"A STUDY OF CLINICAL AND LAB PROFILE OF FEVER WITH THROMBOCYTOPENIA"

BY

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Dissertation submitted to the
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In Partial fulfillment
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IN

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Under the guidance of

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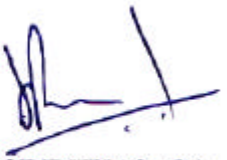

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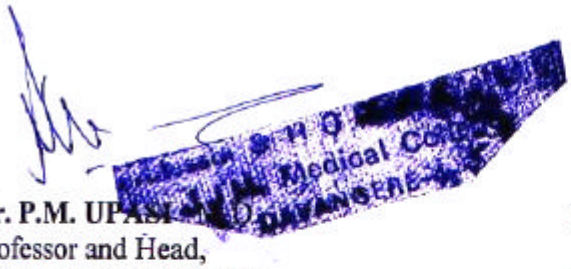

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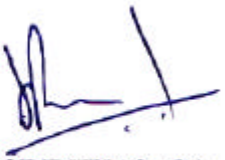

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LIST OF ABBREVATIONS USED

APP	Acute phase protein
APR	Acute phase response
CMV	Cytomegalo virus
CRP	C-Reactive protein
DIC	Dieseminated 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

KEY TO MASTER CHART

N	Normal	AB	Abnormal
-	Negaative	ANTI/SU	Antibiotics/Supportive
HOA	History of abortion	DM	Diabetes mellitus
C	Crepts	E	Expired
P/H	Past history	F	Female
VM	Vivax malaria	FM	Falciparum malaria
ENT	Enteric fever	G	Good
Pl	Pallor	I	Icterus
BH	Bapuji Hosptial	M	Male
NS	Nothing significant	M/BP	Myalgia/body pain
+	Positive	P	Pseudomonas
LS	Loose stools	PA	Pain abdomen
Pu	Purpura	Pe	Petichae
E	E-Coli	PF	Plasmodium falciparum
mds	Malarid-ds	PT	Platelet transfusion
SY	Symptomatic treatment	Pv	Plasmodium vivax
PE/Pu	Petichea/purpure	SPB	Spontaneous Bleeding

ABSTRACT

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

Methods: 100 patients aged >12 years with fever and thrombocytopenia between March 2004 – September 2005 were included for this study.

Results : Infection was the commonest cause of thrombocytopenia and malaria was the commonest infections. Bleeding manifestations were seen in 49% of patients. 63% of patients had Petichae/ purpura as the commonest bleeding manifestation followed by spontaneous bleeding in 37%. Good recovery was noted in 82% while 18% had mortality. Septicemia accounted for 78% of deaths, followed by dengue/VHF 22%.

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

Key words :

Infection, Malaria, Petichae/purpura, Spontaneous bleeding, Mortality.

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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 Gabrial Daniel Fahreneheit, 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). IL-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.

An AM temperature of >37.2°C (98.9°F) or a P.M. temperature of > 37.7°C (99.9°F) would define fever[1].

Thrombocytopenia is defined as platelet count <150,000/µl. This is due to decreased production, increased destruction (immunogenic and non immunogenic), 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, HIV and military TB are some of the common causes of fever with thrombocytopenia.

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

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

OBJECTIVES

- 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 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° F(37°C) its special significance Vis-à-vis the normal temperature. He described the normal diurnal variation of the body 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 work "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 12hour 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 describeod 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 a murine macrophages and human lymphoma cell[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

1. 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:

Thermo regulation is controlled by a continum 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 therma l 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 possesses two known pyrogens: Lipopolysacharide (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 Lipopolysacharide 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 peptidoglycon, Lipotechoic acid and a group of rhamnose glucose polymers. The basic structure responsible for peptidoglycan's pyrogenicity is muramy1 peptide (MDP-N-acety1 muramy1-L-alanine-d-isoglutamine).

Gram positive bacteria release exotoxins, which can also cause fever. Exotoxins act by binding to Major Histocompactibility 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 superantigen.

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 produed 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, IL-1b, TNF-y, TNF-B, IFN-a, IL-6. These now include a family of cytokines using the cell-signalling apparatus gp130.

Cells using this receptor are pyrogenic and currently include IL-6, IL-11, oncostatin-M, Ciliary Neurotrophic Factor (CTNF), 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 Organnum Vasculosum Laminae Terminalus (OVLT), posses 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 PGE2a 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 pathogenessis 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 IL -1

The molecule IL-1 (either IL-1a or IL-1B) is a bonafide EP. At present there are two genes coding for two different IL-1 forms: IL – 1a and IL – 1B. The pH 7 form has been designated IL –1 B and the PH – 5 form has been designated IL – 1a.

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 – 1 B is readily secreted from activated cells, where as IL – 1a remains cell associated and hence IL – 1a is likely to be more relevant for local diseases than for systemic disorders[7].

One of the most potent proinflammatory 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-glycopotein complex designated leucocyte function antigen.

Tumour 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 aminoacid 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-βcells 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 PGE_2 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-1β induction of IL-6, suggesting that IL-6 might be the final common pathway for such fever.

The Interferons (IFN) :

Intererons 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-α 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-α 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-γ can also be pyrogenic in humans, it stimulaltes IL-1 production.

IL-2 infusions induce fever in humans, induces TNF and IFN-γ.

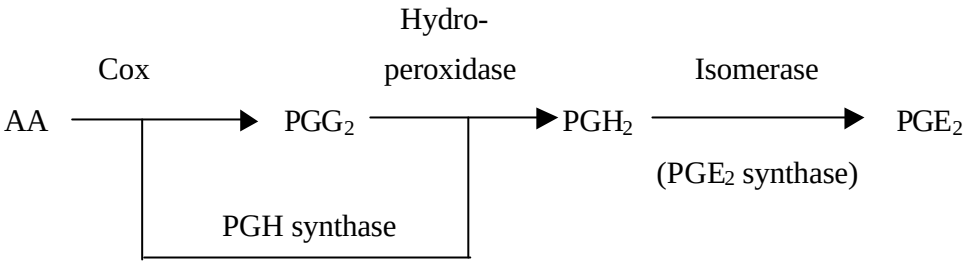
The concept of cytokines inducing other cytokines is vital to the understanding of how non-infectious disease produce fever. Vasculitis, rheumatoid arthritis, lupus, trauma, hemorrhage, thrombophlebites, drug fever and cancer are examples of non-infectious diseases in which both fever and acute phase response are common. Fever is also a prominent side effect of cancer chemotherapy, and the treatment of transplant rejection with antibodies to CD_3 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 E_2 : 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 and essential role in fever production. Induction of PGE_2, is triggered by peripheral signals evoked by early, non cytokine factors elicitied 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 PGE_2.

Prostaglandin E_2 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.



The pathway of PGE_2 synthesis from arachidonic acid.

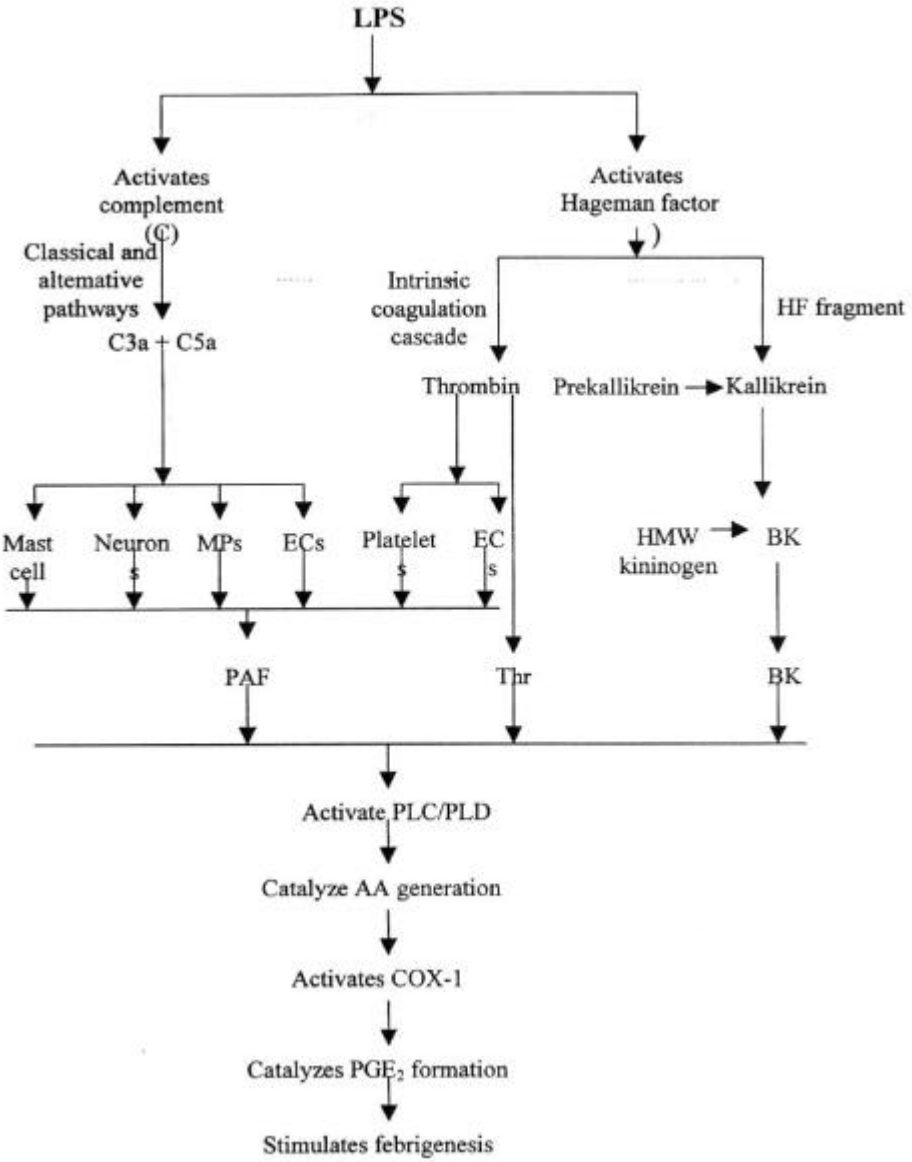
Humoral mechanism :

The anaphylatoxic components of the complement, C_{3a} and C_{5a} 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 C_1 and C_3. Within seconds of binding to its receptors on MP_3, C_{3a} 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.



The hypothesized successive endogenous mediators evoked by intravenous LPS potentially contributing to the generation of PGE2 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 41C 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, α-melancoyte 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.

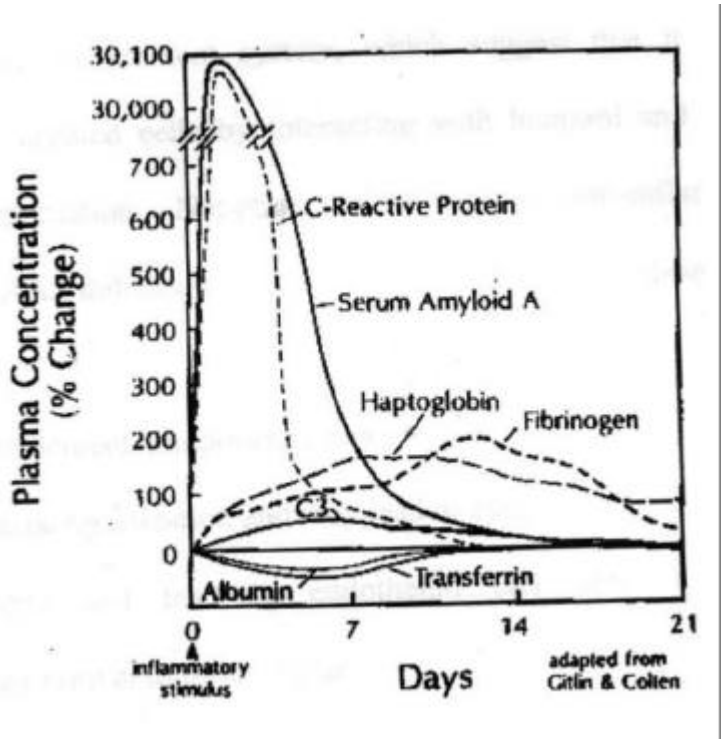
Fever is only one of the changes in homeostatic settings that occur during the APR. Other changes include Somnolence, Anorexia, changes in plasma protein synthesis and altered synthesis of many endocrine hormones. In addition negative nitrogen balance, gluconeogenesis, decreased levels of zinc and iron 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, streneous 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, α2-HS glycoprotein.



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 have 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].

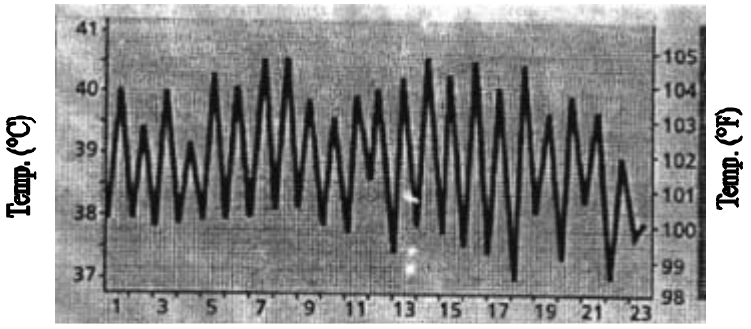
Eg: Localized pyrogenic infections and bacterial endocarditis; Malaria (commonly with leukopenia) may present as quotidian (daily spike), tertian (spike every third day), or quartan (spike every fourth day) types.

A double quotidian pattern with two daily spikes occurs sufficiently often to be helpful in salmnelloses, 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 Bruciella 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.

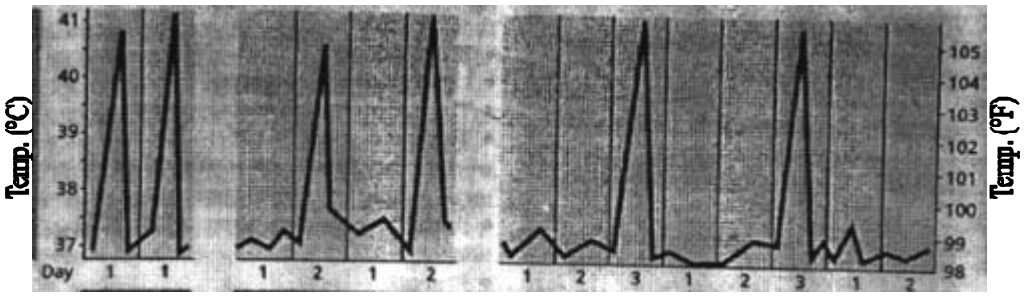
Fever should be regarded as a reliable clinical sign and with the fever pattern mentioned above, it is possible to suggest a diagnosis within the group of disease and this can lead to specific therapy and ultimate cure.

REMITTENT FEVER :

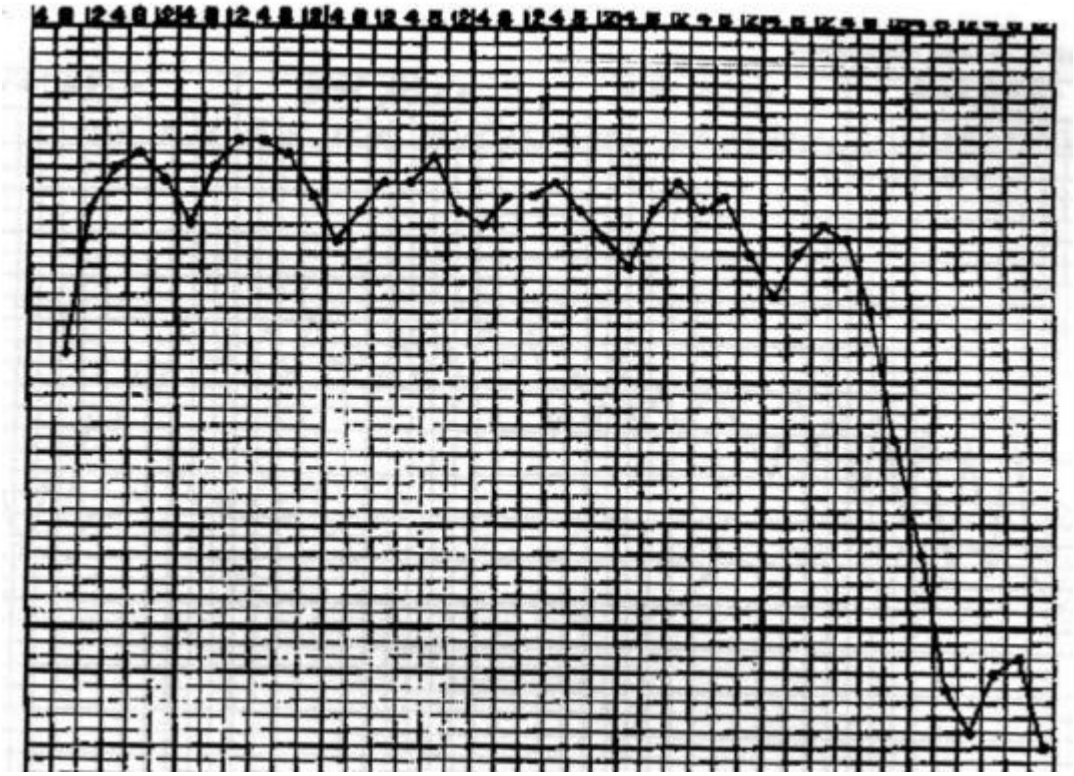


Days

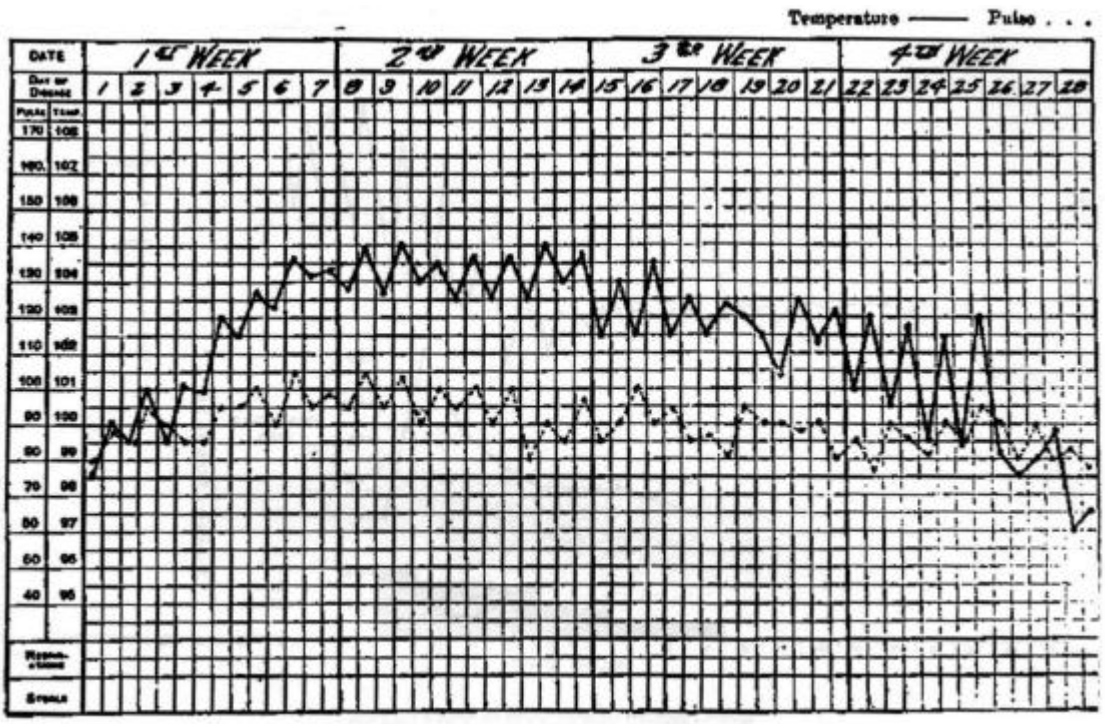
INTERMITTENT FEVER :



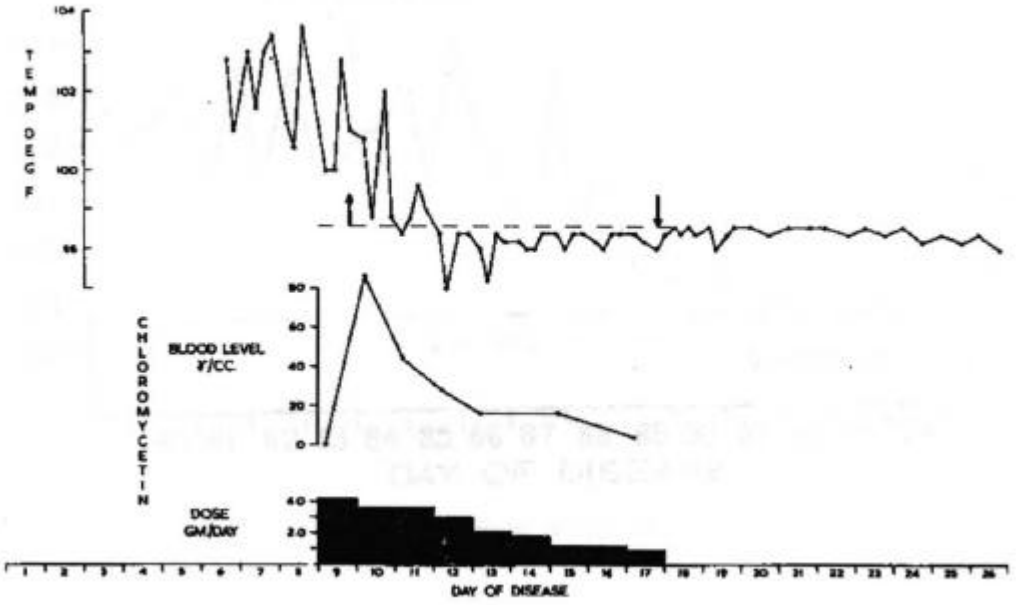
Quotidian Tertian Quartan



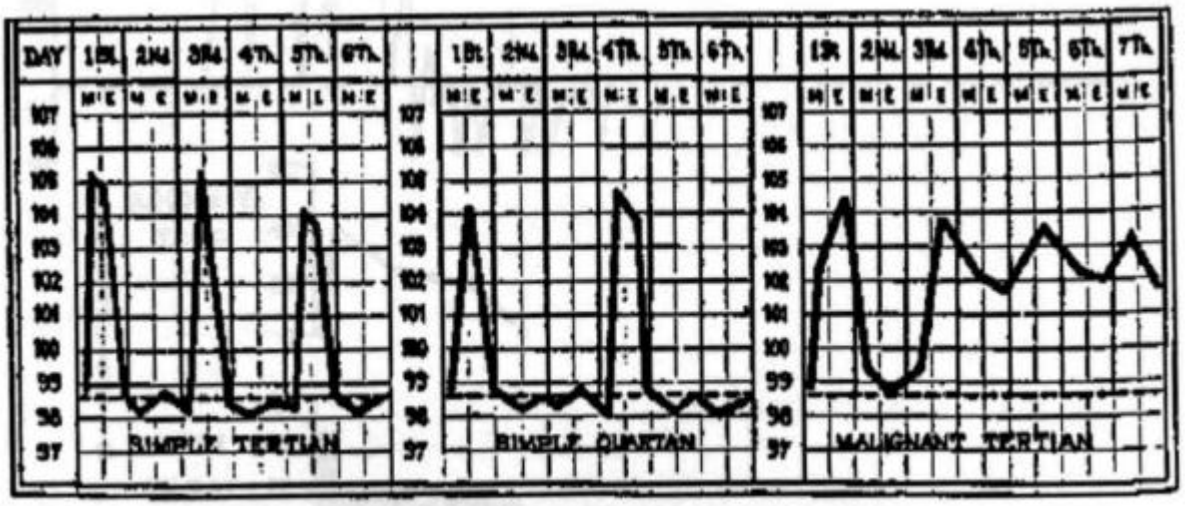
Continuous fever



Febrile course in uncomplicated typhoid fever.



Typhoid fever in specifically treated case.



Malaria fever patterns.

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 count 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 (51_{Cr}) or indium (111_{In})[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 :

CMV, Dengue, Parvo-B_{19}, HSV, HIV, Hantana virus etc[14].

Mechanism :

Viruses produced 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 –ve and gram +ve 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 caused thrombocytopenia by marrow infiltration (lymphoma, leukaemia)[14].

CLINICAL COMPLIATIONS OF THROMBOCYTOPENIA :

- Platelet count of >1 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 <50,000 have easy bruising, manifested by skin purpura after minor trauma.[13,14]
- Platelet count of <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.

a) Complete haemogram :

1. ESR : > 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 suggests septicemia also should be examined for malarial parasites.

b) Rapid spot test :

For plasmodium vivax and plasmodium falciparum species

It is very sensitive for detection of malaria.

c) WIDAL – tube method for identification of enteric fever.

d) IgM ELISA dengue – will be positive

after 5^{th} day of fever and rising titres are indicative of dengue.

e) IgM ELISA leptospiral antibodies – In very acute, toxic presentation with conjunctival suffusion with renal and liver parameters being abnormal.

f) Blood culture – at least 3 blood culture samples should be taken, special technique are required, for fastidious organisms to grow and incubation has to be continued for at least 2 weeks.

g) Bone marrow examination :

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.

i) Malarial infections

ii) Leptospiral

iii) Dengue infections

iv) 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

MEHTOD OF STUDY :

This study was done on patients, who were admitted to Chigateri General Hospital and Bapuji Hospital attached to J.J.M. Medical College, Davangere during the period of May 2004 to December 2005.

We prospectively collected a series of 100 patients with fever and thrombocytopenia.

Inclusion criteria :

- The patients of both sexes aged > 12 years.
- Patients admitted with fever and found to have thrombocytopenia are included in the study.

Exclusion criteria :

- Patients <12 years are excluded
- Patients with fever and no thrombocytopenia are not included.
- Patients with thrombocytopenia and no fever are not included

Once the patients admitted with fever and those who had thrombocytopenia, a careful history was recorded, general physical examination was done. Detailed examination of various systems was done. Routine

investigation was done, the specific and special investigations were done as and when indicated.

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 ventilations, haemodialysis etc.) For bleeding complications platelet transfusions was done if platelet count was <20,000/cumm.

The causes of fever with thrombocytopenia are so numerous, 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.

RESULTS AND OBSERVATION

A total number of 100 patients admitted over a period of one and a half years in our hospital were studied.

No particular age group was considered , but the study subjects were in the age group of 18-79 years.

The sex of the patient was not taken into consideration for the study. Out of 100 cases of fever with thrombocytopenia, 58 were males and 42 were females.

The duration of hospitalization varied between 3 days to 21 days. The average duration of hospitalization was 7 days.

Out of 100 patients of fever with thrombocytopenia, all of them had definitive diagnosis with malaria (41%) as the commonest cause, followed by enteric fever (24%); septicemia (19%), dengue (14%) and leptospirosis (2%).

In malaria, vivax malaria (48%) was commonest followed by falciparum malaria (32%) and mixed malaria (20%).

In our study 65% of the patients had platelet count in the range of 50,000 – 1,00,000, followed by 23% and 12% of the patients had platelet count in the range of 20,000 – 50,000; 0-20,000 respectively.

Common range of platelet count at the time of admission was 61-80 thousands in 36 cases, followed by platelet count in range of 81-100

thousands in 24 cases; 21-40 thousands in 16 cases ;41-60 thousands in 12 cases and 0-20 thousands in 12 cases.

Clinical manifestation of thrombocytopenia was there in 49 patients and there was no clinical manifestation of thrombocytopenia in 51 patients .

Out of 49 patients, 31 patients (67%) had petichae / purpura and spontaneous bleeding was seen in 18 patients(33%).

Out of 100 patients, 82 of them had good recovery and 18 of them expired.

In 82 cases, who had good recovery, 30 cases were followed up and platelet count were near normal after discharge and further follow up.

In 18 mortality cases, 14 (72%) were due to septicemia and multiorgan dysfunction syndrome and 4 (18%) were due to dengue fever.

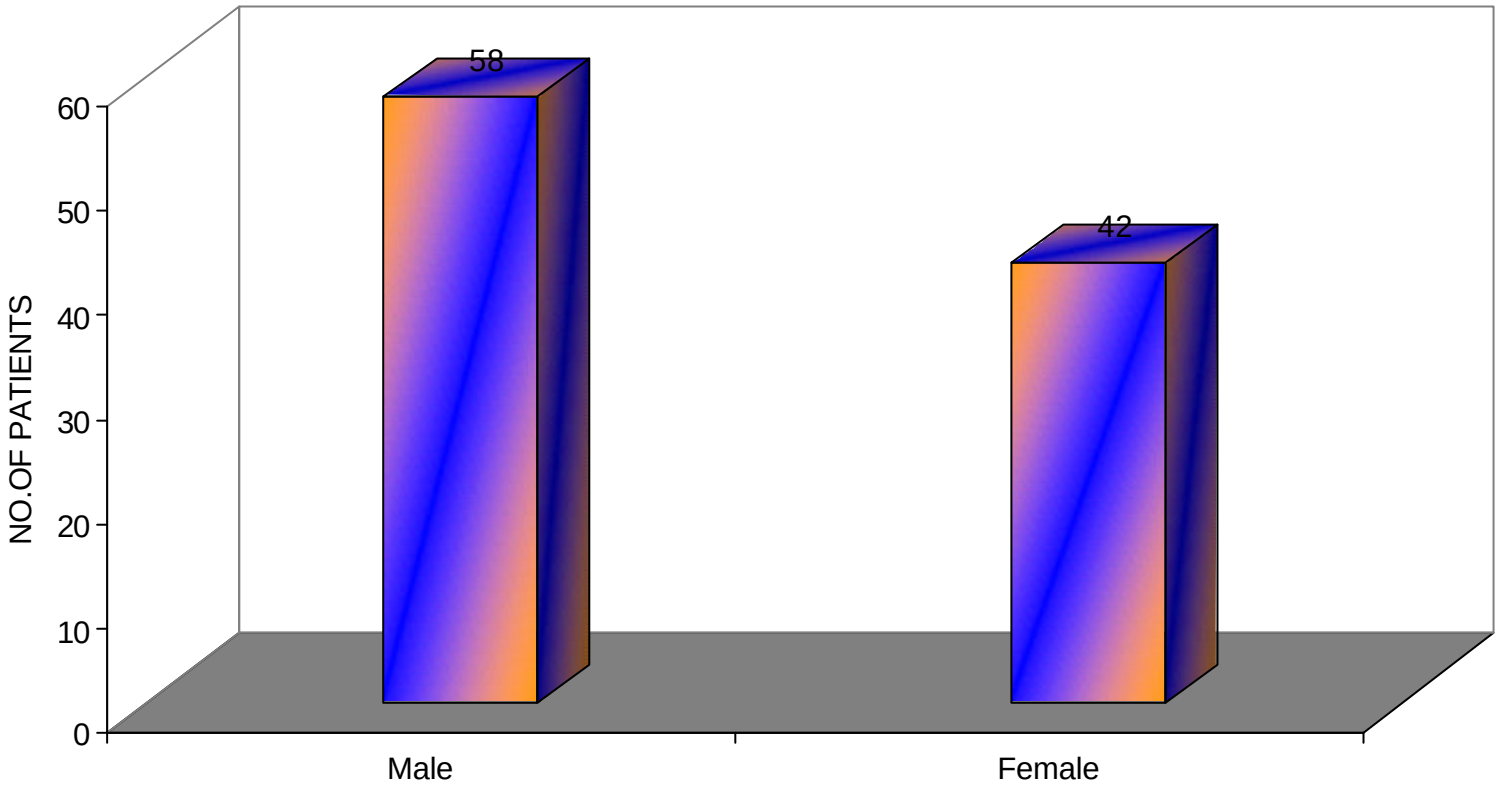
In 18 mortality cases, majority of platelet count was in the range of 10000-20000 cell/cumm in 7 cases.

TABLE – 2

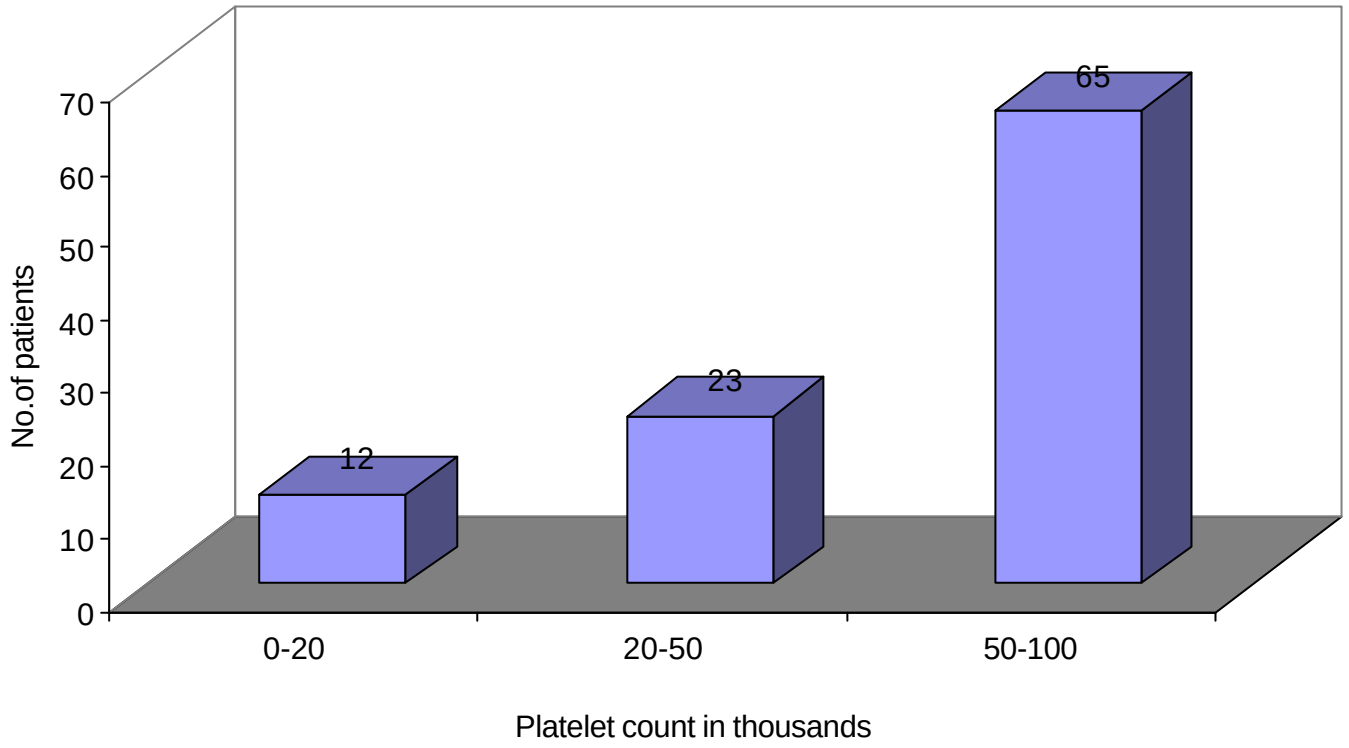
PRELIMINARY DATA OF THE STUDY

Total no. of patients	100
Age range in years	18-79 years
Male and female	58:42
Range of duration of hospitalization (days)	3:21
Average duration of hospitalization (days)	7
Definitive diagnosis	100
Malaria as the common cause	41
In malaria, vivax as common cause	20
61,000-80,000 was common range of platelet count at time of admission	36
Clinical manifestation of thrombocytopenia	49
Bleeding manifestations of thrombocytopenia (petichae/ purpura : spontaneous bleeding)	31:18
Good recovery	82
Mortality	18
Septicemia as the common cause of mortality	14
10-20,000 was the range of platelet count in mortality cases	7
Good recovery cases followed up	30

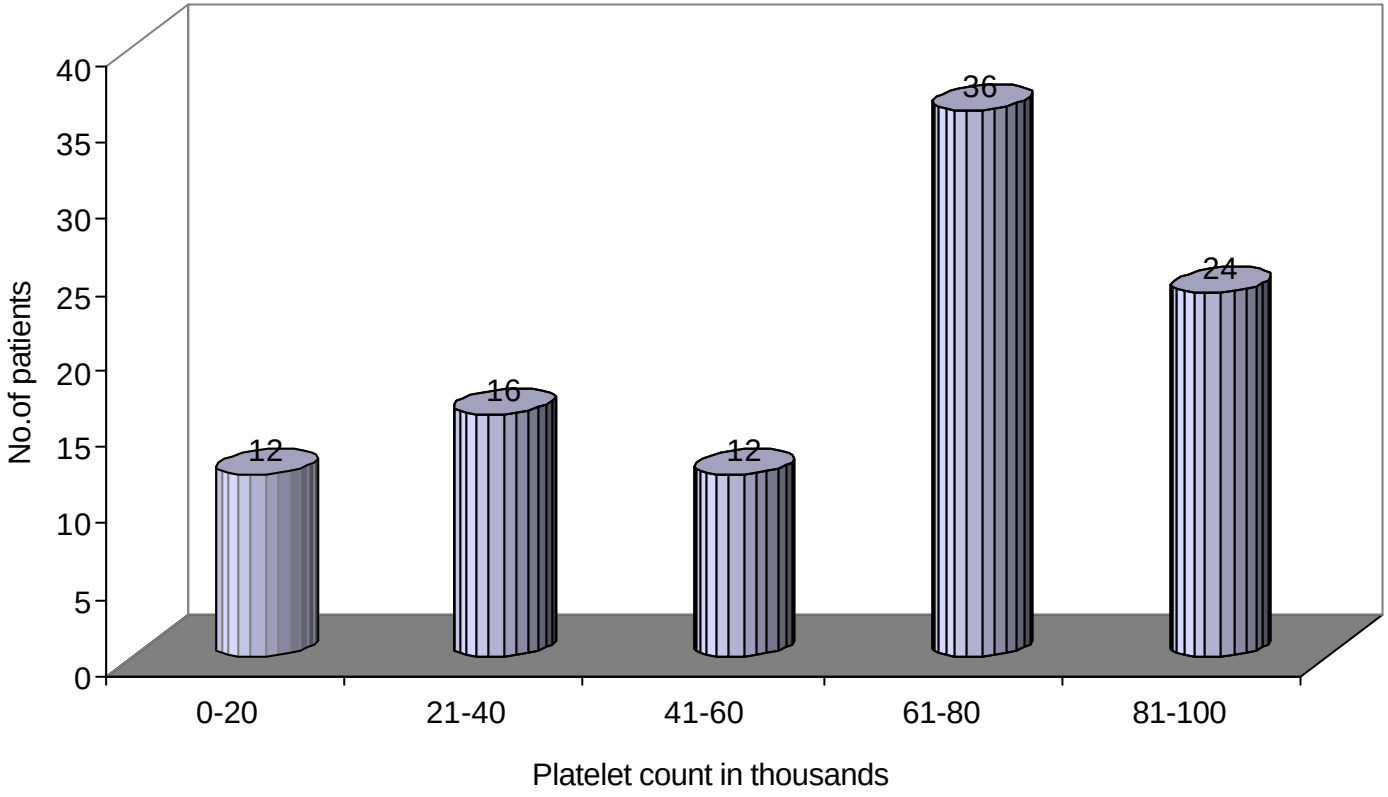
GRAPH SHOWING INCIDENCE OF MALE AND FEMALE PTIENTS IN OUR STUDY



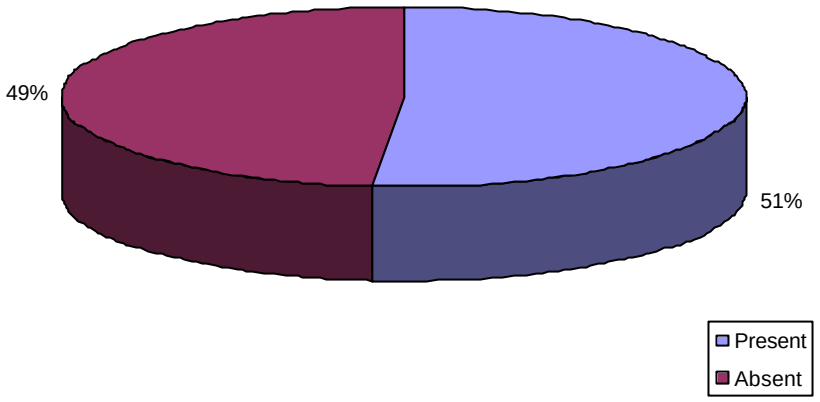
Graph showing distribution of platelet count in present study



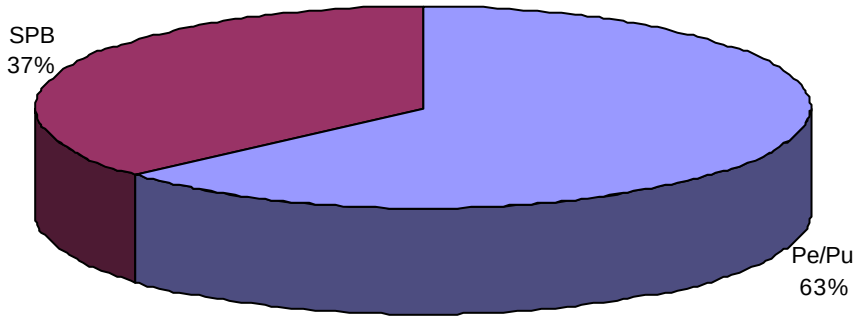
GRAPH SHOWING DISTRIBUTION OF PLATELET COUNT AT THE TIME OF ADMISSION



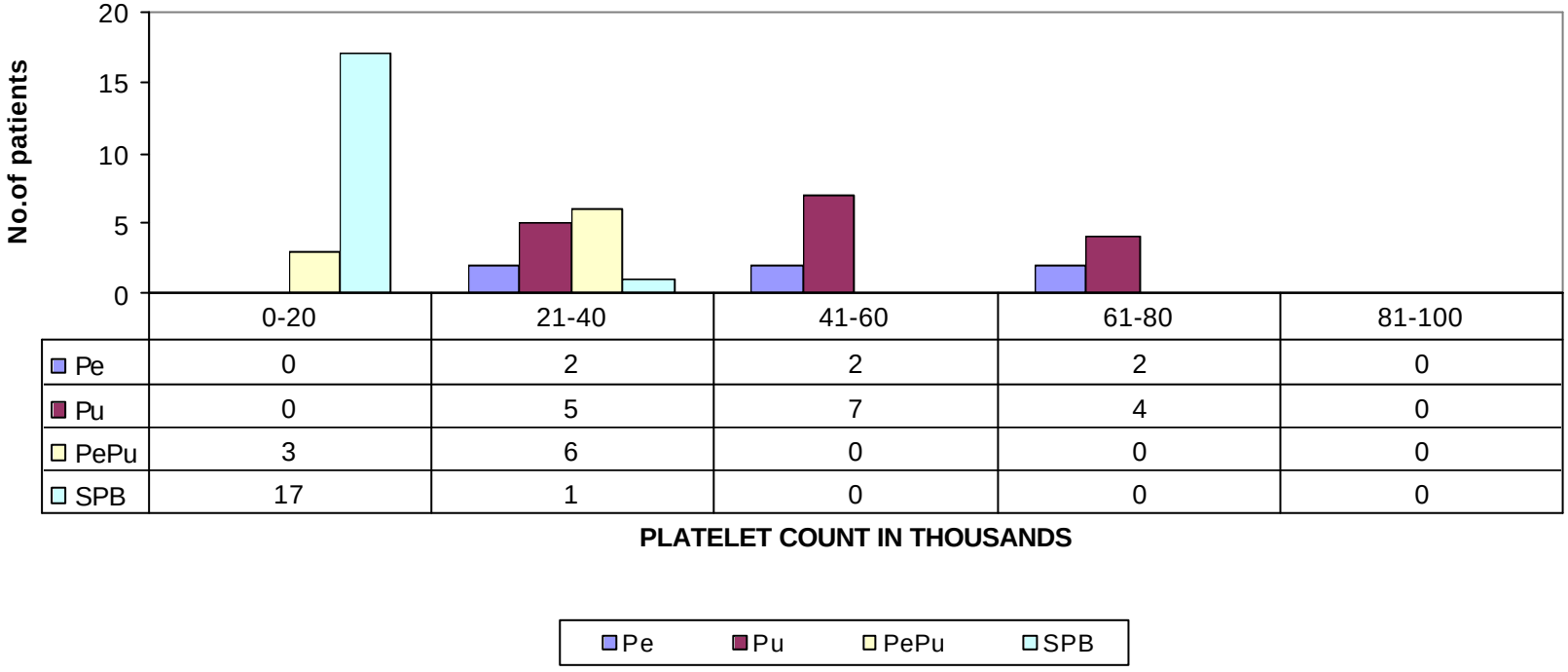
GRAPH SHOWING CLINICAL MANIFESTATIONS OF THROMBOCYTOPENIA



Graph showing bleeding manifestations



GRAPH SHOWING CLINICAL MANIFESTATION OF THROMBOCYTOPENIA IN RELATION TO PLATELET COUNT



	0-20	21-40	41-60	61-80	81-100
Pe	0	2	2	2	0
Pu	0	5	7	4	0
PePu	3	6	0	0	0
SPB	17	1	0	0	0

Incidence of various causes of fever with thrombocytopenia (Present study):

Out of 100 cases, a definitive a diagnosis could be made in all of them.

Among them malaria was the major cause accounting for 41 cases and 41% of the total cases. In malaria, vivax malaria accounted for 20 cases and 48% of the malaria cases, followed by falciparum malaria accounted for 13 (32%) cases and mixed malaria accounted for 8 (20%).

Second major cause was enteric fever 24 (24%) cases, followed by septicemia 19 (19%), dengue 14 (14%) cases and leptospirosis 2 (2%) cases.

TABLE – 3

INCIDENCE OF VARIUOS CAUSES OF FEVER WITH THROMBOCYTOPENIA

Disease category	No.of patients	Percentage
Malaria	41	41
Enteric fever	24	24
Septicemia	19	19
Dengue	14	14
Leptospirosis	2	2
Total	100	100

GRAPH SHOWING INCIDENCE OF VARIOUS CAUSES OF FEVER WITH THROMBOCYTOPENIA

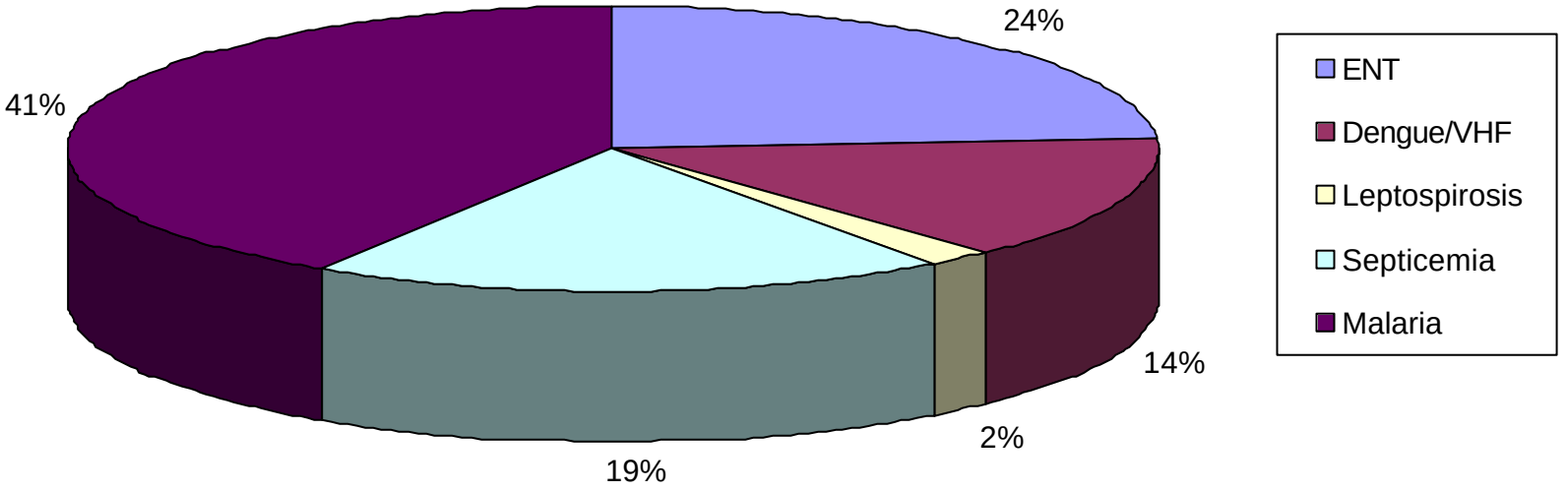
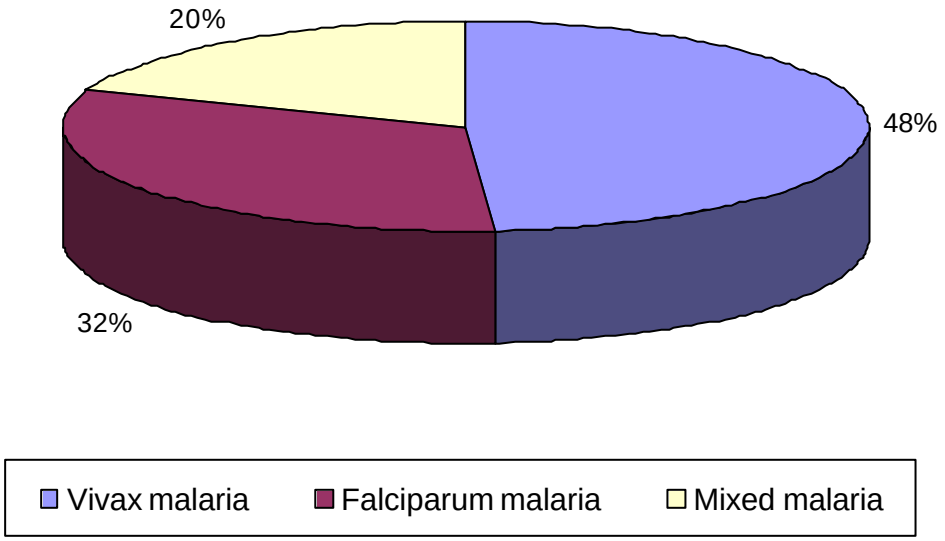


TABLE – 4

TYPES OF MALARIA WITH THROMBOCYTOPENIA

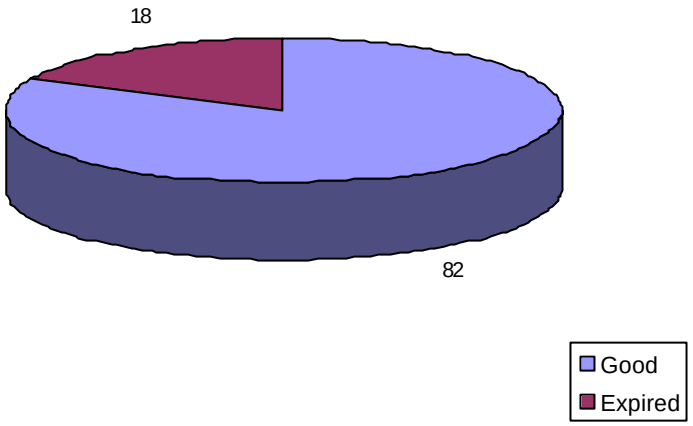
Type of malaria	No.of patients	Percentage
Vivax malaria	20	48%
Falciparum malaria	13	32%
Mixed malaria	08	20%

GRAPH SHOWING TYPES OF MALARIA WITH THROMBOCYTOPENIA

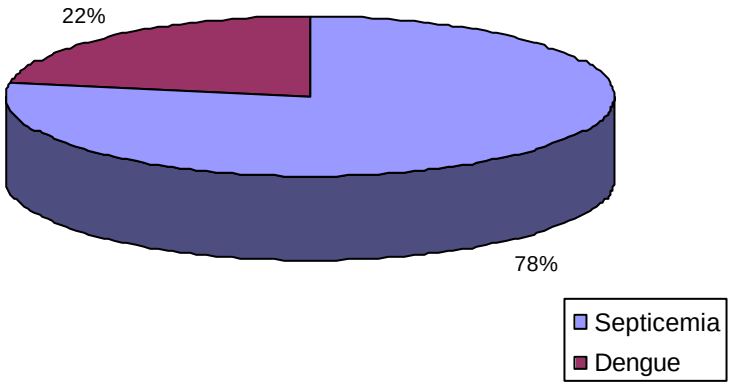


Out of 100 cases, 82 cases had good recovery and 18 cases expired during stay in hospital.

GRAPH SHOWING CLINICAL OUTCOME OF FEVER WITH THROMBOCYTOPENIA



GRAPH SHOWING DISEASES THAT CONTRIBUTED TO MORTALITY (n=18)



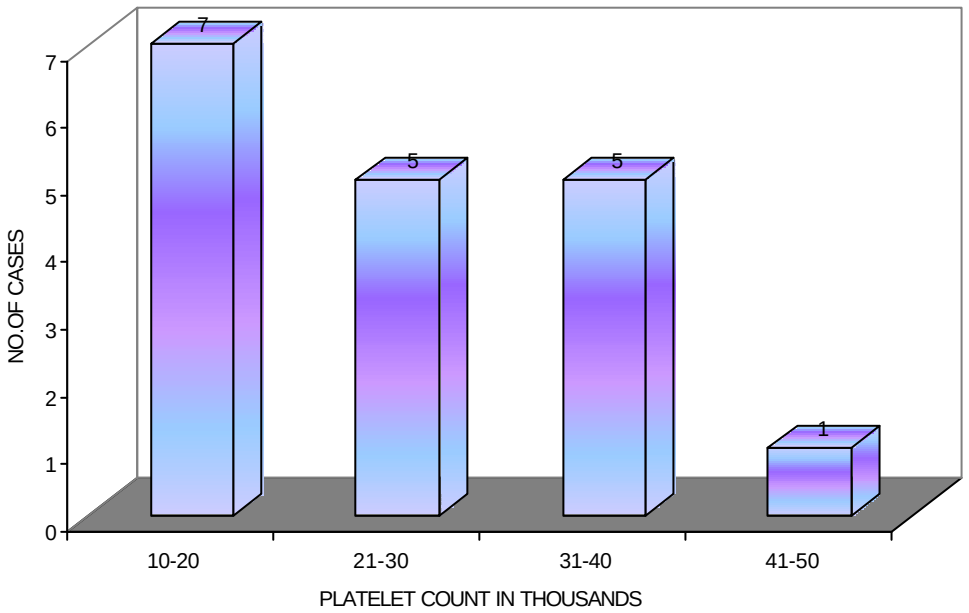
Out of 18 mortality cases, 14 cases were due to septicemia accounting for 78% of death was the common cause. 4 cases were due to dengue accounting for 22% of death.

TABLE – 5

CAUSES OF MORTLAITY IN OUR STUDY

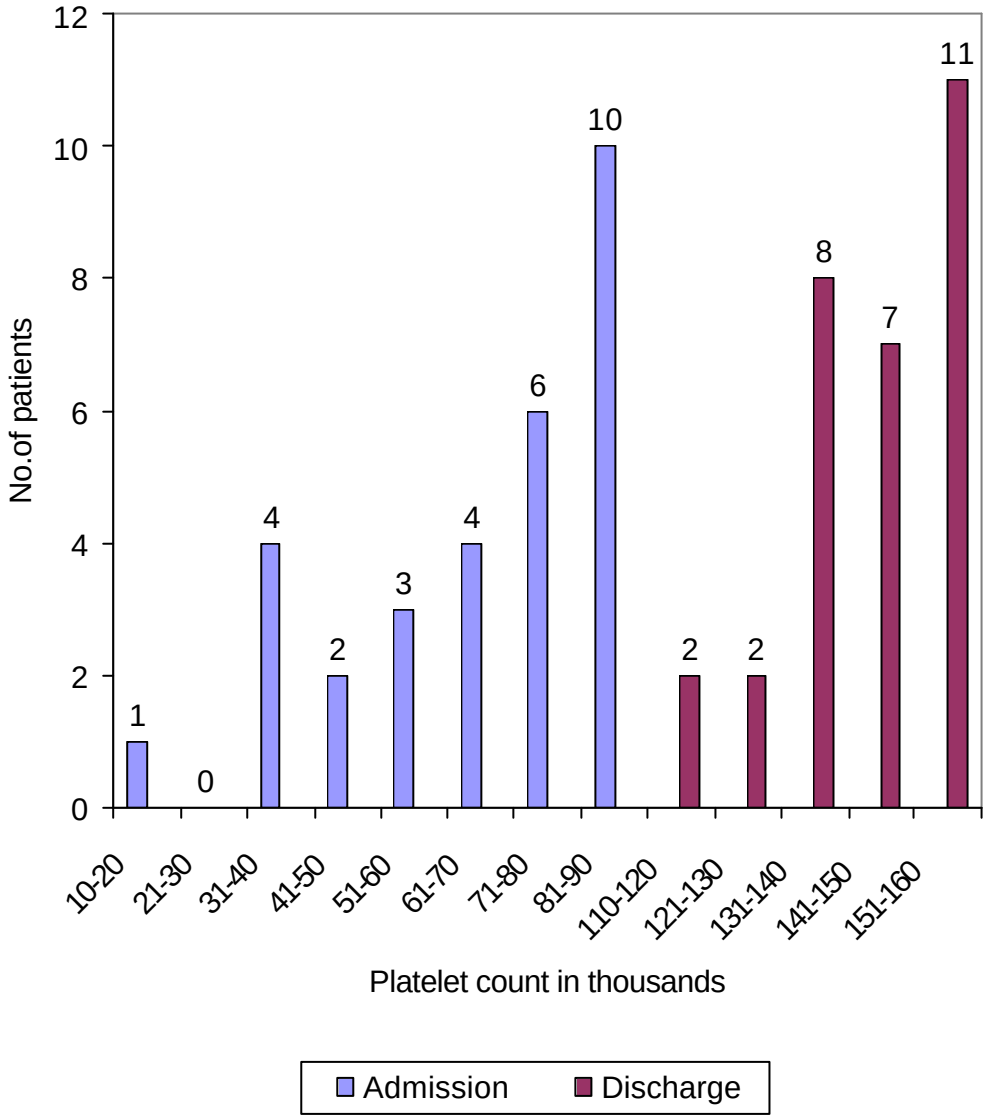
Disease category	No.of patients	Percentage
Septicemia	14	78%
Dengue	4	22%
Total	18	100

GRAPH SHOWING DISTRIBUTION OF PLATELET COUNT IN MORTALITY CASES



Out of 82 cases, who had good recovery – 30 cases 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.

GRAPH SHOWING COMPARISON BETWEEN PLATELET COUNT AT TIME OF ADMISSION AND DISCHARGE (n=30)



DISCUSSION

For a study of fever with thrombocytopenia, patients must satisfy the above mentioned criteria's, 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, megaloblastic anaemia, malaria, haematological malignancy with 16, 15, 13, 10, 4 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%) had 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 form 68% - 100%.

In our study infections (100%) was the established diagnosis as compared to other study in which along with infection (68%), haematological conditions (15%), was also documented.

This may be due to seasonal and regional variations. But infection was the commonest cause of fever with thrombocytopenia.

Among infections, malaria (41%) 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 (19%) was the third most common cause of fever with thrombocytopenia but malaria (9.2%) was the fifth common cause in other study.

In our study haematological condition did not present as fever with thrombocytopenia but in other study it accounted for 15%.

In both studies enteric fever was the second common cause, our study accounted for 24% while other study accounted for 14.7%.

Dengue / VHF infections constituted 14% in our study as compared to 13.8% in other study.

In our study 100% diagnosis was there but in other study 18.3% cases remained undiagnosed.

Disease category	Nair study		Present study	
	No.of cases	Percentage	No.of cases	Percentage
Septicemia	29	26.6	19	19
ENT	16	14.7	24	24
Dengue/VHF	15	13.8	14	14
Haematological conditions	17	15.6	0	0
Malaria	10	9.2	41	41
Others	20	18.3	2	2

In our study thrombocytopenic signs was present in 49% as compared to 41.3% in other study.

In our study Petichae / purpura (63%) was the commonest bleeding manifestations followed by spontaneous bleeding (37%). In other study spontaneous bleeding was the commonest bleeding manifestation (68%) followed by petichae / 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 65% as compared to 56.8% in other study. Platelet count in the range of 20-50000 was seen in 23% 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 it was very difficult to follow up because of cost, affordability, so only 30% of patients were followed up and it 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 as the commonest cause, among infections malaria was the common cause because of seasonal and regional variations. Definitive increase in platelet count was noted after the underlying cause was treated. Septicemia accounted for 78% of mortality in our study followed by dengue 22%.

Infectious diseases group formed the major portion of patients presenting with fever and thrombocytopenia lacking any specific signs. Malaria, enteric fever, dengue, 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.

Thrombocytopenic signs	**Nair study**		**Present study**	
	No.of cases	**Percentage**	**No.of cases**	**Percentage**
Present	45	41.3	49	49
Absent	64	58.7	51	51

Bleeding manifestations	**Nair study**		**Present study**	
	No.of cases	**Percentage**	**No.of cases**	**Percentage**
Petichae / Purpura	10	22.22	31	63
SPB	31	68.00	18	37
Others	4	9.88	0	0

Distribution of platelet count in thousands	**Nair study**		**Present study**	
	No.of cases	**Percentage**	**No.of cases**	**Percentage**
0 – 20	19	17.5	12	12
20- 50	28	25.7	23	23
50-100	62	56.8	65	65

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. But in our study, n=41, no patients had normal platelet count[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 patients with vivax malaria[17].

SUMMARY

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

1) The age range of the patient was 18-79 years, with male and female ratio being 58:42. These factors any way were not considered in our study.

2) The duration of hospitalization was 3-21 days, with an average period of hospitalization being 7 days.

3) A definitive diagnosis was made in all of them.

4) Among the diagnosed cases, malaria formed the largest group with 41%, vivax malaria formed the largest group followed by falciparum malaria and mixed malaria with 48%, 32%, 20% respectively.

5) Other cases diagnosed were Enteric fever 24 cases; Septicemia 19 cases Dengue 14, Leptospirosis 2 cases, constituting 24%, 19%, 14%, and 2% respectively.

6) Common range of platelet count at the time of admission was 61-80,000 in 36 cases, followed by 81-100 thousands in 24 cases , 21-40

thousands in 16 cases, 41-60 thousands in 12 cases and 0-20 thousands in 12 cases.

7) Clinical manifestation of thrombocytopenia was present only in 49 cases and in 51 cases it was not present.

8) Out of 49 cases which had thrombocytopenic manifestations petichae/ purpura was present in 31 cases accounting for 67% and spontaneous bleeding in 18 cases accounting for 33%.

9) In general, 82 cases had good recovery and 18 cases had mortality.

10) In 82 cases who had good recovery 30 were followed up and platelet count were near normal (1.3 – 1.5 lakhs) at the time of discharge.

11) In 18 mortality cases, 14 were due to septicemia – accounting for 78% and 4 were due to dengue accounting for 22% of the total cases.

12) Common range of platelet count in mortality cases was in range of 10-20 thousands in 7 cases, followed by 21-30 thousands in 5 cases, 31-40 thousands in 5 cases and 41-50 thousands in 1 cases.

13) During discharge and follow up of 30 patients in our study platelet count showed increasing trends and were near normal (around 1.5 lakhs/ cumm).

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, malaria 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 40,000 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.

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ANNEXURE - I

PROFORMA

"A CLINICAL AND LABORATORY PROFILE OF FEVER WITH THROMBOCYTOPENIA"

Name	:	I.P.NO.	:
Age	:	D.O.A.	:
Sex	:	D.O.D.	:
Occupation	:	Hospital	:
Address	:		

Duration of hospitalization (Days)

Interval between admission and discharge (Days)

1. PRESENTING COMPLAINS:

1. Fever
2. Malaise, Myalgia
3. Loss of weight
4. Cough
5. Expectoration
6. Dyspnea
7. Haemoptysis
8. Chest pain
9. Palpitation
10. Loss of appetite
11. Difficulty in swallowing
12. Diarrhea
13. Constipation
14. Pain abdomen

15. Distension of abdomen
16. Haemetemesis
17. Malena
18. Dysuria
19. Haematuria
20. Puffiness of the face
21. Headache
22. Giddiness
23. Nausea
24. Vomiting
25. Joint pains
26. Morning stiffness
27. Swelling of the joints
28. Bleeding gums
29. Drowsiness
30. Convulsions
31. Sore throat
32. Rash
33. Itching
34. Bleeding tendencies

II. HISTORY OF PRESENTING SYMPTOMS

1. Fever:
    - Duration (Days):
    - Grade — Mild/Moderate/Severe
    - Chills — Yes/No
    - Rigors — Yes/No
    - Diumal variation — Yes/No
    - Character — Continuous/lntermittent/Remittent/Relapsing
2. Malaise — Yes/No
    - Myalgia — Yes/No
3. Loss of weight — Yes/No
    - Duration (Days) :
4. Cough
    - Onset — Abrupt/Insidious
    - Duration (Days) : — Yes/No
    - Dry cough — Yes/No
5. Expectoration
    - Duration (Days) :
    - Type — Mucoid/Purulent/Blood tinged
    - Foulsmelling — Yes/No
    - Amount — Scanty/Copious
    - Postural drainage — Yes/No
6. Dyspnea — Yes/No
    - Onset — Abrupt/Insidious
    - Duration (Days) :
    - Grade

7. Haemoptysis — Yes/No
   - Duration (Days) :
   - Amount — Scanty/Copious
8. Chest Pain — Yes/No
   - Onset — Abrupt/Insidious
   - Duration (Days) :
   - Site :
   - Aggravating/relieving factors
9. Palpitation — Yes/No
10. Loss of appetite — Yes/No
   - Duration (Days):
11. Difficulty in Swallowing — Yes/No
   - Duration (Days):
   - For type of food — Solids/Liquids
   - Progress :
12. Nausea — Yes/No
13. Vomiting — Yes/No
   - Duration (Days):
   - Amount — Scanty/Copious
   - Type — Projectile/Non-projectile
   - Character — Watery/containing food particles/ Bilious/Blood tinged
14. Diarrhea — Yes/No
   - Duration (Days):
   - Amount — Scanty/copious
   - Character — Watery/Mucoid/Blood tinged
15. Constipation — Yes/No
   - Duration (Days):
16. Pain abdomen — Yes/No
   - Onset — Abrupt/Insidious
   - Duration
   - site
17. Distension of abdomen
   - Duration (Days) : Yes/No
18. Hemetemesis : Yes/No
19. Malena : Yes/No
20. Dysuria : Yes/No

- Difficulty in voiding : Yes/No
- Hesitency : Yes/No
- Oliguria : Yes/No
- Retention : Yes/No
- Incontinence : Yes/No

21. Haematuria : Yes/No
22. Puffiness efface : Yes/No
    - Onset : Abrupt/Insidious
    - Duration (Days): :
23. Headache : Yes/No
    - Onset : Abrupt/Insidious
    - Duration (Days) :
    - Localised/Diffuse :
    - Character : Throbing/Pricking/Dullaching/etc.
24. Giddiness : Yes/ No
25. Drowsiness : Yes/No
26. Convulsions : Yes/No
    - Duration(Days) :
    - Type : Generalized – Tonic/Clonic
      Partial seizures / etc.
27. Joint pains : Yes/No
    - Onset : Abrupt/ Insidious
    - Duration(Days) :
    - Site : Major/Minor - Joint
    - Early morning stiffness : Yes/No
    - Recurrent attacks : Yes/No
    - Symmetrical involvement : Yes/No
    - Restriction of Movement : Yes/No
28. Swelling of the joints : Yes/No
    - Duration (Days) :
    - Site :

- Deformities (If any) :

29. Bleeding gums : Yes/No
30. Sore throat : Yes/No
31. Rash : Yes/No
    - Onset : Abrupt/Insidious
    - Duration (Days) :
    - Site :
32. Itching : Yes/No
33. Bleeding tendencies : Yes/No
    - Site :

III. PAST HISTORY

1. History of prolonged fever
2. Rheumatic fever
3. Tuberculosis
4. Malaria
5. Bleeding tendencies
6. Autoimmune diseases
7. Malignancy
8. Contact with animals
9. Jaundice
10. Veneral diseases
11. Blood transfusion
12. Surgery
13. Hypertension
14. Diabetes

IV. DRUG HISTORY

Detailed history of drugs ingested in the past

V. IMMUNIZATION HISTORY

VI. TRAVEL HISTORY

VII. FAMILY HISTORY :

VIII. PERSONAL HISTORY

1. Appetite :
2. Diet : Veg/Non-Veg
3. Sleep : Disturbed/Normal
4. Bladder :
5. Bowel :
6. Habits

Smoking : Duration :
Frequency :
Amount/Day :

Alcohol consumption : Duration :
Frequency :
Amount/day :

IX. OBSTETRIC AND MENESTRUAL HISTORY :

X. GENERAL PHYSICAL EXAMINATION :

1. Built :
   Weight :
   Height
   Nourishment :
   Scalp hair :

   Skin :
   Conjunctiva :
   Sclera :
   Oral cavity :
   Upper respiratory tract :

2. Neck

   Lymph node enlargement Yes/No
   Thyroid enlargement Yes/No
   Parotid enlargement Yes/No

3. Pallor : Yes/No
   - Grade : Mild/Moderate/Severe
4. Icterus :
5. Cyanosis :
6. Clubbing :

- Grade :

7 Lymphadenopathy :

- Duration (Days)
- Localized/Generalized
- Consistency

8. Odema

Duration (Days)

Localized/Genealized

9. Vital signs

Temperature

Pulse/Minute

Peripheral pulses

JVP

B.P (in mm of Hg)

Respiratory rate

XI. SYSTEMIC EXAMINATION

1. RESPIRATORY SYSTEM EXAMINATION

INSPECTION:

a. Shape of the Chest

Barrel shaped chest etc.,

Hollowing, bulging, flattening or retraction

Sub-costal angle

Shoulders and spine

Spino-scapular distance

b. Respiratory movements

Respiratory Rate :

Rhythm :

Character :

Equality :

Inter-costal retraction :

c. Mediastinum

Trail's sign :

Apical impulse :

d. Miscellaneous

Scars/Sinuses :

Pulsations :

Dilated veins :

PALPATION

a. Trachea :

b. Apex beat :

c. Tactile vocal fremitus :

d Miscellaneous : tenderness/Palpable rales/Ronchi/rub

PERCUSSION

		Right	**Left**
Supraclavicular	:		
Infraclavicular	:		
Mammary	:		
Axillary	:		
Infra-axillary	:		
Suprascapular	:		
Scapular	:		
Infrascapular	:		

AUSCULTATION

a. Breath sounds Normal/Diminished

b. Type Vesicular/Broncho-vesicular/
Vesicular with prolonged expiration

c. Area of bronchial breath sounds:

d. Adventitious sounds Ronchi/Crepitating/Rub

e. Miscellaneous

- Bronchophony :
- Aegophony :
- Whispering pectoriliquy :
- Succusion splash :
- Coin test :
- Post-tussive suction :
- Post tussive rales :

2. <u>CARDIO-VASCULAR EXAMINATION</u>

INSPECTION:

a. Precordium :

b. Apex impulse :

c. Other pulsations :

d. Dilated veins :

e. Scars, sinuses :

PALPATION

a. Apex beat :

b. Parastemal heave :

c. Thrills :

d. Other pulsations :

e. Percussion :

AUSCULTATION:

a. Heart sounds :

b. Murmurs :

c. Other sounds : Pericardial rub/Opening snap/Ejection clicks/etc.

3. <u>GASTROINTESTINAL SYSTEM EXAMINATION</u>

INSPECTION

a. Shape of the abdomen :

b. Umbilicus :

c. Skin over the abdomen :

d. Scars/Sinuses :

PALPATION

a. Tenderness/Guarding/Rigidity:

b. Liver :

- Palpable : Yes/No
- Size : Centimeters below the right costal margin in mid clavicular line
- Consistency : Soft/Firm/Hard

c. Spleen

Palpable : Yes/No

Size : Centimeters below the right costal margin in mid clavicular line

- Consistency : Soft/Firm/Hard

d. Any other palpable mass :
(Size/Surface/Consistency/)

e. Fluid thrill :

PERCUSSION :

a. Shifting dullness : Present/Absent

b. Dullness over any lump (If palpable)

AUSCULTATION

a. Bowel sounds :

b. Rub/Bruit :

EXTERNAL GENITALIS :

<u>CENTRAL NERVOSU SYSTEM EXAMINATION</u> :

a. Higher mental function :

b. Cranial nerves :

c. Motor system :

d. Sensory system :

e. Co-ordination :

f. Signs of meningeal irritation : Neck stiffness/Kernig's sign

g. S.L.R and Laseuge's sign :

h. Skull and spine :

i. Gait :

EXAMINATION OF THE JOINTS

GENERAL OBSERVATIONS

a. Gait :

b Posture :

INSPECTION

a. Site : Major/Minor joints

b. Alterations in the shape : Yes/No

c. Swelling of the Joint : Yes/No

d. Deformities (If any) :

e. Symmetrical involvement : Yes/no

PALPATION

a. Tenderness : Yes/No

b. Joint Crepitus : Yes/No

c. Limitation of movements : Yes/no

d. Fractures (if any)

EXTRA-ARTICULAR FEATURES

a. Sub cutaneous nodules : Rheumatoid nodules/Nodules in SLE/ gouty tophi

b. Cutaneous vasculitic lesions :

XII. INVESTIGATIONS

A. Routine Investigations

1. Routine Haematological examination

Hb gm % :

PCV (Haematocrit) (%) :

RBC count (/cumm) :

Total count (/cumm) :

Differential count :

Platelet count :

Erythrocyte Sedimentation Rate :

Peripheral blood smear :

2. Urine analysis

Albumin :

Sugar :

Microscopy :

3. FBS/ RBS :

4. Blood chemistry

Blood urea :

Serum creatinine :

5. Liver function test

Serum total bilirubin :

Serum total proteins :

Serum albumin :

Serum globulin :

Albumin/Globulin ratio :

S.G.O.T. :

S.G.P.T. :

Serum alkaline phosphatase :

6. Chest x-ray :

7. Ultrasound abdomen :

B. Specific investigations :

- Widal test
- Peripheral smear for malarial parasites
- Rapid spot test for malaria
- IgM ELISA for dengue

- IgM ELISA for Leptospiral
- HIV 1 and 2
- Blood culture and sensitivity
- Urine culture and sensitivity

XIII. TREATMENT :

XIV. FOLLOW UP :

XV. SUMMARY AND CONCLUSION

MASTER CHART

Sl.No.	Name	DATA					SYMPTOMS									P/H		GPE		SYSTEM EXAMINATION					INVESTIGATIONS																												Diagnosis	Treatment	Outcome	PL at the time of discharge(cells/ cumm) in lakhs
																				GIT																																						
		Age (years)	Sex	I.P. NO.	Hospital	Duration of stay in hospital (days)	Fever	Chills & rigors	Headache	Jaundice	Cough & Breath-lessness	Altered sensorium	Bleeding	Others	Any treatment history	Temperature	Pallor, Icterus, Clubbing, Cyanosis, Lymphadenopathy, Oedema	Bleeding manifestations	Spleno-megaly	Hepato-megaly	RS	CVS	CNS	Hb (gm%)	TLC (cells/ cumm)	PL at the time of admission (cells/ cumm)	ESR (mm/hr)	Urine routine	Peripheral smear for MP	Rapid spot test (for malaria)	RBS (mg%)	B. urea (mg%)	S.creatinine (mg%)	S.bilirubin (mg%)	SGOT/SGPT (U/L)	S.albumin	BT/CT	Widal	IgM ELISA Dengu	IgM Leptospira	Bone marrow	Blood culture	HIV				
1	HEMA	23	F	480107	BH	3	+	+	+	+	-	+	-	NS	-	+	P+,I+	-	+	+	NS	NS	NS	8.8	4500	78000	18	N	-	PV	133	33	0.9	4.8	296	5.3	N	+	-	-	-	-	VM	Clokit/mds	G	ND
2	SAVITHRAMMA	27	F	470177	BH	5	+	+	+	+	-	+	-	NS	-	+	P+,I+	-	+	+	NS	NS	NS	9.2	6200	80000	22	N	-	PV/PF	128	48	1.2	5.2	323	5.8	N	+	-	-	-	-	VM/FM	FALCIGO/mds	G	ND
3	SIVAKUMAR	25	M	480324	BH	4	+	-	+	+	-	+	-	NS	-	+	P+,I+	-	+	+	NS	NS	NS	6.2	3800	68000	23	N	-	PF	121	33	0.8	2.3	186	5.2	N	+	-	-	-	-	FM	FALCIGO/mds	G	ND
4	ABY JOSEPH	19	M	4802325	BH	9	+	+	-	-	-	+	-	NS	-	+	P	-	-	-	NS	NS	NS	8.3	4200	90000	20	N	-	PV	128	34	0.9	2.8	196	5.3	N	-	-	-	-	-	VM/FM	Clokit/mds	G	1.5
5	SAKAMMA	48	F	480331	BH	3	+	-	+	+	-	-	-	M/LS	-	+	PI	-	+	+	NS	NS	NS	9.3	6300	89000	19	N	-	-	121	36	0.7	2.8	213	5.4	N	+	-	-	-	-	ENT	OQ	G	ND
6	AHOOPAJLAUBI	20	F	480326	BH	5	+	+	-	-	-	+	+	NS	-	+	-	Pu	-	-	NS	NS	NS	10.6	7200	55000	14	N	-	PV	141	38	0.6	2.9	215	5.5	N	+	-	-	-	-	VM/FM	Clokit/mds	G	ND
7	RAVIRAJ	32	M	480760	BH	8	+	+	+	-	-	+	+	NS	-	+	P	Pu	-	-	NS	NS	NS	9.8	5800	44000	12	N	-	PF	132	33	1.1	3.1	216	4.8	N	+	-	-	-	-	FM	FALCIGO/mds	G	1.3
8	T.SIDDAIAH	53	M	471812	BH	4	+	+	+	-	-	+	-	LS	-	+	P	-	-	-	NS	NS	NS	10.3	4200	69000	11	N	-	-	148	34	0.8	0.9	38	5.6	N	+	-	-	-	-	ENT	OQ	G	ND
9	TIPPESWAMY	73	M	471856	BH	4	+	-	+	+	-	-	-	NS	-	+	PI	-	+	+	NS	NS	NS	8.9	3100	72000	10	N	+	PV	152	28	0.6	3.2	218	5.7	N	-	-	-	-	-	VM/FM	Clokit/mds	G	ND
10	MALLIKARJUNA	22	M	471879	BH	4	+	+	-	-	-	+	-	NS	-	+	-	-	-	-	NS	NS	NS	7.8	5300	75000	13	N	-	-	130	29	0.3	0.8	40	5.3	N	+	-	-	-	-	ENT	XONE	G	ND
11	SANTOSH	20	M	471894	BH	21	+	+	+	-	-	+	-	NS	-	+	P	-	+	-	NS	NS	NS	8.6	6100	78000	14	N	+	PF	140	27	0.4	5.6	326	5.2	N	-	-	-	-	-	FM	FALCIGO/mds	G	1.45
12	MANJUNATH	27	M	481127	BH	8	+	-	+	-	-	+	-	LS	-	+	PI	-	+	+	NS	NS	NS	9.8	4200	90000	18	N	-	-	120	26	0.8	5.8	348	5.1	N	+	-	-	-	-	ENT	XONE	G	ND
13	KALLESH	24	M	481122	BH	3	+	+	-	+	-	+	-	PA	-	+	I	-	+	+	NS	NS	NS	10.2	3800	96000	15	N	-	-	111	33	0.9	5.8	352	5.8	N	+	-	-	-	-	ENT	XONE	G	ND
14	GAYTRAMMA	35	F	481136	BH	4	+	+	+	+	-	+	-	NS	-	+	PI	-	+	+	NS	NS	NS	9.7	4300	89000	18	N	+	PV	132	38	0.8	5.2	226	5.2	N	+	-	-	-	-	VM	Clokit/mds	G	ND
15	GANESH	25	M	481168	BH	5	+	+	+	+	-	-	-	NS	-	+	-	-	-	-	NS	NS	NS	9.6	4800	88000	22	N	+	PV	128	36	0.7	5.3	278	4.8	N	+	-	-	-	-	VM	Clokit/mds	G	ND
16	SALAM	22	M	481607	BH	4	+	+	-	+	-	+	-	LS/PA	-	+	PI	-	-	-	NS	NS	NS	9.4	6000	68000	18	N	-	-	130	38	0.9	0.9	56	4.8	N	+	-	-	-	-	ENT	XONE	G	ND
17	SALOHEWS BABY	22	F	481627	BH	18	+	-	+	+	-	+	-	LS	-	+	PI	-	+	+	NS	NS	NS	9.8	8000	90000	16	N	-	-	118	42	0.6	0.8	48	5.2	N	+	-	-	-	-	ENT	XONE	G	1.4
18	RASIDA BEI	26	F	481656	BH	3	+	+	+	-	-	+	-	PA	-	+	-	-	-	-	NS	NS	NS	11.5	8800	78000	14	N	-	-	132	54	0.8	1.3	98	5.4	N	+	-	-	-	-	ENT	XONE	G	ND
19	PUTTARAJ	35	M	481692	BH	7	+	-	+	-	-	+	-	NS	-	+	-	-	-	-	NS	NS	NS	12.3	8100	81000	22	N	+	PV	126	28	0.7	3.8	168	5.6	N	-	-	-	-	-	VM	Clokit/mds	G	1.6
20	ZURIUIR ALUISH	25	M	481827	BH	3	+	+	+	-	-	+	-	NS	-	+	-	-	-	-	NS	NS	NS	13.6	7800	84000	20	N	+	PV	124	32	0.8	3.2	236	5.3	N	-	-	-	-	-	VM	Clokit/mds	G	ND
21	VEERAPPA	72	M	409406	BH	6	+	+	+	+	-	+	+	M/BP	-	+	PI	Pe	+	+	NS	NS	NS	9.8	5200	52000	18	N	-	-	130	40	0.3	3.8	178	5.2	N	-	+	-	-	-	DENGUE	SY/PT	G	ND
22	MURTHY	18	M	429442	BH	6	+	+	+	+	-	+	+	NS	-	+	PI	Pu	+	-	NS	NS	NS	9.6	5400	68000	16	N	+	PV	138	38	0.7	2.4	186	5.4	N	-	-	-	-	-	VM	Clokit/mds	G	ND
23	GAJABAI	25	F	429495	BH	9	+	+	+	+	-	+	+	NS	-	+	PI	Pu	-	+	NS	NS	NS	8.8	5300	40000	20	N	-	PV	134	42	0.6	2.6	136	5.6	N	+	-	-	-	-	VM	Clokit/mds	G	1.32
24	MAHARUDRAPPA	26	M	429546	BH	7	+	+	+	+	-	+	+	M/BP	-	+	PI	Pe/Pu	+	+	NS	NS	NS	8.8	5200	33000	18	N	-	-	136	26	0.8	4.4	256	5.4	N	-	+	-	-	-	DENGUE	SY/PT	G	1.2
25	RAJESHWARI	54	F	429559	BH	7	+	+	-	+	-	+	-	LS	-	+	PI	-	-	-	NS	NS	NS	9.2	5100	78000	16	N	-	-	122	33	0.9	3.6	248	5.2	N	+	-	-	-	-	ENT	XONE	G	1.4

		DATA					SYMPTOMS								P/H	GPE			SYSTEM EXAMINATION					INVESTIGATIONS																							
																			GIT																												
Sl.No.	Name	Age (years)	Sex	I.P. NO.	Hospital	Duration of stay in hospital (days)	Fever	Chills & rigors	Headache	Jaundice	Cough & Breath-lessness	Altered sensorium	Bleeding	Others	Any treatment history	Temperature	Pallor, Icterus, Clubbing, Cyanosis, Lymphadenopathy, Oedema	Bleeding manifestations	Spleno-megaly	Hepato-megaly	RS	CVS	CNS	Hb (gm%)	TLC (cells/ cumm)	PL at the time of admission (cells/ cumm)	ESR (mm/hr)	Urine routine	Peripheral smear for MP	Rapid spot test (for malaria)	RBS (mg%)	B. urea (mg%)	S.creatinine (mg%)	S.bilirubin (mg%)	SGOT/SGPT (U/L)	S.albumin	BT/CT	Widal	IgM ELISA Dengu	IgM Leptospira	Bone marrow	Blood culture	HIV	Diagnosis	Treatment	Outcome	PL at the time of discharge(cells/ cumm) in lakhs
26	PADMAVATHI	21	F	429756	BH	8	+	+	-	-	-	+	+	HOA	-	+	P	SPB	+	+	NS	NS	NS	9.8	18000	41000	18	N	-	-	130	38	0.8	5.4	224	5	N	-	-	-	-	P	-	Septicemia	ANTI/SU	E	ND
27	ANANDGOWDA	24	M	429764	BH	6	+	+	-	+	-	+	+	NS	-	+	PI	SPB	+	+	NS	NS	NS	9.6	16000	38000	22	AB	-	-	130	64	1.6	5.8	336	5.1	N	+	-	-	-	P	-	Septicemia	ANTI/SU	E	ND
28	BASAVARAJAPPA	55	M	429839	BH	8	+	+	+	-	-	+	+	V/M	-	+	-	Pe	-	-	NS	NS	NS	9.8	6000	62000	20	N	+	PF	128	66	1.8	4.8	228	5.2	N	-	-	-	-	-	-	FM	FALCIGO/mds	G	1.4
29	ANEVARMULLA	60	M	429831	BH	8	+	-	+	-	-	+	+	NS	-	+	-	Pu	+	-	NS	NS	NS	9.2	5800	58000	18	N	+	PF	130	58	1.4	3.9	326	4.8	N	-	-	-	-	-	-	FM	FALCIGO/mds	G	1.36
30	MANJULA	24	M	429872	BH	4	+	-	-	-	-	+	-	NS	-	+	-	-	-	-	NS	NS	NS	8.8	5200	72000	18	N	-	-	133	42	0.8	0.8	36	4.8	N	+	-	-	-	-	-	ENT	XONE	G	ND
31	SRIDAR	72	M	429913	BH	4	+	-	+	+	-	+	-	NS	-	+	I	-	-	+	NS	NS	NS	9	6000	78000	16	N	+	PV	148	36	0.6	2.3	136	4.8	N	-	-	-	-	-	-	VM	Clokit/mds	G	ND
32	RAVINDRA	41	M	429946	BH	5	+	+	+	+	-	+	+	DM/N	-	+	PI	SPB	-	+	NS	NS	NS	6.8	16000	23000	22	N	-	-	168	64	2.4	5.6	346	4.2	AB	-	-	-	-	E	-	Septicemia	ANTI/SU	E	ND
33	GOWRAMMA	35	F	429920	BH	5	+	+	-	+	-	+	+	HOA	-	+	PI	SPB	+	-	NS	NS	NS	7.4	14000	13000	28	N	-	-	152	78	1.8	4.8	276	4.2	AB	-	-	-	-	E	-	Septicemia	ANTI/SU	E	ND
34	SRIKANTAMMA	32	F	429986	BH	10	+	+	+	-	-	+	-	LS	-	+	-	-	+	+	NS	NS	NS	8.8	6000	61000	20	N	+	-	140	36	0.8	0.9	36	4.8	N	+	-	-	-	-	-	ENT	XONE	G	1.4
35	KALSAR	23	F	429986	BH	10	+	-	+	+	-	-	-	LS	-	+	I	-	-	-	NS	NS	NS	9.2	8000	78000	18	N	+	-	139	38	0.7	0.8	42	5	N	+	-	-	-	-	-	ENT	XONE	G	1.5
36	RAVIWBRA	19	F	472701	BH	10	+	-	+	-	-	-	-	LS/PA	-	+	PI	-	+	+	Cryt	NS	NS	8.6	11000	81000	16	N	+	-	128	40	0.8	0.6	54	5.2	N	+	-	-	-	-	-	ENT	XONE	G	1.48
37	ANITHA	18	F	472475	BH	3	+	+	+	+	-	+	+	M/BP	-	+	PI	Pu	-	+	NS	NS	NS	8.8	3400	61000	14	N	-	-	130	52	0.9	2.8	198	-	-	-	+	-	-	-	-	DENGUE	SY/PT	G	ND
38	SHAUDDIN	35	M	472095	BH	7	+	+	+	-	+	+	+	NS	-	+	-	Pu	-	-	C	NS	NS	8.9	6000	48000	18	N	+	Pv	140	48	0.8	0.8	36	-	-	-	-	-	-	-	-	VM	Clokit/mds	G	1.52
39	NAGARAJ	23	M	472036	BH	4	+	+	-	-	-	+	+	NS	-	+	-	Pu	-	-	NS	NS	NS	9.1	5200	66000	20	N	-	PF	138	44	0.7	0.9	40	-	-	-	-	-	-	-	-	FM	FALCIGO/mds	G	ND
40	ANNAPPA	25	M	472061	BH	5	+	+	+	-	+	-	-	M/BP	-	+	-	-	-	-	NS	NS	NS	9.4	3800	90000	22	N	-	-	122	48	0.9	0.7	38	-	-	-	+	-	-	-	-	DENGUE	SY	G	ND
41	GOVINDAPPA	35	M	472102	BH	6	+	+	+	+	-	-	-	NS	-	+	PI	-	+	-	C	NS	NS	9.6	4800	70000	24	N	-	PF	133	50	1.8	3.2	186	-	-	-	-	-	-	-	-	FM	FALCIGO/mds	G	ND
42	THIMMA REDDY	56	M	472120	BH	7	+	+	-	-	-	+	-	NS	-	+	-	-	-	-	NS	NS	NS	8.6	4400	88000	26	N	-	-	134	48	0.9	0.6	36	-	-	+	-	+	-	-	-	Leptospi	PENICILLIN	G	ND
43	SHAKEERABEE	48	F	472172	BH	8	+	+	-	+	+	+	+	NS	-	+	I	PePu	-	+	C	NS	NS	9.4	3800	40000	28	N	-	-	136	46	0.8	4.2	326	-	-	+	-	-	-	-	-	DENGUE	SY	G	1.36
44	VEERAMMA	18	F	472197	BH	9	+	+	-	-	-	-	-	PA	-	+	-	-	-	-	NS	NS	NS	8.5	6000	91000	30	N	-	-	138	44	0.6	0.8	48	-	-	-	-	-	-	P	-	ENT	XONE	G	1.56
45	SURENDRA SINGH	65	M	477348	BH	3	+	+	+	-	+	-	-	NS	-	+	-	-	-	-	NS	NS	NS	8.6	3600	98000	12	N	-	-	140	48	1	0.8	32	-	-	-	+	-	-	-	-	DENGUE	SY	G	ND
46	PRABU	23	M	477398	BH	3	+	+	+	+	-	+	-	M/BP	-	+	I	-	+	-	C	NS	NS	8.9	7200	78000	18	N	-	Pv	142	36	0.8	4.4	226	-	-	-	-	-	-	-	-	VM	I10KIT	G	ND
47	MYLAPPA	50	M	477406	BH	6	+	+	-	+	-	+	-	NS	-	+	I	-	-	+	NS	NS	NS	9.4	6800	87000	26	N	-	PF	144	78	2.8	3.8	156	-	-	-	-	-	-	-	-	FM	FALCIGO/mds	G	ND
48	GIRISH	25	M	477441	BH	7	+	+	+	+	+	-	+	NS	-	+	I	PePu	-	-	C	NS	NS	9.3	5200	23000	14	N	+	Pv	148	36	0.8	3.6	186	-	-	-	-	-	-	-	-	VM	FALCIGO/mds	G	1.2
49	RAZKH SAB	43	M	477445	BH	3	+	-	+	-	-	-	+	NS	-	+	-	SPB	-	-	NS	NS	NS	9.2	18000	18000	16	N	-	-	138	104	4.8	0.8	38	-	-	-	-	-	-	P	-	Septicemia	ANTI/SU	E	ND
50	RAMAPPA	45	M	477569	BH	5	+	-	+	-	+	-	+	LS	-	+	-	SPB	-	-	C	NS	NS	9.3	24000	21000	22	N	-	-	120	133	5.4	0.9	42	-	AB	-	-	-	-	E	-	Septicemia	ANTI/SU	E	ND

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		Age (years)	Sex	I.P. NO.	Hospital	Duration of stay in hospital (days)	Fever	Chills & rigors	Headache	Jaundice	Cough & Breath-lessness	Altered sensorium	Bleeding	Others	Any treatment history	Temperature	Pallor, Icterus, Clubbing, Cyanosis, Lymphadenopathy, Oedema	Bleeding manifestations	Spleno-megaly	Hepato-megaly	RS	CVS	CNS	Hb (gm%)	TLC (cells/ cumm)	PL at the time of admission (cells/ cumm)	ESR (mm/hr)	Urine routine	Peripheral smear for MP	Rapid spot test (for malaria)	RBS (mg%)	B. urea (mg%)	S.creatinine (mg%)	S.bilirubin (mg%)	SGOT/SGPT (U/L)	S.albumin	BT/CT	Widal	IgM ELISA Dengu	IgM Leptospira	Bone marrow	Blood culture	HIV				
51	RUDRAMUNI	35	M	477593	BH	6	+	-	+	-	-	-	-	LS	-	+	-	-	-	-	NS	NS	NS	9.4	16000	78000	10	N	-	-	121	38	0.9	0.6	34	-	-	-	-	-	P	-	Septicemia	ANTI/SU	G	ND
52	VEERANNA	70	M	477602	BH	7	+	-	-	-	-	-	-	LS	-	+	-	-	-	-	NS	NS	NS	9.2	14000	31000	8	N	-	-	131	162	5.2	0.7	38	-	AB	-	-	-	P	-	Septicemia	ANTI/SU	E	ND
53	SOMAPPA	28	M	477637	BH	8	+	-	+	-	+	-	-	LS	-	+	-	-	-	-	C	NS	NS	9	6000	86000	10	N	-	Pv	128	38	0.9	0.6	34	-	-	-	-	-	-	-	VM	ANTI/SU	G	ND
54	SOUMYA	18	F	477658	BH	9	+	-	+	-	-	-	-	NS	-	+	-	-	-	-	NS	NS	NS	8.8	5800	72000	14	N	-	Pv	126	44	0.7	0.5	36	-	-	-	-	-	-	-	VM	ANTI/SU	G	1.48
55	LAKSHMAMMA	65	F	477745	BH	3	+	-	+	+	-	-	+	NS	-	+	I	SPB	-	+	NS	NS	NS	8.7	18000	18000	16	N	-	-	133	148	5.4	3.8	138	-	AB	-	-	-	E	-	Septicemia	ANTI/SU	E	ND
56	NINGAPPA	32	M	477776	BH	5	+	-	+	-	-	-	-	LS	-	+	-	-	-	-	NS	NS	NS	8.6	5200	71000	12	N	-	-	138	42	0.6	0.8	36	-	-	-	-	-	-	-	ENT	XONE	G	ND
57	YASHODAMMA	61	F	477781	BH	8	+	-	+	-	+	-	-	NS	-	+	-	-	-	-	C	NS	NS	8.5	5800	88000	10	N	-	-	126	44	0.8	0.9	40	-	-	-	-	-	-	-	ENT	XONE	G	1.52
58	SHARADAMMA	50	F	482255	BH	7	+	-	+	-	-	-	-	PA	-	+	-	-	-	-	NS	NS	NS	8.4	5600	81000	10	N	-	-	133	36	0.9	0.6	38	-	-	-	-	-	-	-	ENT	XONE	G	1.47
59	SHANTHAMMA	44	F	482256	BH	9	+	+	+	+	-	-	+	PA	-	+	I	SPB	+	-	NS	NS	NS	8.3	14800	17000	14	N	-	-	134	90	1	5.4	356	-	-	-	-	-	E	-	Septicemia	ANTI/SU	G	1.48
60	SHANTHA	28	F	482337	BH	8	+	+	-	-	-	+	-	LS	-	+	-	-	-	-	NS	NS	NS	8.4	5200	71000	16	N	-	-	136	48	0.8	0.8	38	-	-	+	-	-	P	-	ENT	XONE	G	1.4
61	SHAKUNTHALA	35	F	482451	BH	4	+	+	-	-	+	+	-	NS	-	+	-	-	-	-	C	NS	NS	9.1	6000	68000	18	N	+	-	138	52	0.7	0.6	36	-	-	+	-	-	-	-	VM	Clokit/mds	G	ND
62	BHEVANAMMA	35	F	482469	BH	5	+	+	+	-	-	+	-	NS	-	+	-	-	-	-	NS	NS	NS	9.2	6100	28000	20	N	+	-	118	136	5.6	0.4	42	-	AB	-	-	-	-	-	VM	Clokit/mds	E	ND
63	RASUL BM	44	M	482254	BH	7	+	+	+	-	-	+	-	NS	-	+	-	-	-	-	NS	NS	NS	9.6	5800	71000	22	N	+	PF	90	98	2.3	0.4	44	-	-	-	-	-	-	-	FM	FALCIGO/mds	G	ND
64	RAGAVENDRA	36	M	482777	BH	8	+	+	+	-	+	+	+	NS	-	+	-	Pu	-	-	C	NS	NS	9.8	4400	40000	20	N	-	-	160	56	0.8	0.9	41	-	-	-	-	+	-	-	Leptospi	PENICILLIN	G	1.3
65	RAVIKUMAR	20	M	482724	BH	9	+	+	+	-	-	+	-	BP/M	-	+	-	-	-	-	NS	NS	NS	9.9	3600	59000	18	N	-	-	148	40	0.7	0.8	36	-	-	-	+	-	-	-	DENGUE	SY	G	1.38
66	BASAVARAJ	20	M	483020	BH	3	+	+	+	-	+	+	-	NS	-	+	-	-	-	-	C	NS	NS	9.6	3800	81000	16	N	+	Pv	152	33	0.8	0.9	38	-	-	-	-	-	-	-	VM	Clokit/mds	G	ND
67	VINODA	52	F	483630	BH	5	+	+	+	-	-	+	+	LS	-	+	-	SPB	-	-	NS	NS	NS	8.8	18900	18000	15	N	-	-	132	148	4.8	0.8	40	-	AB	-	-	-	P	-	Septicemia	ANTI/SU	E	ND
68	KARISIDDAPPA	79	M	483630	BH	6	+	+	+	+	-	+	+	DM/LS	-	+	PI	Pu	+	-	NS	NS	NS	9.8	14000	28000	18	N	-	-	133	138	2.9	4.8	326	45.3	AB	-	-	-	E	-	Septicemia	ANTI/SU	E	ND
69	YOGARAJ	30	M	483770	BH	8	+	+	+	+	-	+	+	NS	-	+	PI	Pu	+	+	NS	NS	NS	8.1	4800	56000	16	N	+	PF	126	25	0.7	5.2	386	5.1	N	-	-	-	-	-	FM	FALCIGO/mds	G	1.28
70	SUSHEELAMMA	55	F	483802	BH	7	+	-	+	+	-	+	-	NS	-	+	PI	-	+	+	NS	NS	NS	9.2	14200	81000	20	N	-	Pv	128	24	0.6	3.6	364	5.2	N	-	-	-	-	-	VM	Clokit/mds	G	1.49
71	BASAPPA	52	M	484077	BH	5	+	+	-	+	-	-	-	PA	-	+	PI	-	+	+	NS	NS	NS	9.2	6400	78000	14	N	-	-	124	26	0.8	2.8	286	5.4	N	+	-	-	-	-	ENT	XONE	G	ND
72	K.G. SWAMY	30	M	484018	BH	5	+	+	-	+	-	+	-	PA	-	+	PI	-	+	+	NS	NS	NS	8.4	5200	86000	18	N	-	-	120	30	0.6	4.7	278	5.3	N	+	-	-	-	-	ENT	XONE	G	ND
73	RANGA REDDY	18	M	484045	BH	4	+	+	-	-	-	-	+	PA	-	+	P	SPB	-	-	NS	NS	NS	8.6	16600	18000	20	N	-	-	132	132	2.7	6.9	236	5.1	AB	-	-	-	P	-	Septicemia	ANTI/SU	E	ND
74	SAVITHRAMMA	28	F	491204	BH	6	+	+	+	-	-	-	-	NS	-	+	P	-	-	-	NS	NS	NS	9.6	5200	78000	16	N	-	Pv	131	38	0.8	1	48	5.3	N	-	-	-	-	-	VM	Clokit/mds	G	1.48
75	CHANDRAHAS	24	M	491003	BH	7	+	-	+	+	-	+	-	NS	-	+	I	-	+	-	NS	NS	NS	7.8	5200	91000	16	N	-	-	136	34	0.8	3.6	148	5.4	N	+	-	-	E	-	ENT	XONE	G	1.5

Sl.No.	Name	DATA					SYMPTOMS								P/H	GPE			SYSTEM EXAMINATION					INVESTIGATIONS																										Diagnosis	Treatment	Outcome	PL at the time of discharge(cells/ cumm) in lakhs
																			GIT																																					
		Age (years)	Sex	I.P. NO.	Hospital	Duration of stay in hospital (days)	Fever	Chills & rigors	Headache	Jaundice	Cough & Breath-lessness	Altered sensorium	Bleeding	Others	Any treatment history	Temperature	Pallor, Icterus, Clubbing, Cyanosis, Lymphadenopathy, Oedema	Bleeding manifestations	Spleno-megaly	Hepato-megaly	RS	CVS	CNS	Hb (gm%)	TLC (cells/ cumm)	PL at the time of admission (cells/ cumm)	ESR (mm/hr)	Urine routine	Peripheral smear for MP	Rapid spot test (for malaria)	RBS (mg%)	B. urea (mg%)	S.creatinine (mg%)	S.bilirubin (mg%)	SGOT/SGPT (U/L)	S.albumin	BT/CT	Widal	IgM ELISA Dengu	IgM Leptospira	Bone marrow	Blood culture	HIV											
76	YSOTAPPA	20	M	491490	BH	3	+	+	-	+	-	-	+	BP/M	-	+	I	Pu	+	-	NS	NS	NS	9	3800	33000	18	N	-	-	138	36	0.7	3.8	178	5.2	N	+	+	-	-	-	-	DENGUE	SY	E	ND
77	SATHISH	21	M	491219	BH	4	+	+	+	+	-	-	+	BP/M	-	+	I	SPB	+	+	NS	NS	NS	8.4	4000	18000	20	N	-	-	132	31	0.6	4.5	278	5.2	N	+	+	-	-	-	-	DENGUE	SY	E	ND
78	SAMAD SAB	35	M	491189	BH	3	+	+	+	+	-	+	+	NS	-	+	PI	SPB	-	-	NS	NS	NS	6.4	6000	18000	18	N	-	-	134	35	0.6	3.8	236	5.4	N	-	-	+	-	-	-	Septicemia	PENICILLIN	G	ND
79	VEENA	21	F	491589	BH	10	+	-	-	+	-	+	+	HOA	-	+	PI	Pu	-	-	NS	NS	NS	6.2	19200	21000	20	N	-	-	136	137	4.8	4.6	248	5.2	AB	-	-	-	-	+	-	Septicemia	ANTI/SU	E	ND
80	ASHA BEE	15	F	491514	BH	7	+	-	+	+	+	+	+	NS	-	+	PI	Pu	-	I	C	NS	NS	9.8	6000	48000	16	N	+	PF	137	28	0.7	2.3	218	5.3	N	-	-	-	-	-	-	FM	FALCIGO/mds	G	ND
81	MANJUNATH	18	M	491565	BH	6	+	-	-	-	-	-	-	PA	-	+	P	-	-	-	NS	NS	NS	8.6	5200	69000	16	N	-	-	135	29	0.6	0.8	36	5.4	N	+	-	-	-	-	-	ENT	XONE	G	ND
82	DRAKSYANAMMA	20	F	49144	BH	4	+	-	-	+	-	-	-	PA	-	+	PI	-	-	-	NS	NS	NS	9.6	5200	72000	18	N	-	PF	133	25	0.8	2.4	84	5.1	N	-	-	-	-	-	-	FM	FALCIGO/mds	G	ND
83	LAKSHMI	40	F	491254	BH	5	+	-	+	+	+	+	-	NS	-	+	I	-	-	-	NS	NS	NS	8.4	3200	81000	20	N	-	-	131	28	0.7	2.6	316	5.2	N	+	+	-	-	-	-	DENGUE	SY	G	ND
84	JYOTHI	35	F	491315	BH	6	+	-	-	+	+	+	-	BP/M	-	+	I	-	-	I	C	NS	NS	8.6	6200	61000	20	N	+	PF	130	38	0.8	0.9	36	5.2	N	-	-	-	-	-	-	FM	FALCIGO/mds	G	ND
85	NANDINI	24	F	491310	BH	7	+	-	-	-	-	-	-	NI	-	+	-	-	-	-	NS	NS	NS	9.8	5400	78000	18	N	+	PF	120	46	0.9	1.2	52	5.1	N	-	-	-	-	-	-	FM/VM	FALCIGO/mds	G	ND
86	ANITHA	19	F	491312	BH	9	+	+	+	-	-	-	+	NS	-	+	I	Pu	-	-	NS	NS	NS	9.9	4200	67000	16	N	-	Pv	121	52	0.9	0.9	56	5.1	N	+	-	-	-	-	-	VM/ENT	XONE	G	ND
87	RAVI	18	M	491101	BH	10	+	+	-	+	-	+	+	NS	-	+	-	PuPe	-	-	NS	NS	NS	9.6	18800	18000	18	AB	-	-	133	89	1.8	3.7	226	5.1	N	+	-	-	-	P	-	Septicemia	ANTI/SU	E	ND
88	YOGESH	25	M	491002	BH	12	+	+	+	+	-	+	+	NS	-	+	I	PuPe	-	-	NS	NS	NS	9.4	17600	21000	20	N	-	-	134	92	1.4	2.8	184	5.1	N	-	-	-	-	P	-	Septicemia	ANTI/SU	G	ND
89	ARUNA	20	M	491216	BH	8	+	+	-	-	-	-	+	BP/M	-	+	-	Pe	-	-	NS	NS	NS	8.8	3200	41000	26	N	-	-	133	60	0.9	1.2	64	5.6	N	+	+	-	-	-	-	DENGUE	SY	G	ND
90	MAMATHA	19	F	491110	BH	7	+	+	+	-	-	-	+	BP/M	-	+	-	Pe	-	-	NS	NS	NS	9.2	3600	33000	28	N	-	-	128	36	0.8	1.4	58	5.8	N	+	+	-	-	-	-	DENGUE	SY	G	ND
91	RAGAVENDRA	21	M	491594	BH	6	+	+	-	+	-	+	+	NS	-	+	I	Pe	-	+	NS	NS	NS	8.6	9800	38000	26	N	-	-	130	84	1.8	2.6	64	5.8	N	+	+	-	-	P	-	Septicemia	ANTI/SU	G	ND
92	RASHMI	30	F	491184	BH	5	+	-	+	+	-	+	+	NS	-	+	I	Pu	-	-	NS	NS	NS	8.8	18600	31000	28	N	-	-	148	196	5.2	3.6	217	5	N	-	-	-	-	P	-	Septicemia	ANTI/SU	E	ND
93	RAMAPPA	55	M	491216	BH	8	+	+	+	+	-	+	+	PA	-	+	PI	Pe	-	+	NS	NS	NS	8.6	5200	69000	12	N	-	-	120	56	0.9	1	38	5.2	N	+	-	-	-	-	-	ENT	XONE	G	ND
94	SHIVAMMA	35	F	491080	BH	9	+	-	+	-	+	-	+	BP/M	-	+	-	SPB	-	-	NS	NS	NS	8.8	3600	18000	14	N	-	-	140	78	1.8	1.8	74	5.2	N	-	-	-	-	-	-	DENGUE	SY	G	ND
95	RANGAMMA	28	F	491112	BH	7	+	-	+	-	-	-	-	PA	-	+	-	-	-	-	NS	NS	NS	8.6	5200	71000	16	N	-	-	121	56	0.9	0.9	56	5.4	N	-	-	-	-	-	-	ENT	XONE	G	ND
96	LALITHA	25	F	491210	BH	9	+	-	-	-	-	-	-	PA	-	+	-	-	-	-	NS	NS	NS	8.8	4800	91000	18	N	-	-	111	48	0.8	0.8	48	5	N	-	-	-	-	-	-	ENT	XONE	G	ND
97	RAJU	18	M	491316	BH	10	+	+	-	-	-	-	+	BP/M	-	+	-	SPB	-	-	NS	NS	NS	9.3	3800	17000	16	N	-	-	130	86	2.1	2.3	126	5	N	+	+	-	-	-	-	DENGUE	SY	E	ND
98	SUSHMA	19	F	491318	BH	11	+	-	-	-	+	-	+	BP/M	-	+	-	PePu	-	-	NS	NS	NS	8.6	6000	38000	18	N	+	PF	128	94	1.4	1.8	130	5	N	+	-	-	-	-	-	VM/FM/ENT	FALCIGO/mds	G	ND
99	CHANDRU	20	M	491254	BH	12	+	-	-	-	-	-	-	BP/M	-	+	-	-	-	-	NS	NS	NS	8.8	5400	67000	16	N	+	PF	116	96	1.2	2	126	5	N	+	-	-	-	-	-	VM/FM/ENT	FALCIGO/mds	G	1.38
100	RAKESH	24	M	491210	BH	6	+	-	-	+	-	-	-	NS	-	+	PI	-	-	+	NS	NS	NS	8.6	6000	60000	22	N	+	Pv	130	58	1.2	2.3	128	5.1	N	-	-	-	-	-	-	VM	Clokit/mds	G	1.42