Sickle Cell Anemia in Homoeopathic Practice

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Introduction:

Sickle cell anemia now-a-days has attracted the attention of all medical branches to bring about a positive, scientific and rational curative medical aid to the patient, due to its global existence and high incidence of complications, mortality and morbidity rate. Initially sickling disorder was thought to be confined to Negro race, but now it is clearly demonstrated to be global distribution with highest prevalence in the tropical Africa and among blacks in countries that participated in the slave trade. However, it is understood as a World wide prevailing disease, but it is highly seen in Western India. As it is a genetic disorder, this disease cannot be cured, but the mortality rate can be reduced.

Literature review:

Sickle cell anaemia is a hereditary chronic form of anaemia in which abnormal sickle or crescent-shaped erythrocytes are present. The distorted sickle shaped red blood cells that results when the hemoglobin (has an altered amino-acid structure of polypeptide) is deoxygenated, molecules of membrane and the process gets reversed when reoxygenated. The distortion of red cell membrane may become permanent to make the red cells to assume sickle shape constantly. (1)

A normal adult can be depicted as having the hemoglobin genotype AA, sickle cell trait AS, sickle cell anemia or homozygous sickle cell disease by SS. The inheritance when both parents have trait can be shown thus.

1. Sickle cell trait (AS): these individuals may be asymptomatic throughout the life period but there are chances of manifestation of complain due to environmental and exciting factors.
2. Sickle cell disease (SS): these individuals are suffering from disease which presented by a group of complains in gradual course from infant age.

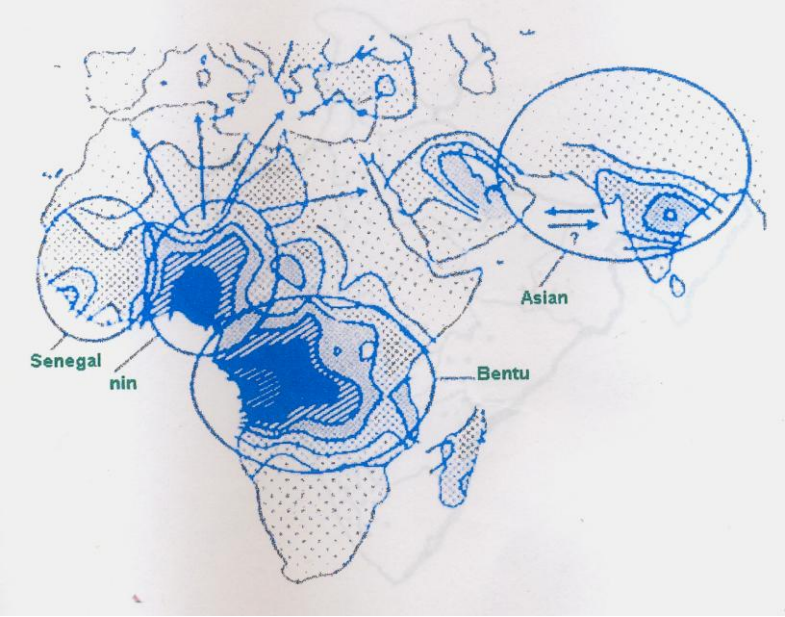
Historical review:

- Sickle cell anaemia alone is the most common heritable hematologic disease affecting humans. Long before they were recognized in the Western hemisphere, sickling disorders were known in Africa by onomatopoeic names denoting the recurrent, unrelenting and painful nature of the crisis. Although in one Ghanaian family, symptoms of sickle cell anaemia could be traced to the year 1670. Disorders of hemoglobin synthesis went unrecognized by the scientific community until 1910.
- In the year 1910, J. B. Herrick, a Chicago cardiologist, demonstrated the presence of peculiar, elongated sickle shaped red blood corpuscles in a 20 years old Negro student from Grenada of West Indies in case of severe anaemia. The fascinating account of the events that followed and of the individuals who contributed to the still unfolding story of sickle cell anaemia is presented in detail by Conley. Herrick's report led, not only to the recognition of hundreds of abnormalities of hemoglobin synthesis but also to a series of remarkable scientific advances involving protein chemistry, cell biology, physiology and genetics. (2)
- In 1911, a second case of sickle shaped RBC was found in a 25 years old Islack woman in a case of anaemia at the university of Virginia hospital. (3)
- In 1922, the term sickle cell anaemia was introduced by Mason & Taliaferro indicated the inherited nature of the disease and proposed that a single non-sex-linked abnormal gene acting as Mandelin dominant, is probably controlling such inheritance. In the next year Syndenstricker described the active and latent phase of disease and attributed the anaemia to be due to the excessive blood destruction resulting from sickling. He also introduced the word crisis in association with sickle cell anaemia.
- In 1924, Grahm observed recurrent paroxysm of acute illness characterized by fevers, prostration, pain in extremities, joints and evidence of marked blood destruction. (4)
- In 1927, Hahn & Gillespie explained the basis of abnormal behaviour of the sickle cell to be due to abnormalities of its hemoglobin in deoxygenated state and emphasized the intricate role diminished oxygen tension and reduced pH, etc. in accelerating the sickling person.
- In 1928, Hahn proposed the term sickle trait where the red cells although susceptible to sickling is unaccompanied by anaemia. (5)
- In 1934, Diggs & Ching Obendorf recognized 'Priapism' as one complication of sickle cell anaemia. (6)

- In 1940, Sherman reported that sickling develops much more rapidly in blood maintained at body temperature, and that the changes in shape occurred very slowly in preparations kept at low temperature.
- In 1945, Murphy and Shapiro noted increased prothrombin activity in sickle cell anaemia which is further increased crisis.
- In 1946, Cooly described hemolytic crisis occurring in patients with sickle cell anaemia in association with streptococcal infection. (8)
- In 1949, Neel described the relationship of sickle cell trait and sickle cell anaemia, which was convincingly shown through genetic study as heterozygote and homozygote respectively.
- In 1957, Gree-Burgh, et al. had explained two chief manifestations of sickle cell anaemia. (7)
- In 1964, Kilon F.M.et. al. first described high fluid intakes & urinary volumes occur in "SS" disease in children. In 1967, Nell et.al. marked the same in adults.
- In 1969, Kwak observed that the other side enuresis & nocturnal are common in children nearly 71% and 67% in adult group as compared to 33% control adult group.
- In 1972, Seeler & Shwiaki, reviewed the clinical feature of acute splenic sequestration & found that profound anaemia, splenomegaly and high reticulocyte count, leucocytosis & thrombocytopenia are common manifestation & immediate blood transfusion becomes highly. (9)
- In 1981, Pattison et.al. of London first described the patients having SS diseases are parvovirus infections which are responsible aplastic crisis of bone marrow.
- In 1982, Anderson et. al. described the same as Pattison et.al. (10)
- In 1985, Samuels-Reid & Scott published through the studies that there is no clear relationship between onset of painful crisis before or during the menstrual cycle. But there is a clear increased risk of painful crisis during pregnancy, especially in 3rd trimester & postpartum period being approximately five times that in the non-pregnant state for the same individual. (11)
- In 1990, Schumacher et.al. stated that muscle injury complicates sickle crisis.
- In 1991, Valerian Marcet & Kerr described that myofibrosis are seen complication of sickle cell anemia. (12)
- During the last four decades many investigations were done by many devoted workers in the field of biochemistry genetic study.

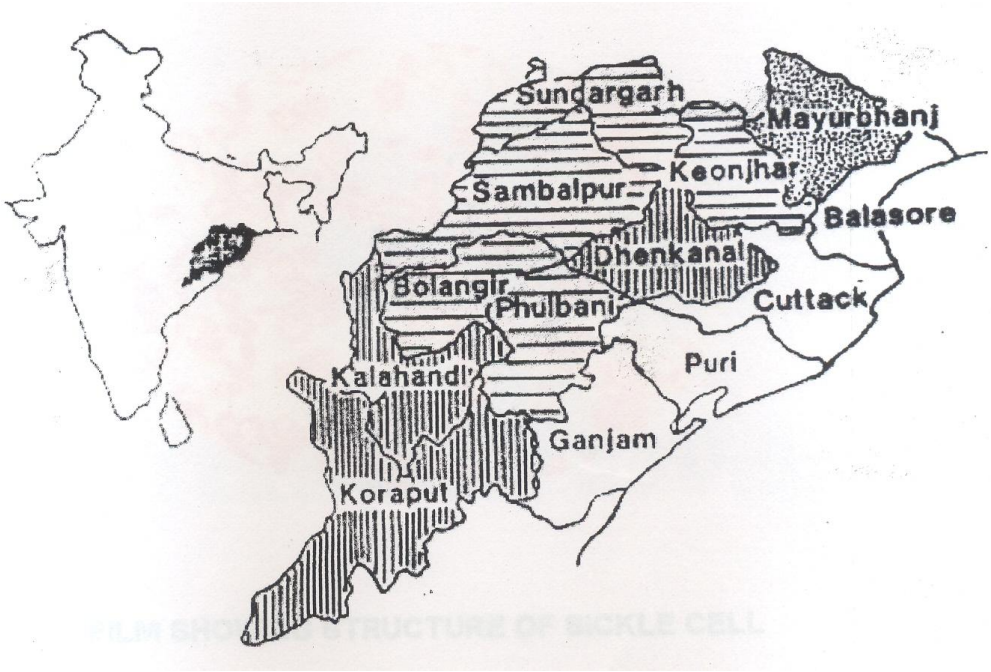
Prevalence: (geographical distribution)

- Results of studies of DNA polymorphism linked to the S- gene suggest that it arouse from three independent mutations in tropical Africa.
- The most common S-chromosome is found in Benin (neighboring Nigeria) and central West Africa.
- The second haplotype is prevalent in Senegal & the Africa West coast and third haplotype is seen in the Central African Republic (Bantu-speaking Africa).
- The same three haplotypes are associated with the gene in black Americans & Jamaicans.
- The Hb-S gene in the Eastern Province of Saudi Arabia & in Central India in association with the different DNA structures not encountered in Africa and probably represents a fourth independent occurrence of sickle cell mutation.
- Only the Benin and Senegal haplotypes are prevalent among North Africans, Greece & Italians, suggesting that the mutation spread to the Mediterranean basin from West Africa.
- In some parts of Africa as many as 45% of population have sickle cell trait. In the United States, Latin America and the Caribbean, approximately 8% of blacks carry the sickle gene. In United genes the expected incidence of sickle cell is 1 in 625.
- In recent years, it has been detected in India among non-tribal and consanguineous groups widely distributed over Assam, Rajasthan, Uttar Pradesh, Bihar, West Bengal, Madhya Pradesh Gujarat, Maharashtra, Tamilnadu, Andhra Pradesh and Orissa.
- In Orissa, among all the zone of western zone hold the lion's share of S-gene victims. (13)



Distribution in World

Distribution in India

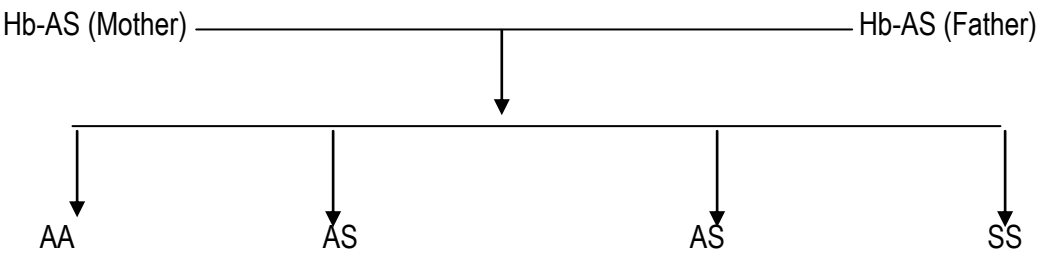


Distribution in Orissa

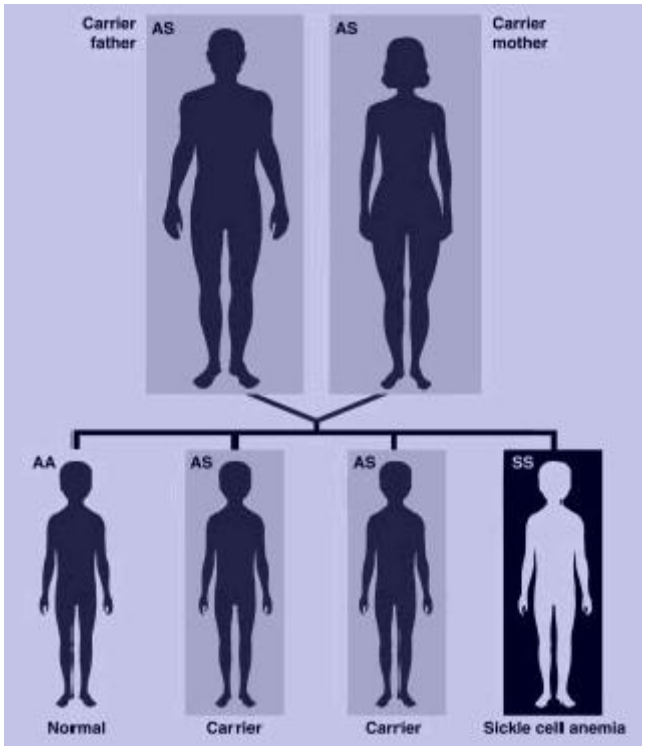
Incidence:

- The first detected case was in North America by Dr. Herrick (1910). He was believed that sickle cell anaemia was confined to Negroes, but subsequently its worldwide distribution has been reported by Margoleis (1951). Later Lahwan H. (1952) reported the presence of sickle cell anaemia cases in Greece, Gelphic (1970), in Saudi Arabia, Fleming et al (1979) in Nigeria, Villegas et. al. (1981) in Spain, and so on. In India Lehman H. Cutbush M (1952) first reported existence of sickle cell trait among tribal of Nilgiri in southern India.
- B.K. Nanda (1967) first reported sickle cell cases from western Orissa in the Agharia community. B.C. Kar (1987) made analysis of regional distribution of sickling positive cases & reported as 1.6% among the population of costal district & 13.09% in hilly district of Orissa were found Sickling positive.
- In survey report of C.C.R.H. (1993) indicates that sickle cell haemoglobinopathy is not only limited to some castes or tribe but is prevalent in nearly all castes.
- Now a day the sickle cell anaemia has a worldwide prevalence. Sickle cell disease is an inherited blood disorder characterized by defective hemoglobin. It affects millions of people throughout the world. In India it has been increasing day by day. Basically, the disease is more prevalence in Western Orissa and part of Chhatishgarh.
- Sickle cell disease is an inherited disease caused by a genetic mutation. Genes are found on structures in the cells of our body called chromosomes. There are normally 46 total or 23 pairs, of chromosomes in each cell of our body. The 11th pair of chromosomes contains a gene responsible for normal hemoglobin production.
- A mutation or error in this gene is what causes sickle cell disease. This mutation is thought to have originated in areas of the world where malaria was common, since people with sickle trait do not get malaria. The sickle trait actually protects them from the parasite that causes malaria, which is carried by mosquitoes. Malaria is most often seen in Africa and in the Mediterranean area of Europe, India.
- Children who inherit the genetic mutation from both parents will have sickle cell disease. Children who inherit the mutation from only one parent will not have the disease, but will carry the trait for it and can pass it on to their children. (14)

The genetic basis of sickling was first suggested by Cook and Meyer in 1915. Then Taliaferro and Huck in 1923 led conclusion that the sickle cell phenomenon was inherited as a Mendelion autosomal dominant character. When one of the partner of parent is heterozygous for the sickle cell gene and other is normal, the offspring would have equal chance of either sickle cell gene (AS) trait or normal genotype (AA). If both partner of parent have gene (AS), there is chance of 1 in 2 offspring having sickle cell trait (AS), and 1 in 4 offspring having normal gene (AA) or having gene (SS) called sickle cell disease.



The inheritance shown that 'AA'-stand for normal offspring, 'AS'- stand for sickle cell trait, and 'SS' stands for sickle cell anaemia offspring.



Pathogenesis:

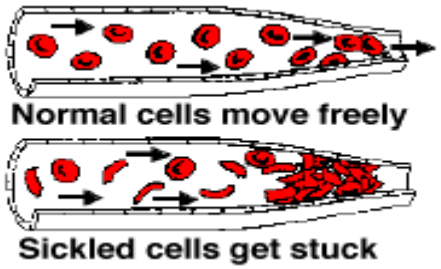
The most important and widely prevalent type of haemoglobinopathy is due to the presence of sickle haemoglobin (HbS) in the red blood cells. The red cells with HbS develop 'sickling' when they are exposed to low oxygen tension. Sickle syndrome has the highest frequency in black race and in central Africa where falciparum malaria is endemic. Patient with HbS are relatively protected against falciparum malaria. (15)

Sickle syndrome occurs in 3 different forms:

I. Heterozygous state for HbS: sickle cell trait (AS)

II. Homozygous state for HbS: sickle cell anaemia (SS)

III. Double heterozygous states: sickle β thalassamia (16)

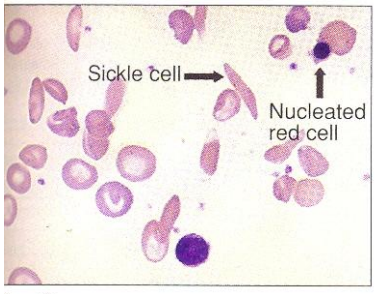


The basic molecular lesion in HbS is substitution of valine for glutamic acid at the 6 residue position of the β globin, producing Hb α β. In sickling 'SS' patients the red cells are predominance HbS and small part consists of non HbS haemoglobin . HbF(2-20% of the total haemoglobin. During deoxygenaton the red cells containing HbS change from biconcave disc shape to an elongated crescent shaped or sickle shaped cell. This process termed sickling occurs both in the intact red cells in vitro in free solution. The mechaism responsible for sickling upon deoxygenation of HbS containing red cells is the polymerization of deoxygenated HbS which aggregates to form elongated rod –like polymers. These elongated fibres align an distort the red cells into classic sickle shape. (17)

The clinical manifestation of homozygous sickle cell disease are wide spread. The symptoms begin to appear after 6th month of life when most of the HbF is replaced by HbS. Infection and folic acid deficiency result is more severe clinical manifestation .

Clinical manifestations of sickle cell anaemia:

1. Constitutional:
    a) Impaired growth and development.
    b) Increased susceptibility to infection.



2. Anaemia

    a) Severe haemolytic anaemia

    b) Aplastic anaemia.

3. Vaso-occlusive phenomena.

    a) Micro infarcts

    b) Macro infarcts.

1. Constitutional manifestations:

a. Impaired growth and development:

- Delayed growth and development
- A general failure to thrive
- In females delayed puberty is marked.
- "SS" patients have a marked impairment of splenic function preventing effective clearance of circulating bacteria creating an increased tendency to develop serious infections particularly due to pneumococcus. Splenomegaly also has been reported frequently in sickle cell anaemia in children as well as in adults.
- In general there may be weakness, pain in abdomen, pain in limbs, painful swelling of hand and feet, growth retardation, bony abnormalities and episodes of jaundice. Hepatomegaly may be marked in some cases. (18)

b. Increased susceptibility towards infection:

Susceptibility to infection is due to defective splenic function, defective opsonisation, and defective immunity pattern. The common infections in sickle cell anaemia are protozoal infections like malaria, infection by salmonella causing osteomyelitis, pneumococcal infection, influenza, urinary tract infection, etc. (19)

2. Anaemia-

a) Severe hemolysis produces two stages: i) chronic haemolytic anaemia, ii) Hyperhaemolytic crisis.

Chronic haemolytic anaemia is due to reduced cell survival, because in homozygous "SS" group the destruction of red blood cell is independent of cell age and the mean red blood cell survival is about 10 to 15 days and appears after 4-6 months of life. Due to this repeated cell destruction bone marrow fails to compensate it and maintenance the equilibrium of red blood cells as a result anaemia develops. There are usually severe haemolytic anaemia with onset of aplastic crisis. The symptoms of anaemia are generally mild since gives up oxygen more readily than HbA to the tissues.

b) Hyperhaemolytic crisis: During this crisis there is an acute increase in break down od red cells leading to a swift fall in hemoglobin resulting in a marked increase in the severity of anaemia. In this period the scleral icterus is more marked and the patient may have abdominal pain and splenomegaly.

c) Aplastic crisis: There is a transient aplasia of erythropoetic tissue followed by development of severe anaemia. This is known as aplastic crisis. It is due to folic acid deficiency and infection which brings about a transient reduction in red blood cell production, especially human parvovirus infection, which causes an abrupt suppression of erythropoiesis. (20)

***3.* Vaso-occlusive phenomena:**

Patients of SS develop recurrent vaso- occlusive episodes throughout their lives due to obstruction to capillary blood flow by sickle red cells upon deoxygenetion or dehydration. Vaso-obstruction affecting different organs and tissues results in infarcts which may be 2 types:

i. Microinfarcts affecting the abdomen , chest, back and joints which are the cause of recurrent painful crises in SS patients

ii. Macroinfarcts involving the spleen ,bones, lungs, kidneys, liver and skin which result in anatomic and functional damage to these organs.

In addition to the features of anaemia and infarction, the patients with 'SS' have impaired growth and development and increased susceptibility to infection due to markedly impaired splenic function . (21)

Micro infarcts:

Common association during vaso-occlusive crisis:

Recurrent painful and febrile condition in crisis:

It is the commonest mode of presentation of vaso-occlusive crisis patients with sickle cell disease. These are plagued throughout their lives with recurrent attacks of pain in the joints and bones, which are thought to be due to stasis of blood in tendons synovial tissues and bones. The pain crisis is more painful in childhood and the frequency tends to decrease with the advancing years. The duration of pain varies from several days to several weeks but in majority cases the attack last for 4 to 6 days. Attack of fever is minor from where the crisis is of short duration. In severe cases the temperature range from 101-104 degree F. and the fever appear several hours after pain. During painful crisis, liver is usually enlarged.

During painful crisis there is significant changes in red cell count, Hb Conc., Haematocrit values, mean corpuscular volume (MCV) or reticulocyte count as compared to steady state. There is increase in total leucocyte count during the painful crisis.

Other laboratory features of Sickle Cell Anaemia:

1. Moderately severe normocytic, normochromic, anaemia is seen.
2. The MCV and MCHC are normal.
3. Blood smears contain variable number of sickled RBC.
4. The platelet count is increased (approx. 440 x 10) reflecting reduced or absent splenic sequestration.
5. The ESR is consistently low even in the presence of anaemia, because of the failure of sickle cell to undergo rouleaux formation. (22)

Macro infarcts: (leading to chronic organic damage)

By the time that the patients reach adulthood there is often objective evidence of anatomical and functional damage to various tissues due to the cumulative effect of recurrent vaso-occlusive episodes. Almost all organs may be involved but the commonest sites are the lungs kidney, liver, skeleton and skin.

i) Cardiopulmonary - Congestive cardiac failure, myocardial infarction, cardiomegaly, dyspnoea, chest pain, cough. (

ii) Hepatobiliary- Icterus, Gallstone, hepatic infarction, liver abscess, fibrosis. (23)

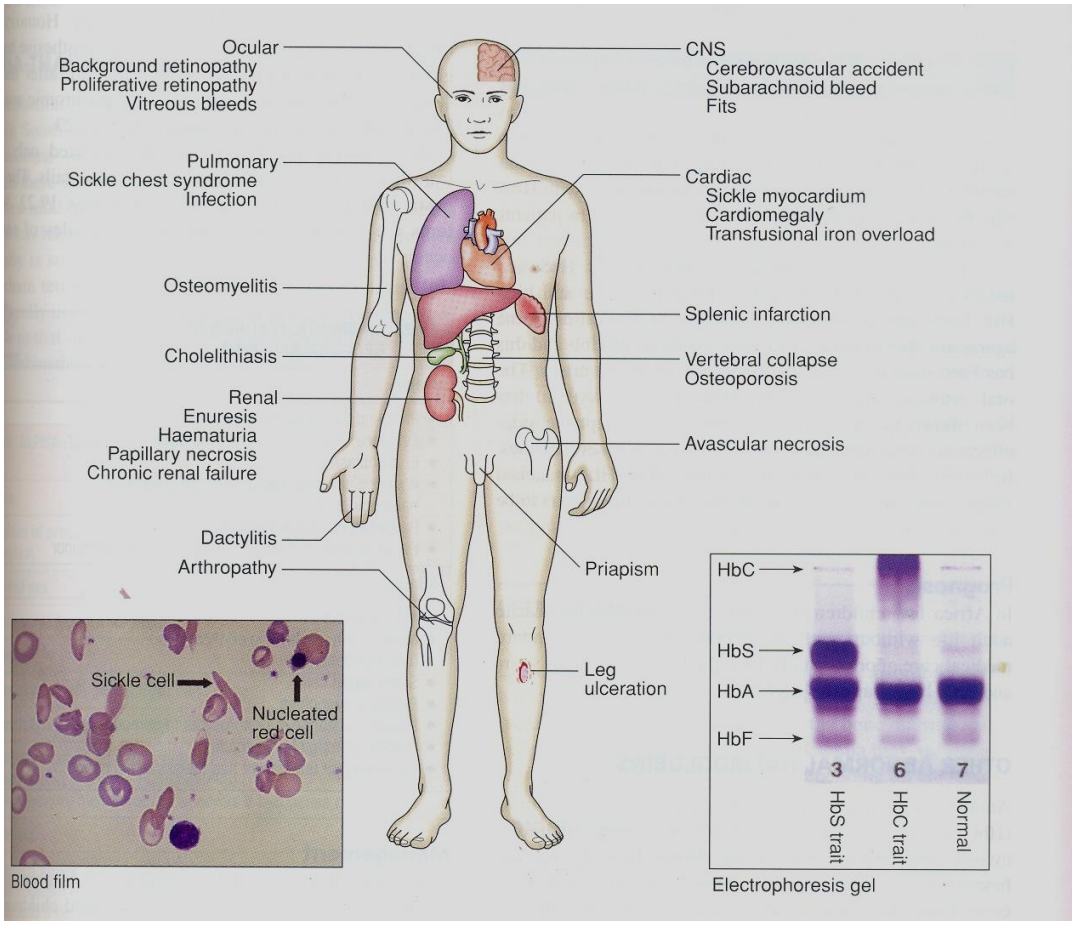
iii) Genitourinary – cortical or medullary necrosis, enuresis in children and nocturia in adults, priapism, hematuria. (24)

iv) Skeletal – Increased trabeculation and sclerosis of bones, aseptic necrosis of head of femur, osteoarthritis. (25)

v) Ocular – Retinal infarction, peripheral vessel disease, vitreous hemorrhage, retinitis proliference, retinal detachment. (26)

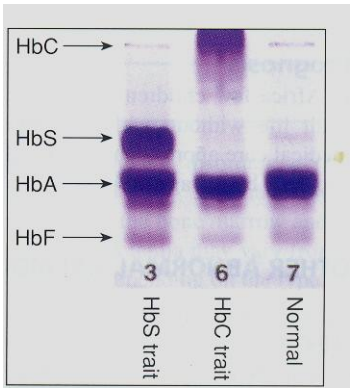
vi) Skin – chronic skin ulcer in the distal lower extremities. (27)

vii) Neurological – cerebral thrombosis, subarachnoid hemorrhage, hemiplegia, convulsion and visual disturbances. (28)



Diagnosis:

The diagnosis of sickle cell anaemia considered in any black patient with a haemolytic anaemia. The history of painful crisis, arthropathy, ankle ulcer etc. are very helpful for diagnosis. If a patient has relatively mild form the disease the diagnosis may not required. Examination of peripheral blood smear reveals, normochromic normocytic red blood cells. The presence of irreversible sickled form cells is very helpful for diagnosis and presence of normoblast suggest the absence of effective splenic function. The patient with homozygous sickle cell anaemia have about 2 to 20% HbF and 2 to 4% HbA Apart from that the solubility test for sickling, electrophoresis for distinguish between 'AS & 'SS'. (29)



Electrophoresis

Laboratory Investigation:

1. Sickling test of patient and family members.
2. Haemoglobin electrophoresis.
3. Haemogram – Hb%, DC, ESR, TLC, TRBC, Reticulocyte count, Platelet count, PCV, MCV.
4. Urine – bile salt and bile pigment.
5. Serum Bilirubin.
6. Liver function test.
7. Radiological investigation for bone abnormality and deformity. (30)

Prognosis

The highest mortality rate of sickle cell anaemia cases are seen in world literature during the first 5 yrs of life. The cause of death among 241 Jamaican patients with 'SS' disease indicate that acute chest syndrome, renal failure, acute splenic sequestration and meningitis were most common.

During the last 40 yrs considerable improvement have seen in sickle cell anaemia patient. The increasing number of patients are surviving and bearing offspring's. There has been decline in the mortality of 'SS' mothers during pregnancy and child birth. (31)

Prevention:

Sickle cell disease and other clinically significant sickle syndrome can be prevented in two general ways.

A. Genetic counseling of identified heterozygous can alert couples at risk about the possibility of heaving affected offspring.

B. Prenatal diagnostics services for pregnancies at risk for sickle cell anemia, is achieved by:

i) Fetal blood sampling by fetoscopy for assays of haemoglobin synthesis & analysis by gene mapping techniques of D.N.A. from amniotic fluid cells, obtained after amniocentesis between 14 & 20 weeks of gestation.

ii) Analysis of D.N.A. from chrionic villi obtained by transcervical biopsy between 8 & 10 week of gestation , suggest the geno- type of offspring &birth of' 'SS' +ve offspring can be prevented by pregnancy .

Aim and objectives:

1. To find out the efficacy of homoeopathic drugs in the treatment of Sickle-cell Anaemia.
2. To study different age, sex, habitat, socio-economic group, body built.
3. To analyse the various types of precipitating factors.
4. To study the various symptomatology in sickle cell patients.
5. To study the incidence of Hb SS and Hb AS patients.
6. To study increased Hb% after Homoeopathic treatment.
7. To observe the action of different homoeopathic medicines in various groups with their reliable indications, suitable potencies and repetition schedule.

Materials and methods:

Source of Case Materials

The present study includes the cases of sickle cell anaemia who were admitted to the I.P.D. & O.P.D. of Dr. Abhin Chandra Homoeopathic Medical College and Hospital, Bhubaneswar/ authors own clinic.

A prospective controlled study was conducted during the period from 1992 to 2007. 992 patients of varying ages, sexes, socio-economic status, and different body built and habitats were taken for study.

Research Design

1. **Selection of Research Strategies:**
   Among the patients admitted to above institution/clinic during above mentioned period, whose ever fitted to any of the following diagnostic criteria were taken up for the primary screening for detection of sickle cell anaemia.

2. **Selection of Research Setting:**
   Diagnostic criteria

   (A) Inclusion

   Patients of varying ages, both sexes, different body built, occupation, and habitats having the following symptoms, signs and radiological features were diagnosed as suffering from sickle cell anaemia and were given treatment:

   a. Symptoms

   Subjective
   - Recurrent attack of sudden pain in the extremities with or without fever.
   - Recurrent attack of fever and headache
   - Great restlessness with anxiety
   - Sudden attack of fever, pain with joints swelling

Objective

- Chest pain or breathlessness
- Abdominal colic with gas like discomfort.
- Anorexia and weakness general
- Skin eruption with yellowish discolouration.
- Epistaxis

Sign

- Anaemia
- Jaundice
- Enlargement of spleen
- Enlargement of liver

Laboratory Finding

Sickling of R.B.C.(Positive)

- Hb% - low
- Hb. Electrophoresis – AS / SS
- Hb. HPLC – AS / SS

(B) Exclusion

(1) Sickling of R.B.C. (negative)

(2) Hb Electrophoresis – (AS / SS)- (negative)

(3) Hb HPLC finding (AS /SS) (negative)

(4) Hb. HPLC finding (AS/ SS) (negative).

3. Sampling:

a) Sample size: In a view of the design of this study, the sample size was set at 992

b) Sampling Method – simple random method was adopted for selection of the case.

4. Use of Controls:

The above diagnostic criteria were adopted for the use of controls too.

5. Study of Instruments:

To minimize, interviewer and respondent bias, a standard case recording proforma was prepared. Following laboratory tests were done.

(1) D.C.
(2) Hb. Electrophoresis
(3) Hb. HPLC
(4) Hb%

Detailed method of study of the selected cases:

(i) A careful detailed history was taken as to the specific symptoms, duration of illness, past history of diseases and presence of any predisposing factors, were properly evaluated by clinical examination of all systems. Then the above laboratory tests were done.

Short Description for Collection of Data

Case Recording

One standard case recording format was prepared for maintaining the clinical profiles of the patients which incorporated the bio-data as well as other specific information about the patients including their age, sex, habitat habits, socio-economic status, their presenting features from four dimensions location (including extension of pain) /sensation,/modalities/concomitants and their mode of onset and ,progress, past history including injuries, family / personal, obstetrical and gynecological / treatment histories; physical / mental generalities etc.

The routine examination of blood was done in each case such as D.C., Hb. Electrophoresis, Hb HPLC, hemoglobin etc.

Categorization of patients:

The patients were categorized into 2 groups on the basis of medicine administrated to them which are as follow:

A. **Group I. (test group):** Patients were administered drug evolved on the basis of its repertorisation (only homoeopathic medicine)

B. **Group II (control group):** Patients administered placebo.

Repertorisation:

After careful case taking and diagnosis prescription through repertorisation was done. Different steps of repertorisation i.e. evaluation of symptoms, analysis, synthesis and finally building up the reportorial totality were strictly followed in each and every cases. Every repertorisation was done by taking the help of computer in both i.e. RADAR and Hompath classic.

Administration of medicine:

After thorough repertorisation selection of the similimum was done giving due importance to the following factors.

a) Constitution of the patient
b) Miasmatic background
c) Past and family history
d) Presenting complaints

After considering the above factors most similimum was selected and administered in different potencies according to the case before hand.

After repertorisation with respective repertories on the basis of its adoptability, the drug evolved in the panel on the basis of totality of symptoms a similimum was prescribed to each patient.

Selection of Potency:

Selection of potency was done by following strict homoeopathic criteria laid down by different stalwarts. Selection of the potency was done basing entirely upon the susceptibility of the patient which in turns depends on the following criteria.

i. Age of the patient
ii. Sex of the patient
iii. Nature and duration of the illness
iv. Involvement of vital organ
v. Moral and intellectual faculty of the patient
vi. Occupation/status of the patient
vii. Lastly degree of the similarity between disease symptoms with that of medicinal symptoms.

In general selections of potencies were done depending upon the following categorization.

a) Indicated medicines were administered in centesimal (30, 200, & 1M) & 50 millesimal potencies.
b) Selection of potencies, dose and administration of medicines were done on the guidelines prescribed by Hahnemann in his Organon of Medicine, vide Aphorisms 272, 292, 248 (along with its foot note), 269-270 (along with its foot notes) & 272-273; by Roberts vide chapter XIII of the book, "The Principles and Art of Cure by Homeopathy" (1976, PP 113-122).

Repetition Schedule:

Indicated medicines were given in single or in repeated doses on the basis of the principles laid down by Hahnemann in his organon of Medicine Vide Aphorisms 272,292, Kent in his lecture XXXV and XXXVI of "Lectures on Homeopathic Philosophy" (1967, PP 224-241). Robert in the chapter – XIV, XVI of "The principles & Art of Cure by Homeopathy" (1967, PP 124-134, 144-149).

Follow up:

After prescribing drugs & providing other instructions, the patients were asked to report at suitable intervals (preferably) after every 4 weeks in the O.P.D./clinic and when required they were admitted in the I.P.D for proper follow up.

At each unit a detail follow up record were maintained as regards improvement or worsening of each symptoms and subsequent drugs were prescribed

Assessment of results:

It is difficult to formulate definite assessment criteria, in of view pathogenesis and unpredictable course of the disease. However the cases were assessed in terms of their presenting symptom physical signs and pathological findings.

Administration of Medicines:

Oral route was chosen for the administration of medicine.

Short description for Analysis of data

The results were documented after the administration of medicine as per Kent's Repertorial totality. The parameters fixed for documentation were as follows;

(A) Positive Responses

i. **Marked improvement –**

- In this parameter the patients remain absolutely free from any kind of complaints in relation with sickle cell anaemia 'Hb AS' or 'Hb SS' comprising of the symptoms like marked pallor, fever, joints pain, extremities pain or swelling, loss of appetite etc. for a period of 10 to 15 years during the treatment, with earlier history of attacks being at short intervals or long intervals respectively.

ii. **Moderate Improvement –**

- In this parameter the patients remain free from relation with sickle cell anemia 'Hb AS' or 'Hb SS' for a period from 1 to 2 years during regular follow-up while undergoing treatment with an earlier history of complaints as above are taken as moderate improved.

iii. **Mild Improvement –**

In the parameter the increase in spacing between the paroxysm / crisis associated with removal or reduction in intensity and duration of complaints are taken as mildly improved.

(B) Negative Responses

i. **No improvement -** Patient did not improve with indicated medicine although prescribed for sufficient of period of time.
ii. **Aggravation** - Condition of the patients was increased during course of treatment.
iii. **Dropped out** – The patient did not stick to the treatment for sufficient period of time.

Age group

For recording of data following age groups were made:

a) 1 – 10 yrs.
b) 11 -20 yrs.
c) 21 – 30 yrs.
d) 31 yrs and above.

For recording of data for socio-economic group:

Lower socio-economic – Annual income less than 10,000.
Middle socio-economic – Annual income ranging from more than 10,000 to 100,000
Higher socio-economic – Annual income more than 100, 000.

Observations:

Table -1 (Sex distribution)

Sex	No. of patients	%
Male	512	51.61
Female	480	48.387
Total	992	100.00

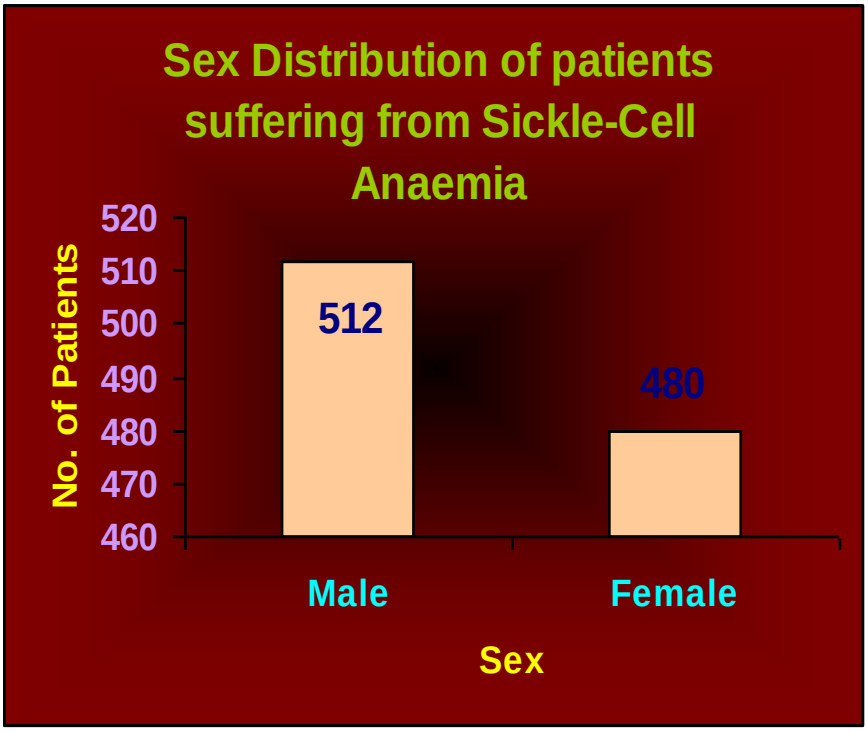


Table- 2 (Age distribution)

Age groups	No. of patients	%
1-10 yrs	482	48.588
11-20 yrs	210	21.693
21-30 yrs	200	20.161
31yrs above	100	10.080
Total	992	100.00

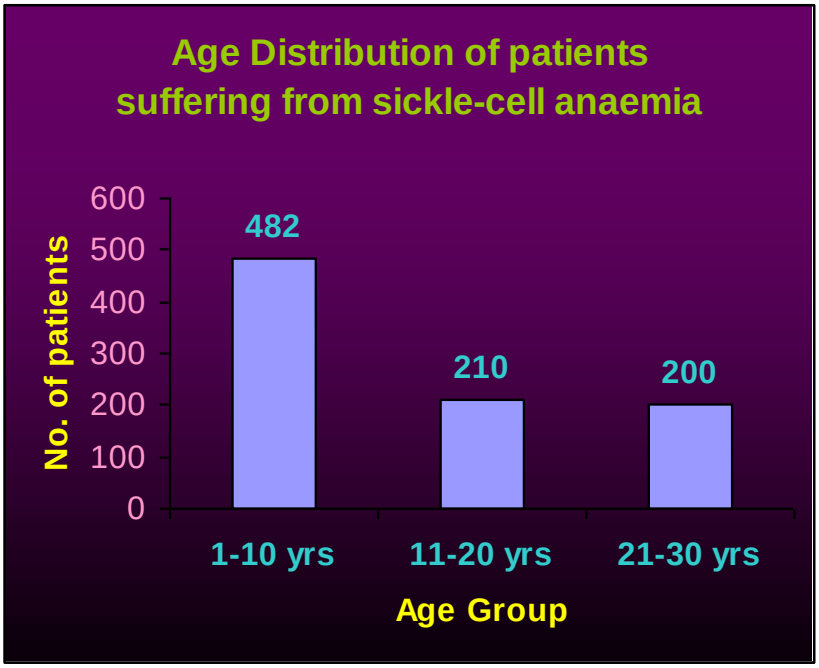


Table-3 (Socio-Economic Distribution)

Category	L.S.E.G	%	M.S.E.G	%	H.S.E.G	%
Male	262	52	158	45	92	65
Female	240	48	191	55	49	35
Total	502	100	349	100	141	100

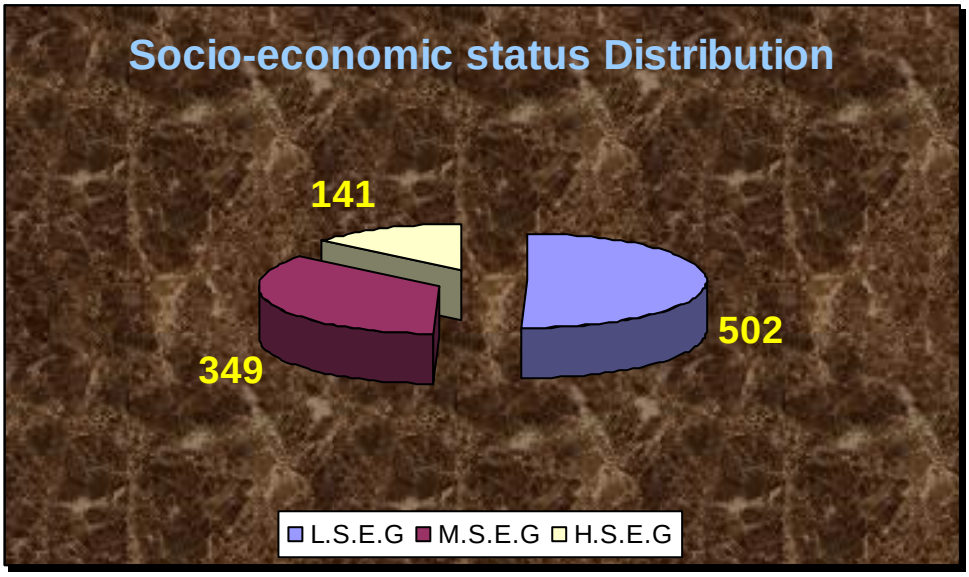


Table -4 (Distribution of general Built status of sickle cell anemia)

Types	No. of patients	%
Lean and thin	708	71.370
Moderate	204	20.564
Obese	80	8.064

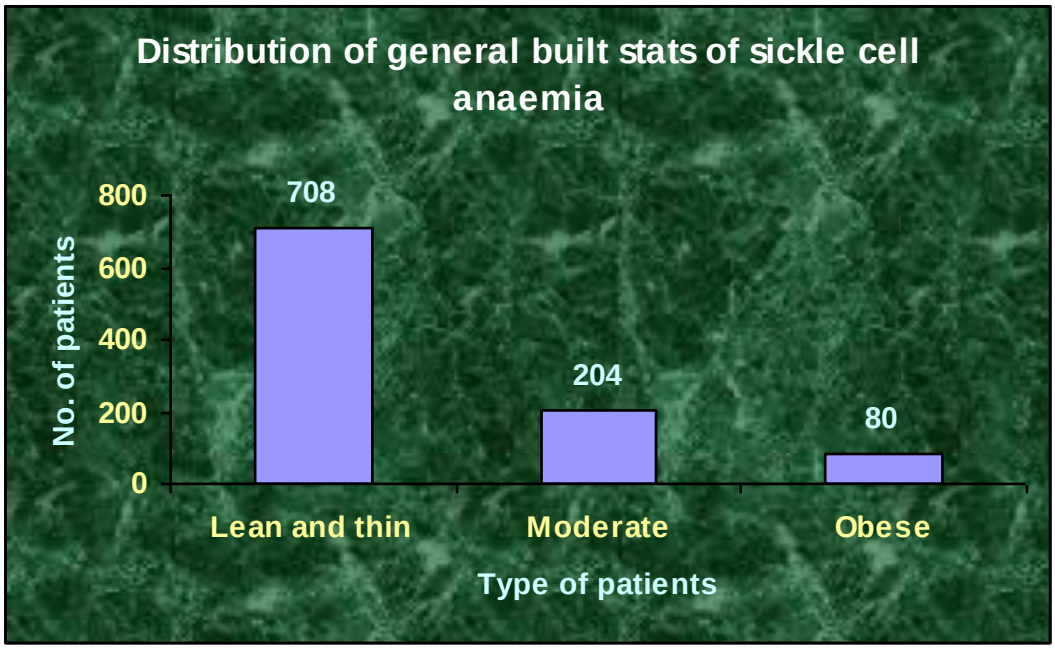


Table-5 (Distribution according to nativity)

Nativity	No. of patient	%
Rural	690	69.564
Urban	302	30.44
Total	992	100.00

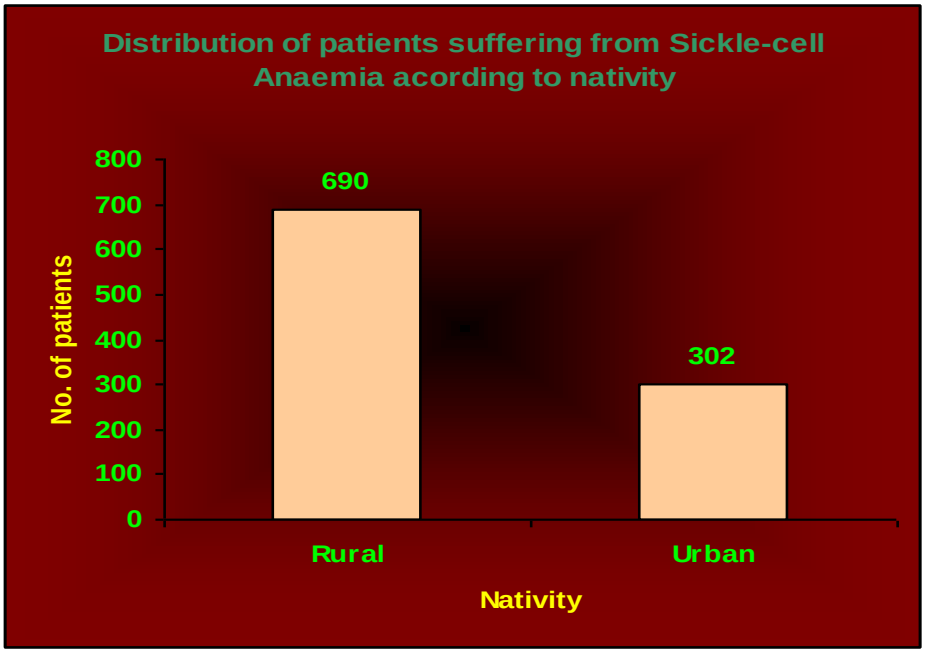


Table-6 (Distribution according to Pathological investigation)

Diagnostic test	No. of patients	%
Sickling	992	100
Hb Electrophoresis	992	100

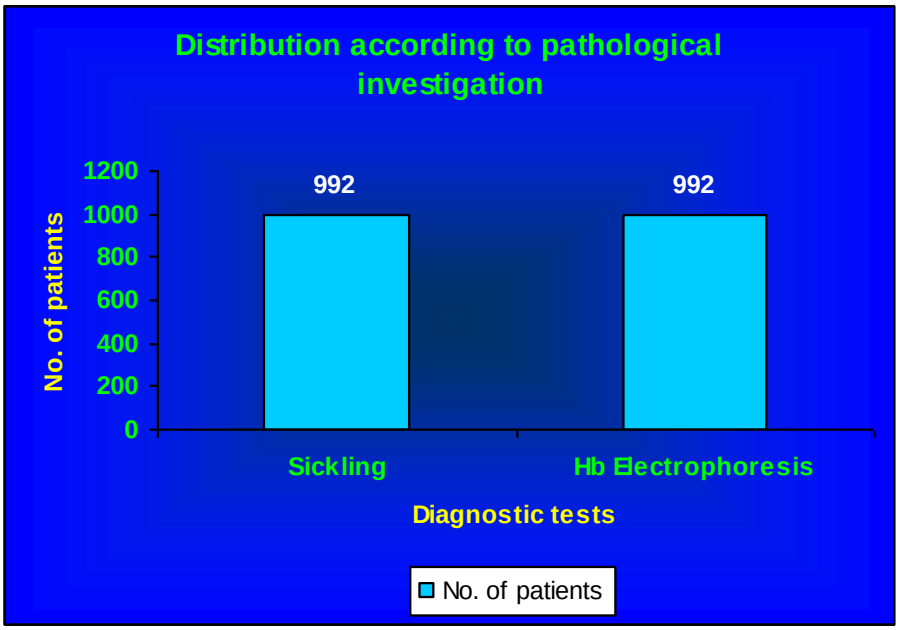


Table-7 (Assessment of symptomatic improvement)

Category of symptoms Test group	No. of patients	No. of cases improved	%
1. Anaemia	900	792	88
2. Extremities pain	702	647	92.16
3. Abdominal pain	607	543	89.45
4. Acute chest syndrome	109	90	82.56
5. Enlargement of spleen	798	392	49.12
6. Jaundice	590	409	69.32
7. Constipation	452	409	90.48
8. Vertigo	703	692	98.43
9. Impotency	80	62	77.5
10. Damage of bone	4	2	50
11. Early Gall stone	5	0	0
12. Damage of kidney	0	0	0
13. Damage of eyes	0	0	0

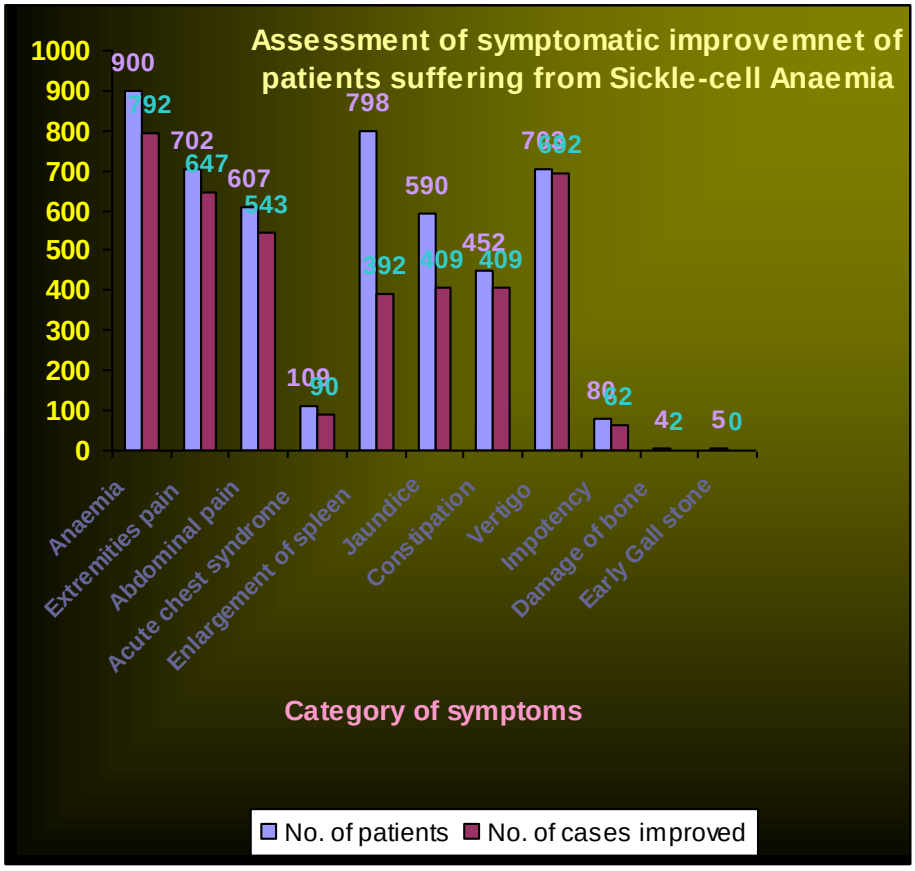


Table-8 (General response of the treatment)

Group	+ve response	-ve response	Total
Test group	809	91	900
Control	09	83	92
Total	818	174	992

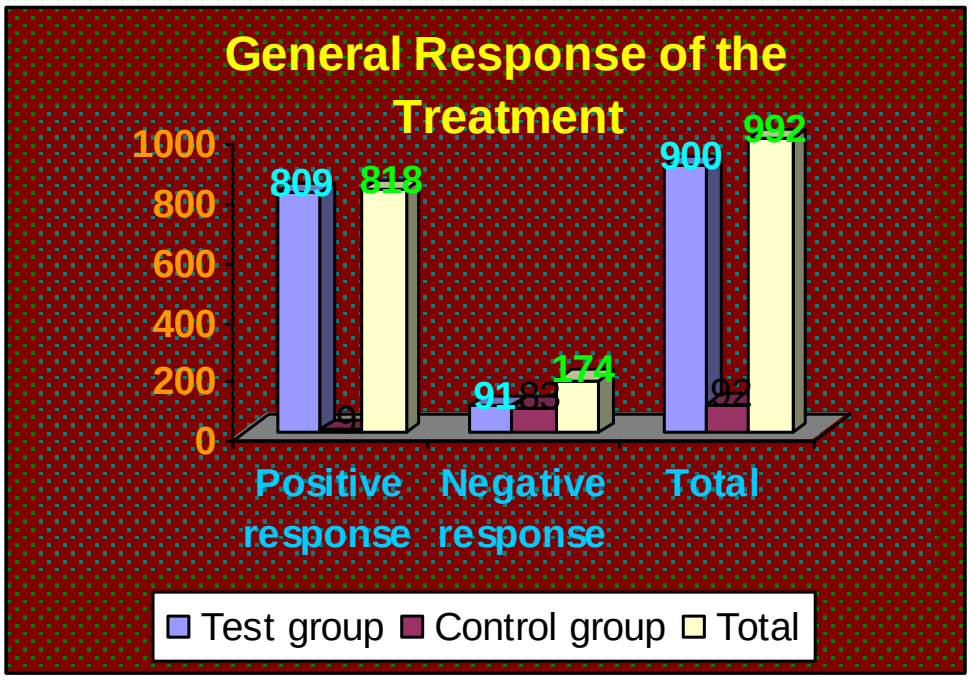
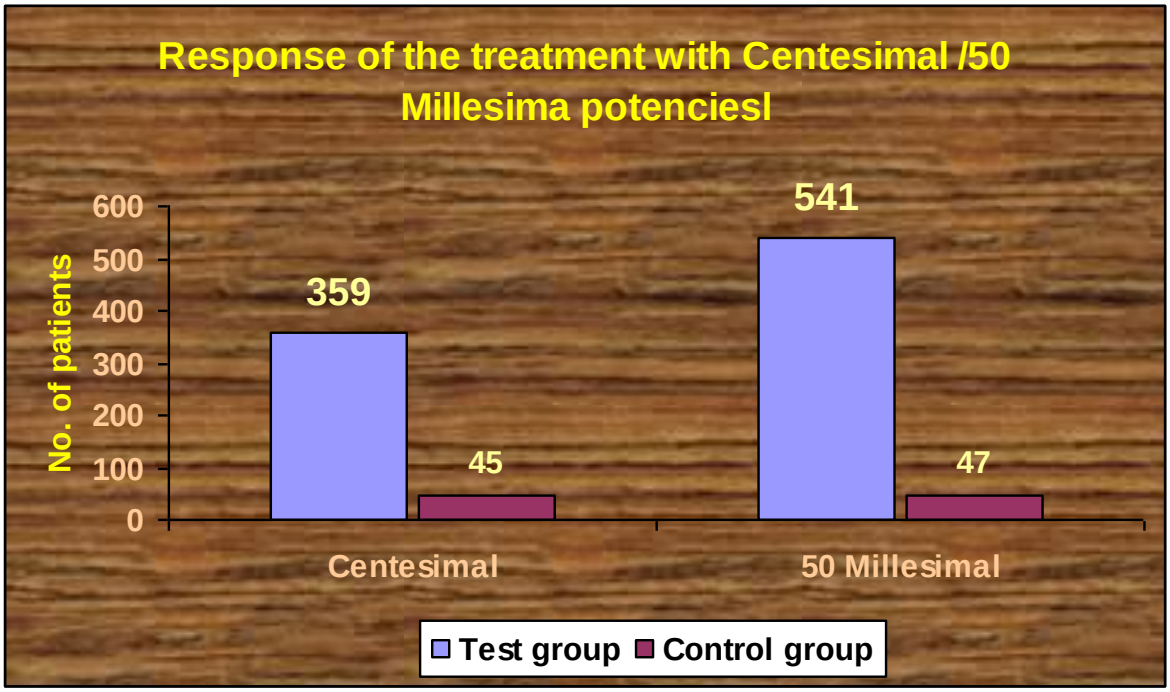
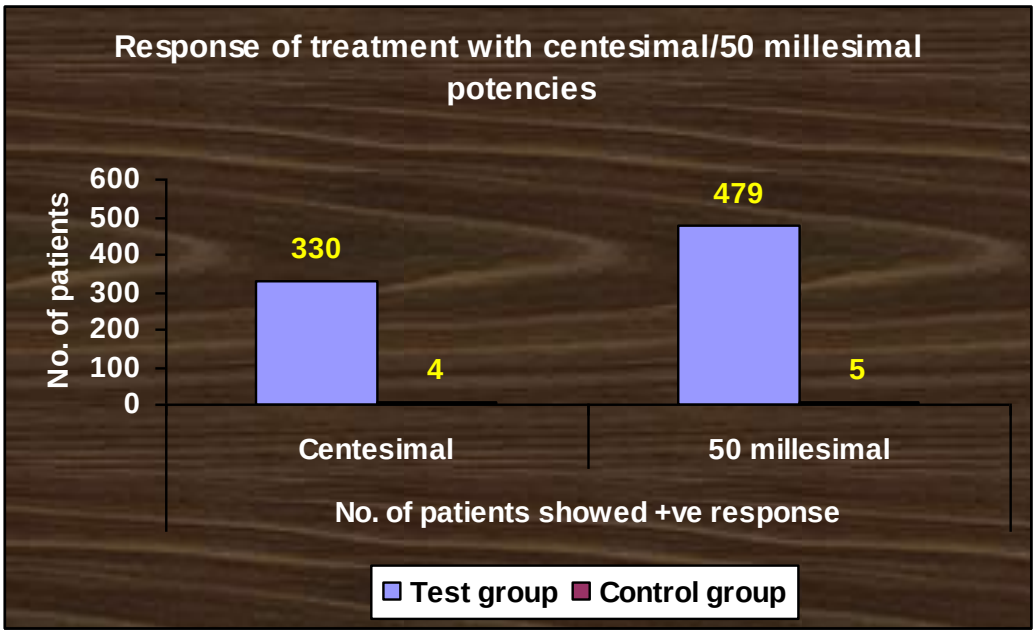


Table-9 (Response of the treatment with centesimal/ fifty millesimal potencies)

Response	No. of patients		No. of patients showed +ve response		No. of patients showed –ve response	
	Centesimal	50 Millesimal	Centesimal	50 millesimal	Centesimal	50 millesimal
Test group	359	541	330	479	29	62
Control group	45	47	4	5	41	42
Total	404	588	334	484	70	104




Response of treatment with centesimal/50 millesimal potencies
No. of patients
600
500
400
300
200
100
0
330
4
479
5
Centesimal
50 millesimal
No. of patients showed +ve response
Test group
Control group

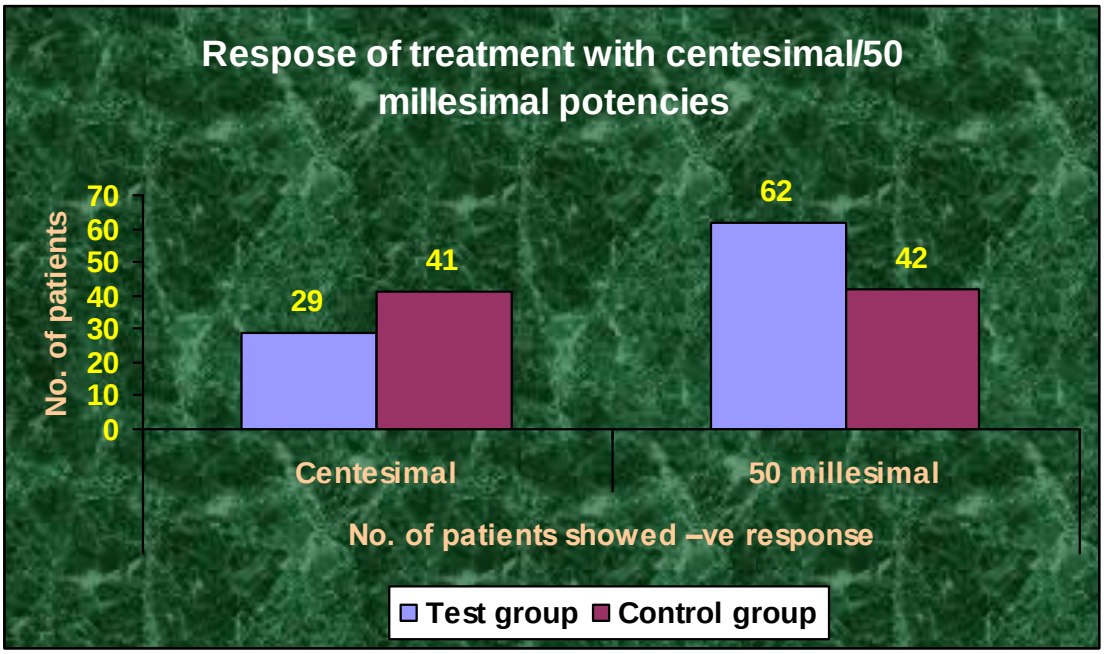

Respose of treatment with centesimal/50 millesimal potencies
No. of patients
70
60
50
40
30
20
10
0
29
41
62
42
Centesimal
50 millesimal
No. of patients showed –ve response
Test group
Control group

Table-10 (Frequencies of medicines appeared in cured cases)

Prescribed Medicines	Number of times appeared
Bryonia	757
Rhus tox	709
Ars. alb	302
Sulphur	259
Natrm. mur	209
Calc. carb	198
Lycopodium	172
Tuberculinum	153

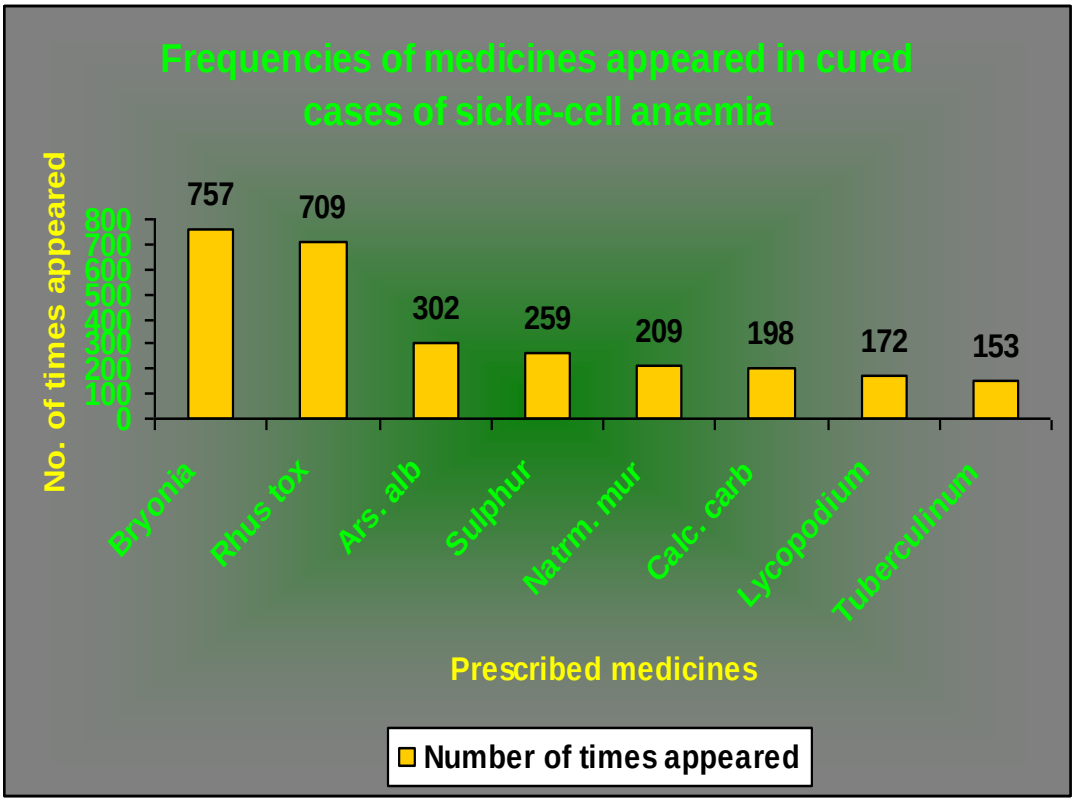


Table-11 (Analysis of various types of precipitating factors)

Category	No. of cases	%
Aggravation from exertion	357	35.987
Aggravation from cold/winter	402	40.524
Aggravation from heat/summer	201	20.262
Aggravation from wet/rainy	230	23.185
Aggravation at evening/night	102	10.282

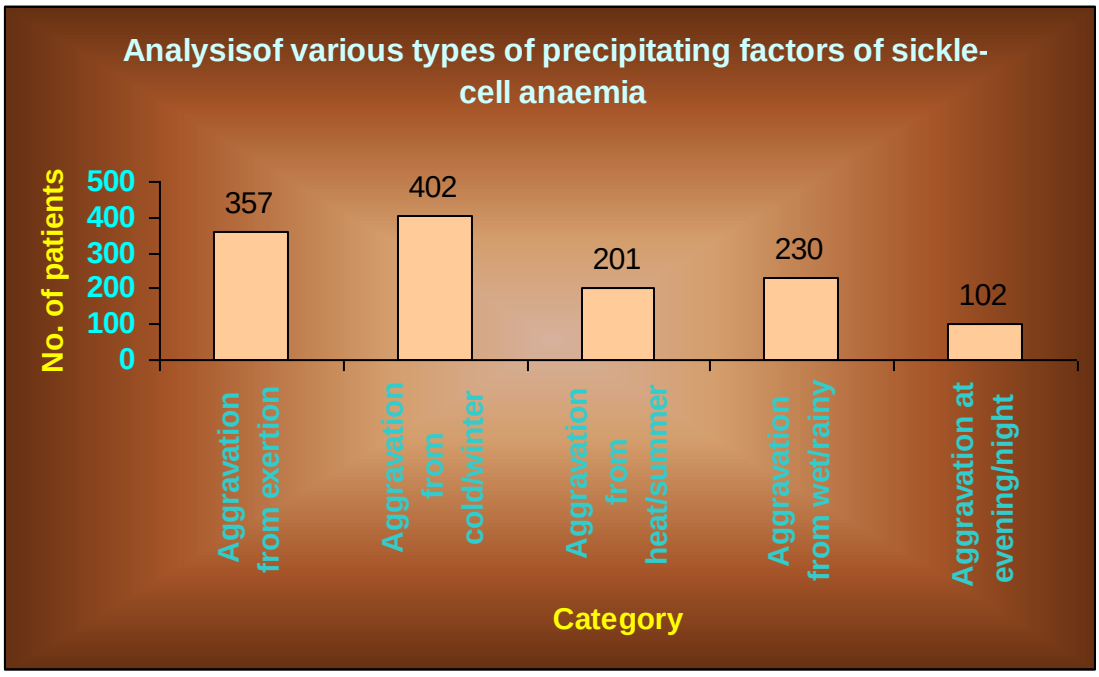


Table-12 (Incidence of previous treatment of the patients)

Category	No. of cases	%
No treatment	88	8.8709
Homoeopathic treatment	210	21.169
Allopathic treatment	542	54.637
Ayurvedic treatment	6	0.604
Mixed treatment	54	5.4435
Total	992	100.000

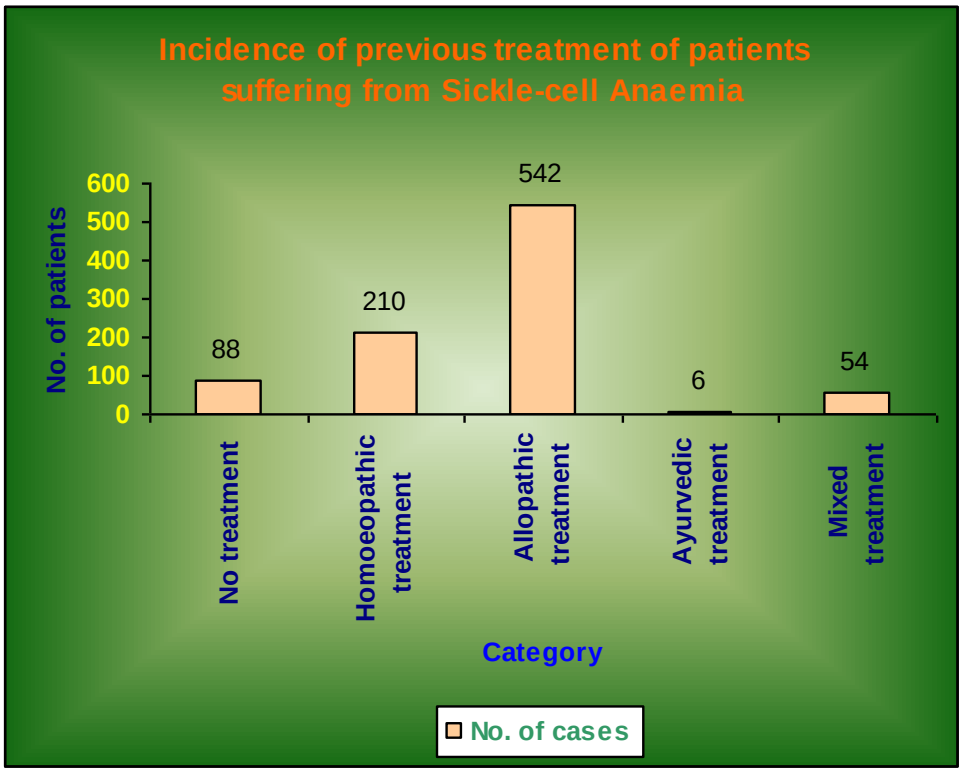


Table-13 (Observation of laboratory findings)

Category of Lab. finding	Before treatment	After treatment
Haemoglobin	900	809
Sickling test(+ve)	900	900
Hb Electrophoresis	AS-504 SS-396	AS-504 SS-396

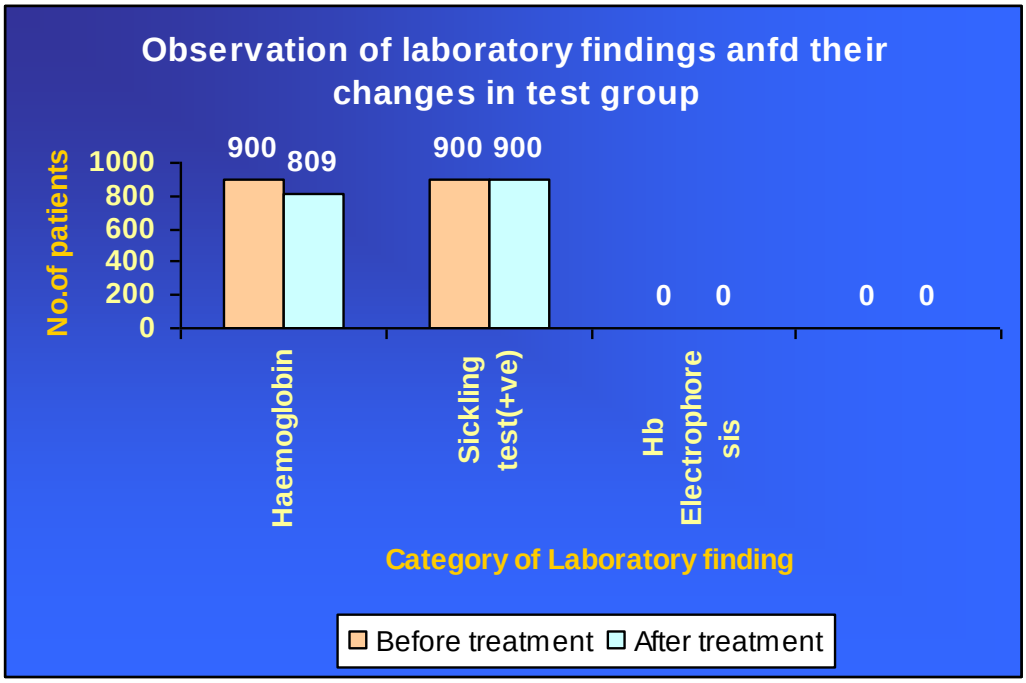
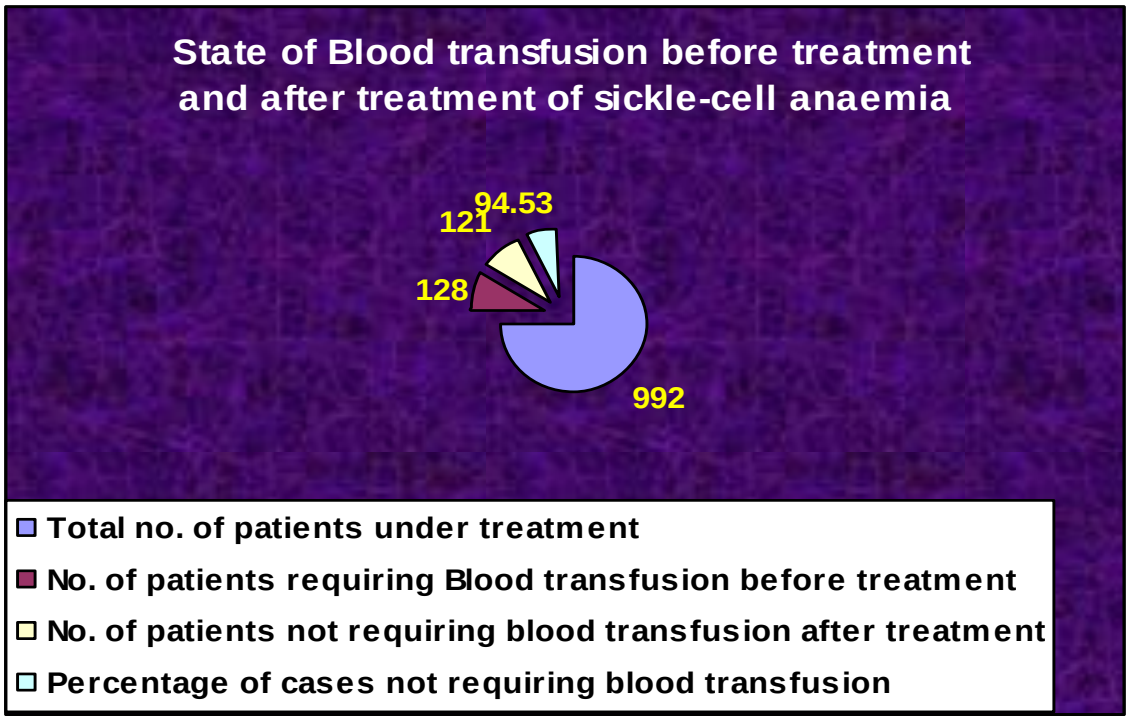


Table-14 (State of Blood transfusion before treatment and after treatment of sickle-cell anaemia)

Total no. of patients under treatment	No. of patients requiring Blood transfusion before treatment	No. of patients not requiring blood transfusion after treatment	Percentage of cases not requiring blood transfusion
992	**128**	**121**	**94.53**



Discussion

1. The study on sex aspect **Table – 1** reveals that there is male dominance of 52% compared to female 48%. This validates the observation of Baum et. al. (1987). The higher prevalence may be due to care for male child in the community than female or due to exposure to adverse environmental condition like excess labour, exposure to excess heat and cold.

2. The study on age distribution of case **Table – 2** reveals that the children between the age group of 1-10 years of age there is majority of cases i.e.49% than the incidence is more between the age group of 10-20 years of age i.e. 21%. Next to that, the incidence is more between the age group of 20-30 years of age i.e. 20% and least incidence above 31 years of age i.e. 10%.

   This type of higher incidence of sickle cell anaemia among the children of age 1-10 years of age was also observed by researcher Power 1975, Dr. Cealaer et. al. (1985) and Brainbridge et. al. (1988).

   The increased incidence among child age group is due to the inadequate resistance and increased susceptibility various infections or growing stage requires more building material for growth of the body or it is a genetic disorder and inherits from parents and in early part the manifestations usually to be exhibited.

3. **Table – 3** shows the study on socio-economic status. It is observed that it is more marked among lower socio-economic group i.e. 51 %. Next to that it is the middle socio-economic group i.e. 35 % and less common higher socio-economic group of people i.e. 14 %. It validates the observation of Seargent (1974).

   Looking to magnitude of the disease among different socio-economic group it is observed that the disease is milder in higher socio-economic group. The reason for observations are due to better living style and less exposure to heat & cold better and less physical labour and better food which contain different nutrients including B12 which is required for erythropoeisis.

4. Regarding built of the sickle cell patients, from **Table - 4** it is observed that 71 % of patients are thin and 21 % are with moderate built and 8 % are obese. It validates the observation of Seargent (1992) with slight higher side at thin built. Another important observation is being made that those who are 'SS' disease.

5. From distribution **Table – 5** regarding nativity of the patients it is understood that 70% are from rural and 30% are from urban. More patients are from rural. It is perhaps due to large number of people live in rural area than urban area. Another reason may be a fact that consanguineous marriage is practiced at rural area than urban areas.

6. From pathological investigation distribution **Table - 6** it is observed that all are having sickling test positive (+ve) and Hb-electrophoresis test +ve. It is because these two pathological tests are included in the inclusion criteria.

7. From assessment of symptomatic improvement **Table – 7** it is observed that there is conspicuous improvement is seen in anaemic, pain in extremities, pain in abdomen, jaundice, constipation, vertigo, etc. but little improvement in cases with damage to bone and gall stones.

8. Referring to the **Table – 8,** the data was put to χ^2 test, the value χ^2_{cal} is 370.21 but tabulated value of chi-square for 1 degree of freedom at 10% level of significance is 2.71. So, here χ^2_{cal} is > χ^2_{tab} . hence, the test is significant, i.e. there is significant difference. So, we have to reject the null hypothesis (H_0) and accept the alternative hypothesis (H_l). it is concluded that constitutional medicine is more effective than placebo in the treatment of Sickle Cell Anaemia.

9. Referring to the **From Table -9**, the data was put to χ^2 test, the value χ^2_{cal} is 2.816 but tabulated value of χ^2 for 1 degree of freedom at 10% level of significance is 2.71. So, here χ^2_{cal} is > χ^2_{tab} . Hence, the test is significant, i.e. there is significant difference. So, we have to reject the null hypothesis (H_0) and accept the alternative hypothesis (H_l). It is concluded that 50 millesimal scale of potency is more effective than Centesimal scale of potency in the treatment of Sickle Cell Anaemia.

10. On referring to the **Table – 10** showing frequency of medicine appeared in cured case, it is observed that Bryonia alb. and Rhus tox. have evolved on the drug of choice for sickle cell anaemia in tackling acute exacerbation crisis. Tuberculinum & Sulphur has indicated as intercurrent remedy.

11. Studying the various types of precipitating factors it is observed from the **Table-11**, that cold aggravation and aggravation from exertion have been noticed on quite large number of patients. Apart from this aggravation from heat, aggravation from wet weather and evening and night are also precipitating factor for acute exacerbation of the case.

12. Looking to the previous treatment of the patients from the distribution **Table – 12** it is understood that large number of patients are resorting to allopathic treatment next to that is homoeopathy. This reveals the popularity and predominance of the system in the locality.

13. Observing to the **Table – 13** of laboratory findings and their changes before and after it is envisaged that definitely there is increase in Hb% in 90% of cases. But sickling test and Hb electrophoresis finding remains unchanged.

14. Referring to the **Table – 14** of blood transfusion it is concluded that Homoeopathic treatment is sure to about blood transfusion in sickle cell patients.

Conclusion:

1. Male sex is more victim of sickle cell anaemia.
2. It is more prevalent between 1 to 10 yrs of age group.
3. It is noticed more among low socio-economic group of people.
4. Sickle cell patients are more prone to be of thin built and specifically 'SS' group are thin.
5. It is more marked in rural area than urban.
6. Anaemia, pain in various parts of the body, jaundice, fever etc. are well abated by Homoeopathic medicines and there by crisis is well controlled.
7. Acute remedies are Bryonia alb., Rhus tox., etc. and chronic remedies are Ars.alb., Calc.c., Lyco. etc. the intercurrent remedies are Tuberculinum, Sulphur etc.
8. Precipitating factors are cold, heat exertion, wet weather and it should be avoided by the patient.
9. Hemoglobin percentage is increased with homoeopathic remedies without any chosen drug of our counter part.
10. Blood transfusion is abated with homoeopathic medicament.
11. Fifty millesimal acts better than centesimal potency.
12. Homoeopathic medicines do act curatively in combating sickle cell anaemia which is statistically established in large no. of samples i.e. 992.

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