CHILDREN WITH BICYTOPENIA AND PANCYTOPENIA - CLINICAL, ETIOLOGICAL SPECTRUM, OUTCOME AND FOLLOW UP IN A TERTIARY CARE CENTRE.

Dissertation submitted for

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BRANCH VII - PAEDIATRIC MEDICINE

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MADURAI MEDICAL COLLEGE, MADURAI

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**CHILDREN WITH BICYTOPENIA AND PANCYTOPENIA - CLINICAL, ETIOLOGICAL SPECTRUM, OUTCOME AND FOLLOW UP IN A TERTIARY CARE CENTRE**" is the bonafide work of **Dr. M.VIJAY ANAND** in partial fulfillment of the university regulations of the Tamil Nadu Dr. M.G.R Medical University, Chennai, for M.D Degree **Branch VII – PAEDIATRIC MEDICINE** examination to be held in May 2018.

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DECLARATION

I **Dr. M.VIJAY ANAND** solemnly declare that the dissertation entitled **"CHILDREN WITH BICYTOPENIA AND PANCYTOPENIA- CLINICAL, ETIOLOGICAL SPECTRUM, OUTCOME AND FOLLOW UP IN A TERTIARY CARE CENTRE"** has been conducted by me at the Institute of Child Health and Research centre, Madurai, under the guidance and supervision of my unit Chief Prof**. Dr. S. SHANMUGASUNDARAM M.D., D.C.H.**

This is submitted in partial fulfillment of the award of the degree of M.D. Pediatrics for the May 2018 examination to be held under The Tamil Nadu Dr. M.G.R Medical University, Chennai. This has not been submitted previously by me for any Degree or Diploma from any other university.

Place: Madurai **DR.M.VIJAY ANAND**

Date:

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CONTENTS

INTRODUCTION

INTRODUCTION

Cytopenia is defined as reduction in any of the cellular elements of blood, i.e., red cells, white cells or platelets. When there is reduction in any of the two cell lines it is called bicytopenia. When there is decrease in all the three types of cell lines it is called pancytopenia[8].

The etiology of bicytopenia and pancytopenia varies widely in children, ranging from bone marrow suppression by a viral infection to infiltration of marrow by malignant cells. There is considerable overlap between the causes of bicytopenia and pancytopenia[8].

Peripheral smear study becomes essential if cause of bicytopenia and pancytopenia was not apparent from clinical history and examination. If this didn't reveal the cause bone marrow aspiration or biopsy is needed. Clinically anemia leads to fatigue, breathlessness and cardiac symptoms. Thrombocytopenia leads to bruising and mucosal bleeding, and leucopenia leads to increased susceptibility to infection[67].

There are many studies in literature in children with pancytopenia, but there are only few studies in literature in children with bicytopenia. So far, no was study done in children with bicytopenia and pancytopenia in south tamil nadu.

So we have conducted this study, to assess the clinical profile and etiology in children admitted with bicytopenia or pancytopenia in our Institute. This study helps us to find out the common causes of bicytopenia and pancytopenia in our population.

We will also follow up the children who are admitted with bicytopenia or pancytopenia for 18 months and assess the outcome in children admitted in our hospital with bicytopenia or pancytopenia.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

HEMOPOIESIS:

The blood cells are formed in the bone marrow from a single type of cell called the pluripotent hematopoietic stem cell, from which all the cells of the circulating blood are ultimately derived. As these cells reproduce, a small portion of the original pluripotent cells will reside in the bone marrow. Almost major part of the reproduced cells, differentiate in to other cell types. The intermediate-stage cells are almost like the pluripotent stem cells, which are committed to produce only a particular line of cells called committed stem cells. The different committed stem cells produce specific types of blood cells in colonies, when grown in culture. CFU-E is used to designate the type of stem cell which is committed to produce erythrocytes. Similarly, CFU-GM is the colony-forming units that produce granulocytes and monocytes[1,2,3,4,5,6].

Growth and reproduction of almost all the stem cells are controlled by multiple proteins called growth inducers. One of these, interleukin-3, promotes growth and reproduction of virtually all the different types of committed stem cells. The growth inducers promote only growth. But the differentiation of the cells is done by another set of proteins called differentiation inducers. Each one of these differentiation inducers causes one type of committed stem cell to differentiate one or more steps toward

a final adult blood cell. Formation of the both the growth inducers and differentiation inducers is controlled by factors outside the bone marrow. For instance, in the case of RBCs, exposure of the blood to low oxygen for a long time causes growth induction, differentiation, and production of greatly increased numbers of RBCs. In the case of some of the white blood cells, infectious diseases cause growth, differentiation, and eventual formation of specific types of white blood cells that are needed to combat each infection. Aside from the cells committed to form RBCs, two major lineages of WBCs are formed, the myelocytic and the lymphocytic lineages. The myelocytic lineage starts with the myeloblast. The lymphocytic lineage begins with the lymphoblast. The granulocytes and monocytes are formed only in the bone marrow. Lymphocytes and plasma cells are produced mainly in the various lymphogenous tissues especially the lymph glands, spleen, thymus, tonsils, and various pockets of lymphoid tissue in bone marrow. The WBC's formed in the bone marrow are stored within the marrow until they are needed in the circulatory system. Then, when the need arises, various factors cause them to be released. The lymphocytes are mostly stored in the various lymphoid tissues, except for a small number that are temporarily being transported in the blood. Megakaryocytes are also formed in the bone marrow. These megakaryocytes fragment in the bone marrow; the small

fragments, known as platelets or thrombocytes, then pass into the blood. They are very important in the initiation of blood clotting[66,2,5.]

The disorders which primarily or secondarily affecting bone marrow manifest as peripheral cytopenia.

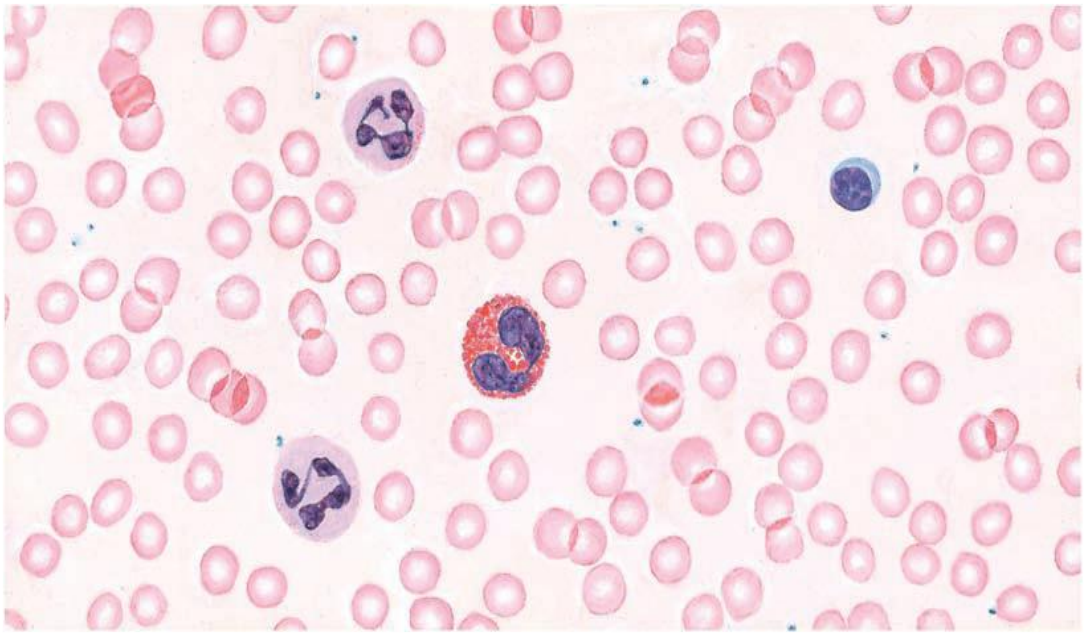


Figure -1: Normal human peripheral smear showing RBC, Neutrophil, Lymphocyte, Platelets and Eosinophils -Wright stain (high magnification)

To begin with, mild impairment in bone marrow activity is inapparent and cytopenia may become evident only during time of stress or increased demand. Bicytopenia and pancytopenia is not a disease but hematological finding caused by various underlying disease processes.

ANEMIA:

Anemia is defined as decreased hemoglobin content or RBC count below the normal range for age and gender. Anemia can be classified based morphology (RBC indices) and etiology[26].

MORPHOLOGICAL CLASSIFICATION[26]:

1) MICROCYTIC HYPOCHROMIC ANEMIA:

MCV, MCH, MCHC below normal for age. It is due to defect in red cell formation, where hemoglobin synthesis is impaired to a great extent. Most common causes are iron deficiency anemia and thalassemia.

2) NORMOCHROMIC NORMOCYTIC ANEMIA:

MCV, MCH, MCHC are within normal limits. Size and hemoglobin content of RBC are normal. It is caused by substantial blood loss, hemolysis and impaired RBC production by bone marrow in conditions like aplastic anemia, chronic infection and chronic renal failure.

3) MACROCYTIC ANEMIA:

MCV is above the upper limit of normal. Hemoglobin concentration is normal. The best example is megaloblastic anemia, due to B12 or folic acid deficiency[26].

ETIOLOGICAL CLASSIFICATION OF ANEMIA[26]:

A) DECREASED RED CELL PRODUCTION:

1) Stem cell failure

-Aplastic anemia

-Anemia of leukemia

2) Progenitor cell failure

-Chronic renal failure

-Chronic diseases

3) Precursor cell failure

-Megaloblastic anemia

-Iron deficiency anemia

-Thalassemia

-Hemoglobinopathies

B) INCREASED RED CELL DESTRUCTION:

1) Acquired causes

- Acute blood loss

- Hypersplenism

-Antibody mediated

-Micro and macroangiopathic

2) Hereditary causes:

-membrane defects

-enzyme defects

-globin defects [26]

LEUCOPENIA[26]:

Leucopenia is defined as reduction in leukocyte count below normal range for that age.

NORMAL VALUES

Infants: 6000 – 18000 / mm^3 of blood

Children: 5000 – 15000/mm^3 of blood

CAUSES OF LEUCOPENIA

1) Infections – typhoid fever, paratyphoid fever, early phases of viral infections like dengue fever, infectious hepatitis.
2) Overwhelming sepsis: in severe sepsis, consumption of neutrophils exceeds production.

3) Replacement of hematopoietic tissue in bone marrow by neoplastic infiltrative cells as in acute leukemia, lymphoma, myelofibrosis, etc.
4) Aplastic anemia – Hypoplasia of bone marrow.
5) Cytotoxic therapy
6) Hypersplenism
7) Starvation and malnutrition

Leucopenia occurs mainly due to neutropenia[26].

THROMBOCYTOPENIA[26]:

Thrombocytopenia is defined as decrease in platelet count below the normal range.

NORMAL LEVELS: 1.5 – 4.5 lakhs /mm^3 of blood

CAUSES OF THROMBOCYTOPENIA:

1) Idiopathic thrombocytopenic purpura
2) Aplastic anemia
3) Hypersplenism
4) Acute leukemia
5) Cytotoxic chemotherapy
6) Radiation treatment[26].

BICYTOPENIA:

Decrease in any two cell lineage of these three cells RBC's, WBC's or platelets is called bicytopenia. Bicytopenia can be a life threatening or temporary condition. In particular viral infections, malignancy, drugs, chemotherapy and radiotherapy may cause bicytopenia.

CAUSES OF BICYTOPENIA:

Zahide yalaki et al studied bicytopenia in pediatric patients and reported that the causes of bicytopenia in their study were 64.2% due to infection, 14.2% acute leukemia, 7.1% idiopathic thrombocytopenic purpura, 7.1% medicine use, 3.5% megaloblastic anemia and 3.5 % due to chronic illness anemia. They also revealed that the organisms causing bicytopenia in their study were salmonella, brucella, EBV, Hepatitis A, B, C, Mumps and Parvovirus[10.]

In a study done by Saadla Haroon Durrani et al on incidentally diagnosed bicytopenia in children age ranged from 1 year to 17 years, the most common type of bicytopenia was found to be anemia and thrombocytopenia. Among which the common cause was acute lymphoblastic leukemia, followed by mixed nutritional deficiency anemia, iron deficiency anemia, megaloblastic anemia and malaria. According to their study the second common type of bicytopenia was

thrombocytopenia and leukopenia which was found in marrow hypoplasia and visceral leishmaniasis. Finally anemia and leucopenia caused by hemolytic anemias were reported[11.]

In a study done in Rawalpindi by Muddassar Sharif et al on etiological spectrum of pancytopenia and bicytopenia, 62.9% had bicytopenia and 37.1% had pancytopenia on blood complete picture. 41.9% patients were diagnosed to have megaloblastic anemia on bone marrow examination and it was the leading cause of bicytopenia and pancytopenia in their study. Infective etiology was the cause of bicytopenia and pancytopenia in 19%, followed by aplastic anemia in 13.3% and acute leukemia in 10.5% cases in their study[12].

Shano naseem et al[8] done a study in pediatric patients with bicytopenia and pancytopenia found that the common non-malignant conditions causing bicytopenia in their study were idiopathic thrombocytopenic purpura (ITP) (5.2%), followed by megaloblastic anemia (3.7%), marrow hypocellularity (2.9%) and visceral leishmaniasis (2.0%). Commonest malignant condition associated with bicytopenia in their study was acute leukemia (66.9%). Of the acute leukemias, acute lymphoblastic leukemia was more common. The common non-malignant conditions associated with pancytopenia were aplastic anemia (33.8%), followed by megaloblastic anemia (13.7%). Most common malignant condition associated with pancytopenia was acute leukemia (26.6%).

PANCYTOPENIA:

Reduction in all the 3 types of cellular components in peripheral blood is termed pancytopenia and this involves anemia, leucopenia, and thrombocytopenia. Pancytopenia could be a result of either primary or secondary disorders of bone marrow. A detailed hematological and clinical study of the patients is important to find the underlying etiology of pancytopenia[68].

CAUSES OF PANCYTOPENIA[26]:

Bone marrow failure Hypoplastic/aplastic anemia Inherited, viral, idiopathic, drugs(Methotrexate, Linezolid)
Bone marrow infiltration Acute leukemias, haemophagocytic syndromes, Myeloma, Myelodysplastic syndromes, Lymphoma, Acquired immunodeficiency syndrome
Ineffective hematopoiesis Megaloblastic anemia
Peripheral pooling/destruction Portal hypertension, Malaria, Felty's syndromes, Myelofibrosis

CLASSIFICATION OF PANCYTOPENIA[4,68,2]:

1) HYPOCELLULAR MARROW:

-Inherited marrow failure syndromes

-Acquired aplastic anemia

-Hypoplastic variant of myelodysplastic syndrome

-Paroxysmal nocturnal hemoglobinuria

2) CELLULAR MARROW:

A) Primary bone marrow disease

-Acute leukemia

-MDS (Myelodysplastic syndrome)

B) Secondary to systemic disease

-Autoimmune disorders (SLE)

-Vitamin B12 or Folate deficiency

-Storage disease (Gaucher's, Niemann Pick diseases)

-Overwhelming infection

-Sarcoidosis

-Hypersplenism

3) BONEMARROW INFILTRATION

-Metastatic solid tumors

-Myelofibrosis

-Hemophagocytic lymphohistiocytosis

-Osteopetrosis

INHERITED PANCYTOPENIA SYNDROMES [68]**:**

1. Fanconi anemia
2. Shwachman-Diamond syndrome
3. Dyskeratosis congenita
4. Congenital amegakaryocytic thrombocytopenia
5. Reticular dysgenesis
6. Down syndrome
7. Dubowitz syndrome
8. Seckel syndrome
9. Schimke immunoosseous dysplasia
10. Cartilage-hair hypoplasia
11. Noonan syndrome

ETIOLOGY OF ACQUIRED APLASTIC ANEMIA [68] [2]:

- Radiation, drugs, and chemicals

  - Predictable: chemotherapy, benzene

  - Idiosyncratic: chloramphenicol, antiepileptics, gold;

  3, 4-methylenedioxymethamphetamine

- Viruses:

  Cytomegalovirus, Epstein-Barr virus, Hepatitis B, Hepatitis C, Hepatitis non-A, non-B, non-C (seronegative hepatitis), HIV

- Immune diseases:

  - Eosinophilic fasciitis
  - Hypoimmunoglobulinemia
  - Thymoma
  - Paroxysmal nocturnal hemoglobinuria

- Marrow replacement:

  - Leukemia
  - Myelodysplasia
  - Myelofibrosis
  - Autoimmune

- Others:

  - Cryptic dyskeratosis congenita (no physical stigmata)
  - Telomerase reverse transcriptase haploinsufficiency

Shazia menon et al studied the etiological spectrum of pancytopenia in children based on the bone marrow examination. They revealed that the common cause of pancytopenia was aplastic anemia (23.9%), followed by megaloblastic anemia (13.04%), leukemia (13.05%), enteric fever (10.8%), malaria (8.69%) and sepsis (8.69%).

Gunvanti B. Rathod et al did clinico-hematological analysis of pancytopenia in Pediatric patients and stated that megaloblastic anemia 26.5% was the most common cause of pancytopenia followed by aplastic anemia 20.0%, leukemia 17.5%, idiopathic thrombocytopenic purpura 10%, iron deficiency anemia 9.5%, anemia of chronic disorder 1.5% and finally malaria 3.5%.

Mirza Asif Baig et al evaluated bone marrow aspirate in pediatric patients with pancytopenia and reported that ALL 71.7% was the most common cause of pancytopenia followed by aplastic anemia 22.6%.

Shiv Ram Krishna Dubey et al studied the clinico-etiological spectrum of pancytopenia in hospitalized children and stated that the common causes of pancytopenia were megaloblastic anemia (47%), aplastic anemia (25.8%) and leukemia (17.6%).

In their study, clinico-aetiological profile of pancytopenia in paediatric practice by Amieleena Chhabra et al, megaloblastic anemia 31.8% was the most common cause of pancytopenia. Causes like acute

lymphoblastic leukemia, acute myeloid leukemia, non-Hodgkin's lymphoma, Langerhans cell histiocytosis and myelodysplastic syndrome constituted 25.2% cases. Aplastic anaemia seen in 18.68% cases was another important cause of pancytopenia in their study. In their study infections such as kala azar, malaria, enteric fever, bacterial septicemia caused pancytopenia in 19.7% of patients. Other infections include tuberculosis and dengue fever. The miscellaneous group included Gaucher's disease presenting as hypersplenism.

Chate Sambhaji et al studied the clinical and hematological profile of pancytopenia in children and found that megaloblastic anemia 30.4% was the most common cause of pancytopenia followed by aplastic anaemia in 26% cases. Causes like acute lymphoblastic leukemia, acute myeloid leukemia, Langerhans cell histiocytosis, myelodysplastic syndrome constituted 19% cases. Infections such as malaria, enteric fever, kala azar, bacterial septicemia and HIV with parvovirus caused pancytopenia in 17.3% patients. Other rare causes include Gaucher's disease, hypersplenism due to extra hepatic portal vein obstruction and hemophagocytosis secondary to still's disease.

Jitender Mohan Khunger et al from India did a clinico hematological study of 200 cases of pancytopenia age ranged from 2 to 70 years, and found that megaloblastic anemia was commonest cause (72%), followed by aplastic anemia (14%) and subleukemic leukemia

(5%). This study also found out hypersplenism due to malaria in 1 % of cases and disseminated tuberculosis with pancytopenia in 1% of cases.

Baus et al, Yadav et al, Singh et al have also reported pancytopenia in cases of disseminated tuberculosis. The above studies prove high prevalence of tuberculosis in India and pancytopenia was an important finding seen in tuberculosis.

Shishir Kumar Bhatnagar et al studied the etiological profile of pancytopenia in children and reported that megaloblastic anemia 28.4% was the single most common cause of pancytopenia, followed by aplastic anemia (20%) and acute leukemia (21%). Infections such as enteric fever, malaria, kala-azar and bacterial septicemia caused pancytopenia in 21% of patients[16].

Fahim Manzoor et al studied clinical hematological profile of pancytopenia in children and adults and found that the most common cause of pancytopenia was megaloblastic anemia (56%) , followed by hypoplastic/aplastic anemia (14%). Post viral illness cases comprised dengue and H1N 1 swine flu.

ETIOPATHOGENESIS OF CYTOPENIAS:

SEPTICEMIA:

Pediatric septicemia usually comprises a spectrum of diseases or disorders caused by various viruses, bacteria, fungi, parasites or toxic products of these microorganisms.

In children pathogenic bacteria enter in to blood and produce severe infection without much localization called septicemia, commonly caused by staphylococcus aureus, Klebsiella and Escherichia coli. Hospital acquired infection are due to CONS and pseudomonas. Child usually presents with refusal of feeds, lethargy, fever, breathlessness, abdominal distension, and vomiting. In a child with systemic bacterial infection meningitis and pneumonia is common. If child has fast breathing, chest retractions and grunting, it suggests pneumonia. If child has incessant cry, vacant stare, convulsions and bulging fontenelle, it suggests meningitis. Blood culture gives the definite diagnosis; samples are taken before first dose of antibiotics. Sepsis screen comprises of total count <5000/mm^3, ANC <1800/mm^3, immature to total neutrophil ratio >20%, CRP >1 mg/dl and microESR >15mm in first hour. Sepsis screen is positive if any of the above 2 parameters are positive. Empirical antibiotics given once diagnosis is made, after culture reports specific antibiotics are given. Outcome depends on the type of organism, its antibiotic sensitivity and adequacy of specific and supportive treatment[67].

Garewal et al in their study reported that the septicemia commonly gram negative sepsis causes bi/pancytopenia in children, due to bone marrow necrosis. Disseminated intravascular coagulation also leads to cytopenia in sepsis[62].

ENTERIC FEVER:

Enteric fever commonly called typhoid fever is most commonly caused by a gram negative bacilli salmonella typhi. It is one of the common causes of fever in developing countries like India, due to poor sanitation and water supply. Clinically its insidious onset, presents with low grade fever, coated tongue, anorexia, vomiting, diarrhea, abdominal pain and hepatosplenomegaly. Blood culture is the gold standard for diagnosis. Widal test has low sensitivity and specificity. Complete blood counts commonly show leucopenia, sometimes in small children leucocytosis also seen. Thrombocytopenia is seen in severe cases of typhoid fever. Typhoid fever is treated with antibiotics like ceftriaxone or cefixime or ciprofloxacin or azthiromycin for 14 days. Prognosis is good in appropriately treated cases and case fatality rate is less than 1%.

Cytopenia in enteric fever is caused by varied mechanisms. Bone marrow may undergo histiocytic hyperplasia along with hemophagocytosis or complete necrosis. Immune mediated hemolysis or

leucopenia, hypersplenism and transient disseminated intravascular hemolysis can occur in enteric fever [67] [65] [64] [63].

MEGALOBLASTIC ANEMIA[67,58,47]:

Ineffective erythropoiesis, leukopoiesis and thrombopoiesis due to enhanced programmed cell death in absence of vitamin B12 or folic acid and decreased survival of precursors in peripheral blood are most commonly implicated in causing pancytopenia in megaloblastic anemia.

MALARIA:

Malaria is common in developing countries like India. It is transmitted by bite of female anopheles mosquito and caused by protozoa, Plasmodium (P) falciparum, P.vivax, P.ovale, P.malariae. Fever is the most common presenting symptom. It is also associated with chills, headache, nausea, vomiting, lethargy, weakness and body ache. Severe cases may have impaired consciousness, convulsions, respiratory distress, shock and bleeding manifestations. Malaria is diagnosed by peripheral smears thick and thin film study, which is the gold standard test for diagnosis of malaria. Rapid diagnostic test, immunochromatographic test is used in places where expert microscopic diagnostic facilities are not available. Once diagnosis is confirmed, antimalarials are given. WHO recommends multidrug treatment for P.falciparum infections. Hematological parameters are altered in children with malaria. Severe

anemia can occur due to red blood cell destruction by hyperparasitemia. In majority of cases it is normocytic normochromic anemia, and some cases show microcytic hypochromic anemia. Anemia is commonly caused by plasmodium falciparum. WBC counts is variable (normal or leucopenia) in children with malaria. Thrombocytopenia is a characteristic finding in malaria, it is seen in both falciparum and vivax malaria. Aouba et al reported hemophagocytic syndrome resulting from P. vivax infection as the cause of pancytopenia. Arya et al also reported that pancytopenia can occur in P. falciparum malaria. Malaria causes anemia and thrombocytopenia due to direct invasion by parasite, immune hemolysis, disseminated intravascular coagulation, hypersplenism and hemophagocytosis. So in malaria, anemia and thrombocytopenia are classical features. Changes in WBC counts are less dramatic, as there is conflicting reports from various studies[67].

APLASTIC ANEMIA:

Aplastic anemia comprises a group of disorders of hematopoietic stem cells that results in suppression of erythroid or myeloid or megakaryocytic cell lines. Aplastic anaemia is one of the most serious causes of pancytopenia. Marrow failure leading to pancytopenia may result from immune-mediated or non-immune mediated damage or suppression of either pluripotent stem cells or committed progenitor cells. Child presents with pallor with or without congestive heart failure due to

anemia. Bleeding tendencies like petechiae, ecchymosis, gum bleeding and nose bleeding occur due to thrombocytopenia. Fever, sepsis and pneumonia occur due to neutropenia. Complete blood count shows pancytopenia. Bone marrow is hypocellular, which is replaced with fat cells and lymphocytes. Treatment is supportive, with transfusion of packed cells for anemia, platelets transfusion for thrombocytopenia and antibiotics for neutropenia. But the definitive therapy is hematopoietic stem cell transplantation. Long term survival with transplantation is 70%. Prognosis depends on the severity of cytopenias. Mortality is due to heart failure or severe sepsis or bleeding[67].

FANCONI ANEMIA:

Fanconi anemia is an inherited autosomal recessive disorder. It is one of the congenital syndromes associated with bone marrow failure. Associated features are absent thumb, absent radius, microcephaly, renal anomalies, short stature, café u lait spots and skin pigmentation[67].

ACUTE LEUKEMIAS:

Leukemia is the most common cancer in children. Of which, ALL is the most common childhood malignancy.

ACUTE LYMPHOBLASTIC LEUKEMIA (ALL):

ALL incidences are 3-4 cases per 1 lakh children. Peak incidence between 2 to 5 year and boys has higher rates than girls. Progenitor B cell ALL constitutes 80 to 85% and T cell ALL constitutes 15%. Clinical features of ALL are due to bone marrow infiltration with leukemic cells (bone marrow failure) and extramedullary involvement. Common clinical features include pallor, fatigue, petechiae and infections. Clinical findings like lymphadenopathy, hepatomegaly and splenomegaly are present in >60% cases. Bone pain or joint pain and bone tenderness is also a common clinical feature. Tachypnea in ALL may occur due to severe anemia. Children with ALL may present with pancytopenia or bicytopenia. Diagnosis is confirmed by peripheral smear study and bone marrow aspiration. Bone marrow showing >25% lymphoblast is diagnostic. ALL is treated with combination chemotherapy and radiotherapy. Prognosis depends on many factors. In developed countries >80% children with ALL are long term survivors. In developing countries survival is poor due to infections. Despite treatment, 20-30% children with ALL usually relapse. Common sites of relapse are bone marrow (20%), central nervous system (5%) and testis (3%) [(67)].

ACUTE MYELOID LEUKEMIA:

AML constitutes 15 -20% of leukemia in children. It can occur at any age, but incidence is more during adolescence. AML can occur after ionizing radiation. Down syndrome is the most common genetic risk factor for AML. According to FAB classification, AML is divided into eight types M0 to M7. Clinically AML patients usually have higher white cell count at presentation, along with anemia and thrombocytopenia. Clinical features in AML are pallor, fever, fatigue and bleeding. Clinical findings like lymphadenopathy and hepatosplenomegaly are not common as in ALL. Sometimes diagnosis of AML is preceded by prolonged preleukemic phase, characterized by lack of one of the cell lineages, i.e. refractory anemia, neutropenia or thrombocytopenia, this is called myelodysplastic syndrome. Sometimes patient have hypoplastic marrow which may later develop into acute leukemia. AML is treated by combination chemotherapy. Long term survival rate is 50% and only 70-80% achieves remission with current regimens. Increased relapse rate occurs due to chemotherapy resistance and increased risk of death occurs due to infections and hemorrhage[67].

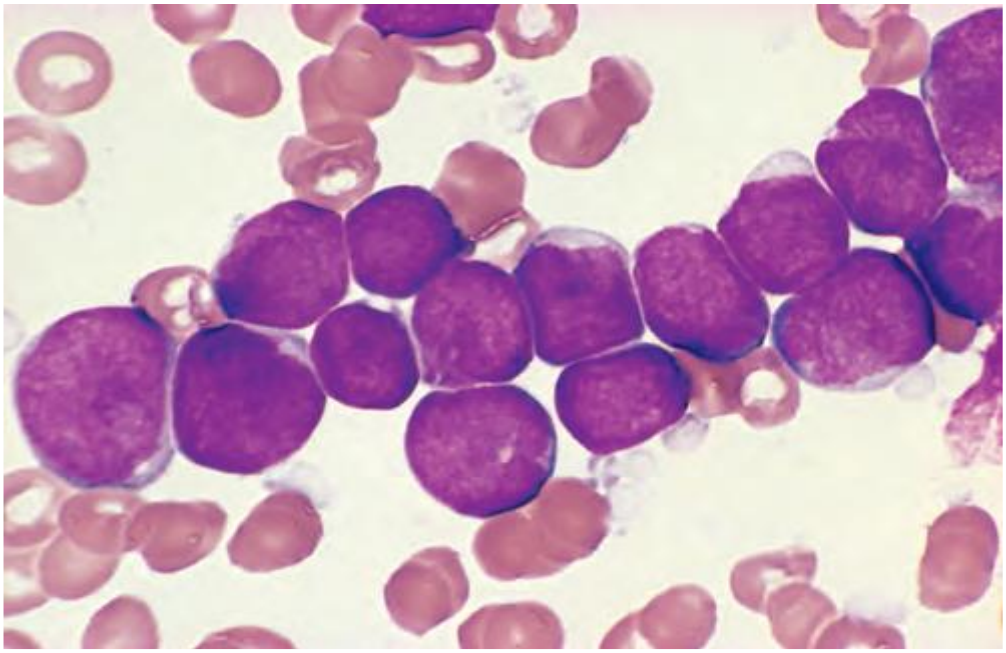


Figure-2- Acute Lymphoblastic Leukemia, Lymphoblasts with condensed nuclear chromatin, small nucleoli, and scant agranular cytoplasm (Robbin's pathology)

The mechanisms of marrow failure in these diseases are unclear but probably involve active suppression of normal hematopoiesis as well as bone marrow infiltration by these abnormal cells.

The mechanism by which AML mediates this complication is not clear, but one widely accepted explanation is that AML depletes the hematopoietic stem cells of the bone marrow through displacement.

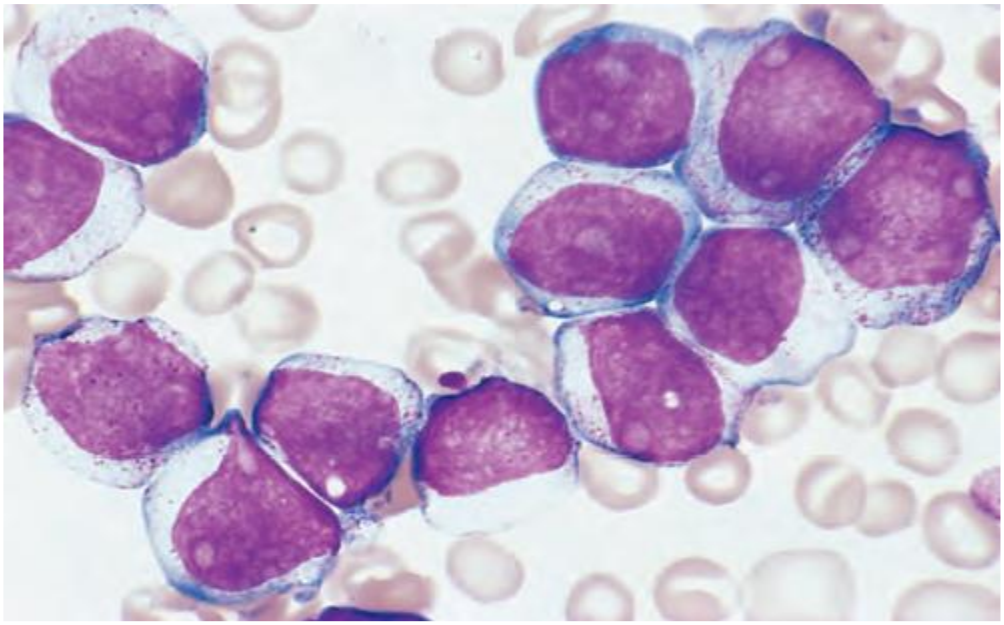


Figure-3- Acute myeloid leukemia without maturation (FAB M1 subtype)

Myeloblasts have delicate nuclear chromatin, prominent nucleoli, and fine azurophilic granules in the cytoplasm (Robbin's Pathology)

DRUG INDUCED CYTOPENIA:

A. Cytotoxics causes bone marrow suppression

B. Chloramphenicol (by dose related effects)

C. Idiosyncratic response (immune mediated):

a. NSAIDs

b. Sulfonamides

c. Anti-epileptics

DRUG INDUCED CYTOPENIAS:

Drug induced myelosuppression is common following anticancer drugs. It is temporary. Nadir is the point at which the lowest blood count is reached following therapy. Nadir usually occurs at 6-10 days following therapy. Recovery occurs at 14-21 days. WBC's are the most sensitive cells and they are first affected, followed by RBC's and platelets. Myelosuppression is commonly caused by cyclophosphamide, cytarabine, 6Mercaptopurine used in treatment of acute lymphoblastic leukemia. If myelosuppression is severe then it is necessary to reduce the dose or stop the drug and allow the bone marrow to recover[2,66,68].

ETIOPATHOGENESIS OF CYTOPENIA IN VIRAL INFECTION:

Viral infections causing cytopenia are Hepatitis B and C viruses, occasionally by cytomegalovirus, Epstein – Barr Virus, HIV, Dengue virus and rarely by Hepatitis A virus. Other rare causes are rubella, influenza, parainfluenza, measles, and mumps.

DENGUE FEVER:

Dengue fever is the most common arboviral infection in India. It is transmitted by bite of Aedes aegypti or by A.albopictus. 70 % of the dengue infections are asymptomatic and 30% symptomatic. It is further classified in to undifferentiated viral fever, probable dengue without

warning signs, probable dengue with warning signs and severe dengue. Incubation period is usually 4 to 10 days. Dengue fever has different clinical presentation and is often unpredictable. Illness begins abruptly, followed by 3 phases. First is febrile phase, in which child have sudden onset of high grade fever, facial flushing, skin erythema, body ache, head ache, nausea, vomiting, hepatomegaly and decrease in white cell count. Febrile phase lasts for 2–7 days. Second is the critical phase after 3–4 days of onset of fever, in this phase there is no fever, child presents with abdominal pain, vomiting, bleeding manifestations especially mucosal bleeds from GIT, shock, hemoconcentration, thrombocytopenia and severe organ impairment like hepatitis. Finally, recovery phase starts usually after 6-7 days of fever, lasts for 48-72 hours. Gradual reabsorption of fluid takes place, general well being improves, appetite returns, diuresis occurs, some experiences generalized pruritis, bradycardia and rashes. Hematocrit also stabilizes in this phase. Total count starts to rise after critical phase first and recovery of platelet count takes longer time. Dengue fever is diagnosed by NS1 antigen detection during first 5 days of infection, IgM ELISA after 5 days, IgG ELISA method after 10-15 days. Treatment depends on the phase of illness. Symptomatic and supportive therapy is given. Packed red blood cells and platelet transfusions may be needed in dengue with hemorrhagic manifestations. Early diagnosis, appropriate fluid therapy and careful

monitoring, decrease the mortality to less than 1 %. Prognosis depends on duration and severity of peripheral circulatory failure [67].

Complete blood count (CBC) and peripheral smear study is very useful in early diagnosis of dengue infection.CBC usually shows raised hematocrit, leucopenia (<4000/mm^3), with relative lymphocytosis (>40%) and thrombocytopenia (<1.5lakh/mm^3). Raised hematocrit is due to plasma leakage caused by cytokine release. Leucopenia and thrombocytopenia is due to bone marrow suppression caused by virus. On peripheral smear examination, atypical lymphocytes are seen. These atypical lymphocytes had large cell size, increased amount of cytoplasm with characteristic tailing pattern of cytoplasm and increased cytoplasmic basophilia. Nuclear chromatin is slightly open. Dengue virus can propagate in bone marrow cultures without direct cytotoxicity, but dengue antigens induce lymphocyte activation and the release of marrow suppressive cytokines.

The reasons for low platelet count in dengue fever are as follows.

-Dengue virus induces bone marrow suppression, leading to low platelet count.

-Studies suggest that dengue virus can even bind to platelets of human blood in the presence of virus-specific antibody. When vascular endothelial cell that are infected with dengue virus gets combined with

platelets they tend to destroy platelets. This is one of the major causes of low platelet count in dengue fever.

- Even the antibodies that are produced after infection of dengue virus can contribute in destruction of platelets, thus lowering the platelet count. Low platelet count in dengue fever may lead to life-threatening condition known as hemorrhagic dengue fever that is categorized by spontaneous bleeding tendency and shock.

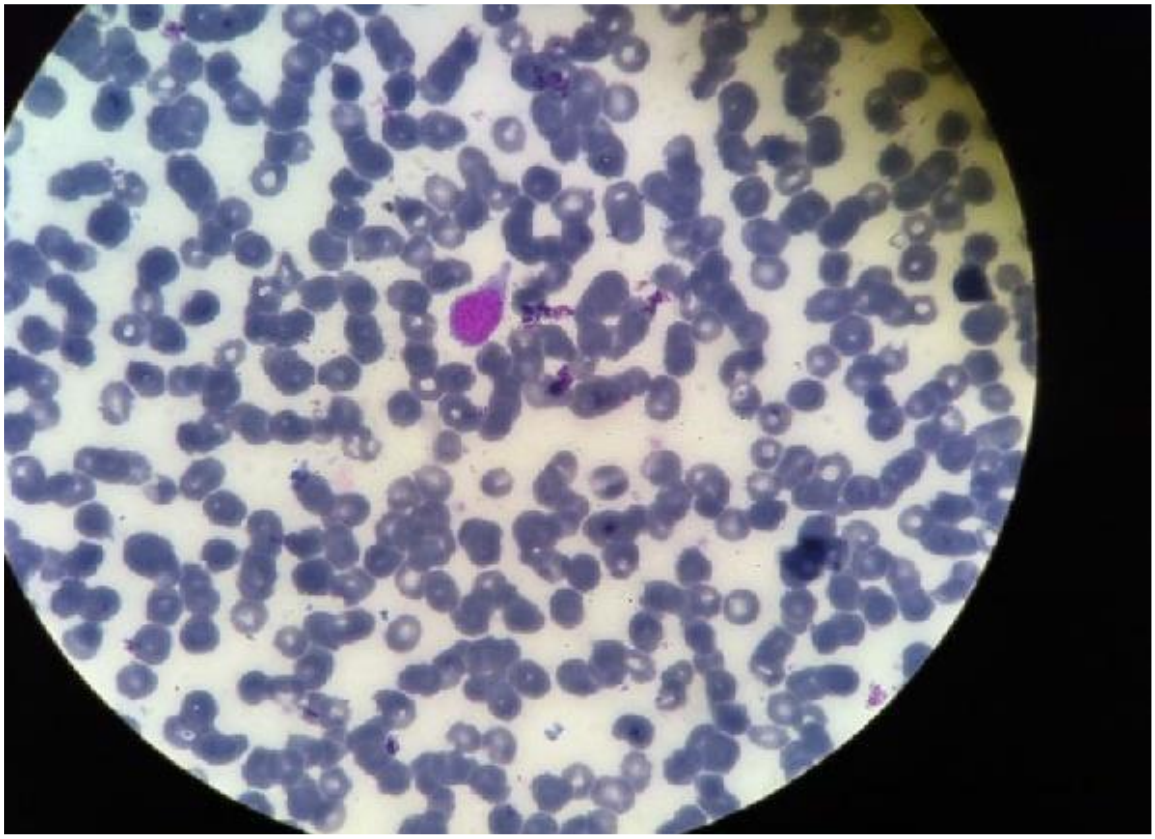


Figure 4 – characteristic atypical lymphocyte seen in dengue infection

TUBERCULOSIS:

Tuberculosis is still a common problem causing morbidity and mortality in developing countries. The diagnosis is based on the clinical features, chest x ray, tuberculin testing and history of contact with adult TB patient. Chest x ray and Mantoux test is useful in diagnosis but not confirmatory. Demonstration of bacilli in various clinical specimens is the gold standard. GenXpert is a real time PCR technique used to identify M.tuberculosis in sputum/Gastric juice sample. The stage of the disease and the site of involvement affect the outcome of the disease. Malnutrition, delay in diagnosis and treatment and central nervous system involvement determines prognosis.

Tuberculosis produces normochromic normocytic anemia. Leucopenia occurs in miliary tuberculosis. Pancytopenia is seen in children with miliary or disseminated tuberculosis. Peripheral count abnormalities will revert to normal after anti tuberculosis therapy[67].

CHRONIC LIVER DISEASE (CLD):

Chronic liver disease includes wide group of disorders, including:

1) prolonged cholestasis of infancy like biliary atresia and neonatal hepatitis,

2) Chronic hepatitis due to viral hepatitis B, C& D, autoimmune hepatitis, drug induced hepatitis due to anticancer drugs, antiepileptics and antituberculous drugs,

3) Metabolic/genetic disorders like glycogen storage disease, Gaucher's disease, galactosemia and mucopolysaccharidosis,

4) Copper associated disorder- Wilson disease,

5) Iron associated disorder –Hemochromatosis,

6) Vascular- Buddchiari syndrome, venoocclusive disease,

Clinically child presents with abdominal distension, jaundice, hemetemesis, signs of portal hypertension, shrunken or enlarged liver, firm splenomegaly, failure to thrive and muscle wasting. Liver transaminases and bilirubin levels are increased in children with acute liver injury e.g., due to hepatitis virus. In liver cell failure synthetic functions of the liver is affected, so hypoalbumenemia, reversal of albumin and globulin ratio and elevation of prothrombin time occurs.

Elevation of alkaline phosphatase and gamma glutamyl transpeptidase suggests cholestasis. Ultrasonogram (USG) of abdomen, USG colour Doppler of spleen-portal vein axis is helpful in diagnosis of portal hypertension.

Causes of hematological abnormalities in children with CLD are, portal hypertension induced splenic sequestration, bone marrow suppression and increased blood loss. Thrombocytopenia is due to portal hypertension induced hypersplenism, reduction in thrombopoietin, bone marrow suppression by hepatitis B and C viruses and consumptive coagulopathy (disseminated intravascular coagulation).Anemia due to increased blood loss or hemorrhage. Pancytopenia is due to bone marrow suppression by hepatitis viruses[67].

SYSTEMIC LUPUS ERYTHEMATOSIS (SLE):

SLE is an autoimmune disorder; autoantibodies are formed against self antigens. It affects various organs like skin, kidney, blood vessels and nervous system. Children with SLE usually presents with fever, rash, and arthritis. Glucocorticoids form the first line of treatment in SLE. Hematological manifestations like anemia, leucopenia and thrombocytopenia are common in SLE. Leucopenia is due to peripheral destruction of granulocytes by auto antibodies and decreased bone marrow production. Thrombocytopenia (<1 lakh/cumm) is due to

decreased marrow production, peripheral destruction by antiplatelet antibobies and splenic sequestration. Autoimmune hemolytic anemia is due to warm auto antibodies of IgG type[67].

HEMOPHAGOCYTIC LYMPHOHISTIOCYTOSIS:

It is an uncommon hematologic disorder clinically manifesting as fever, hepatosplenomegaly, lymphadenopathy, jaundice and rash. It is due to excessive activation of T lymphocytes and macrophages. Histiocytosis, hemophagocytosis and pancytopenia are the pathological findings. Primary HLH (familial HLH) is autosomal recessive. Familial HLH mostly caused by mutation in genes: PRF1 and UNC13D. Secondary HLH or Acquired HLH occurs after strong immunologic activation by systemic infection, immunodeficiency, or underlying malignancy[68].

WISKOTT-ALDRICH SYNDROME:

An inherited X-linked recessive immunodeficiency disorder characterized by leucopenia, thrombocytopenia, petechiae, bleeding and eczema[67]

BERNARD SOULIER SYNDROME:

This is an inherited bleeding disorder due to qualitative defects in platelets characterized by thrombocytopenia and giant platelets. Clinically child presents with increased bleeding tendencies like easy bruising, gum bleeding and epistaxis[67].

AGE AND GENDER DIFFERENCE IN CHILDREN WITH BICYTOPENIA AND PANCYTOPENIA:

Muddassar Sharif et al, done a study on etiological spectrum of pancytopenia and bicytopenia in children between 2 months to 12 years in 105 patients and found that there were 56 (53. 3%) male and 49 (46.7 %) female patients with Male: Female ratio 1.2:1 in their study.

Shano naseem et al, in their study in pediatric patients with bicytopenia/pancytopenia in 990 children had a male: female ratio of 2.9:1.

Zahide Yalaki et al, in their study in experience with bicytopenia in patients treated at the Ankara hospital pediatric clinic, reported that in their study 57.1% patients were male and 42.9% were female.

Shazia menon et al, studied etiological spectrum of pancytopenia in children based on bone marrow examination in 230 cases. In their study, there were 60.86% male and 40.14% female patients with male to female ratio of 1.6:1. Most cases seen were in between 6-10 years age group 56.52%, while 26.08% were between 2 months to 5 years of age and 17.39% were between 11 and 15 years.

Ghanshyam singh et al, studied the clinco hematological profile of childhood pancytopenia with special reference to non malignant presentation and stated that 51.6% belong to 1 to 5 year age group.

Gunvanti B. Rathodm et al, studied the clinico-hematological analysis of pancytopenia in pediatric patients, stated that 65% were males and 35% females, with male to female ratio of 1.85:1, their ages ranged from one month to 14 years. Maximum numbers of patients 39% were in the age group of 6 month to 5 years, followed by 34% in the age group of 6 to 10 years while 27% were those exceeding 11 years of age. They also reported that all age group had a male predominance.

CLINICAL FEATURES IN CHILDREN WITH BICYTOPENIA AND PANCYTOPENIA:

The cardinal clinical features of cytopenia are anemia, bleeding, and infection. Red blood corpuscles survive much longer than platelets or neutrophils.

The platelet count is the first to be affected. The presenting symptoms in child with thrombocytopenia are, mucocutaneous bleeding commonest being epistaxis, followed by gum bleeds, haematuria, GI bleeding, rarely intracranial bleeding and easy bruising with minimal trauma.

Next to be affected is the myeloid series. Infections are caused by commensal organisms of the skin or gastrointestinal tract. Early manifestation of neutropenia is fever due to lung or skin infections.

Unfortunately, patients with cytopenias may develop overwhelming septicemia without any focal sign of infection; the only clinical features being malaise and fever. The commonest offending organisms include coliforms, klebsiella spp, pseudomonas species, and staphylococci

Anemia develops slowly unless there is significant bleeding. The typical symptoms of tiredness, easy fatigability, facial puffiness, edema, and exercise intolerance may not be striking in the initial phase.

Evidence of erythropoietic failure characterized by pallor, fatigue, and tachycardia is often late because the life span of the erythrocyte (120 days) far exceeds that of platelets (10 days) or white cells (variable, but measured in hours for granulocytes). Mucous membranes and nail beds may be pale.

Lymphadenopathy, splenomegaly, and severe weight loss are uncommon and may suggest other underlying disorders. Short stature, congenital anomalies (particularly of the thumbs and forearms), areas of hyper- or hypopigmentation, dystrophic nails, immunologic abnormalities, or pulmonary disease are the features of other possible inherited bone marrow failure disorders.

CLINICAL PROFILE IN BICYTOPENIA:

Zahide yalaki et al, studied bicytopenia in pediatric patients and reported that 67.8% had fever on admission, 14.2% had weakness and 14.2% had rashes. On their physical examination, 14.2% had petechiae, 10.7% had hepatomegaly and 3.5% was with short stature.

Shano naseem et al from India, in their study on pediatric patients with bicytopenia/pancytopenia found that the main presenting features in children with bicytopenia were fever (69.2%) and pallor (40.9%), other common ones being petechial rash, bleeding manifestations and bone pains. In their study the common physical findings were hepatomegaly (69.2%) and splenomegaly (60.5%).

In a study done in Rawalpindi, by Muddassar Sharif et al on etiological spectrum of pancytopenia and bicytopenia in children between 2 months to 12 years, fever (82.9%) was the commonest presentation. Hepatosplenomegaly was seen in 27.6%, isolated splenomegaly in 4.8% while 5.7% patients had generalized lymphadenopathy.

HEMATOLOGICAL PROFILE IN BICYTOPENIA:

Zahide yalaki et al, studied pediatric patients with bicytopenia and reported that 85.7% patients had neutropenia, 57.1% patients had anemia and 71.4% thrombocytopenia.

Shano naseem et al from India, in a study on pediatric patients with bicytopenia, reported that thrombocytopenia and anemia (77.5%) was the most common form of bicytopenia, followed by anemia and leukopenia in 17.3% and thrombocytopenia and leukopenia in 5.5% cases.

CLINICAL PROFILE IN PANCYTOPENIA:

Shano naseem et al from India, in their study on pediatric patients with bicytopenia and pancytopenia found that the main presenting features in children with pancytopenia were fever (65.5%) and pallor (59%). Other common symptoms consisted of bleeding, petechial rash and bone pains. On clinical examination, hepatomegaly was seen in 51.8% and splenomegaly in 37.4% cases.

Shazia menon et al, studied the etiological spectrum of pancytopenia in children based on the bone marrow examination and found that the most common presenting symptom was pallor in 87% and fever in 65% cases.

Ghanshyam singh et al, studied the clinco hematological profile of childhood pancytopenia with special reference to non malignant presentation and reported that the most common presenting compliant was fever 71.9% followed by pallor 33.35%.

Gunvanti B. Rathod et al, did a clinico-hematological analysis of pancytopenia in pediatric patients and found that the most common symptom was pallor in 81.5% cases and fever in 65% cases.

Shiv Ram Krishna Dubey et al, in their study on clinico-etiological spectrum of pancytopenia in hospitalized children, found that the common clinical presentations were pallor (81%), fever (68%) and petechial haemorrhages (51%).

Chate sambhaji et al, also studied the clinical and hematological profile of pancytopenia in children and found that fever (67.3%) was most common clinical feature followed by pallor (63%) and bleeding manifestations (54.3%). Hepatomegaly was present in 56.5% patients and splenomegaly was present in 41.3% patients.

Shishir Kumar Bhatnagar et al, in their study on etiological profile of pancytopenia in children, reported that the skin bleeds in the form of petechiae, bruises and ecchymosis were the commonest bleeding manifestations. Also epistaxis, melena, gum bleeds and hematuria were observed.

INVESTIGATIONS IN CHILDREN WITH BI/PANCYTOPENIA:

- Complete hemogram using automated analyzer (Hb, TC, DC, Platelet, PCV, MCV, MCH, MCHC, RDW, MPV, RBC COUNT & ESR)
- Peripheral smear study with reticulocyte count
- Bone marrow aspiration/biopsy
- Urine routine
- Blood urea, S.creatinine
- Liver function test SGOT, SGPT, Bilirubin (total, direct, indirect)
- Prothrombin time, APTT
- Enteric culture, non -enteric culture, urine culture and sensitivity
- Smear for malaria parasite
- Sputum AFB, Gene Xpert for M.tuberculosis
- Widal test
- NS1 Ag test, IgM Elisa for dengue fever
- Flow cytometry
- Metabolic diseases workup.
- Vitamin B12, Folate levels in blood
- USG Abdomen
- Chest X ray (if necessary)
- Coombs test

- S.electrolytes
- Serologic testing/PCR Hepatitis, EBV, HIV, other virus
- Other relevant investigations to identify etiology

MANAGEMENT IN CHILDREN WITH BICYTOPENIA AND PANCYTOPENIA [67, 68]:

The basic management of patients with Bicytopenia and Pancytopenia involves identification and reversal of the underlying cause. It must be emphasized that bleeding and infection due to cytopenias is a medical emergency.

A. Supportive care:

This is the most important aspect of management of bicytopenia and pancytopenia. Anemia is corrected by transfusion of packed red cells to maintain haemoglobin (Hb) level above 8-9 gm/dl. Intramuscular injections and teeth brushing should be avoided in thrombocytopenic patients. Active bleeding should be promptly managed with the help of infusion of platelet concentrates in the form of platelet packs.

B. Prevention of infection:

Careful maintenance of skin hygiene, good dental care, and rectal hygiene is absolutely essential. Severe neutropenia by itself is not an indication for hospitalization as with each admission in the hospital the patient is exposed to the risk of becoming colonized with antibiotic

resistant micro organisms. Strict isolation in a sterile environment (equipped with laminar flows) together with measures for skin and gastrointestinal tract decontamination and consumption of sterile food have been shown to reduce the episodes of infection.

Prophylactic oral antibiotics, such as ciprofloxacin or norfloxacin reduce the incidence of gram negative sepsis. Scrupulous hand washing by medical and health care personnel routinely before examining any patient of pancytopenia is a simple modality for infection prophylaxis.

The availability of recombinant growth factors like granulocyte colony stimulating factor (GCSF) or granulocyte macrophage colony stimulating factor (GM-CSF), and recombinant erythropoietin have enabled more specific management with improved outcome of the pancytopenic patients. The exact role of newer cytokines like recombinant human interleukin – 3 (IL-3) and interleukin-6 will gradually become better established in near future.

Immunosuppressive therapy with anti lymphocyte globulin (ALG) and/or cyclosporine has proved to be effective in achieving remission in aplastic anemia. Bone marrow transplantation (BMT) is a therapeutic option for suitable subsets of younger patients who have HLA matched siblings.

OUTCOME IN BICYTOPENIA AND PANCYTOPENIA:

Outcome depends on, early diagnosis of etiology and appropriate treatment and supportive care. Spontaneous recovery can occur in bicytopenia due to any transient illness. Viral diseases generally cause neutropenia attacks in the first 24-48 hours and it lasts for about 3 to 6 days. Tantawy et al,reported in their study that it takes 7 days to recover from neutropenia. Many other studies reported that temporary neutropenias may take 16 days to 2 months to recover to normal.

Unlike bicytopenia, spontaneous recovery from pancytopenia rarely occurs. If left untreated, severe pancytopenia has an overall mortality rate of approximately 50% within 6 months of diagnosis and more than 75% overall mortality. Infection and hemorrhage are the major causes of morbidity and mortality.

Zahide yalaki et al studied bicytopenia in pediatric patients and reported that the recovery period of bicytopenia was on average 6.5±2.1 day.

AIM AND OBJECTIVES OF THE STUDY

AIM AND OBJECTIVES OF THE STUDY

The primary objective of this study is to find out the clinical and etiological spectrum in children admitted with bicytopenia and pancytopenia in the Institute of child health and research centre, Government Rajaji hospital, Madurai. The secondary objective of this study is to follow up the children admitted with bicytopenia and pancytopenia for 18 months to find out the outcome and prognosis.

MATERIALS AND METHODS

MATERIALS AND METHODS

We conducted the study in the Institute of child health and research centre, Department of Pediatrics, Madurai Medical College, Government Rajaji hospital, Madurai. Ethical Committee of Madurai Medical College approved the study. This was a hospital based prospective study done over a period of 1 year and 6 months from April 2016 to July 2017.

Informed consent was taken from the parents before the study. From the total 9675 children, in the age group of 2 months to 12 years admitted in our institute during the study period, 264 of them had bicytopenia, 36 of them had pancytopenia, and they were included in this study. Detailed history, physical examination findings on admission was noted, complete hemogram, peripheral smear findings, Erythrocyte sedimentation rate(ESR), C-reactive protein , Culture reports, viral tests (IgM Elisa for Dengue, HbsAg, Anti HCV), Liver function tests, bone marrow aspiration/biopsy results, other relevant investigation reports were all recorded in profoma.

Complete hemogram was assessed by SYSMEX automated hematology analyzer. Blood counts obtained from automated analyzer were cross checked with pathologist peripheral smear report.

Cytopenia was defined as Hemoglobin <10gm%, Total leukocyte count <4000/mm^3, Platelet count <1lac/mm^3. Bicytopenia is defined as reduction in any of the two above parameters. Pancytopenia is defined as reduction in all three parameters.

Known acute leukemia and lymphoma, aplastic anemia, chronic idiopathic thrombocytopenic purpura patients who were diagnosed before the study period and on regular treatment in our institute during the study period were excluded from the study.

Bone marrow aspiration and biopsy was done as per the clinical indication. It was done in all pancytopenia cases, in bicytopenia cases with anemia, thrombocytopenia and leucocytosis. It was also done in children who had atypical cells or blast cells in peripheral smear report.

The diagnosis was established by morphological examination of bone marrow smears or biopsy and wherever required immunohistochemistry and cytogenetic analysis were done.

All bicytopenia and pancytopenia cases were followed up every fortnight with clinical examination and complete blood count in our hematology outpatient clinics on Saturday. During follow up we tried to find out the time interval between onset of cytopenia and diagnosis of leukemia or lymphoma i.e., we tried to find out the time duration of

bicytopenia and pancytopenia before it manifests as leukemia/lymphoma/aplastic anemia. We also assesed the short term outcome of bicytopenia and pancytopenia i.e., recovered or relapsed or treatment failure/death.

RESULTS

RESULTS

During the study period, out of 9675 admissions in the age group 2 months to 12 years for various clinical conditions in the Institute of child health and research centre, Government Rajaji hospital, Madurai, 300 children had either bicytopenia or pancytopenia with a frequency of 3.3 percent of total admissions were taken up for this study.

Of the 300 children 264 (88%) had bicytopenia and 36 (12%) had pancytopenia. As shown in Table-1, out of the 264 children with bicytopenia, 155 (59%) were male and 109(41%) were female with male-female ratio of 1.4:1.

Then, out of the 36 patients with pancytopenia 20 (56%) were male and 16(44%) were female, with male-female ratio of 1.25: 1.

TABLE -1: GENDER DIFFERENCE IN BI/PANCYTOPENIA

PARAMETERS	**MALE**	**FEMALE**	**TOTAL**
BICYTOPENIA	155(59%)	109(41%)	264
PANCYTOPENIA	20(56%)	16(44%)	36
TOTAL	175(58%)	125(42%)	300

FIGURE.1: GENDER DIFFERENCE IN BICYTOPENIA

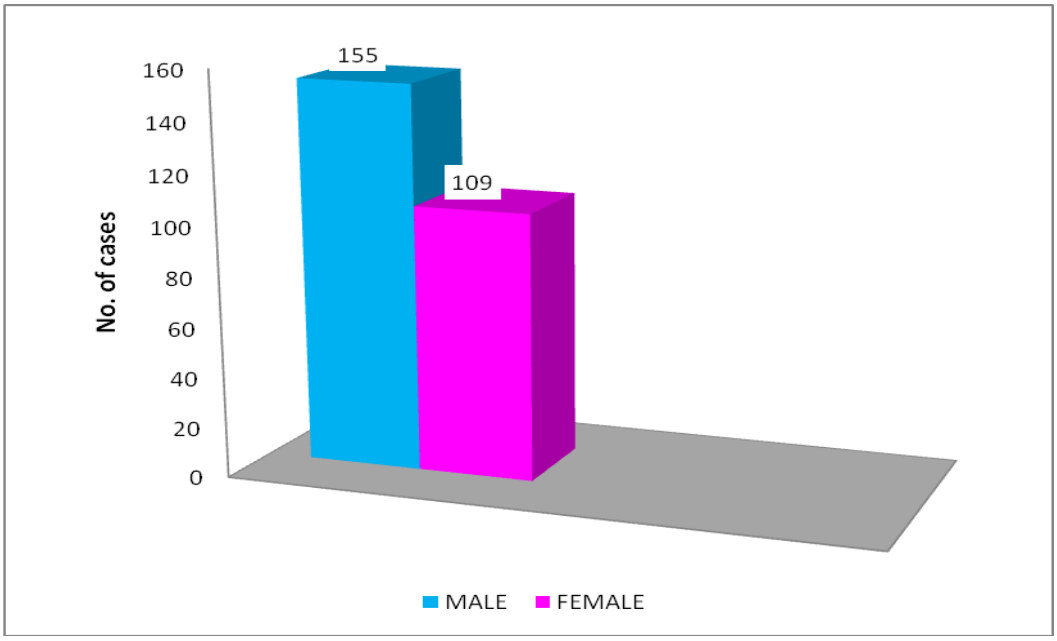
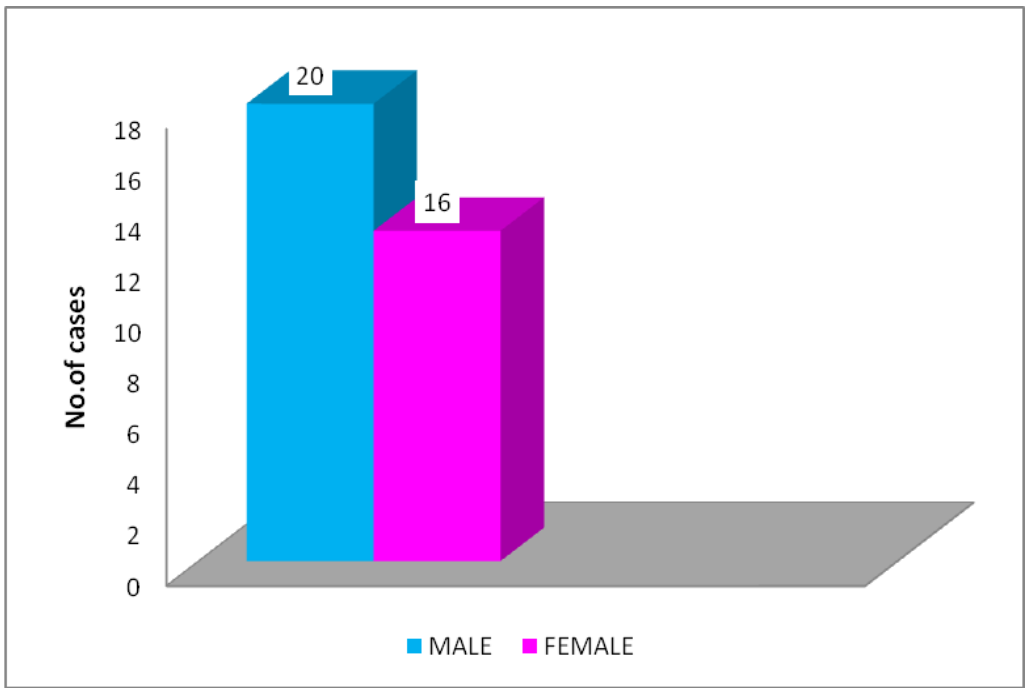


FIGURE.2: GENDER DIFFERENCE IN PANCYTOPENIA



Over all of 300 patients, 175 (58%) were male and 125(42%) were female with male-female ratio of 1.25: 1.

Of the total 264 children with bicytopenia, as per Table-2, maximum number of patients 142(54%) were in the age group of 7 to 12 years, followed by 108 (41%) in the age group of 1 to 6 years and least number 14(5%) in the age group of 2 months to 11 months.

Of the total 36 children with pancytopenia, as per Table-2, maximum number of patients 22 (61%) were in the age group of 1 to 6 years, followed by 8 (22%) in the age group of 2 months to 11 months and least number 6 (16%) in the age group of 7 to 12 years. Bicytopenia was more common in 7 to 12 years (54%). Pancytopenia was more common in 1 to 6 years of age (61%).

TABLE-2: AGE GROUP DISTRIBUTION IN BICYTOPENIA AND PANCYTOPENIA

AGE GROUP	**PANCYTOPENIA**	**BICYTOPENIA**
2MONTHS-12MONTHS	8(22%)	14(5%)
1-6 YEARS	22(61%)	108(41%)
7-12 YEARS	6(16%)	142(54%)
TOTAL	36	264

FIGURE 3- AGE GROUP DISTRIBUTION OF BICYTOPENIA

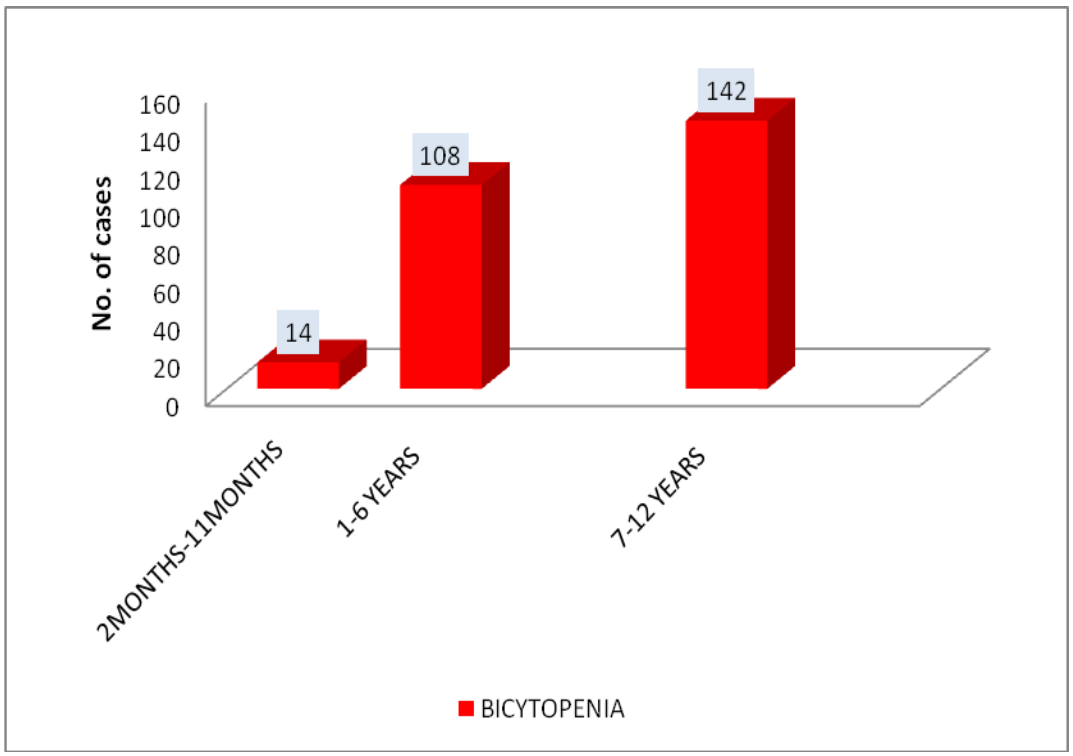
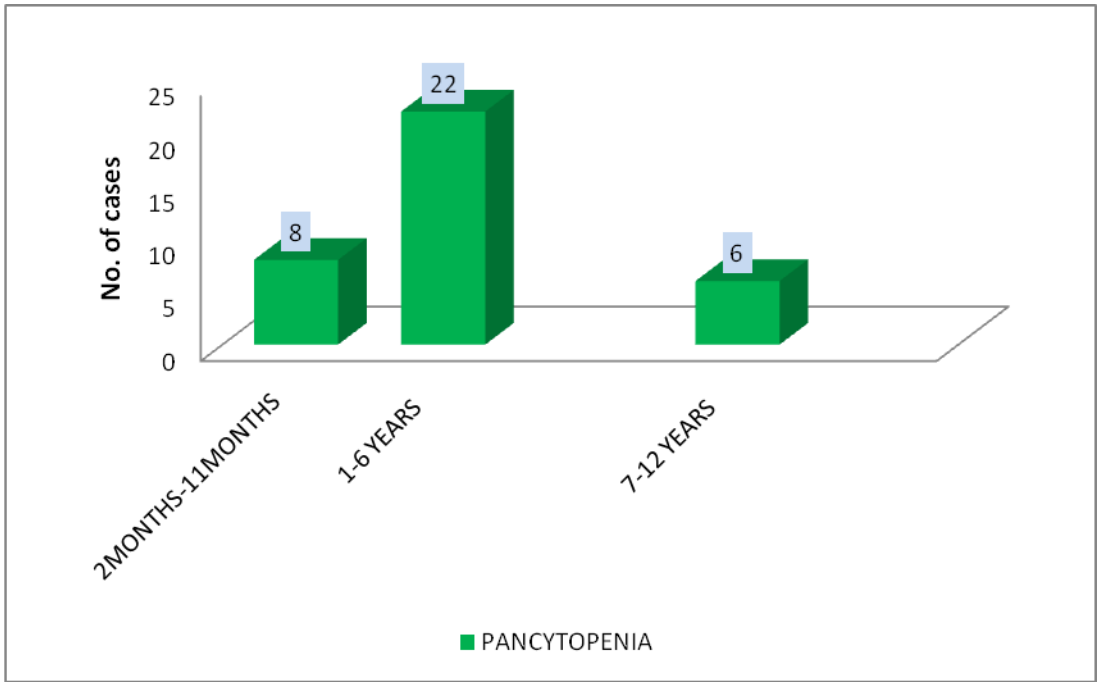


FIGURE 4- AGE GROUP DISTRIBUTION OF PANCYTOPENIA



HEMATOLOGICAL PROFILE:

As shown in Table-3, decreased hemoglobin count, decreased leukocyte count and decreased platelet count was seen in all the 36 cases of pancytopenia. In the bicytopenia patients, 259 cases presented with decreased platelet count, 179 cases presented with decreased total leucocyte count and 89 cases presented with decreased haemoglobin count. Hematologically, 264 children had bicyotpenia in complete hemogram testing using automated analyzer and confirmed by peripheral smear study.

Of the 264 children with bicytopenia, as shown in Table-4, 175 (66%) had leucopenia and thrombocytopenia, 84 (32%) children had anemia and thrombocytopenia, 5 cases (2%) had anemia and leucopenia. Most common form of bicytopenia in our set up was leucopenia and thrombocytopenia.

Circulating blasts were seen in 4(11%) cases of pancytopenia and 53(20%) cases of bicytopenia.

Bone marrow biopsy findings was hypercellular marrow with blast cells in cases of leukemia, hypoplastic bone marrow was seen in aplastic anemia. Peripheral smear study showed thrombocytopenia with giant platelets in Bernard Soulier syndrome.

TABLE-3: HEMATOLOGICAL PROFILE IN BICYTOPENIA AND PANCYTOPENIA

PARAMETERS	PANCYTOPENIA	BICYTOPENIA
Hemoglobin < 10 g/dl	36(100%)	89(34%)
Total leukocyte <4000/micro L	36(100%)	179(68%)
Platelet Count <1 lakh /micro L	36(100%)	259(98%)
Circulating Blasts	4(11%)	53(20%)

FIGURE-5: HEMATOLOGICAL PROFILE IN BICYTOPENIA

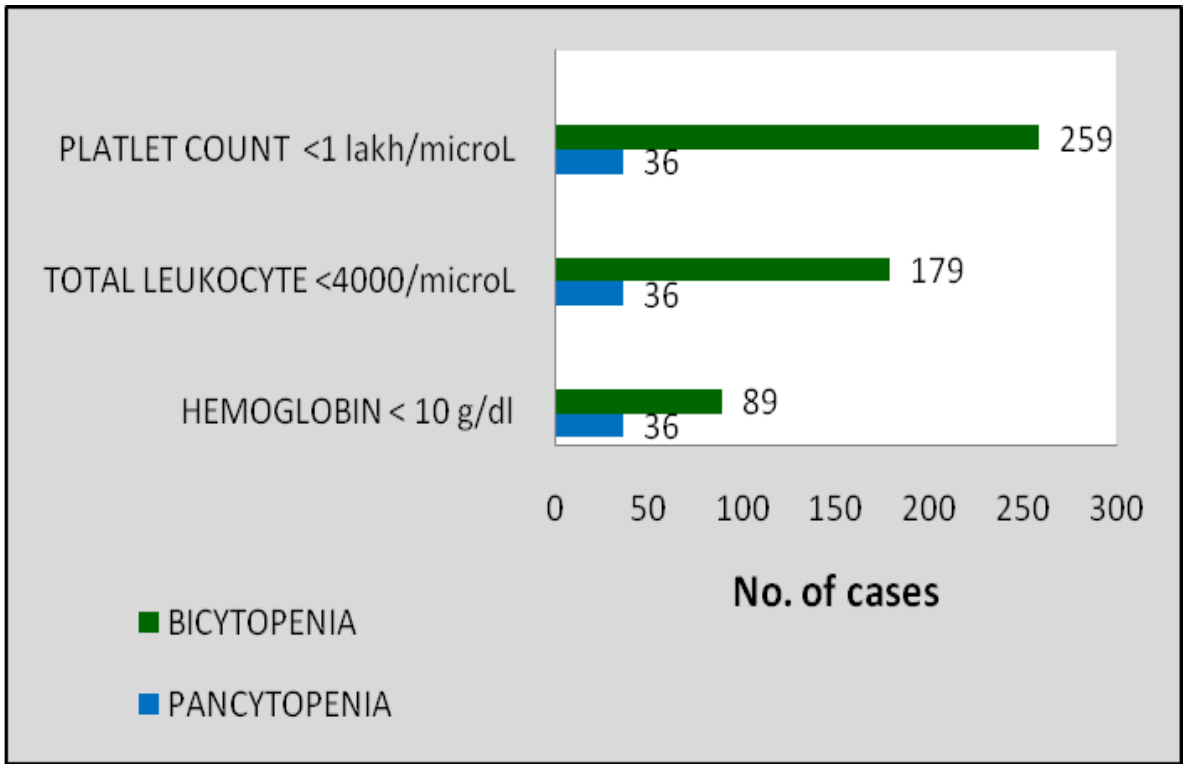
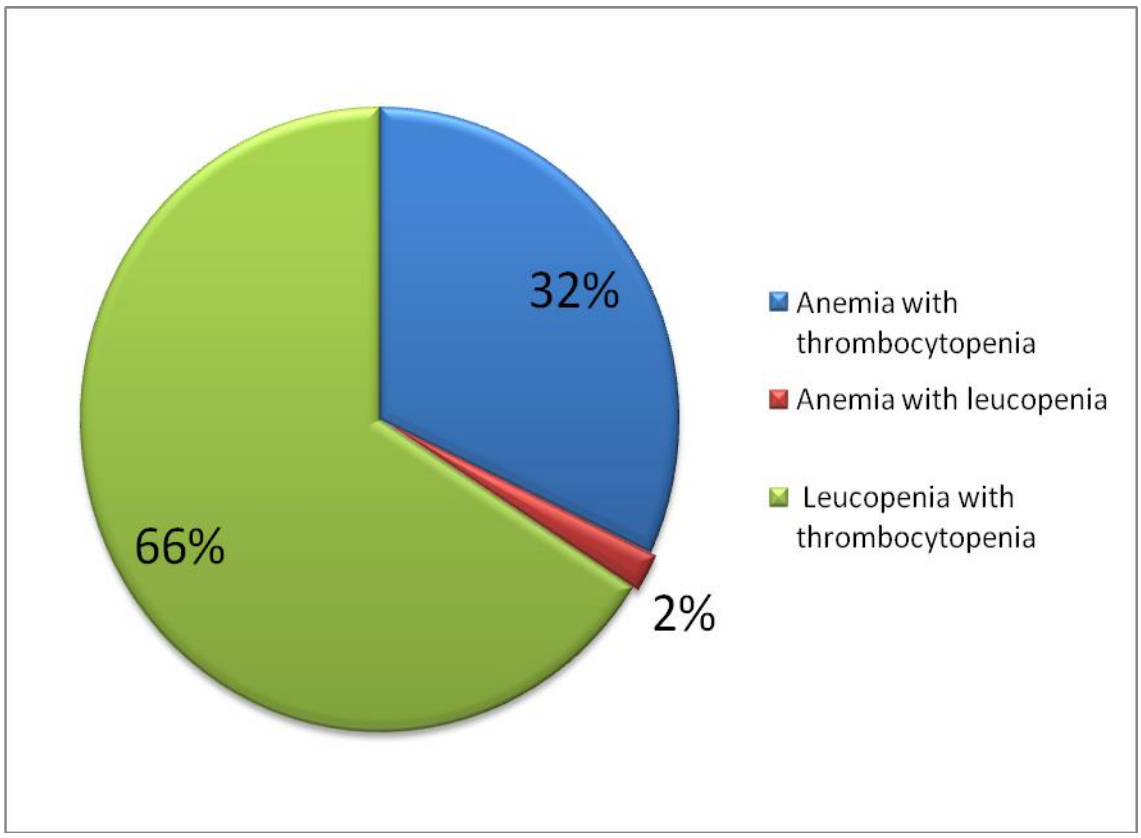


TABLE-4: PERIPHERAL BLOOD FINDINGS IN CHILDREN WITH BICYTOPENIA

TOTAL CASES OF BICYTOPENIA= 264 CASES

1	Leucopenia with thrombocytopenia	175 (66%)
2	Anemia with thrombocytopenia	84 (32%)
3	Anemia with leucopenia	5 (2%)

FIGURE-6: PERIPHERAL BLOOD FINDINGS IN CHILDREN WITH BICYTOPENIA



CLINICAL PROFILE:

Table- 5 and table- 6 summarize the clinical profile of children with bicytopenia and pancytopenia analyzed in our study.

BICYTOPENIA:

The main presenting symptoms as per table-5 in bicytopenia were fever in 247 (94%) patients, followed by abdominal pain 116 (44%) and vomiting 90 (34%). Other common symptoms were loss of appetite, lethargy, abdominal distension, joint pain and petechial rashes.

The common physical findings were pallor 99 (38%), followed by hepatomegaly 97 (37%) and splenomegaly 58 (22%).

PANCYTOPENIA:

The main presenting symptom as per table-5 in pancytopenia was fever in 31 (86%) cases followed by bleeding manifestations in 10 (28%) cases and lethargy in 8 (28%).Other common symptoms consisted of vomiting, loss of appetite and abdominal distension. Most common physical finding in children with pancytopenia was pallor 36 (100%), followed by hepatomegaly 13 (26%), splenomegaly 8 (22%), and lymphadenopathy in 32 (12%).

TABLE-5: FREQUENCY OF SYMPTOMS IN BICYTOPENIA AND PANCYTOPENIA

PRESENTING SYMPTOMS	PANCYTOPENIA	BICYTOPENIA
1)Fever	31 (86%)	247(94%)
2)Abdominal pain	4(11%)	116(44%)
3)Vomiting	6(17%)	90(34%)
4)Bleeding manifestations	10(28%)	24(9.7%)
a)Petechial rashes	3(8%)	8(3%)
b)Hemetemesis	-	4(2%)
c)Malena	2(6%)	2(0.7%)
d)Bleeding from gums	2(6%)	5(2%)
e)Epistaxis	3(8%)	5(2%)
5)Joint pain/leg pain	3(8%)	9(3%)
6)Jaundice	1(3%)	2(0.7%)
7)Loss of weight /appetite	7(19%)	23(9%)
8)Abdominal distension	7(19%)	17(6%)
9)Lethargy	8(22%)	18(7%)
10)Convulsion	4(11%)	3(1%)
11)Loose stools	2(6%)	7(3%)

FIGURE- 7: FREQUENCY OF SYMPTOMS IN BICYTOPENIA

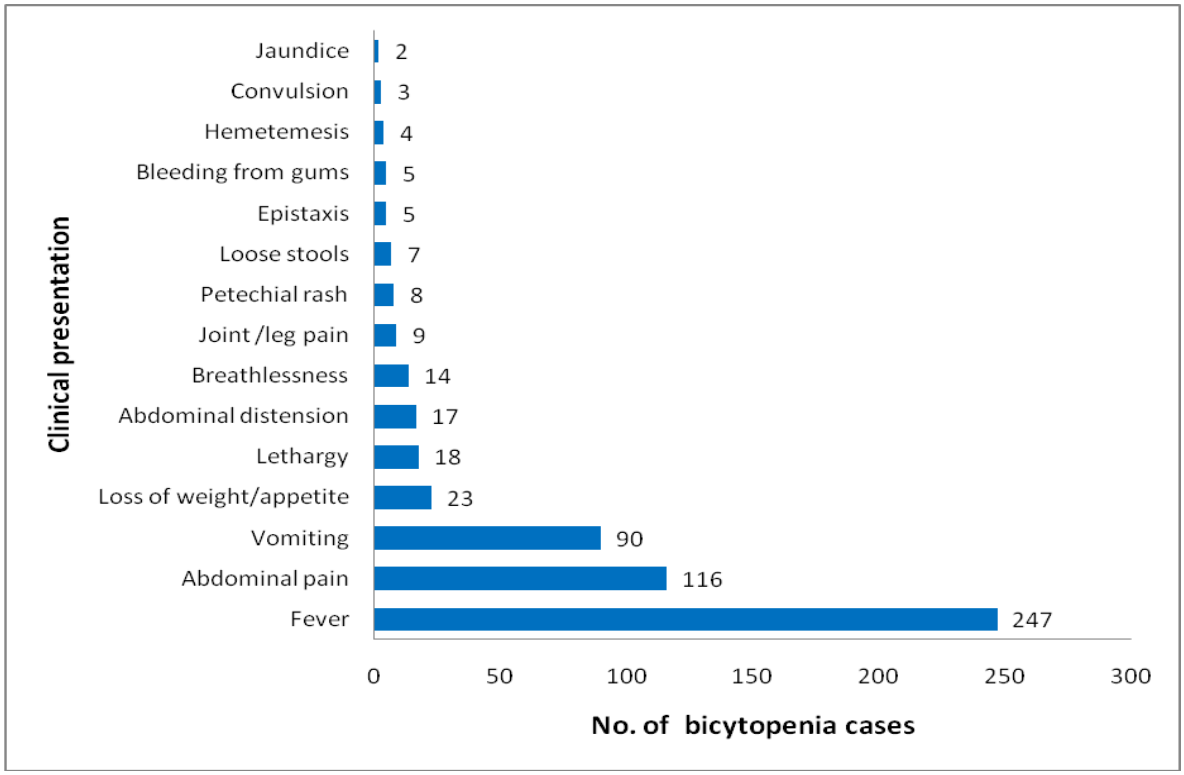


FIGURE- 8: FREQUENCY OF SYMPTOMS IN PANCYTOPENIA

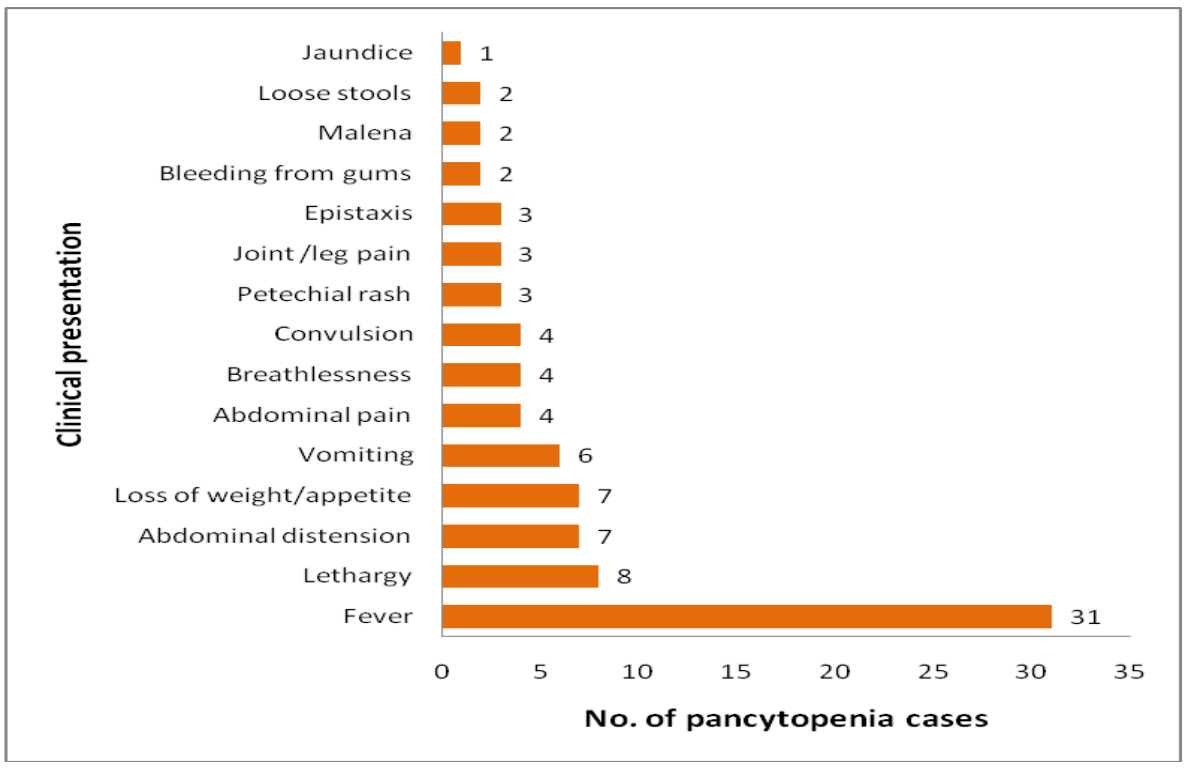


TABLE-6: FREQUENCY OF SIGNS IN BICYTOPENIA AND PANCYTOPENIA

CLINICAL SIGNS	PANCYTOPENIA	BICYTOPENIA
Pallor	36(100%)	99(38%)
Lymphadenopathy	4(11%)	32(12%)
Icterus	2(6%)	2(0.7%)
Hepatomegaly	13(36%)	97(37%)
Splenomegaly	8(22%)	58(22%)

ETIOLOGICAL PROFILE:

Based on the clinical examination findings, peripheral blood and bone marrow findings, the bicytopenic and pancytopenic children were divided into 4 groups

- Malignant
- Non malignant- infectious
- Non malignant-non infectious
- undiagnosed

FIGURE-9: FREQUENCY OF SIGNS IN BICYTOPENIA

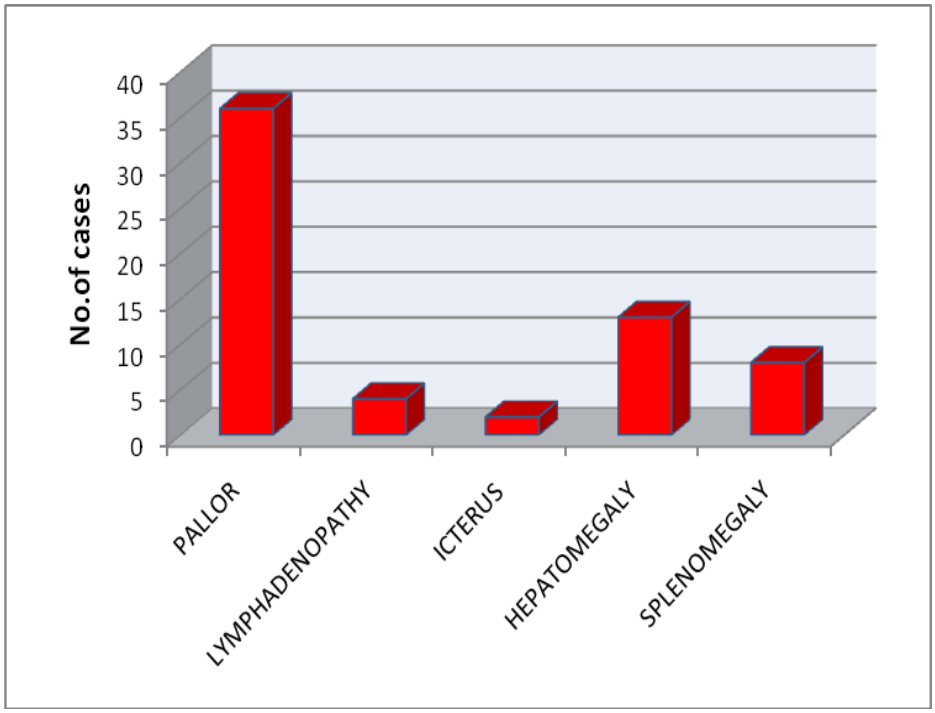
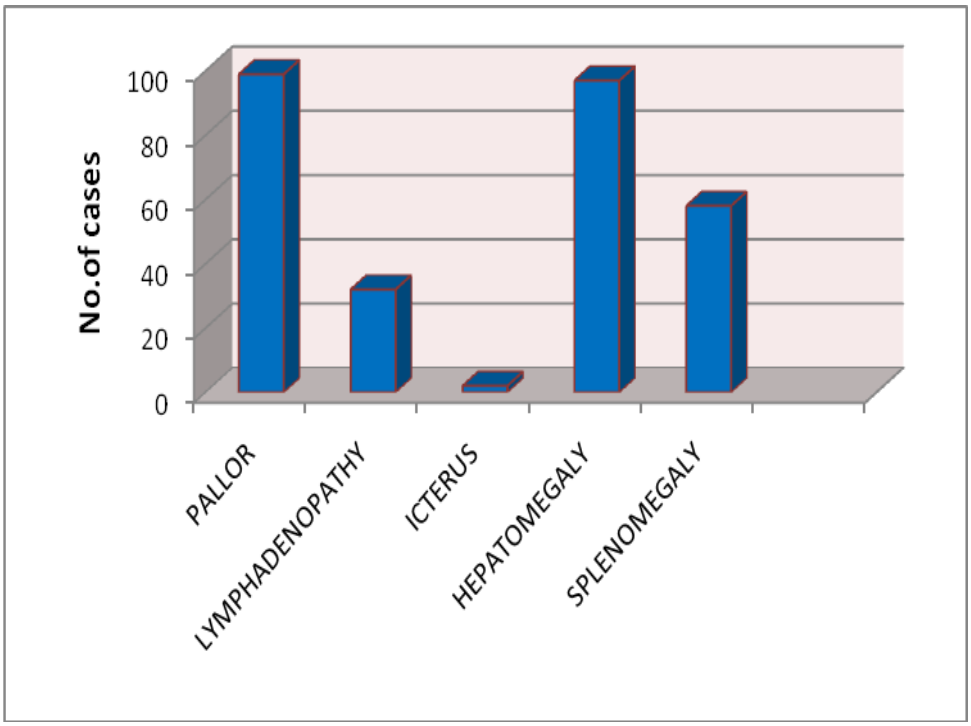


FIGURE-10: FREQUENCY OF SIGNS IN PANCYTOPENIA



ETIOLOGICAL PROFILE OF BICYTOPENIA:

As shown in table-7, out of the 264 children with bicytopenia 184(70%) cases were diagnosed as non malignant-infectious, 63(24%) cases were diagnosed as malignant, 9(3%) cases were diagnosed as non malignant non infectious, 8(3%) were undiagnosed.

As per Table-8, Most common non malignant infectious conditions causing bicytopenia were dengue fever 162(61%), followed by septicemia 18(6%) and enteric fever 4(1.5%).

Most common malignant condition causing bicytopenia was acute lymphoblastic leukemia 50(19%) followed by acute myeloid leukemia 10(4%) and lymphoma 3(1%).

Most common non malignant noninfectious conditions causing bicytopenia was chronic liver disease with portal hypertension in 3(1%) cases.

TABLE-7: ETIOLOGICAL PROFILE OF BICYTOPENIA

ETIOLOGICAL PROFILE	NO.OF CASES OF BICYTOPENIA
Non malignant- infectious	184 (70%)
Malignant	63 (24%)
Non malignant- non infectious	9 (3%)
Undiagnosed	8 (3%)

TABLE -8: ETIOLOGICAL PROFILE OF BI/PANCYTOPENIA

ETIOLOGY	PANCYTOPENIA	BICYTOPENIA
MALIGNANT		
ALL	4(11%)	50(19%)
AML	-	10(4%)
LYMPHOMAS		3(1%)
INFECTIOUS		
Dengue fever	4(11%)	162(61%)
Septicemia	13(36%)	18(6%)
Enteric fever	-	4(2%)
Tuberculosis	3(8%)	-
Falciparum malaria	1(3%)	-
NON INFECTIOUS		
Aplastic anemia	3(8%)	-
Chronic liver disease with portal hypertension	1(3%)	3(1%)
Neonatal hepatitis	1(3%)	1(0.3%)
SLE	1(3%)	-
Bernard soullier syndrome	-	1(0.3%)
Hereditary spherocytosis	-	1(0.3%)
Wiskott aldrich syndrome	-	1(0.3%)
Gauchers disease	-	1(0.3%)
Megaloblastic anemia	-	1(0.3%)
Undiagnosed	5(14%)	8(3%)

FIGURE-11: ETIOLOGICAL PROFILE OF BICYTOPENIA

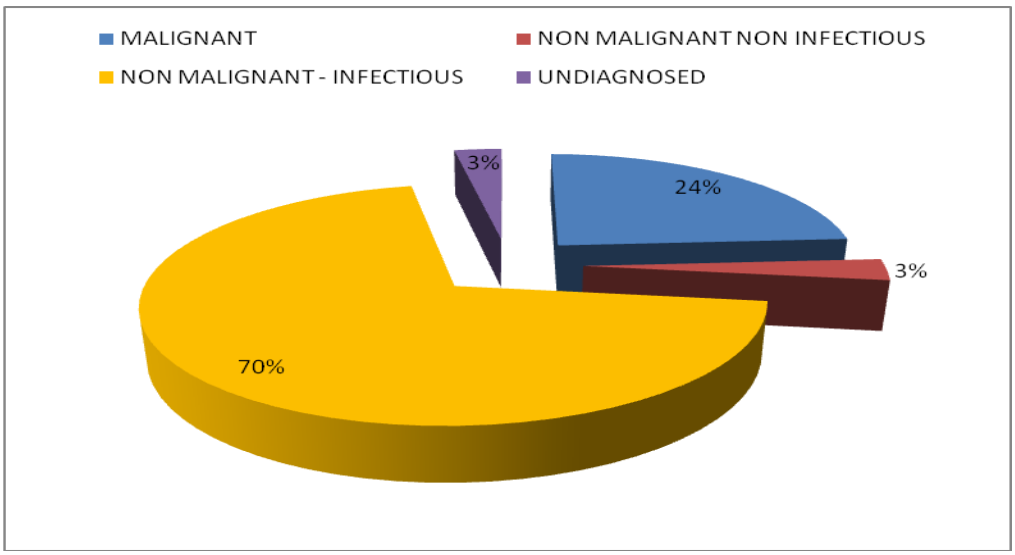
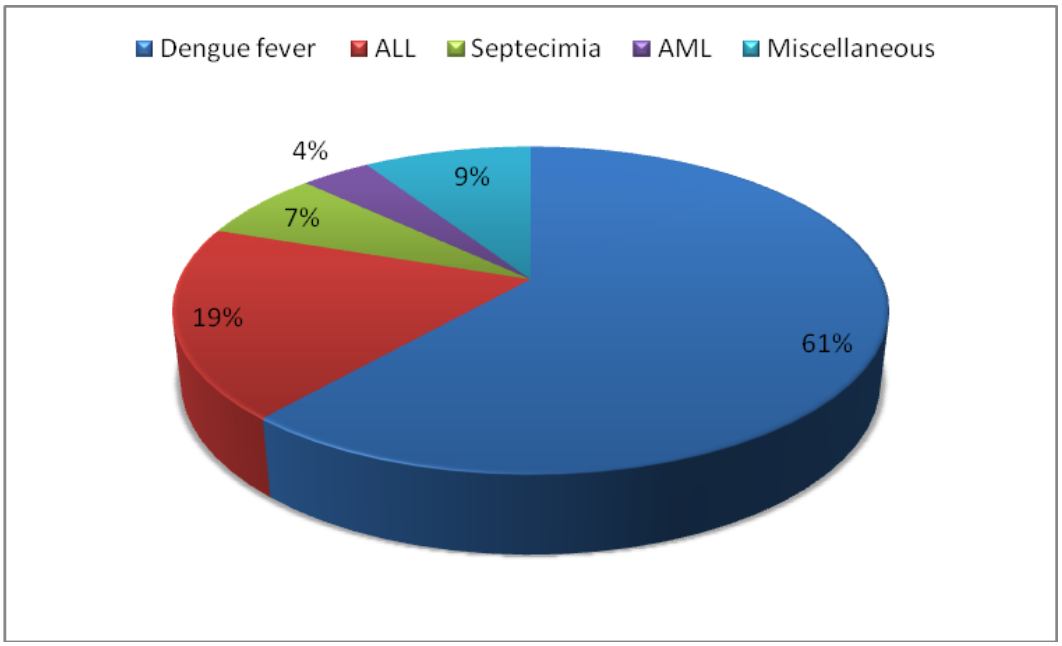


FIGURE-12: COMMONEST CAUSES IN BICYTOPENIA



Of the total 18 cases of septicemia, 9 cases were due to CONS sepsis, 7 cases were due to gram negative klebsiella sp, in 2 cases, clinical features of sepsis present but cultures showed no growth.

TABLE -8.1 - ETIOLOGICAL PROFILE IN BICYTOPENIA

S.No	BICYTOPENIA	ETIOLOGY	No. OF CASES
1	Leucopenia with thrombocytopenia Total -175 (66%)	Dengue viral fever	161 (92%)
		Septicemia	10 (6%)
		Enteric fever	4 (2%)
2	Anemia with thrombocytopenia Total -84 (32%)	ALL	50 (60%)
		AML	9 (11%)
		Septicemia	7 (8%)
		Lymphoma	3 (4%)
		CLD with portal hypertension	2 (2%)
		Bernard soulier syndrome	1 (1%)
		Wiskott Aldrich syndrome	1 (1%)
		Gaucher's disease	1 (1%)
		Neonatal hepatitis	1 (1%)
		Dengue Viral fever	1 (1%)
		Undiagnosed	8 (10%)
3	Anemia with leucopenia Total -5 (2%)	AML	1(20%)
		Chronic liver disease with portal hypertension	1(20%)
		Megaloblastic anemia	1(20%)
		Hereditary spherocytosis	1(20%)
		Septicemia	1(20%)

In our study, as per table 8.1 the most common causes of leucopenia with thrombocytopenia are dengue fever 161(92%) followed by septicemia 10(6%) and enteric fever 4(2%). So the most common form of bicytopenia, leucopenia with thrombocytopenia was caused by infections (100%). So if the child had leucopenia with thrombocytopenia on admission, infectious etiology should be considered first.

In our study the common causes of anemia with thrombocytopenia 84(32%) are acute lymphoblastic leukemia 50(60%), followed by acute myeloid leukemia 9 (11%), septicemia 7(8%), lymphoma 3(4%), chronic liver disease with portal hypertension, Bernard soulier syndrome, gaucher disease, neonatal hepatitis and dengue fever. So, if child presents with anemia and thrombocytopenia, malignancy (75%) should be considered first.

In our study anemia and leucopenia 5(2%) are caused by AML, chronic liver disease with portal hypertension, megaloblastic anemia, hereditary spherocytosis, septicemia.

ETIOLOGICAL PROFILE OF PANCYTOPENIA:

As shown in table -9, out of the 36 children with pancytopenia, 21(58%) cases were diagnosed as non malignant-infectious, 6(17%) cases were diagnosed as non malignant non infectious,4 (11%) cases were diagnosed as malignant and 5(14%) were undiagnosed.

As shown in Table -8, Most common infectious condition causing pancytopenia was septicemia 13(36%), followed by dengue fever 4(11%), Tuberculosis 3(8%) and falciparum malaria(3%). Most common malignant condition causing pancytopenia was acute lymphoblastic leukemia 4(11%). Most common non malignant noninfectious condition causing pancytopenia was aplastic anemia 3(6%), followed by chronic liver disease with portal hypertension 1(3%), SLE 1(3%) and Neonatal hepatitis 1(3%).

TABLE-9: ETIOLOGICAL PROFILE IN PANCYTOPENIA

ETIOLOGICAL PROFILE	NO.OF CASES OF PANCYTOPENIA
Non malignant - infectious	21(58%)
Non malignant non infectious	6(17%)
Malignant	4(11%)
Undiagnosed	5(14%)

FIGURE-13: ETIOLOGICAL PROFILE IN PANCYTOPENIA

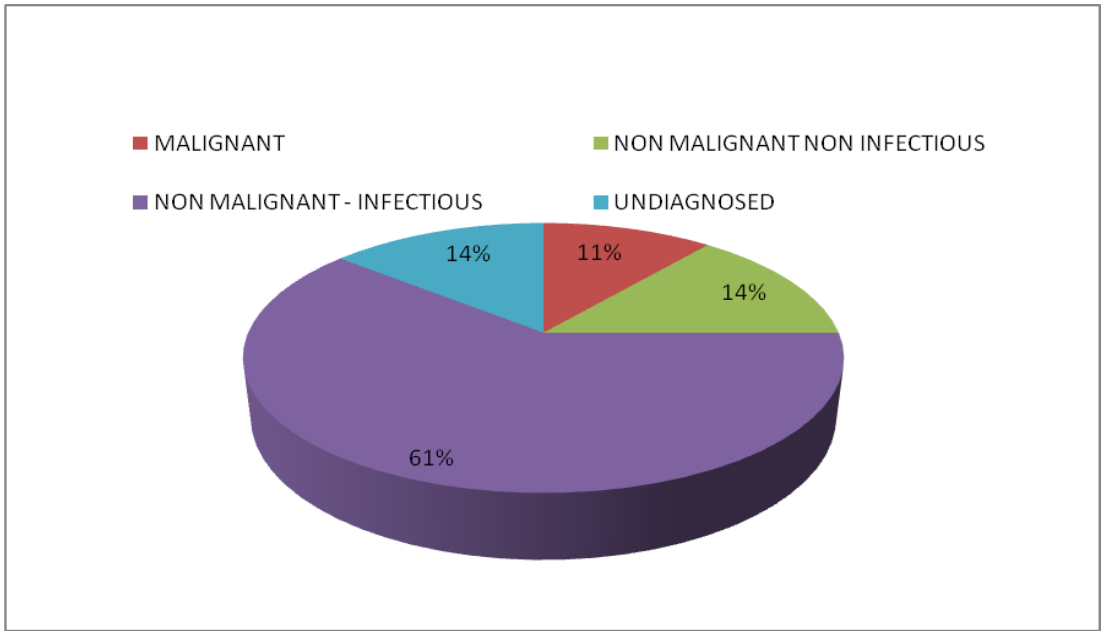
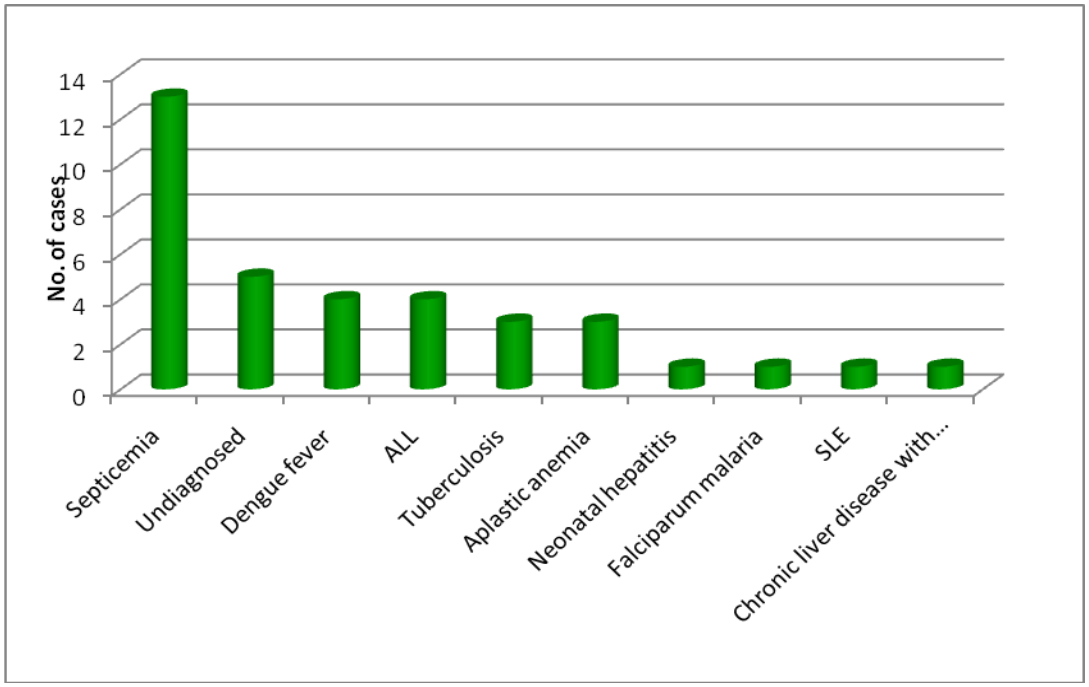


FIGURE-14: COMMONEST CAUSES IN PANCYTOPENIA



Of the total 18 cases of septicemia, 5 cases were due to CONS (coagulase negative staphylococcus aureus), 4 cases were due to gram negative klebsiella pneumoniae, and 4 cases had clinical features of sepsis but cultures showed no growth.

OUTCOME AND FOLLOW UP:

As shown in table 10, the outcome of children with bicytopenia/pancytopenia was as follows,

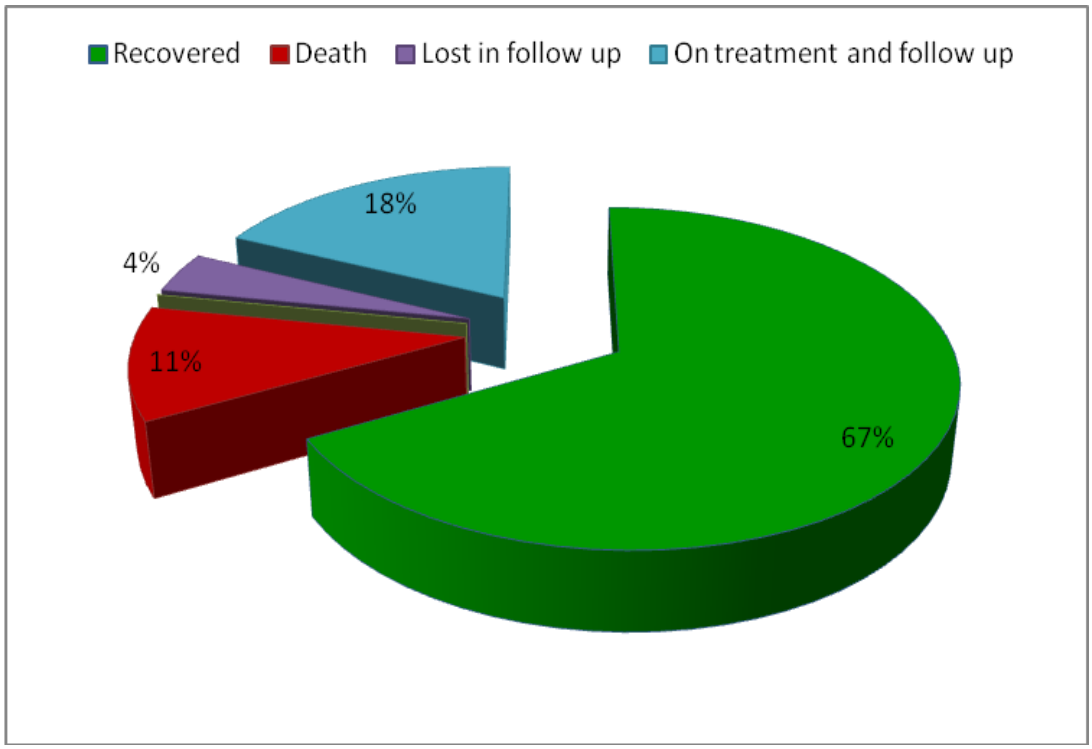
TABLE-10: OUTCOME IN CHILDREN WITH BICYTOPENIA AND PANCYTOPENIA

OUTCOME	PANCYTOPENIA	BICYTOPENIA
Recovered	10 (28%)	176(67%)
Death	12(33%)	30(11%)
Lost in follow up	2(6%)	10(4%)
On treatment/ follow up	12(33%)	48(18%)
Total	36	264

Recovered means child free from illness after therapy and normalization of peripheral blood counts. Death includes those children who died during treatment either due to disease or its complications.

OUTCOME AND FOLLOW UP IN BICYTOPENIA: As given in table-10, of the total 264 cases, 176(67%) children recovered, and 30 (11%) cases died. 48 (18%) were undergoing treatment and 10 cases (4%) were lost in follow up.

FIGURE- 15: OUTCOME OF CHILDREN WITH BICYTOPENIA



Outcome in children with bicytopenia depend on the etiology. As shown previously, the common infectious conditions causing bicytopenia were dengue fever 162(61%), septicemia 18(6%) and enteric fever 4(1.5%). Of the 162 cases of dengue fever, 161 cases (99%) recovered completely and 1 case died due to refractory shock, uncontrolled GI bleeding ie, malena.

OUTCOME IN DENGUE FEVER	NO. OF CASES
DIED	1(1%)
RECOVERED	161(99%)
TOTAL	162

Of the total 18 cases of septicemia, 9(50%) cases died during treatment and 9(50%) cases recovered after treatment. The cause of death in septicemia was shock and disseminated intravascular coagulation.

As shown previously, Most common malignant condition causing bicytopenia was acute lymphoblastic leukemia 50(19%) followed by acute myeloid leukemia 10(4%). Of the total 50 cases of ALL, 12 (24%) children died during treatment. Most common cause of death in leukemic children was febrile neutropenia, septicemia and septic shock. 3(6%) children were lost in follow up. 35(70%) cases were undergoing regular treatment and on regular follow up. Of the total 10 cases of AML, 4 (40%) cases died during treatment and remaining 6(60%) children undergoing treatment and follow up.

OUTCOME	ALL	AML
DIED	12(24%)	4(40%)
LOST IN FOLLOW UP	3(6%)	-
ON TREATMENT & FOLLLOW UP	35(70%)	6(60%)

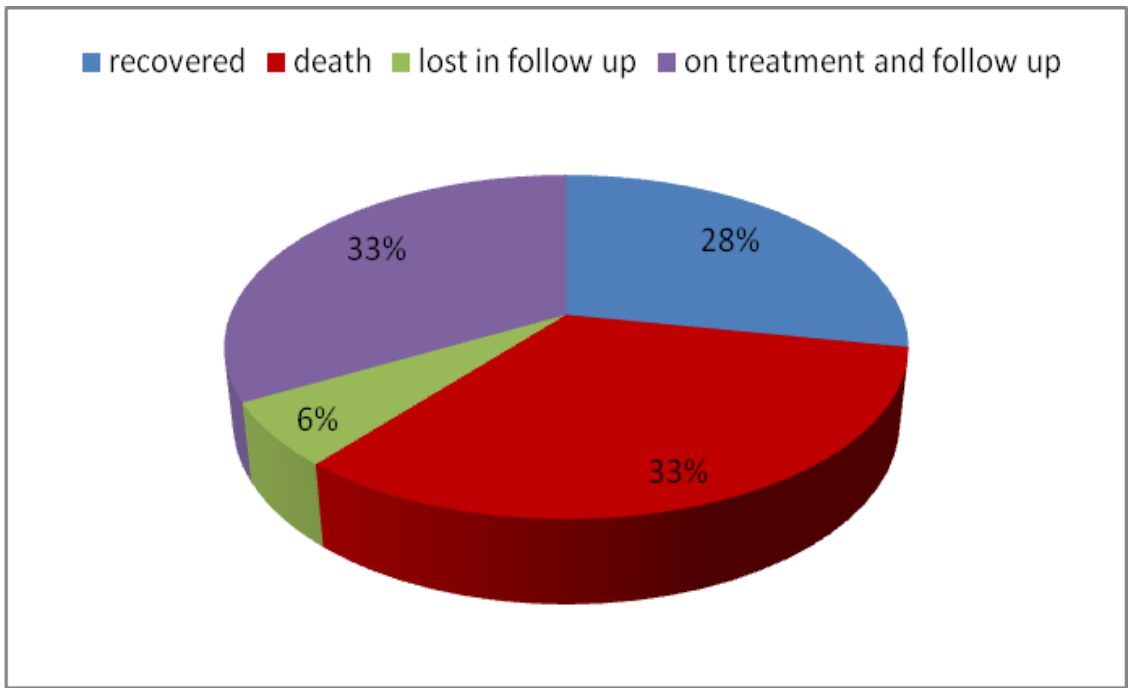
FOLLOWUP RESULTS OF BICYTOPENIAC CASES:

Of the 264 cases of bicytopenia, in 8 cases the etiological diagnosis of bicytopenia was not confirmed after admission. Of the 8 cases, 2 cases were lost in follow up, 2 cases had only fever and bicytopenia and they recovered with antibiotics and antipyretics. 4 cases died during treatment due to various reasons like multiorgan dysfunction, OPC poisioning, congestive cardiac failure, platelet disorder and anemia of chronic disease. The cause of bicytopenia in the above cases could not be explained and all the 4 cases died within 24 hours of admission.

OUTCOME IN PANCYTOPENIA:

As given in table-10, of the total 36 cases, 12 (33%) were on treatment and follow up, 10 cases (28%) recovered, 12(33%) died, and 2 cases (6%) were lost in follow up. As shown previously, the common infectious conditions causing pancytopenia were septicemia 13(36%), followed by dengue fever 4(11%), and Tuberculosis 3(8%). Most common malignant condition causing pancytopenia was acute lymphoblastic leukemia 4(11%).

FIGURE 16: OUTCOME OF CHILDREN WITH PANCYTOPENIA



Of the total 13 cases of septicemia, 9 (69%) cases died during treatment, 4(31%) cases recovered.

TABLE - OUTCOME IN CHILDREN WITH PANCYTOPENIA

Outcome in pancytopenia	Septicemia	Dengue fever	ALL	Tuberculosis	others
Died	9(69%)	-			3
Recovered	4(31%)	4(100%)		2(66%)	2
On treatment and follow up		-	4(100%)	1(33%)	7
Total	13	4	4	3	12

Of the 4 cases of Dengue fever, all 4 were recovered. Of the 4 cases of ALL, all 4 were under treatment and follow up. Of the 3 cases of tuberculosis, 2 cases recovered with antituberculus treatment and 1 case was on treatment and follow up.

Of the total 12 deaths in pancytopenia , 9 deaths due to septicemia, 1 death due to chronic liver disease with portal hypertension,1 death due to neonatal hepatitis and 1 death due to unknown cause.

CASE REPORTS OF IMPORTANT PANCYTOPENIA CASES:

All children with pancytopenia, were followed up. On follow up, 1 case died within 24 hours of admission, so the cause of pancytopenia was not known and could not explained.

In one case initial peripheral smear study and bone marrow aspiration was inconclusive showed pancytopenia for evaluation. On follow up after 2 months, complete hemogram showed MCV -114 fl. Peripheral smear study showed normochromic macrocytes with normal platelets. Bone marrow aspiration showed megaloblastic and normoblastic erythropoiesis and vitamin B12 levels were low 122pg/m(normal 187-883pg/m), diagnosis of megaloblastic anemia was made. So, this case was started on vitamin B12 therapy, but this case was lost in follow up so outcome could not be assessed.

In one case with Grade 4 PEM, complete blood count showed pancytopenia, while other investigations could not be done as child was lost in follow up.

During follow up, a 2 year old male child, admitted with pancytopenia, initial bone marrow aspiration showed dimorphic anemia / megaloblastic anemia, so treated with vitamin B12 and blood transfusion. Initially cytopenia was corrected, later after 3 months child again admitted with pancytopenia blood transfusions given and discharged. After 6 months the child admitted with bleeding per rectum, hematuria, and epistaxis. His peripheral blood counts showed pancytopenia and bone marrow examination showed hypoplastic marrow, markedly hypocellular suggestive of aplastic anemia, now the child is undergoing treatment.

An 8 year old female child, presented with fever and pancytopenia, bone marrow showed normoblastic maturation and no blast cells. ESR 160mm/hr, mantoux test positive, mother open case on treatment. Disease was confirmed by gastric juice gene xpert Analysis, started treatment with ATT. But after 3 months child admitted again with fever and hemetemesis, blood transfusions given and child discharged. After 6 months child again got admitted with fever, pallor and hepatosplenomegaly. Her peripheral smear showed pancytopenia and bone marrow examination showed myeloblasts 50% with prominent

nucleoli and Auer rods in cytoplasm suggestive of AML. Now the child is on cancer chemotherapy for AML and she is showing good response to treatment.

An 11 months old female child, admitted with fever and vomiting for 5 days, on examination child had pallor, moderate hepatosplenomegaly. Her peripheral smear showed pancytopenia, NS1 Ag was positive, IgM antibody for dengue positive. Child was treated as dengue fever, platelet count started increasing and child was afebrile for 2 days. On day 9 of illness, child again developed fever. Further fever workup done, septicemia, malaria, Scrub typhus, Tuberculosis and malignancy ruled out. Since diagnosis was not made, hemophagocytic lymphohistiocytosis was suspected, further investigations revealed hypertriglyceridemia (1054mg/dl) and increased serum ferritin (5124ng/ml). So, the child was diagnosed as secondary haemophagocytic lymphohistiocytosis, started on oral prednisolone and child completely recovered with treatment.

A 3 year old male child admitted with fever, pallor and hepato splenomegaly. Peripheral smear study showed normochromic normocytic anemia, leucopenia, no blast cells and platelets reduced in number. Bone marrow was diluted with peripheral blood. Child was absconded from hospital. Later after 4 months child again admitted with fever and

bicytopenia. Peripheral smear study showed normochromic normocytic anemia, lymphoblasts 15% and platelets decreased. Bone marrow showed hypercellular marrow with lymphoblasts 65%, suggestive of acute lymphoblastic leukemia. Now child is on treatment and follow up.

DISCUSSION

DISCUSSION – BICYTOPENIA

There are only very few data reported on clinical and etiological spectrum in children with bicytopenia. The published studies have also not stated about the outcome and prognosis in these children. Bicytopenia can be due to transient viral illness or life threatening malignancy. Different population groups show difference disease patterns based on their genetic pattern, prevalent infections in that location, and also their nutritional status. Etiological profile also shows difference with research methodology.

AGE AND GENDER DIFFERENCE:

During our study period, of the total admissions in our institute, 2.7% of children have bicytopenia. Out of that almost $3/5^{th}$ are male children and remaining $2/5^{th}$are female children, with male to female ratio 1.4:1. There is slight male predominance in children with bicytopenia in our study. This result is similar to the study done by Zahide et al.

In children with bicytopenia, little more than half are in age group 7 -12 years, followed by children in age group 1 -6 years and small percentage in age group 2 months to 12 months. Bicytopenia is more common between 1 to 12 years. It is rare in infancy. This result is similar to the study done by Zahide et al.

HEMATOLOGICAL PROFILE IN BICYTOPENIA:

In our study, out of the all children admitted with bicytopenia, 2/3rd has leucopenia and thrombocytopenia, almost 1/3rd has anemia and thrombocytopenia and very few children has anemia and leucopenia. So the most common bicytopenia in our study is leucopenia and thrombocytopenia. This in contrary to the study done by Shano naseem et al, in which, the most common bicytopenia was anemia and thrombocytopenia. The reason for this disparity is, our study is a prospective study, done in children admitted in pediatric ward with bicytopenia in southern part of Tamil nadu and Shano naseem et al study was a retrospective study, done in pathology department in northern part of india. The etiological profile in children with bicytopenia differs in both the studies. Most common cause of bicytopenia in our study is dengue fever. The second most common cause of bicytopenia in our study is acute lymphoblastic leukemia

In our study the most common causes in children with leucopenia and thrombocytopenia are dengue fever in 9/10th of cases and remaining 1/10th are caused by septicemia and enteric fever. Infections are the cause of leucopenia with thrombocytopenia in almost all children. So if child presents with leucopenia and thrombocytopenia, infectious etiology should be investigated first.

In our study the most common causes in children with anemia and thrombocytopenia are acute lymphoblastic leukemia, followed by acute myeloid leukemia and septicemia and lymphoma.

In 3/4th of children the cause of anemia with thrombocytopenia is malignancy. So if child presents with anemia and thrombocytopenia, malignancy should be investigated first. Remaining 1/4th are due to septicemia and others.

So in our study, infections commonly present with leucopenia and thrombocytopenia and malignancy commonly presents with anemia and thrombocytopenia.

In our study anemia and leucopenia are caused by AML, chronic liver disease with portal hypertension, megaloblastic anemia, hereditary spherocytosis, septicemia.

CLINICAL SPECTRUM IN BICYTOPENIA:

In our study the common presenting symptoms in children with bicytopenia are fever, followed by abdominal pain and vomiting. Other less common symptoms are bleeding manifestations like petechial rashes, gum bleeding, epistaxis, hemetemesis and malena, loss of weight/appetite, lethargy and abdominal distension. So in our study the most common presenting symptom in children with bicytopenia is fever,

which is similar to the studies done by Zahide yalki et al and Shano Naseem et al.

In our study, common clinical signs in children with bicytopenia are pallor, followed by hepatomegaly and splenomegaly. On comparison, with shano naseem et al study the incidence of pallor is almost similar in both, but the incidence of hepatomegaly and splenomegaly is lower in our study.

COMPARISION OF CLINICAL FINDINGS IN BICYTOPENIA

STUDY	FEVER	HEPATOMEGALY
Present study(n=264)	247(94%)	97(37%)
Shano Naseem et al(n=347)	240(69.2%)	240(69.2%)
Zahide Yalaki et al(n=28)	19(67.8%)	3(10.7%)

ETIOLOGICAL SPECTRUM IN BICYTOPENIA:

In our study, almost 2/3rd cases of bicytopenia are due to infectious etiology, of the remaining 1/3rd majority are due to malignant etiology, very few due to non malignant non infectious etiology. In our study, most common infections causing bicytopenia are dengue fever followed by septicemia and enteric fever. Of the total cases of septicemia, approximately half is due to CONS sepsis and almost 1/3rd is due to klebsiella pneumonia sp.

In our study the common malignancies presented with bicytopenia are ALL followed by AML. Most of the acute leukemias presents as bicytopenia (anemia with thrombocytopenia) rather than pancytopenia.

In our study the common non malignant non infectious etiology causing bicytopenia are chronic liver disease with portal hypertension, followed by neonatal hepatitis, Bernard soulier syndrome, hereditary spherocytosis, Wiskott Aldrich syndrome, Gaucher's disease and megaloblastic anemia.

Our study results are similar to the study done by Zahide et al, in which 2/3rd of bicytopenia was due to infection followed by acute leukemia. Our study report is similar to the study done by shano naseem et al, in which the commonest malignant condition causing bicytopenia was acute lymphoblastic leukemia.

COMPARISION OF ETIOLOGY IN BICYTOPENIA:

STUDY	Most common cause	Second common cause
Present study(n=264)	Dengue fever 162(61%)	Acute leukemia 60(23%)
Shano Naseem et al (n=347)	Acute leukemia 232 (66.9%)	Idiopathic thrombocytopenic purpura
Zahide Yalaki et al(n=28)	Viral infections 18(64.2%)	Acute lymphoblastic leukemia 4(14.2%)

OUTCOME AND FOLLOW UP IN BICYTOPENIA:

In our study, almost 2/3rd children with bicytopenia recovered. Of the remaining 1/3rd majority of children is on regular treatment and follow up as they have malignancy.

In dengue fever cases almost all recovered. Recovery rate in children with bicytopenia is more because most cases are due to infections.

In ALL patients, 2/3rd is on regular treatment & follow-up and the remaining 1/3rd died. In AML patients 3/5th are on regular treatment and follow up, and the remaining 2/5th died. In leukemia, almost all died due to febrile neutropenia, septicemia and shock.

In cases of septicemia, almost half of the children who presented with bicytopenia died due to septic shock and DIC and remaining half recovered after treatment. So, in septicemia if child presents with bicytopenia the mortality is high, almost 50%.

DISCUSSION – PANCYTOPENIA

Pancytopenia itself is not a disease entity; it means reduction in all 3 peripheral blood lineages red blood cells, leukocytes, platelets. There is only minimal number of studies done in India evaluating pancytopenia in children. The published studies have also not stated about the outcome.

AGE AND GENDER DIFFERENCE:

During our study period, of the total admissions in our institute, 0.37% of children have pancytopenia. Of that almost $3/5^{th}$ are male children and $2/5^{th}$ are female children, with male to female ratio 1.25:1. There is slight male predominance in children with pancytopenia in our study. This result is similar to the study done by Shazia menon et al and Gunavanthi et al in pediatric pancytopenic patients.

In children with pancytopenia, $3/5^{th}$ of children are in the age group 1-6 years. Of the remaining, $1/5^{th}$ is in the age group 2months -12months and $1/5^{th}$ are in age group 7 to 12 years. In our study, pancytopenia is common between 1 to 6 years of age. This result is similar to the study done by Ghanshyam singh et al in children with pancytopenia with special reference to non malignant presentation.

CLINICAL SPECTRUM IN PANCYTOPENIA:

In our study the most common presenting symptoms in children with pancytopenia are fever followed by bleeding manifestations like petechial rashes, gum bleeding, epistaxis and malena , lethargy, abdominal distension and loss of weight/appetite . So in our study the most common presenting symptom in children with pancytopenia is fever, which is similar to the studies done by Ghanshyam singh et al and Shano Naseem et al.

In our study bleeding manifestations is the second most common presenting symptom. Bleeding manifestations are more common in pancytopenia than bicytopenia in our study.

In our study, almost all children with pancytopenia had pallor, 1/3rd of children had hepatomegaly and 1/4th had splenomegaly. This result is similar to the study done by shazia menon et al study in children with pancytopenia in which more than 3/4th had pallor. Pallor is the most common physical finding in children admitted with pancytopenia.

COMPARISION OF CLINICAL FINDINGS IN PANCYTOPENIA

STUDY	PALLOR	FEVER	HEPATOMEGALY
Present study(n=36)	36(100%)	31(86%)	13(36%)
Dubey SRK et al(n=170)	137(81%)	116(68%)	72(44.8%)
Shano Naseem et al(n=139)	82(59%)	91(65.5)	72(51.8%)

ETIOLOGICAL SPECTRUM IN PANCYTOPENIA:

In our study, 3/5th cases of Pancytopenia are due to infectious etiology, 1/5th due to non malignant non infectious etiology, and the remaining 1/5th are either due to malignant etiology or undiagnosed.

In our study, the common infections causing pancytopenia are septicemia in 1/3rd of cases followed by dengue fever, tuberculosis and falciparum malaria. Thus most common cause of pancytopenia in our study is septicemia.

Of the total cases of septicemia, 1/3rd cases is due to CONS (Coagulase negative staphylococcus aureus), 1/3rd cases is due to gram negative klebsiella pneumoniae, and 1/3rd cases has clinical features of sepsis but cultures show no growth.

In our study the common non malignant non infectious etiologies causing pancytopenia are aplastic anemia followed by chronic liver

disease with portal hypertension, neonatal hepatitis and SLE. Non malignant non infectious etiology is the second common cause of pancytopenia in our study.

In our study most common non malignant non infectious etiology causing pancytopenia is aplastic anemia.

In our study common malignancy causing pancytopenia is acute lymphoblastic leukemia in 1/10th cases.

Many studies have been done in various part of the world in pancytopenia and each study has given different underlying etiology for pancytopenia. **Shano Naseem et al.,** from India in their study in pancytopenia children found that aplastic anemia was the most common etiology causing pancytopenia, followed by acute leukemia. In our study also acute leukemia is the common malignant etiology for pancytopenia.

Bhatnagar et al, from India in their study in pancytopenia children found that Megaloblastic anemia was the most common etiology causing pancytopenia, followed by infections and acute leukemia. They found enteric fever as the commonest infectious cause. Most common cause of pancytopenia in our study is septicemia. Most common malignancy presenting with pancytopenia in our study is acute lymphoblastic leukemia.

Gupta et al, from India in their study in pancytopenia children found that aplastic anemia was the most common etiology causing pancytopenia, followed by acute leukemia. They found Infections as the third common etiology of pancytopenia, of which Kala azar is common.

In our study the common causes of pancytopenia are septicemia in 1/3rd cases followed by dengue fever (1/10th), Acute lymphoblastic leukemia(1/10th) and aplastic anemia(1/10th) of cases.

TABLE- COMPARISON OF ETIOLOGICAL SPECTRUM IN PANCYTOPENIA

STUDY	**Study population**	**Most common cause**	**Second common cause**
Present study(n=36)	Children	Infections	Acute lymphoblastic leukemia
Shano Naseem et al (n=139)	Children	Aplastic anemia	Acute leukemia
Bhatnagar et al(n=109)	Children	Megaloblastic anemia	Infections and acute leukemia
Gupta et al (n=105)	Children	Aplastic anemia	Acute leukemia-2nd , Infections -3rd

OUTCOME AND FOLLOW UP IN PANCYTOPENIA:

In our study, Almost 1/3rd of patients presented with pancytopenia recovered completely, 1/3rd of patients died, and the remaining 1/3rd are on regular treatment and follow up.

In cases of septicemia, almost 2/3rd of the children who presented with pancytopenia died due to septic shock and DIC, late referral and remaining 1/3rd children recovered. In cases of septicemia if child presents with pancytopenia the mortality is high >66%.

In Dengue fever, all recovered. In ALL, all are under treatment and follow up. In tuberculosis, 2/3rd recovered with antituberculus treatment, 1/3rd is on regular treatment and follow up.

Of the total deaths in pancytopenia, 3/4th of deaths due to septicemia, remaining 1/4th are due to death due to chronic liver disease with portal hypertension, neonatal hepatitis.

Death rate is more in children with pancytopenia than children with bicytopenia.

Late referral and delay in diagnosis is associated with high mortality.

IMPORTANCE OF FOLLOW UP IN PANCYTOPENIA:

All pancytopenia cases were followed up.

During follow up, 1 case died within 24 hours of admission so the cause of pancytopenia is not known.

In a case the cause of pancytopenia is not made during the first admission, on follow up after 2 months diagnosis of megaloblastic anemia is made.

A case was initially diagnosed as megaloblastic anemia, treated with vitamin B12 and blood transfusions; later after 6 months, it turned out to be aplastic anemia.

A case was initially diagnosed as tuberculosis started on anti tubercular drugs, on follow up after 6 months it turned out to be acute myeloid leukemia.

A child was initially diagnosed as dengue fever after treatment at first showed recovery, later again developed fever, after ruling out the other causes of fever in that child, on follow-up after 25 days; diagnosis of hemophagocytic lymphohistiocytosis is confirmed.

A case with pancytopenia and hepatosplenomegaly is lost in follow up after first admission, after 4 months it turned out as acute lymphoblastic leukemia.

All the above mentioned children are diagnosed early during follow up and they are now responding well to treatment. So, all children with unexplained pancytopenia or bicytopenia should be followed up.

CONCLUSION

CONCLUSION

We conclude from our study that, Bicytopenia is more common than pancytopenia in our locality. Both bicytopenia and pancytopenia have slight male predominance. Most of the children with bicytopenia and pancytopenia presented with fever.

Infections were the most common etiology in children admitted with bicytopenia and pancytopenia. Dengue fever was the commonest cause of bicytopenia in our study. It is due to increased incidence and outbreaks of dengue fever in our locality.

In a child with fever, if leucopenia and thrombocytopenia also present, probable etiology could be dengue fever, enteric fever and septicemia. So if a febrile child presents with leucopenia and thrombocytopenia consider infectious etiology.

In a child with fever, if anemia and thrombocytopenia also present, probable etiology could be acute leukemia. So if a febrile child presents with anemia and thrombocytopenia, do peripheral smear study and bone marrow aspiration to rule out leukemia.

In our study, most common cause of pancytopenia was septicemia, caused by coagulase negative staphylococcus aureus and klebsiella

pneumonia. In all children admitted with pancytopenia, blood cultures should be sent before first dose of antibiotics.

In our study the other common causes of pancytopenia are aplastic anemia and acute lymphoblastic leukemia. So in all children with pancytopenia bone marrow aspiration should be done to rule out acute leukemia and aplastic anemia. So in a child with fever if pancytopenia also present, probable etiology could be septicemia followed by aplastic anemia, ALL.

In children with clinical features of sepsis, if bicytopenia and pancytopenia present, it is associated with high mortality.

Although bicytopenia & pancytopenia look ominous, this study showed the common causes were infections. High index of suspicion was necessary for early diagnosis and initiation of appropriate treatment will have an impact on prognosis, as most of the causes were treatable and curable.

At the same time all unexplained bicytopenia and pancytopenia should be followed up meticulously, as it may turn out to be malignancy at a later date.

LIMITATIONS

LIMITATIONS OF THE STUDY:

- Investigations like, iron studies, vitamin B12, folate levels, Genetic analysis for storage disorders, viral antigen and antibody testing for CMV, EBV, Parvo virus were not available in our hospital setup, so in few cases etiological diagnosis could not be made.
- The study period was one and half years, which is not sufficient for follow up of unexplained bicytopenia, pancytopenia, and also predict the final outcome in children with acute leukemia and other chronic disorders.

RECOMMENDATIONS

RECOMMENDATIONS

- In children admitted with bicytopenia especially leucopenia with thrombocytopenia and pancytopenia, treat it as infection till results are awaited.
- In children admitted with bicytopenia especially anemia and thrombocytopenia, bone marrow aspiration should be done.
- Follow up of unexplained bicytopenia and pancytopenia is essential as it may later turn into leukemia.
- Long term follow up studies are needed in children presenting with bicytopenia and pancytopenia to predict the final outcome and prognosis.

ANNEXURES

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DATA COLLECTION PROFORMA

Name: Age/ sex: DATE:

Address with phone no:

Informant/Reliability:

COMPLAINTS: 1)

2)

HOPI:

H/O Fever: Yes/NO

H/O Easy fatiguablity: Yes/NO

H/O Pallor: Yes/NO

H/O Petechial rashes: Yes/NO

H/O malena: Yes/NO

H/O bleeding from gums: Yes/NO

H/O epistaxis: Yes/NO

H/O joint pain/leg pain: Yes/NO

H/O jaundice: Yes/NO

PAST H/O:

TREATMENT H/O:

AN/N/PN H/O:

DEVELOPMENTAL H/O:

IMMUNISATION H/O:

FAMILY H/O: CONTACT H/O:

GENERAL EXAMINATION: Conscious, Febrile/Not, Pallor, Icterus , Cyanosis, Pedal Edema, Lymphadenopathy, Neurocutaneous Markers

VITALS:

HR: GCS: RR : CRT:
BP: URINE OP: TEMP:
ANTHROPOMETRY: HT: WT: HC:
LIVER SPAN: AG

SYSTEMIC EXAMINATION:

Per Abdomen:

CVS:

RS:

CNS:

INVESTIGATIONS:

-Hb-

-RBC COUNT

-TC

-DC

-PLT

-PCV

-MCV

-MCH

-MCHC

-RDW

-PDW

-RETIC COUNT

-ESR

-PERIPHERAL SMEAR STUDY

-BONE MARROW STUDY

-OTHER RELEVENT INVESTIGATIONS:

SUMMARY: BI/PAN-CYTOPENIA:

SEX:

AGE GROUP:

PERIPHERAL BLLOD FINDINGS:

CLINICAL PRESENTATION:

ETIOLOGY:

OUTCOME ASSESMENT:

CYTOPENIA CORRECTED/NOT CORRECTED:

HOW LONG IT TAKES TO GET CORRECTED:

COMPLETELY RECOVERED OR DEATH:

WHY NOT CORRECTED:

FOLLOW UP OF BI/PANCYTOPENIA:

DATE: VISIT NO:

CLINICAL EXAMINATION FINDINGS-

CBC

COMPLICATIONS

இணைப்பு - 1

நோயாளி தகவல் மற்றும் ஒப்புதல் படிவம்

இரத்தத்தில் சிவப்பு, வெள்ளை மற்றும் தட்டனுக்கள் குறைபாடு உள்ள குழந்தைகளின் நோயின் தன்மையையும் அதற்கான காரணத்தையும் கண்டறிவதைப் பற்றிய ஆய்வு

பங்கேற்பதற்கான தகுதிகள்

இரத்தத்தில் சிவப்பு, வெள்ளை மற்றும் தட்டனுக்கள் குறைபாடு உள்ள 2 மாதத்திலிருந்து 12 வயதுகுட்பட்ட குழந்தைகள்

கீமோகுளோபின் அளவு 10க்கும் கீழே உள்ளவர்கள்

வெள்ளை அணுக்கள் 4000 க்கும் கீழே உள்ளவர்கள்

தட்டனுக்கள் 100000 க்கும் கீழே உள்ளவர்கள்

பரிசோதனைகளின் விபரம்

நோயாளிகளின் இரத்தத்தில் சிவப்பு, வெள்ளை மற்றும் தட்டனுக்கள் அளவை பரிசோதித்தல், நோயின் தன்மை, எலும்பு மஞ்சை, பெரிபரல்ஸ்மியர் மற்றும் சுயவிபரம் தொடர்புடைய ஆய்வு

இரத்த பரிசோதனைகளின் ஆபத்துக்கள்

பெரும்பாலானவர்களுக்கு, இரத்தம் எடுப்பதற்காக ஊசிகுத்துவது எவ்வித மோசமான பிரச்சனைகளையும் ஏற்படுத்துவதில்லை. ஆயினும், சிலநேரங்களில் அவை இரத்தம் எடுக்கப்பட்ட இடத்தில், இரத்தக்கசிவு, இரத்தம் கன்றிப்போதல், அசௌகரியம், நோய்த் தொற்றுக்கள் மற்றும் வலி ஆகியவற்றை உண்டாக்கலாம். அவ்வாறு ஏற்படும் பட்சத்தில் அதற்கான தகுந்த சிகிச்சை வழங்கப்படும்.

இணைப்பு - 2

நோயாளி தகவல் மற்றும் ஒப்புதல் படிவம்

ஆய்விடத் தகவல் மற்றும் தொடர்பு விவரங்கள்

இரத்தத்தில் சிவப்பு, வெள்ளை மற்றும் தட்டனுக்கள் குறைபாடு உள்ள குழந்தைகளின் நோயின் தன்மையையும் அதற்கான காரணத்தையும் கண்டறிவதைப் பற்றிய ஆய்வு

ஆரோக்கிய அனுபவங்களை அறிவிக்க, ஏதேனும் மற்ற கேள்விகளைப் பற்றி கேட்பதற்கான ஆய்வு மருத்துவர்களின் தொடர்பு விவரங்கள்

ஆய்வு மருத்துவரின் பெயர்	:	மரு. விஜய் ஆனந்த்
முகவரி	:	அரசு இராஜாஜி மருத்துவமனை மற்றும் மதுரை மருத்துவக்கல்லூரி பனகல் ரோடு, மதுரை-625020
தொடர்பு எண்	:	9629290198

ஓர் பங்கேற்பாளராக உங்கள் உரிமைகளைப் பற்றி கேட்பதற்கான தொடர்பு விவரங்கள்

தலைவர்
இண்ஸ்டிடியூஷனல் ரிவ்யூ போர்டு / இண்டிபெண்டன்ட் எத்திக்ஸ்கமிட்டி
அரசு இராஜாஜி மருத்துவமனை
மற்றும் மதுரை மருத்துவக்கல்லூரி
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தமிழ்நாடு, இந்தியா.

தொடர்பு எண் 91 452 2532535 2521021

இணைப்பு - 3

நோயாளி தகவல் மற்றும் ஒப்புதல் படிவம்

இரத்தத்தில் சிவப்பு, வெள்ளை மற்றும் தட்டனுக்கள் குறைபாடு உள்ள குழந்தைகளின் நோயின் தன்மையையும் அதற்கான காரணத்தையும் கண்டறிவதைப் பற்றிய ஆய்வு

இந்தப் பக்கத்தை கையொப்பமிடுவதன் மூலமாக பின்வருவனவற்றை நான் உறுதி செய்கிறேன்.

மேற்படி ஆய்விற்கான ..... தேதியிடப்பட்ட இந்த நோயாளி தகவல் மற்றும் ஒப்புதல் படிவத்திலுள்ள அனைத்துத் தகவல்களையும் நான் படித்துப் புரிந்து கொண்டிருக்கிறேன் எனவும் அதைப்பற்றி சிந்திக்க எனக்கு கால அவகாசம் இருந்தது எனவம் நான் உறுதியளிக்கிறேன்.

என் குழந்தையின் கையிலிருந்து இரத்தம் மற்றும் எலும்பு மஞ்சை எடுப்பதற்காக ஊசிகுத்தும்பொழுது இரத்தம் எடுக்கப்பட்ட இடத்தில், இரத்தக்கசிவு, இரத்தம் கன்றிப்போதல், அசௌகரியம், நோய்த் தொற்றுக்கள் மற்றும் வலி ஆகியவற்றை உண்டாக்கலாம். அவ்வாறு ஏற்படும் பட்சத்தில் அதற்கான தகுந்த சிகிச்சை வழங்கப்படும் என்பதையும் நான் அறிந்து சம்மதிக்கிறேன்.

கேள்விகள் கேட்பதற்கான வாய்ப்பு எனக்கு இருந்தது மேலும் எனது கேள்விகள் அணைத்தும் எனது திருப்திக்குத் தக்கவாறு பதிலளிக்கப்பட்டிருக்கின்றன.

இந்த ஆய்வில் என் பங்கேற்பு தன்னார்வம் சார்ந்தது எனவும் பங்கேற்பை எப்போது வேண்டுமானாலும் எவ்விதக் காரணமும் அளிக்காமல் என் மருத்துவக் கவனிப்பு அல்லது சட்ட உரிமைகள் பாதிக்கப்படாமல் விலகிக் கொள்ள நான் சுதந்திரமானவர் என்பதை நான் புரிந்து கொள்கிறேன்.

வேண்டிக் கொள்ளப்பட்ட படி ஆய்வு நடைமுறைகளை பின்பற்றவும் மற்றும் மருத்துவர், செவிலிகள் அல்லது மற்ற ஊழிய உறுப்பினர்களுக்கு தேவையான தகவல்களை வழங்கவும் நான் தன்னார்வத்துடன் ஒப்புக் கொள்கிறேன்.

இந்த ஆய்விலிருந்து எழும் எந்தவொரு தரவு அல்லது முடிவுகளின் உபயோகத்தினையும். இது போன்ற உபயோகமானது தரவு பாதுகாப்பு அறிக்கையில் குறிப்பிட்டபடி மட்டுமாக உபயோகிக்கப்படும் பட்சத்தில் அவைகளை நான் தடுக்காமலிருக்க நான் ஒப்புக் கொள்கிறேன்.

மேற்கண்ட ஆய்வில் பங்கெடுக்க நான் ஒப்புக் கொள்கிறேன்.

எனக்காக வைத்துக்கொள்வதற்காக இந்த நோயாளி தகவல் மற்றும் ஒப்புதல் படிவத்தின் ஓர் நகலை நான் பெற்றுக் கொள்கிறேன்.

நோயாளி கையொப்பம்

தேதி

(அல்லது நோயாளி கல்லாதவரானால், பாகுபாடற்ற சாட்சியின் முன்னிலையில் நோயாளி வாய்மூலமான ஒப்புதல் கொடுத்திருக்கிறார் என்பதை குறிப்பதற்காக பெருவிரல் ரேகை) நோயாளி தாமாகவே தேதியிடவேண்டும்

நோயாளியின் பெயர் (நோயாளி என் மற்றும் பெயர் முதலெழுத்துக்கள்)

பெற்றோரி / வயது

MASTER CHART KEY

Y	-	Yes
N	-	No
M	-	Male
F	-	Female
TC	-	Total count
PLC	-	platelet count
WBC	-	White blood cells
RBC	-	Red blood cells
T&F	-	Treatment and follow up
R	-	Recovered
D	-	Death
L	-	Lost in follow up
NEC	-	Non Enteric culture
MP	-	Malarial parasite
G/E	-	General Examination
P/A	-	per abdomen

MASTER CHART

Sl.No	NAME	AGE	SEX	HISTORY OF PRESENT ILLNESS																G/E				P/A		INVESTIGATIONS								
				FEVER	ABDOMINAL PAIN	VOMITING	PETECHIAL RASHES	HEMETEMESIS	MALENA	BLEEDING GUMS	EPISTAXIS	JOINT PAIN/LEG PAIN	JAUNDICE	LOSS OF WEIGHT /APPETITE	ABDOMINAL DISTENSION	LETHARGY	CONVULSION	LOOSE STOOLS	BREATHLESSNESS	PALLOR	THY	FEBRILE/NOT	ICTERUS	HEPATOMEGALY	SPLENOMEGALY	HB g/dl	Hematocrit	TC /cubic mm	PLT/cubic mm	BICYTOPENIA			PANCYTOPENIA	PERIPHERAL SMEAR STUDY		
																													anemia with thrombocytopenia	anemia with leucopenia	leucopenia with thrombocytopenia		WBC	PLATELETS	BLAST CELLS
1	Sri yoga harish	5	M	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	5	15	3200	9000	N	N	N	Y	reduced	thrombocytopenia	no
2	Anbarasan	4	M	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	N	N	8	24	2000	21000	N	N	N	Y	reduced	thrombocytopenia	
3	Sudharsan	11	M	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	3	10	1400	5000	N	N	N	Y	Leukopenia	thrombocytopenia	no
4	Arjun	3	M	Y	Y	N	Y	N	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	N	7	21	1200	24000	N	N	N	Y	Leukopenia	thrombocytopenia	no
5	Yogesh	4	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	10	30	2200	13000	N	N	N	Y	reduced	thrombocytopenia	no
6	Sanjay	2.6	M	Y	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	Y	N	Y	N	Y	Y	7	21	2300	10800	N	N	N	Y	Leukopenia	reduced	No
7	Manimaran	7m	M	Y	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Y	N	Y	N	N	N	7	21	2000	75000	N	N	N	Y	reduced	reduced	no
8	Kumaravel	3	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	3	9	3200	65000	N	N	N	Y	reduced	reduced	5%
9	Sridhar	3	F	Y	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	Y	N	Y	N	Y	N	8	24	3900	85000	N	N	N	Y	reduced	thrombocytopenia	no
10	Archana	8	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	Y	N	Y	Y	7	21	2300	98000	N	N	N	Y	reduced	thrombocytopenia	no
11	Rahul	4	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	3600	94000	N	N	N	Y	reduced	thrombocytopenia	NIL
12	Madesh kumar	4	M	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	N	6	18	3300	33000	N	N	N	Y	reduced	thrombocytopenia	NO
13	Siva shri	7	F	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	4	7	3200	12000	N	N	N	Y	reduced	thrombocytopenia	NO
14	Nithya saranya	11	F	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	6	18	1200	19000	N	N	N	Y	reduced	thrombocytopenia	NO
15	Kishore	2 .4	M	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	4	12	1900	9000	N	N	N	Y	reduced	reduced	10%
16	Sudharsan	6	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	3	9	1400	14000	N	N	N	Y	Leukopenia	thrombocytopenia	NO
17	Jeevitha	1. 6	F	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	Y	N	Y	Y	Y	Y	4	12	3000	63000	N	N	N	Y	reduced	thrombocytopenia	NO
18	Raviprasad	2	M	N	N	Y	N	Y	N	N	N	N	N	N	Y	N	N	N	N	Y	N	N	N	N	N	2	7	2200	9000	N	N	N	Y	Leukopenia	thrombocytopenia	NO
19	Hanika	7 m	F	Y	N	Y	N	N	N	N	N	N	N	Y	Y	Y	N	N	N	Y	N	Y	N	Y	N	8	24	3400	60000	N	N	N	Y	Leukopenia	thrombocytopenia	NO
20	Someswaran	4	M	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	4	12	3000	20000	N	N	N	Y	Leukopenia	thrombocytopenia	25%
21	Sudharshana shree	6	F	Y	N	N	jn	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	Y	N	9	27	3500	1.3 lakh	N	Y	N	N	normal	thrombocytopenia	no
22	Saravanakumar	5	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	N	N	N	4	12	4200	83000	Y	N	N	N	normal	thrombocytopenia	no
23	Manoj kumar	4	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	Y	Y	6	18	28900	4000	Y	N	N	N	leucocytosis	thrombocytopenia	65%
24	Archana	7 m	F	Y	N	Y	N	N	N	N	N	N	N	Y	N	Y	N	N	N	Y	N	Y	N	Y	N	5	15	4800	7000	Y	N	N	N	normal	thrombocytopenia	no
25	Rameetha	2m	F	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	Y	N	Y	N	Y	N	9	27	5000	31000	Y	N	N	N	normal	thrombocytopenia	no
26	Hariharan	4	M	N	N	N	N	Y	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	6	18	4400	83000	Y	N	N	N	normal	thrombocytopenia	no
27	Muthulakshmi	7	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	75000	82000	Y	N	N	N	leucocytosis	thrombocytopenia	75%
28	Harish	5	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	9	27	48000	25000	Y	N	N	N	leucocytosis	thrombocytopenia	85%
29	Gurumari	3.6	F	Y	N	N	N	N	N	N	N	Y	N	N	N	Y	N	N	N	Y	Y	Y	N	Y	N	3	9	25700	35000	Y	N	N	N	leucocytosis	thrombocytopenia	25%
30	Bhavakeerthana	3.6	F	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	N	N	N	N	7	21	14000	48000	Y	N	N	N	neutrophilia	thrombocytopenia	no
31	Muthuselvi	10	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	6	19	16100	79000	Y	N	N	N	leucocytosis	thrombocytopenia	60%
32	Mithun	1	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	6	18	9000	43000	Y	N	N	N	normal	thrombocytopenia	no
33	B/o thillaipushpam	2m	M	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	Y	N	4	12	9000	30000	Y	N	N	N	normal	thrombocytopenia	no
34	Ragavan	8	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	7	21	27300	40000	Y	N	N	N	leucocytosis	thrombocytopenia	90%
35	Sujith	4	M	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	7	21	7700	60000	Y	N	N	N	normal	Giant platelets	no
36	Rutharan	2m	M	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	Y	N	6	18	14500	10000	Y	N	N	N	normal	thrombocytopenia	no
37	Kavidharshini	7m	F	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	8	24	16500	48000	Y	N	N	N	leucocytosis	thrombocytopenia	no
38	Barakath nisha	12	F	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	Y	Y	N	N	N	Y	N	2	6	7800	65000	Y	N	N	N	normal	thrombocytopenia	no
39	Sanmathi	2 . 6	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	8	24	13000	74000	Y	N	N	N	normal	thrombocytopenia	no
40	Rithish	2m	M	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	Y	N	Y	N	N	N	7	21	10300	36000	Y	N	N	N	normal	thrombocytopenia	no
41	Muniasamy	4	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	Y	4	12	7900	67000	Y	N	N	N	normal	thrombocytopenia	no
42	Jaffer hussain	8	M	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	Y	N	N	N	N	N	5	15	8300	26000	Y	N	N	N	normal	thrombocytopenia	no
43	Melki santhosh	10	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	4	12	55000	8000	Y	N	N	N	leucocytosis	thrombocytopenia	50%
44	Rajesh	10	M	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	29500	29000	Y	N	N	N	leucocytosis	thrombocytopenia	65%
45	Beema fathima	2 . 4	F	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	Y	N	Y	Y	8	24	32000	37000	Y	N	N	N	leucocytosis	thrombocytopenia	20%
46	Sarvesh	2	M	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	Y	N	Y	N	Y	Y	2	6	13800	18000	Y	N	N	N	leucocytosis	thrombocytopenia	2%
47	Akasthri	10	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	9	27	46000	29000	Y	N	N	N	leucocytosis	thrombocytopenia	5%
48	B/o meena	2m	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	Y	N	Y	Y	7	21	87000	36000	Y	N	N	N	leucocytosis	thrombocytopenia	92%
49	Vishnu	5	M	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	17000	55000	Y	N	N	N	leucocytosis	thrombocytopenia	16%
50	Sriram	2 .6	M	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N	N	Y	N	N	N	N	Y	3	9	7000	54000	Y	N	N	N	normal	thrombocytopenia	no

51	Immanuvel	2 .6	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	N	4	12	4700	17000	Y	N	N	N	normal	thrombocytopenia	15%
52	Dharsan	10 m	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	4	12	4600	45000	Y	N	N	N	normal	thrombocytopenia	no
53	Archana	8	F	Y	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	4	12	25000	16000	Y	N	N	N	leucocytosis	thrombocytopenia	20%
54	perumal	5 .6	M	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	5	15	6400	11000	Y	N	N	N	normal	thrombocytopenia	no
55	B/o thangammal	2m	F	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	Y	N	Y	Y	Y	N	4	12	6400	53000	Y	N	N	N	normal	thrombocytopenia	no
56	Kamalapriyan	8	M	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	Y	Y	7	21	5700	22000	Y	N	N	N	normal	thrombocytopenia	no
57	Muthumarudhupandi	3	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	5	15	21000	10200	Y	N	N	N	leucocytosis	thrombocytopenia	11%
58	Kavin	2 m	M	N	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	Y	N	N	N	Y	N	8	24	30000	65000	Y	N	N	N	leucocytosis	thrombocytopenia	no
59	Vimsala	9	F	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	Y	Y	N	Y	Y	4	13	83000	21000	Y	N	N	N	leucocytosis	thrombocytopenia	25%
60	Keerthanashree	6	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	6	18	8600	90000	Y	N	N	N	normal	thrombocytopenia	6%
61	Rashidha	5	F	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	Y	N	5	15	7200	38000	Y	N	N	N	normal	thrombocytopenia	no
62	Kousalya	4	F	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	N	N	6	18	10200	75000	Y	N	N	N	normal	thrombocytopenia	no
63	Jesline	8 m	F	Y	N	Y	N	N	N	N	N	N	N	Y	N	N	N	Y	N	Y	N	Y	N	N	N	8	24	5600	82000	Y	N	N	N	normal	thrombocytopenia	no
64	Harish pandi	8 m	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	5	15	35000	60000	Y	N	N	N	leucocytosis	thrombocytopenia	25%
65	Manisha	4	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	N	4	12	5300	15000	Y	N	N	N	normal	thrombocytopenia	18%
66	Suryaprakash	5	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	N	5	15	5100	10000	Y	N	N	N		thrombocytopenia	5%
67	Thirukumaran	7	M	Y	Y	N	N	Y	N	N	N	N	N	N	Y	N	N	N	N	Y	N	Y	N	N	Y	8	24	3200	1.3 lakh	N	Y	N	N	leucopenia	normal	no
68	Annalakshmi	9	F	N	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	Y	N	N	Y	Y	Y	6	18	2400	125000	N	Y	N	N	leucopenia	normal	NO
69	Gokulakrishnan	3.6	M	Y	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	Y	N	N	N	10	30	3800	18000	N	N	Y	N	leucopenia	thrombocytopenia	NO
70	Kaniska	2m	F	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N	N	11	33	2100	29000	N	N	Y	N	leucopenia	thrombocytopenia	no
71	Balaji	8	M	Y	N	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	Y	N	Y	N	N	N	11	32	2800	40000	N	N	Y	N			
72	Karthick	8	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	11	33	3800	33000	N	N	Y	N			
73	Yuva shree	5	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	36	3900	26000	N	N	Y	N			
74	Vijay	6	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	14	46	3900	90000	N	N	Y	N			
75	Dinesh karthick	5	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	47	2800	37000	N	N	Y	N			
76	Santhosh	10	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	13	44	2900	48000	N	N	Y	N			
77	Manimaran	12	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	41	2100	83000	N	N	Y	N			
78	Harini	10	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3800	57000	N	N	Y	N			
79	Jegan kumar	8	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	39	3800	37000	N	N	Y	N			
80	Jero	10	M	Y	N	Y	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	N	N	7	21	3100	130000	N	Y	N	N	leucopenia	normal	5%
81	Gopika	9	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	47	1600	79000	N	N	Y	N			
82	Kousalya	10	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	45	3600	90000	N	N	Y	N			
83	Sasuraj	11	M	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3000	87000	N	N	Y	N			
84	Madhumitha	2 . 6	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	Y	N	N	N	8	24	3600	190000	N	Y	N	N			
85	Sethupriyan	12	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	3800	16000	N	N	Y	N			
86	Harish	10	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3400	43000	N	N	Y	N			
87	Yogeshwari	6	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	43	3900	88000	N	N	Y	N			
88	Arun	5	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	3600	90000	N	N	Y	N			
89	Anand	3	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	42	2700	60000	N	N	Y	N			
90	Manoj	7	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	3500	32000	N	N	Y	N			
91	Sundar	3	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	46	2100	64000	N	N	Y	N			
92	Sharmila	7	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3200	76000	N	N	Y	N			
93	Ragasiya	5	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	45	2600	53000	N	N	Y	N			
94	Anish	4	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	39	3100	86000	N	N	Y	N			
95	Akbar ali	7	M	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	2800	57000	N	N	Y	N			
96	Sivanya	5	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3700	49000	N	N	Y	N			
97	Siva	9	M	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3400	54000	N	N	Y	N			
98	Mahilan	6	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	10	35	2900	67000	N	N	Y	N			
99	Sivanadhan	12	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	46	3500	45000	N	N	Y	N			
100	Sadhana	6	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	13	43	3200	55000	N	N	Y	N			
101	Sangavi sri	7	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Y	Y	6	18	25000	82000	Y	N	N	N	leucocytosis	thrombocytopenia	20%
102	Sabarish	3	M	Y	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	Y	Y	Y	N	Y	Y	9	27	35000	25000	Y	N	N	N	leucocytosis	thrombocytopenia	30%
103	Muneeswaran	5.5	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	N	Y	Y	4	12	65000	8000	Y	N	N	N	leucocytosis	thrombocytopenia	68%
104	Sivamani	11	M	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	Y	N	Y	Y	8	24	32000	37000	Y	N	N	N	leucocytosis	thrombocytopenia	40
105	Padmapraveen	9	M	Y	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	Y	Y	N	Y	Y	7	21	13800	18000	Y	N	N	N	normal	thrombocytopenia	NO
106	Mutheeshwaran	3	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	9	27	46000	29000	Y	N	N	N	leucocytosis	thrombocytopenia	5
107	Anushree	2.5	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	Y	N	Y	Y	7	21	87000	36000	Y	N	N	N	leucocytosis	thrombocytopenia	52
108	Sivasankari	6	F	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	17000	55000	Y	N	N	N	leucocytosis	thrombocytopenia	55%
109	Mohammed ayub	9	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	7	21	27300	40000	Y	N	N	N	leucocytosis	thrombocytopenia	9%
110	Kanishkar	8	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	N	4	12	4700	17000	Y	N	N	N	normal in numb	thrombocytopenia	3%
111	Sridhar	5	M	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	8	24	2900	80000	N	N	N	Y	Leukopenia,Hy	thrombocytopenia	NO
112	Lalitha	12m	F	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	Y	N	Y	N	5	15	3000	75000	N	N	N	Y	leukopenia	thrombocytopenia	no

113	Marium	3m	F	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	Y	N	Y	N	7	21	3200	35000	N	N	N	Y	leukopenia	thrombocytopenia	no
114	Aswanth	9m	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	Y	N	N	N	8	24	2500	78000	N	N	N	Y	Leukopenia	thrombocytopenia	no
115	Ragini	1	F	Y	N	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	Y	N	Y	N	N	N	6	18	2800	38000	N	N	N	Y	not done		
116	Haniska	4 m	F	y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	5	15	2100	29000	N	N	N	Y	not done		
117	Aarav	3	M	Y	Y	N	Y	N	N	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	7	21	1800	94000	N	N	N	Y	Leukopenia	thrombocytopenia	no
118	Sanjika	1	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	Y	N	Y	N	N	N	7	21	2500	90000	N	N	N	Y	leukopenia	thrombocytopenia	no
119	Monisha	5	F	Y	N	N	N	N	N	N	N	N	N	Y	Y	N	N	N	N	Y	N	Y	N	Y	N	8	24	2900	65000	N	N	N	Y	leukopenia	thrombocytopenia	no
120	Ragav	9 m	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	N	N	6	18	1400	75000	N	N	N	Y	leukopenia	thrombocytopenia	no
121	Kavitha	1	F	Y	N	Y	N	N	N	N	N	N	N	N	N	Y	Y	Y	Y	Y	N	Y	N	N	N	7	21	3800	18000	N	N	N	Y	not done		
122	Boominathan	9	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	5	15	21000	10200	Y	N	N	N	leucocytosis	thrombocytopenia	60%
123	Sanjai	12	M	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	Y	Y	N	Y	Y	4	12	58300	21000	Y	N	N	N	leukemia	thrombocytopenia	90%
124	Saravanakumar	2	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	6	18	8600	90000	Y	N	N	N	normal in numb	thrombocytopenia	no
125	Selvamari	10	M	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	Y	N	5	15	7200	38000	Y	N	N	N	normal in numb	thrombocytopenia	no
126	Ponraj	10	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	50000	82000	Y	N	N	N	leucocytosis	thrombocytopenia	95%
127	Elakiya	10	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	7	21	80000	25000	Y	N	N	N	leucocytosis	thrombocytopenia	85%
128	Kavitha	8	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	4	12	115000	8000	Y	N	N	N	leucocytosis	thrombocytopenia	90%
129	Muthuselvi	10	F	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	95000	29000	Y	N	N	N	leucocytosis	thrombocytopenia	95%
130	Sangavi	6	F	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	Y	N	Y	Y	8	24	32000	37000	Y	N	N	N	leucocytosis	thrombocytopenia	40%
131	Balavikash	5	M	Y	Y	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	Y	N	Y	N	Y	Y	2	7	13800	18000	Y	N	N	N	normal in numb	thrombocytopenia	2%
132	Rohith viswesh	10	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	9	27	46000	29000	Y	N	N	N	leucocytosis	thrombocytopenia	5%
133	Jero	10	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	N	Y	N	Y	Y	7	21	87000	36000	Y	N	N	N	leucocytosis	thrombocytopenia	92%
134	Amalajeeva	11	M	Y	Y	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	17000	55000	Y	N	N	N	leucocytosis	thrombocytopenia	60
135	Kumaravel	5	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	7	21	27300	40000	Y	N	N	N	leucocytosis	thrombocytopenia	I-90%
136	Karthick	7	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	N	4	12	4700	17000	Y	N	N	N	normal in numb	thrombocytopenia	no
137	Muthumarudhupandi	3	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	5	15	21000	10200	Y	N	N	N	leeucocytosis	thrombocytopenia	60%
138	Sakthiramya	6	F	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	Y	Y	N	Y	Y	4	12	183000	21000	Y	N	N	N	leukemia	thrombocytopenia	90%
139	Sakteeshwarapandiar	2.8	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	6	18	8600	90000	Y	N	N	N	normal in numb	thrombocytopenia	no
140	Suryaprakash	5.5	M	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	Y	N	5	15	7200	38000	Y	N	N	N	normal in numb	thrombocytopenia	no
141	Tamilarasan	5	M	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	N	Y	Y	6	18	28900	4000	Y	N	N	N	leucocytosis	thrombocytopenia	65%
142	Mahalakshmi	7	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	5	15	21000	10200	Y	N	N	N	leucocytosis	thrombocytopenia	60%
143	Kaleshwari	4	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	73500	82000	Y	N	N	N	leucocytosis	thrombocytopenia	95%
144	shanmugapriya	6	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	8	24	28000	25000	Y	N	N	N	leucocytosis	thrombocytopenia	no
145	Hariharan	6	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	4	12	65000	8000	Y	N	N	N	leucocytosis	thrombocytopenia	90
146	Kavitha	9	F	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	Y	Y	N	Y	Y	6	18	95000	29000	Y	N	N	N	leucocytosis	thrombocytopenia	95
147	Laksaya	5	F	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	Y	N	Y	Y	8	24	32000	37000	Y	N	N	N	leucocytosis	thrombocytopenia	40
148	Vetrivel	4	m	y	n	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	Y	Y	N	Y	Y	4	12	7000	25000	Y	N	N	N	normal in numb	Thrombocytopenia	35%
149	Mahasri	3	f	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	Y	Y	3	10	39500	46000	Y	N	N	N	leucocytosis	thrombocytopenia	45%
150	Jegankumar	8	M	Y	N	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	Y	N	N	N	10	30	3800	18000	N	N	Y	N	leucopenia	thrombocytopenia	nil
151	Gopika	9.5	F	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N	N	N	11	33	2100	29000	N	N	Y	N	leucopenia	thrombocytopenia	nil
152	Sabarish	4	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	2800	37000	N	N	Y	N	not done		
153	Manoharan	7	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	40	2900	48000	N	N	Y	N	not done		
154	Karthick	8	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	14	46	2100	83000	N	N	Y	N	not done		
155	Kowsalya	10	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	47	3800	57000	N	N	Y	N	not done		
156	Sethupriyan	12	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	3800	37000	N	N	Y	N	not done		
157	Bargavi	8	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	1600	79000	N	N	Y	N	not done		
158	Probivitha	10	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3600	90000	N	N	Y	N	not done		
159	Balachandran	7	M	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	39	3000	87000	N	N	Y	N	not done		
160	Rohin sammuel	9	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	47	3800	16000	N	N	Y	N	not done		
161	Vignesh	11	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	45	3400	43000	N	N	Y	N	not done		
162	Brindha	8	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3900	88000	N	N	Y	N	not done		
163	Ayannar	9	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	3600	90000	N	N	Y	N	not done		
164	Rahul	12	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	2700	60000	N	N	Y	N	not done		
165	Sakthivel	9	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	43	3500	32000	N	N	Y	N	not done		
166	Srikanthchandru	11	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	2100	64000	N	N	Y	N	not done		
167	Harsha	10	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	42	3200	76000	N	N	Y	N	not done		
168	Sabika	6	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	2600	53000	N	N	Y	N	not done		

169	Harshanth	9.5	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	46	3100	86000	N	N	Y	N	not done		
170	Ilango	12	M	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	2800	57000	N	N	Y	N	not done		
171	Shalini	7	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	45	3700	49000	N	N	Y	N	not done		
172	Sabarivel	4.5	M	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	39	3400	54000	N	N	Y	N	not done		
173	Dharsan	9	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	41	2900	67000	N	N	Y	N	not done		
174	Bharathi	11	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3500	45000	N	N	Y	N	not done		
175	Arunachalam	5	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	11	38	3200	55000	N	N	Y	N	not done		
176	Gandhi	10	M	Y	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	Y	N	N	N	10	30	3500	30000	N	N	Y	N	leucopenia	thrombocytopenia	nil
177	Nishanthini	5	F	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N	N	N	13	39	2100	29000	N	N	Y	N	leucopenia	thrombocytopenia	nil
178	Meenalochini	12	F	Y	N	Y	N	N	N	N	N	N	N	Y	Y	N	N	N	Y	N	Y	N	N	N	10	35	2800	40000	N	N	Y	N	not done		
179	Divyadharshini	12	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	14	46	3800	33000	N	N	Y	N	not done		
180	Smithiha	12	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	43	3900	26000	N	N	Y	N	not done		
181	Sivaperumal	10	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	40	3900	90000	N	N	Y	N	not done		
182	Malaithran	3	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	2800	37000	N	N	Y	N	not done		
183	Rhythin	5	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	42	2900	48000	N	N	Y	N	not done		
184	Muthuzhagan	4	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	14	46	2100	83000	N	N	Y	N	not done		
185	Gopika	6	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	45	3800	57000	N	N	Y	N	not done		
186	Chandralekha	10	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	3800	37000	N	N	Y	N	not done		
187	Sibiraj	8	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	1600	79000	N	N	Y	N	not done		
188	Tharan	4	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3600	90000	N	N	Y	N	not done		
189	Mowlika	5	F	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3000	87000	N	N	Y	N	not done		
190	Kayalshrea	1.5	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	47	3800	16000	N	N	Y	N	not done		
191	Nagalakshmi	10	F	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	3400	43000	N	N	Y	N	not done		
192	Hemanth	4	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3900	88000	N	N	Y	N	not done		
193	Besmitha	10	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	2700	60000	N	N	Y	N	not done		
194	Divya	9	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	3500	32000	N	N	Y	N	not done		
195	Anushka	6	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	2100	64000	N	N	Y	N	not done		
196	Ranjini	6	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	3200	76000	N	N	Y	N	not done		
197	Ashifa	3.5	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	2600	53000	N	N	Y	N	not done		
198	Nachimuthu	9	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	47	3100	86000	N	N	Y	N	not done		
199	Hasbiya	7	F	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	2800	57000	N	N	Y	N	not done		
200	Abdul ajees	10	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	46	3700	49000	N	N	Y	N	not done		
201	Swetha	7	F	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	3400	54000	N	N	Y	N	not done		
202	Vimal	8	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	41	2900	67000	N	N	Y	N	not done		
203	Pothirajan	7	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3500	45000	N	N	Y	N	not done		
204	Vishnuprabakaran	10	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	11	37	3200	55000	N	N	Y	N	not done		
205	Mathanpandi	4.5	M	Y	N	Y	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	Y	N	N	N	10	30	3300	28000	N	N	Y	N	leucopenia	thrombocytopenia	nil
206	Yogesh	7	M	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	Y	N	N	N	N	N	13	39	2100	29000	N	N	Y	N	leucopenia	thrombocytopenia	nil
207	Sriprabaharan	5.5	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	2800	37000	N	N	Y	N	not done		
208	Santhiya	7.5	F	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	41	2900	48000	N	N	Y	N	not done		
209	Rithika	7	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	14	47	2100	83000	N	N	Y	N	not done		
210	Varunkumar	6.5	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	48	3800	57000	N	N	Y	N	not done		
211	Dhiyasri	4	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	45	3800	37000	N	N	Y	N	not done		
212	Karolin	8M	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	1600	79000	N	N	Y	N	not done		
213	Sonu	5.5	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3600	90000	N	N	Y	N	not done		
214	Haridha	10	F	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3000	87000	N	N	Y	N	not done		
215	Supriya	10	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	45	3800	16000	N	N	Y	N	not done		
216	Sadhana	5	F	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	43	3400	43000	N	N	Y	N	not done		
217	Jeevan	4	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	35	3900	88000	N	N	Y	N	not done		
218	Sonaiprabhu	11	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	37	3600	90000	N	N	Y	N	not done		
219	Jeyrahul	12	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	36	2700	60000	N	N	Y	N	not done		
220	Tamaraiselvi	5	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	40	3500	32000	N	N	Y	N	not done		
221	Kodigarasu	5	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	39	2100	64000	N	N	Y	N	not done		
222	Madhusri	6	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	3200	76000	N	N	Y	N	not done		
223	Sivashri	9	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	39	2600	53000	N	N	Y	N	not done		
224	Ganesh	12	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	45	3100	86000	N	N	Y	N	not done		
225	Subashree	5	F	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	2800	57000	N	N	Y	N	not done		
226	Sudeeksam	6	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	48	3700	49000	N	N	Y	N	not done		
227	Bavadharani	12	F	Y	N	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	3400	54000	N	N	Y	N	not done		

228	Malaisamy	5	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	40	2900	67000	N	N	Y	N	not done		
229	Kanimozhi	5	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3500	45000	N	N	Y	N	not done		
230	Kaleshwari	5	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	11	38	3200	55000	N	N	Y	N	not done		
231	Karthick	8	M	Y	N	Y	N	N	N	N	N	N	N	N	N	Y	N	Y	Y	N	N	Y	N	N	N	10	30	3000	35000	N	N	Y	N	leucopenia	thrombocytopenia	nil
232	Harivignesh	12	M	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	N	N	N	11	33	2100	29000	N	N	Y	N	leucopenia	thrombocytopenia	nil
233	Seethalakshmi	7	F	Y	N	Y	N	N	N	N	N	N	N	N	Y	Y	N	N	N	Y	N	Y	N	N	N	10	32	2800	40000	N	N	Y	N	not done		
234	Thangakalyan	7	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	14	46	3800	33000	N	N	Y	N	not done		
235	Surya	12	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	40	3900	26000	N	N	Y	N	not done		
236	Bharathraj	12	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	40	3900	90000	N	N	Y	N	not done		
237	Edwin	3	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	39	2800	37000	N	N	Y	N	not done		
238	Manoj	4.5	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	12	41	2900	48000	N	N	Y	N	not done		
239	Ramya	10	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	Y	N	14	47	2100	83000	N	N	Y	N	not done		
240	Dhanusri	8	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	46	3800	57000	N	N	Y	N	not done		
241	Malini	11	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	43	3800	37000	N	N	Y	N	not done		
242	Suryakumar	7	M	Y	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	1600	79000	N	N	Y	N	not done		
243	Balamurugan	11	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3600	90000	N	N	Y	N	not done		
244	Sanjaykumar	11	M	Y	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	39	3000	87000	N	N	Y	N	not done		
245	Deya	6	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	48	3800	16000	N	N	Y	N	not done		
246	Pradheep	10	M	Y	N	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	42	3400	43000	N	N	Y	N	not done		
247	Divya	9	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3900	88000	N	N	Y	N	not done		
248	Deja	12	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	1600	79000	N	N	Y	N	not done		
249	Harish	6	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3600	90000	N	N	Y	N	not done		
250	Selvakannan	7	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	2600	89000	N	N	Y	N	not done		
251	David raja	7	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	46	3300	80000	N	N	Y	N	not done		
252	Durgedevi	5	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	47	2000	76000	N	N	Y	N	not done		
253	Devasanjan	5.5	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	3600	85000	N	N	Y	N	not done		
254	Surya	10	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	2300	81000	N	N	Y	N	not done		
255	Sadhana	6	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3200	75000	N	N	Y	N	not done		
256	Kesavan	8	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	39	3000	95000	N	N	Y	N	not done		
257	Muthuram	8	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	47	2400	83000	N	N	Y	N	not done		
258	Dhamodharan	10	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	45	3500	85000	N	N	Y	N	not done		
259	Kalpana	10	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	1700	65000	N	N	Y	N	not done		
260	Manoj	8	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	3400	84000	N	N	Y	N	not done		
261	Sachin	11	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	2100	67000	N	N	Y	N	not done		
262	Sivaprathap	9	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	43	3300	83000	N	N	Y	N	not done		
263	Sakthivel	12	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	3000	80000	N	N	Y	N	not done		
264	Pradeep	5	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	42	1800	79000	N	N	Y	N	not done		
265	Kesavan	7	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	2500	72000	N	N	Y	N	not done		
266	Ayyappan	3.5	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	46	1900	69000	N	N	Y	N	not done		
267	Malathy	7	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	2600	90000	N	N	Y	N	not done		
268	Sundarapandi	11	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	45	3600	79000	N	N	Y	N	not done		
269	Mutharasan	4	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	39	1500	68000	N	N	Y	N	not done		
270	Saran	3.5	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	41	3100	73000	N	N	Y	N	not done		
271	Vihashini	3	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	2000	75000	N	N	Y	N	not done		
272	Ranjithkumar	6	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	2600	83000	N	N	Y	N	not done		
273	Abirami	11	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	10	35	1900	88000	N	N	Y	N	not done		
274	Suryamoorthy	11	M	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	46	1700	65000	N	N	Y	N	not done		
275	Andhavan	8	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	43	3400	84000	N	N	Y	N	not done		
276	Arulvijilathaprabha	12	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	2100	67000	N	N	Y	N	not done		
277	Karupusamy	7	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	3300	83000	N	N	Y	N	not done		
278	Punitha	7	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	42	3000	80000	N	N	Y	N	not done		
279	Praveenkumar	10	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	46	1800	79000	N	N	Y	N	not done		
280	Varshidha	8	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	45	2500	72000	N	N	Y	N	not done		
281	Aathiksha	5	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	1900	69000	N	N	Y	N	not done		
282	Akashkumar	7	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	12	40	2600	90000	N	N	Y	N	not done		
283	Nithyasri	7	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	3600	79000	N	N	Y	N	not done		
284	Sivaranjani	12	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	1500	68000	N	N	Y	N	not done		
285	Abisek	10	M	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	14	47	3100	73000	N	N	Y	N	not done		
286	Manikandan	8	M	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	2000	75000	N	N	Y	N	not done		
287	Karthigayani	6	F	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	38	2600	83000	N	N	Y	N	not done		
288	Priyadharshini	10	F	Y	Y	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	11	37	1900	88000	N	N	Y	N	not done		
289	Tharan	4	m	Y	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	13	44	2300	77000	N	N	Y	N	not done		

290	Kavipriya	3	F	Y	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	Y	Y	N	Y	N	6	18	29500	29000	Y	N	N	N	leucocytosis	thrombocytopenia	35%
291	Radhakrishnan	5	M	Y	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	Y	10	30	2800	80000	N	N	Y	N	not done	not done	
292	Sumeya	6	F	Y	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	N	11	33	3500	75000	N	N	Y	N	not done	not done	
293	Karthyayini	7	F	Y	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	N	Y	12	36	3300	90000	N	N	Y	N	not done	not done	
294	Sundareshan	8	M	Y	Y	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	Y	N	Y	Y	13	39	2500	85000	N	N	Y	N	not done	not done	
295	Anitha	9	F	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	12	40	3000	50000	N	N	Y	N	not done		
296	Akash	2	M	Y	N	N	N	N	Y	N	Y	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	5	15	2500	25000	N	N	N	Y	Leukopenia	thrombocytopenia	No
297	Nandhini	11 m	F	Y	N	Y	Y	N	N	N	N	Y	N	N	N	N	N	N	N	Y	N	N	N	Y	Y	12	40	3500	66000	N	N	N	Y	Leukopenia	thrombocytopenia	No
298	Ashok	6	M	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	13	44	3000	75000	N	N	N	Y	not done		
299	Sivayogini	9	F	Y	N	N	N	N	N	N	N	N	N	N	N	Y	N	N	N	N	N	N	N	N	N	12	41	3300	33000	N	N	N	Y	not done		
300	Abinaya	4	F	Y	Y	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	N	14	47	2000	75000	N	N	N	Y	not done		

Sl.No	RBC	BONE MARROW ASPIRATION/BIOPSY STUDY	OTHERS	ETIOLOGY	OUTCOME
1	normocytic normochromic anemia	markedly hypocellular, aplastic anemia		Aplastic anemia	T-Danazol
2	hypochromic microcytic anemia	not done	gene expert gastric juice-tuberculosis,rifampicin sensitive	mycobacterial tuberculosis	R-after ATT
3	hypochromic microcytic anemia	pancytopenia,hypoplastic marrow		Aplastic anemia	T & F
4	hypochromic microcytic anemia	not done	cons positive sepsis sensitive to cipro gentamicin	SJS/TEN,Septecemia	D
5	hypochromic microcytic anemia	mild hypercellular marrow, erythroid hyperplasia, increased megakaryoc	MANTOUX POSITIVE,ESR ELEVATED tuberculous lymphaden	tuberculosis	recovered
6	microcytic hypochromic anemia	ALL- sub leukemic variant	flow cytometry -ALL	ALL	T & F
7	microcytic hypochromic anemia	not done	MRI-parainfectious demylination,ct chest-b/l pneumonitis	septicemia/Klebsiella	D
8	normochromic normocytes	Blasts-25% -ALL	flow cytometry -ALL	ALL	T& F
9	hypochromic microcytic anemia	not done	NEC- gram negative sepsis	septicemia	D
10	microcytic hypochromic anemia	microcytic hypochromic anemia with thrombocytopenia no blast	mantoux positive gastric juice afb m tb esr elevated	tuberculosis	ATT GIVEN ,F- AML
11	microcytic hypochromic anemia	not done	smear for MPpositive	Falciparum malaria	R
12	pancytopenia	severe hypoplasia aplatic anemia		Aplastic anemia	T & F
13	microcytic hypochromic anemia	not done		? nutritional	lost on follow up
14	microcytic hypochromic anemia	no	ANA positive	SLE	T&F
15	normocytic normochromic anemia	hypercellular blast 60 %		?Pancytopenia for evaluation	F - ALL
16	hypochromic microcytes	large atypical cells with degenerative changes,suggestive ALL	flow cytometry -ALL	ALL	T & F
17	microcytic hypochromic anemia	not done	ascitis,usg-neonatal hepatitis,intrahepatic cholestasis,increased LFT	neonatal hepatitis ?CAUSE	D
18	microcytic anemia	not done	usg-chronic liver disease with portal hypertension,splenomegaly	Chronic liver disease with portal hypertension	D
19	microcytic anemia	not done	NEC-GRAM negative sepsis	septicemia	D
20	normocytic normochromic anemia	hypercellular marrow blasts 56% -ALL	flow cytometry -ALL	ALL	T & F
21	normochromic macrocytes hypersegm	megaloblstic and normoblastic erythropoiesis seen	s.vit B12-122pg/m (LOW)sr.homocystine-79.3(HIGH)	Megaloblastic anemia	T & F
22	hypochromic microcytic anemia	not done	IgM ELISA dengue positive	Dengue viral fever /down syndrome	R
23	hypochromic microcytic anemia	Blats-85% ,Acute lymphoblatic leukemia	flow cytometry -ALL	ALL	T& F
24	hypochromic microcytic anemia	not done	NEC-nonfermentive gram negative bacilli sent amikacin	septicemia	D
25	hypochromic microcytic anemia	not done	NEC-CONS SEPSIS,ctbrain-ICH/SAH,LFT-elevated enzymes,bilir	septicemia	D
26	hypochromic microcytic anemia	not done	usg-chronic liver disease with portal hypertension	Chronic liver disease with portal hypertension	T & F
27	hypochromic microcytic anemia	hypercellular marrow with blast 95% no platlet -ALL	Flow cytometry-ALL	ALL	T & F
28	nomochromic normocytic anemia	hypercellular marrow with blast 90% no platlet-ALL	Flow cytometry-ALL	ALL	T & F
29	normochromic normocytes	hypercellular marrow RBC decreased WBC incresaed blast -50%	Flow cytometry-ALL-L2	ALL	T & F
30	hypochromic microcytic anemia	not done	NEC- gram negative sepsis	septicemia	D
31	normochromic normocytes	normoblastic maturation blast 85% AML M3	flow cytometry- AML	AML M3	T & F
32	microcytic hypochromic anemia	microcytic hypochromic anemia with thrombocytopenia	IgM low	Wiskott aldrich syndrome	L
33	microcytic hypochromic anemia	not done		?platlet disorder	D
34	normochromic normocytes	hypercellular marrow blast 90 RBC decreased- ALL L2	flow cytometry-ALL	ALL L2	D-sepsis
35	microcytic hypochromic anemia	thrombocytopenia with giant platlets seen		Bernard souliier syndrome	L
36	normochromic normocytic anemia	not done	NEC-CONS	septicemia	R
37	microcytic hypochromic anemia	not done		OPC poisoning /MODS	D
38	microcytic hypochromic anemia	bicytopenia-no blasts	echo-CCF,	? Nutritional/downs syndrome /ccf/anemia	D
39	microcytic hypochromic anemia	not DONE	IgM ELISA dengue positive	Dengueviral fever/?nutritional anemia	R
40	microcytic hypochromic anemia	not done	NEC-non fermentive gram negative sepsis	septicemia	D
41	microcytic hypochromic anemia	not done		downs syndrome /pem grade 4/nutritional	L
42	microcytic hypochromic anemia	not done		CRF/ANEMIA of chronic disease	D
43	normochromic normocytes anemia	hypercellular marrow blast 95% RBC decreased-ALL	Flow cytometry-ALL	ALL	T & F
44	normochromic normocytes anemia	hypercellular marrow blast 95% RBC decreased-ALL	flow cytometry-ALL	ALL	D
45	normochromic normocytes anemia	hypercellular marrow blast 85% RBC decreased-AL	flow cytometry -ALL	ALL	T & F
46	normochromic normocytes anemia	erythropoiesis,myelopoiesis supressed blast 21%-ALL	flow cytometry -ALL	ALL	D
47	normochromic normocytes anemia	hypercellular marrow blasts 35% -ALL	flow cytometry -ALL	ALL	L
48	normochromic normocytes anemia	NOT DONE	flow cytometry-AML	AML	D
49	microcytic hypochromic anemia	hypercellular marrow blasts 38% -ALL	Flow cytometry -ALL	ALL	T & F
50	microcytic hypochromic anemia	not done	usg-cirrhosis ascitis portal hypertension splenomegaly	Chronic liver disease with portal hypertension	T & F

51	microcytic hypochromic	hyper cellular marrow blast 30% suggestive of ALL	flow cytometry -ALL	ALL	T& F
52	microcytic hypochromic anemia	NOT DONE	usg-GB wall edema ascitis/igm elisa positive	Dengueviral fever/?nutritional anemia	L
53	microcytic hypochromic anemia	hypercellular myeloblasts-60%-AML	LN biopsy-epitheloid granuloma TB/ CT abdomen-mediastinal LN	K/C of TB/AML	T & F
54	microcytic hypochromic anemia	inconclusive -bicytopenia for evaluation		? bicytopenia for evaluation	L
55	microcytic hypochromic anemia	myeloid erythroid reversal erythroid hyperplasia normoblastic maturation	USG -neonatal hepatitis,ascitis	neonatal hepatitis ?cause	D
56	microcytic hypochromic anemia	biopsy-gauchers disease	decreased beta glucocerebrosidase	Gauchers disease	L
57	normochromic normocytic anemia	hypercellular marrow blatst 40%-ALL L2	flow cytometry -ALL	ALL	T & F
58	microcytic hypochromic anemia	not done	usg-liver abscess NEC-Staph aureus	septicemia	T&F
59	microcytic hypochromic anemia	hypercellular blast 95% plt decreased- AML	flow cytometry-AML	AML	T & F
60	dimorphic anemia	hypercellular marrow blasts 35% -ALL	Cytometry-B cell ALL aberrent CD 13 expression	ALL -B CELL	T & F
61	normocytic normochromic anemia	hypercellular,blast 90%-ALL	Flow cytometry-ALL	ALL	T & F
62	microcytic hypochromic anemia	normocellular no blasts platlets decreased	flow cytometry-Burkitt lymphoma	Burkitt lymphoma /chemo induced	T & F
63	microcytichypochromic anemia	not done	NEC- CONS positive sepsis	septicemia	R
64	normocytic normochromic anemia	hypercellular blast 90% plt ,RBC decreased- ALL	flow cytometry -ALL	ALL	D
65	hypochromic microcytic anemia	hypercellular blast 45% plt decreased- ALL	flow cytometry -ALL	ALL	L
66	normocytic normochromic anemia	Hypercellular marrow Blast-45%-ALL	flow cytometry -ALL	ALL	D-renal mets
67	normochromic normocytic anemia	not done	USG-portal vein thrombosis,EHPVO,varicel bleed,grade 3 ,4 varice	liver disease portal hypertension	T & F
68	microcytic hypochromic anemia,spherocytes			Hereditary spherocytosis	T & F
69	normochromic normocytes	not done	NEC-GRAM negative sepsis	septicemia	T & F
70	normochromic normocytes	not done	NEC-CONS SEPSIS	septicemia	R
71		not done	IgM ELISA dengue positive	Dengue viral fever	R
72		not done	IgM ELISA dengue positive	Dengue viral fever	R
73		not done	IgM ELISA dengue positive	Dengue viral fever	R
74		not done	IgM ELISA dengue positive	Dengue viral fever	R
75		not done	IgM ELISA dengue positive	Dengue viral fever	R
76		not done	IgM ELISA dengue positive	Dengue viral fever	R
77		not done	IgM ELISA dengue positive	Dengue viral fever	R
78		not done	IgM ELISA dengue positive	Dengue viral fever	R
79		not done	IgM ELISA dengue positive	Dengue viral fever	R
80	normochromic normocytic anemia	myeloid erythroid reversal erythroid hyperplasia normoblastic maturation	flow cytometry -AML	AML	Died
81		not done	IgM ELISA dengue positive	Dengue viral fever	R
82		not done	IgM ELISA dengue positive	Dengue viral fever	R
83		not done	IgM ELISA dengue positive	Dengue viral fever	R
84		not done	NEC-no growth/ CRP positive	septicemia/pneumonia	R
85		not done	IgM ELISA dengue positive	Dengue viral fever	R
86		not done	IgM ELISA dengue positive	Dengue viral fever	R
87		not done	IgM ELISA dengue positive	Dengue viral fever	R
88		not done	IgM ELISA dengue positive	Dengue viral fever	R
89		not done	IgM ELISA dengue positive	Dengue viral fever	R
90		not done	IgM ELISA dengue positive	Dengue viral fever	R
91		not done	IgM ELISA dengue positive	Dengue viral fever	R
92		not done	IgM ELISA dengue positive	Dengue viral fever	R
93		not done	IgM ELISA dengue positive	Dengue viral fever	R
94		not done	IgM ELISA dengue positive	Dengue viral fever	R
95		not done	IgM ELISA dengue positive	Dengue viral fever	R
96		not done	IgM ELISA dengue positive	Dengue viral fever	R
97		not done	IgM ELISA dengue positive	Dengue viral fever	R
98		not done	IgM ELISA dengue positive	Dengue viral fever	R
99		not done	IgM ELISA dengue positive	Dengue viral fever	R
100		not done	IgM ELISA dengue positive	Dengue viral fever	R
101	hypochromic microcytic anemia	hypercellular marrow with blast 55% decreased platlet -ALL	flow cytometry -ALL	ALL	T & F
102	nomochromic normocytic anemia	hypercellular marrow with blast 60% decreased platlet-ALL	flow cytometry -ALL	ALL	T & F
103	normochromic normocytIC ANEMIA	hypercellular marrow blast 65% RBC ,Platelet decreased-ALL	flow cytometry -ALL	ALL	T & F
104	normochromic normocytiC ANEMIA	hypercellular marrow blast 85% RBC ,Platlet decreased -ALL	flow cytometry -ALL	ALL	T & F
105	normochromic normocytes	WBC normal ,RBC AND PLT decreased	flow cytometry/immuno histochemistry-NHL	NHL/Chemotheraphy induced	R-after chemo
106	normochromic normocytic anemia	hyper cellular marrow blast 30% suggestive of ALL	flow cytometry -ALL	ALL	D-Febrile neutropenia
107	normochromic normocytic anemia	hypercellular marrow blasts 85% -ALL	flow cytometry -ALL	ALL	D-septic shock
108	microcytic hypochromic anemia	Hypercellular marrow myeloblast 85% RBC, plt decreased -AML	flow cytometry-AML	AML	T & F
109	normochromic normocytic anemia	hypercellular marrow blast 90% RBC decreased -ALL	flow cytometry -ALL	ALL	T & F
110	microcytic hypochromic	hyper cellular marrow blast 30% suggestive of ALL	flow cytometry -ALL	ALL	L
111	hypochromic microcytic anemia	pancytopenia for evaluation	B12 LEVELS LOW	?MEGALOBLASTIC ANEMIA	L
112	hypochromic microcytic anemia	not done	NEC- CONS sepsis	septicemia	D

113	hypochromic microcytic anemia	not done	NEC- CONS sepsis	septicemia	D
114	hypochromic microcytic anemia	not done	CRP-Positive	?septicemia/acute CNS Infection	D
115		not done	NEC-GRAM negative sepsis	septicemia/septic shock	D
116		not done	NEC-GRAM negative sepsis	SEPTICEMIA/septic shock	D
117	hypochromic microcytic anemia	not done	cons positive sepsis sent to cipro gentamicin	septicemia	R
118	hypochromic microcytic anemia	pancytopenia for evaluation	NEC-GRAM negative sepsis	septicemia	R
119	hypochromic microcytic anemia	pancytopenia for evaluation	NEC- gram negative sepsis	septicemia	R
120	hypochromic microcytic anemia	pancytopenia for evaluation	NEC-CONS SEPSIS	septicemia	R
121		not done	NEC- gram negative sepsis	septicemia	D
122	normochromic normocytic anemia	hypercellular marrow blast 45% -ALL	flow cytometry -ALL	ALL	T & F
123	microcytic hypochromic	hypercellular blast 95% plt decreased ALL	flow cytometry -ALL	ALL	T & F
124	dimorphic anemia	Hypercellular marrow Blast-45%-ALL	flow cytometry -ALL	ALL	T & F
125	normochromic normocytic anemia	hypercellular,blast 90%-ALL	flow cytometry -ALL	ALL	F-Testicular relapse
126	hypochromic microcytic anemia	hypercellular marrow with blast 95% platlet decreased-ALL	flow cytometry -ALL	ALL	T&F-CNS relapse
127	nomochromic normocytic anemia	hypercellular marrow with blast 70% decreased platlet-AML	flow cytometry -AML	AML	T & F
128	normochromic normocytes anemia	hypercellular marrow blast 95% RBC , PLT decreased-ALL	flow cytometry -ALL	ALL	T & F
129	normochromic normocytes anemia	hypercellular marrow blast 95% RBC ,PLT decreased-AML	flow cytometry-AML	AML	T & F
130	normochromic normocytes anemia	hypercellular marrow blast 85% RBC ,Platlet decreased -ALL	flow cytometry -ALL	ALL	T & F
131	normochromic normocytes anemia	erythropoiesis,myelopoiesis supressed blast 51% -ALL	flow cytometry -ALL	ALL	D-sepsis
132	normochromic normocytes anemia	hypercellular marrow blasts 85% -ALL	flow cytometry -ALL	ALL	T&F
133	normochromic normocytes anemia	hypercellular marrow myeloblasts55% -AML	flow cytometry-AML	AML	D-Septecemia
134	microcytic hypochromic anemia	hypercellular marrow blasts 85% -ALL	flow cytometry -ALL	ALL	D-CNS relapse
135	normochromic normocytes anemia	hypercellular marrow blast 90 RBC decreased ALL	flow cytometry -ALL	ALL	T & F
136	microcytic hypochromic anemia	hyper cellular marrow blast 30% suggestive of ALL	flow cytometry -ALL	ALL	T-for relapse
137	normochromic normocytic anemia	hyper cellular marrow blasts 55% ALL	flow cytometry -ALL	ALL	T& F
138	microcytic hypochromic anemia	hypercellular blast 95% plt decreased AlL	flow cytometry -ALL	ALL	T & F
139	dimorphic anemia	hypercellular marrow blasts 85% -ALL	flow cytometry -ALL	ALL	T & F
140	normocytic normochromic anemia	hypercellular,blast 90% ALL	flow cytometry -ALL	ALL	D-septecemia
141	hypochromic microcytic anemia	Hypercellular marrow,blasts- 47% ALL	flow cytometry -ALL	ALL	T& F
142	normochromic normocytic anemia	Hypercellular marrow blasts 75% -ALL	flow cytometry -ALL	ALL	D-sepsis
143	hypochromic microcytic anemia	Hypercellular marrow,blasts- 57% ALL	flow cytometry -ALL	ALL	D-Pneumonia
144	nomochromic normocytic anemia	normal study	flow cytometry/immuno histochemistry-NHL	NHL/Chemotheraphy induced	T & F
145	normochromic normocytes anemia	hypercellular marrow blast 95% RBC decreased -ALL	flow cytometry -ALL	ALL	T & F
146	normochromic normocytes anemia	hypercellular marrow blast 95% RBC decreased-ALL	flow cytometry -ALL	ALL	T & F
147	normochromic normocytes anemia	hypercellular marrow blast 85% RBC decreased-ALL	flow cytometry -ALL	ALL/DOWNS SYNDROME	T & F
148	normochromic microcytic anemia	hypercellular marrow myeloblast 65% RBC , plt decreased -AML	flow cytometry-AML	AML	T & F
149	normochromic normocytic anemia	hypercellular marrow,blasts 68%,suggestive of ALL	flow cytometry -ALL	ALL	T & F
150	normochromic normocytes	not done	NEC-GRAM negative sepsis	septicemia	D
151	normochromic normocytes	not done	NEC-CONS SEPSIS	septicemia	R
152		not done	IgM ELISA dengue positive	Dengue viral fever	R
153		not done	IgM ELISA dengue positive	Dengue viral fever	R
154		not done	IgM ELISA dengue positive	Dengue viral fever	R
155		not done	IgM ELISA dengue positive	Dengue viral fever	R
156		not done	IgM ELISA dengue positive	Dengue viral fever	R
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174		not done	IgM ELISA dengue positive	Dengue viral fever	R
175		not done	IgM ELISA dengue positive	Dengue viral fever	R
176	normocytic normochromic rbc	not done	NEC-GRAM negative sepsis	septicemia	D
177	normocytic normochromic RBC	not done	NEC-CONS SEPSIS	septicemia	R
178		not done	IgM ELISA dengue positive	Dengue viral fever	R
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205	normocytic normochromic rbc	not done	NEC-GRAM negative sepsis	septicemia	D
206	normocytic normochromic rbc	not done	NEC-CONS SEPSIS	septicemia	R
207		not done	IgM ELISA dengue positive	Dengue viral fever	R
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230		not done	IgM ELISA dengue positive	Dengue viral fever	R
231	normocytic normochromic rbc	not done	NEC-GRAM negative sepsis	septicemia	D
232	normocytic normochromic rbc	not done	NEC-CONS SEPSIS	septicemia	R
233		not done	IgM ELISA dengue positive	Dengue viral fever	R
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285		not done	IgM ELISA dengue positive	Dengue viral fever	R
286		not done	IgM ELISA dengue positive	Dengue viral fever	R
287		not done	IgM ELISA dengue positive	Dengue viral fever	R
288		not done	IgM ELISA dengue positive	Dengue viral fever	R
289		not done	IgM ELISA dengue positive	Dengue viral fever	R

290	normochromic normocytlc anemia	hypercellular marrow blast 55% RBC , Platlet decreased-ALL	flow cytometry -ALL	ALL	D-Febrile neutropenia
291	not done	not done	Widal positive	Enteric fever	R
292	not done	not done	Widal positive	Enteric fever	R
293	not done	not done	Widal positive	Enteric fever	R
294	not done	not done	Widal positive	Enteric fever	R
295		not done	IgM ELISA dengue positive	Dengueviral fever	D-encephalitis
296	hypochromic microcytic anemia	1.dimorphic anemia 2.hypocellular marrow on follow up		?Pancytopenia for evaluation	F-aplastic anemia
297	hypochromic microcytic anemia	microcytic hypochromic anemia, lymphocytosis	IgM ELISA dengue positive,NS1 Ag positive	Dengue viral fever	F-hemophagocytic lymph
298		not done	IgM ELISA dengue positive	Dengue viral fever	R
299		not done	IgM ELISA dengue positive	Dengue viral fever	R
300		not done	IgM ELISA dengue positive	Dengue viral fever	R

LIST OF ABBREVIATIONS

AIDS	-	Acquired immuno deficiency Syndrome
ALL	-	Acute lymphoblastic leukemia
AML	-	Acute myeliod leukemia
BT	-	Bleeding Time
BMI	-	Body mass index
CT	-	Clotting Time
ESR	-	Erythrocyte sedimentation rate
g/dl	-	Grams per deciliter
SLE	-	Systemic Lupus erythematosus
MCV	-	Mean corpuscular volume
MRI	-	Magnetic resonance imaging
PT	-	Prothrombin time
RBC	-	Red blood cell
CFU	-	Colony forming unit
WBC	-	white blood cell
MCH	-	mean corpuscular hemoglobin
MCHC	-	mean corpuscular hemoglobin concentration
EBV	-	Ebstein bar virus
CMV	-	cytomegalo virus.

ITP	-	idiopathic thrombocytopenic purpura
HIV	-	human immunodeficiency virus
CBC	-	complete blood count
NSAID	-	non steroidal anti inflammatory drugs
CLD	-	Chronic liver disease
HLH	-	hemophagocytic lympho histiocytosis
MPV	-	mean platelet volume
PCV	-	packed cell volume
RDW	-	red cell distribution width
ESR	-	erythrocyte sedimentation rate
USG	-	ultrasonogram
APTT	-	activated partial thromboplastin time
PT	-	prothrombin time
PCR	-	polymerse chain reaction
Hb	-	hemoglobin
G-CSF	-	Granulocyte colony stimulating factor
BMT	-	bone marrow transplantation
CONS	-	coagulase negative staphylococcus aureus
CRP	-	C reactive protein
ANC	-	Absolute neutrophi;l count

NEC	-	Non Enteric culture
MP	-	Malarial parasite
G/E	-	General Examination
P/A	-	per abdomen
PS	-	Peripheral smear study.

ETHICAL COMMITTEE APPROVAL LETTER

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ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	Dr.M.Vijay Anand
Course	:	PG in MD., Paediatrics
Period of Study	:	2015 - 2018
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Children with bicytopenia and pancytopenia – clinical, etiological spectrum, outcome and follow up in a tertiary care centre'.
Ethical Committee as on	:	26.10.2016

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

DEAN
Madurai Medical College
Madurai-20

26 OCT 2016

ANTI PLAGIARISM CERTIFICATE

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CERTIFICATE

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