	–	–	NS	C	NS	AS	9.8	28500	37000	46	N	–	–	73	1.3	0.5	78	39	96	N	–	+	–	–	–	–	SEPTICEMIA	G
138	314234	18	F	5	+	+	–	–	+	+	–	–	–	+	P_1I	–	NS	NS	NS	AS	3.1	7200	57000	24	N	–	–	25	0.8	2.8	113	51	50	N	–	–	–	–	–	–	UNEXPLAINED	G
139	313641	17	M	7	+	+	–	+	–	+	–	PA/LS	–	+		–	NS	NS	HSM	NS	14.4	4000	20000	4	N	–	–	25	1	1	88	56	87	N	–	–	–	–	–	–	UNEXPLAINED	G
140	314046	26	M	28	+	+	–	–	–	–	–	PA	–	+	P_1I	–	NS	C	HSM	NS	5.8	5100	78000	48	N	PV	–	110	1.2	11	34	34	105	N	–	–	–	–	–	–	VM	G
141	312744	24	F	5	+	–	+	+	–	–	–		–	+		SB/Pe	NS	NS	NS	NS	11.5	3500	38000	38	N		–	10	0.8	1.2	162	86	126	N	–	–	–	–	–	–	UNEXPLAINED	G
142	302407	30	M	6	+	+	–	–	–	–	+	–	–	+	–	Pe/Pu	NS	C	NS	NS	18	6300	16000	10	N	–	–	32	1	3.2	182	82	122	N	–	–	–	–	–	–	DENGUE	G
143	302132	29	M	5	+	+	–	–	–	–	–	PA	–	+	–		NS	NS	HEP	NS	14.5	3700	29000	20	N	–	–	25	0.8	0.4	1085	520	108	N	–	+	–	–	–	–	DENGUE	G
144	313579	35	M	6	+	+	+	–	–	–	–	PA	–	+	–	–	NS	NS	NS	NS	13.4	3500	44000	28	N	–	–	32	1.2	0.5	65	42	72	N	–	+	–	–	–	–	DENGUE	G
145	303897	51	M	4	+	–	+	–	–	–	–		–	+	P	–	NS	NS	NS	NS	10	3400	50000	10	N	–	–	20	1	2.2	26	32	116	N	–	+	–	–	–	–	DENGUE	G

S. No	IP NO.	DATA			SYMPTOMS									P/H	GPE			SYST EXAM				INVESTIGATIONS																							OUTCOME
		AGE (in years)	SEX	HOSPITAL STAY (days)	FEVER	CHILLS & RIGORS	HEADACHE	JAUNDICE	COUGH & BREATHLESSNESS	ALTERED SENSORIUM	BLEEDING	OTHERS	TREATMENT HISTORY	TEMPERATURE	PALLOR, ICTERUS, EDEMA	BLEEDING MANIFESTATIONS	CVS	RS	PA	CNS	Hb (gm%)	TC (cells/cu mm)	PLATELET COUNT (cells/cu mm	ESR (mm/hr)	URINE ROUTINE	PS FOR MP	QBC FOR MP	BLOOD UREA (mg/dl)	SERUM CREATININE (mg/dl)	SERUM BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALKALINE PHOSPHATASE	COAGULATION PROFILE	WIDAL	DENGUE ELISA IgM/ IgG	LEPTOSPIRA IgM	HIV	BLOOD CULTURE	BONE MARROW	DIAGNOSIS	
146	305249	20	F	6								M/BP				SB/Pe	NS	NS	NS	NS	15	19200	26000	10	N			10	0.5	1.2	333	154	200	N							DENGUE	G
147	303893	30	M	4	+	+	–	–	–	–	+	M/BP	–	+	–	SB/Pe	NS	NS	HSM	NS	17.8	3500	17000	18	N	–	–	31	1.2	0.5	65	42	72	N	–	+	–	–	–	–	DENGUE	G
148	309836	19	F	8	+	+	–	–	–	–	+		–	+	–		NS	C	NS	NS	14.2	3200	52000	10	N	–	–	32	0.7	0.9	312	116	136	N	–	+	–	–	–	–	DENGUE	G
149	311926	37	M	5	+	–	–	–	+	–	–	–	–	+	–	–	NS	NS	NS	NS	14	6600	54000	8	N	–	–	30	0.9	0.8	48	56	88	N	–	+	–	–	–	–	DENGUE	G
150	306369	18	M	2	+	+	–	–	–	–	–	M/BP	–	+	–	Pe/Pu	NS	NS	NS	NS	15.3	3300	18000	10	N	–	–	20	0.7	0.9	152	52	112	N	–	+	–	–	–	–	DENGUE	G
151	249985	17	M	8	+	+	–	–	–	–	–	M/BP	–	+	–	Pe/Pu	NS	NS	HEP	AS	13.1	16100	65000	35	N	–	–	96	1.8	0.9	162	54	168	N	–	+	–	–	–	–	DENGUE	G
152	264877	28	M	8	+	+	+	–	–	+	–		–	+	P		NS	NS	SPL	NS	6	1400	19000	112	N	PV	–	40	1	2	79	88	86	N	–	+	–	–	–	–	VM	G
153	293627	19	M	5	+	+	–	–	–	–	–	M/BP	–	+		–	NS	NS	NS	NS	15	3000	25000	40	N		–	37	1	0.4	231	118	80	N	–	–	–	–	–	–	UNEXPLAINED	G
154	317513	19	M	1	+	+	+	–	–	–	–	LS	–	+	P	SB	NS	NS	NS	NS	4.5	3400	50000	140	N	–	–	31	1.2	0.9	270	105	55	N	–	–	–	–	–	–	UNEXPLAINED	G
155	265273	15	M	15	+	–	–	–	–	–	+	PA/LS	–	+			NS	NS	SPL	NS	9.1	1700	20000	48	N	–	–	22	1	1	243	89	135	N	–	–	–	–	–	–	ENTERIC FEVER	G
156	240400	48	F	4	+	+	+	–	–	–	–	M/BP	–	+	P	–	NS	NS	NS	NS	13.5	5200	32000	20	N	–	–	13	0.7	0.6	667	262	301	N	–	–	–	–	–	–	DENGUE	G
157	234344	36	M	8	+	–	–	–	–	–	–	M/BP	–	+		Pe/Pu	NS	NS	NS	NS	14.9	2200	15000	68	N	–	–	21	0.9	0.6	105	74	51	N	–	+	–	–	–	–	DENGUE	G
158	235695	28	M	6	+	–	+	–	–	–	–		–	+	P	SB	NS	NS	NS	NS	13.8	2200	26000	53	N	–	–	24	0.8	0.5	287	169	171	N	–	+	–	–	–	–	DENGUE	G
159	251011	38	M	7	+	+	–	–	–	–	+	M/BP	–	+			NS	NS	NS	NS	12	6200	10000	28	N	–	–	28	0.9	0.6	112	71	121	N	–	+	–	–	–	–	DENGUE	G
160	239332	55	F	4	+	+	+	–	–	–	–	PA	–	+	–	–	NS	NS	NS	NS	13	6300	38000	28	N	–	–	17	0.7	0.6	209	124	117	N	–	+	–	–	–	–	DENGUE	G
161	280471	18	M	14	+	+	–	–	–	–	–	M/BP	–	+	–	–	NS	NS	NS	NS	17.7	7500	15000	9	N	–	–	27	0.8	0.2	88	40	104	N	–	+	–	–	–	–	UNEXPLAINED	G
162	282174	32	M	3	+	+	–	–	–	–	–	PA	–	+	I	–	NS	C	HSM	NS	12.9	6300	73000	24	UTI	–	–	59	1.8	8	211	110	191	N	–	–	–	–	–	–	SEPTICEMIA	G
163	281443	24	M	6								M/BP				Pe/Pu	NS	NS	NS	NS	12.4	3500	12000	46	N			33	0.7	0.3	78	59	66	N							UNEXPLAINED	G
164	249660	31	F	6	+	–	+	–	–	–	–		–	+	–		NS	NS	HSM	NS	9.1	8300	28000	98	UTI	–	–	37	0.7	1.4	387	249	1069	N	–	–	–	–	–	–	DENGUE	G
165	241380	18	F	5	+	+	+	–	–	–	–	–	–	+	P	–	NS	NS	NS	NS	8	7600	80000	28	N	–	–	27	0.9	1.3	32	26	56	N	–	+	–	–	–	–	DENGUE	G
166	235044	32	M	8	+	+	–	–	+	–	–	PA	–	+		SB/Pe	NS	PE	HEP	NS	16.2	3800	5000	39	N	–	–	29	0.8	1.2	167	97	128	N	–	+	–	–	–	–	DENGUE	G
167	238206	18	M	6	+	+	+	–	–	–	+	PA	–	+	–		NS	PE	NS	NS	16.5	13000	13000	4	N	–	–	24	0.7	0.7	439	89	112	N	–	+	–	–	–	–	DENGUE	G
168	233853	18	M	5	+	–	–	–	–	–	+	M/BP	–	+	–	Pe/Pu	NS	NS	NS	NS	16.2	10200	25000	10	N	–	–	36	1.3	0.9	37	27	52	N	–	+	–	–	–	–	DENGUE	G
169	252118	32	M	4	+	–	+	–	–	–	–	M/BP	–	+	–		NS	NS	NS	AS	14.4	20500	98000	15	N	–	–	41	0.9	0.6	130	96	104	N	–	+	–	–	–	–	DENGUE	G
170	253360	27	M	7	+	+	–	–	+	+	–	PA	–	+	–	–	NS	NS	NS	NS	13.4	4200	51000	28	N	–	–	20	0.7	0.9	215	149	102	N	–	+	–	–	–	–	DENGUE	G
171	234074	21	M	5	+	+	+	–	–	–	–	PA	–	+	–	SB	NS	NS	NS	NS	10	6200	99000	18	N	–	–	30	1	1.8	60	53	142	N	–	+	–	–	–	–	DENGUE	G
172	240728	65	M	3	+	–	–	–	–	–	+		–	+	I		NS	NS	NS	AS	13.2	2100	80000	28	N	–	–	106	2.3	8.3	151	92	181	AB	–	+	–	–	–	–	UNEXPLAINED	G
173	241304	70	F	3	+	+	+	–	–	–	–	–	–	+		–	NS	NS	NS	NS	12.8	15800	21000	10	N	–	–	166	3.2	0.9	29	36	109	N	–	–	–	–	–	–	DENGUE	G
174	237962	35	M	7	+	–	–	–	–	–	–	–	–	+	–	–	NS	NS	NS	NS	15.6	6900	14000	8	N	–	–	25	0.9	2.9	599	252	192	N	–	+	–	–	–	–	UNEXPLAINED	G

S. No	IP NO.	DATA			SYMPTOMS									P/H	GPE			SYST EXAM				INVESTIGATIONS																							OUTCOME
		AGE (in years)	SEX	HOSPITAL STAY (days)	FEVER	CHILLS & RIGORS	HEADACHE	JAUNDICE	COUGH & BREATHLESSNESS	ALTERED SENSORIUM	BLEEDING	OTHERS		TREATMENT HISTORY	TEMPERATURE	PALLOR, ICTERUS, EDEMA	BLEEDING MANIFESTATIONS	CVS	RS	PA	CNS	Hb (gm%)	TC (cells/cu mm)	PLATELET COUNT (cells/cu mm	ESR (mm/hr)	URINE ROUTINE	PS FOR MP	QBC FOR MP	BLOOD UREA (mg/dl)	SERUM CREATININE (mg/dl)	SERUM BILIRUBIN (mg/dl)	SGOT (U/L)	SGPT (U/L)	ALKALINE PHOSPHATASE	COAGULATION PROFILE	WIDAL	DENGUE ELISA IgM/ IgG	LEPTOSPIRA IgM	HIV	BLOOD CULTURE	BONE MARROW	DIAGNOSIS	
175	238802	35	F	5								M/BP						NS	NS	HEP	NS	16.4	8400	25000	39	N			26	0.6	0.6	1129	459	105	N							DENGUE	G
176	267003	51	M	18	+	+	+	–	+	–	–	PA/OG	–	+	I	SB	NS	PE	NS	AS	9.9	16300	34000	34	N	PF/PV	–	71	3.3	29	511	650	129	AB	–	+	–	–	–	–	MM	G
177	266820	20	F	9	+	+	+	+	–	+	+		–	+	P,I	Pe/Pu		NS	NS	HSM	NS	9	2300	8000	48	UTI	PF/PV	+	23	0.5	7.3	61	67	146	N	–	–	–	–	–	–	MM	G
178	268089	30	M	4	+	+	+	+	+	–	–	M/BP	–	+				NS	NS	SPL	NS	10.4	5000	70000	62	N	PV	+	30	0.9	0.7	25	42	66	N	–	–	–	–	–	–	VM	G
179	240450	50	F	5	+	+	+	–	–	–	–	M/BP	–	+	–	Pe/Pu		NS	NS	NS	NS	14	8000	22000	22	N		–	22	0.4	0.8	66	54	87	N	–	–	–	–	–	–	DENGUE	G
180	248372	22	F	6	+	–	+	–	–	–	–	PA/LS	–	+	–	SB/Pe		NS	NS	HEP	NS	12.3	7800	21000	64	N	–	–	27	0.7	0.5	80	58	107	N	–	+	–	–	–	–	DENGUE	G
181	317079	23	F	5	+	–	+	–	+	–	+	M/BP	–	+	I			NS	NS	SPL	NS	11.8	6500	33000	28	N	–	–	22	0.9	4	76	74	176	N	–	+	–	–	–	–	MM	G
182	317076	18	F	4	+	–	+	–	+	–	–		–	+		–		NS	NS	NS	NS	14.1	4800	15000	18	N	–	+	40	0.7	0.9	66	54	112	N	–	–	–	–	–	–	DENGUE	G
183	271283	35	M	4	+	–	+	–	–	–	–	PA/LS	–	+	–	SB		NS	NS	HEP	NS	13.3	21800	89000	15	N	–	–	16	1	0.7	96	87	111	N	–	+	–	–	–	–	SEPTICEMIA	G
184	271230	50	M	5	+	+	+	–	–	–	+	M/BP	–	+	–			NS	C	HEP	NS	13.8	8500	20000	44	UTI	–	–	51	2.3	1.7	94	84	106	N	–	–	–	–	–	–	SEPTICEMIA	G
185	272374	40	M	4	+	+	+	–	–	+	–		–	+	I	–		NS	C	HEP	AS	11.2	13600	75000	48	UTI	–	–	187	7.7	6.7	167	39	108	AB	–	–	–	–	–	–	LEPTOSPIROSIS	E
186	273111	24	M	5	+	+	+	+	–	+	–	PA/LS	–	+		SB/Pe		NS	NS	NS	NS	17.7	7000	12000	10	N	–	–	96	3.3	1.6	108	105	97	N	–	–	+	+	–	–	DENGUE	G
187	275405	25	M	3	+	+	+	–	–	–	+		–	+	–	SB/Pe		NS	NS	NS	NS	15	3100	20000	10	N	–	–	29	0.7	0.5	112	65	88	N	–	+	–	–	–	–	DENGUE	G
188	281838	55	M	8	+	–	+	–	–	–	+	M/BP	–	+	–	SB/Pe		NS	NS	NS	NS	12.9	2800	33000	30	N	–	–	26	0.7	0.6	104	88	126	N	–	+	–	–	–	–	UNEXPLAINED	G
189	281821	60	F	5	+	–	+	–	–	–	+	M/BP	–	+	I			NS	NS	NS	NS	11.9	8600	58000	34	N	–	–	66	1.4	5.5	114	90	175	AB	–	–	–	–	–	–	UNEXPLAINED	G
190	280704	48	M	11	+	–	–	+	–	–	–	OG	–	+		SB		NS	C	NS	AS	12.5	4900	76000	16	N	–	–	84	3.8	0.5	147	141	171	N	–	–	–	–	–	–	UNEXPLAINED	G
191	323727	58	F	5	+	+	+	–	–	+	+	PA	–	+	–			NS	NS	NS	NS	12	5800	21000	14	N	–	–	48	1.1	0.6	78	92	82	N	–	–	–	–	–	–	DENGUE	G
192	323827	28	M	6														NS	NS	NS	NS	13.5	7900	36000	9	N			44	1	0.6	112	88	161	N							DENGUE	G
193	319084	43	M	11	+	+	–	–	–	–	–	–	–	+	P,E	–		NS	NS	NS	NS	6	5800	84000	82	N	–	–	34	0.8	0.8	115	90	92	N	–	+	–	–	–	–	UNEXPLAINED	G
194	318063	35	M	8	+	+	–	–	+	–	–	PA	–	+		–		NS	NS	NS	AS	13	12500	80000	18	N	–	–	30	1.1	1.3	68	40	130	N	–	–	–	–	–	–	UNEXPLAINED	G
195	318482	32	M	10	+	+	–	–	–	+	–	PA	–	+	P	–		NS	NS	HEP	NS	8	13500	82000	48	N	–	–	78	1.8	1.6	68	70	84	N	–	–	–	–	–	–	UNEXPLAINED	G
196	273093	35	F	5	+	+	+	–	–	–	–		–	+	P	SB		NS	NS	SPL	NS	3.5	8500	18000	55	N	PV	–	46	0.6	0.3	22	25	79	N	–	–	–	–	–	–	VM	G
197	272376	20	M	10	+	+	–	–	–	–	+	M/BP	–	+				NS	NS	HSM	NS	12.8	12000	80000	32	N		–	27	1.1	0.5	124	91	95	N	–	–	–	–	–	–	UNEXPLAINED	G
198	266388	54	M	6	+	–	+	–	+	–	–	M/BP	–	+	–	–		NS	NS	NS	NS	10.9	6700	15000	44	N	–	–	31	0.8	0.6	51	31	108	N	–	–	–	–	–	–	SEPTICEMIA	G
199	248017	35	M	4	+	+	+	–	–	–	–		–	+	–	–		NS	NS	HSM	NS	11.8	3400	72000	45	N	PF/PV	–	28	1.1	1.7	38	80	64	N	–	–	–	–	–	–	MM	G
200	256246	18	M	10	+	+	+	–	+	–	–	M/BP	–	+	–	SB/Pe		NS	NS	SPL	NS	13.4	1200	5000	46	N		+	23	0.9	0.6	51	74	130	N	–	–	–	–	–	–	UNEXPLAINED	G

ANNEXURE IV

KEY TO MASTER CHART

-	-	Negative
+	-	Positive
AB	-	Abnormal
AS	-	Altered Sensorium
C	-	Crepts
DM	-	Diabetes mellitus
E	-	E-Coli
E	-	Expired
ENT	-	Enteric fever
F	-	Female
FM	-	Falciparum Malaria
G	-	Good
HEP	-	Hepatomegaly
HOA	-	History of abortion
HSM	-	Hepatosplenomegaly
I	-	Icterus
LS	-	Loose stools
M	-	Male
M/BP	-	Myalgia/body pain
MM	-	Mixed Malaria
N	-	Normal
NS	-	Nothing significant

OG	-	Oliguria
P	-	Pallor
P/H	-	Past history
PA	-	Pain abdomen
PE	-	Pleural Effusion
Pe	-	Petechiae
Pe/Pu	-	Petechiae/Purpura
PF	-	Plasmodium falciparum
Pu	-	Purpura
PV	-	Plasmodium vivax
SB	-	Spontaneous Bleeding
SPL	-	Splenomegaly
UTI	-	Urinary Tract Infection
VM	-	Vivax Malaria