

SICKLE CELL ANAEMIA

Dr. Ashay H. Nandeshwar,
M.D.(Ayu.)

Dr. Leena A. Nandeshwar,
M.D.(Ayu.)

Mr. Kaustubh Chahande

Published by -

VEENA GROUP OF HOSPITAL

www.sicklecellanaemiatreatment.com

Contact : 0922 334 15 76 / 0976 688 32 08

Edition - Ist - Date : 21st January 2014.

Price : Rs. 100/-

INDEX

	Page No.
Chapter - 1	8
Introduction	
Chapter - 2	17
Methodology	
Chapter - 3	35
Immunity & Sickle Cell Anaemia	
Chapter - 4	40
Effect of Blood transfusion on sickle cell anaemia.	
Chapter - 5	41
Sickle Cell Anaemia & Multiple Drug Resistance i.e. M.D.R.	
Chapter - 6	43
Anaemia पाण्डु An Ayurvedic Concept	
Chapter - 7	48
Sickle Cell Anaemia (सिकल सेल अॅनिमियाची दहशत)	

आशिर्वाद

प्रिय डॉ. आशय नंदेश्वर

स.न.वि.वि.

आपले पत्र मिळाले, सिकल सेल अॅनेमिया या आजारीवरील रुग्णांकरीता आवश्यक असलेले सविस्तर माहितीचे पुस्तक प्रकाशित करणार असल्याचे वाचून अतिशय आनंद वाटला. आपल्या भावी आयुष्यात रुग्णोपयोगी पडतील असे साहित्य आपल्या हातून प्रकाशित होत जावो हीच माझ्याकडून शुभेच्छा...

शुभेच्छासर
आपला स्नेह
अरुण
(अणुलक्षित) (अणुलक्षित)

'सिकल सेल' अॅनेमियाची चिकित्सा

दि. २६ जानेवारी २०१४ 'वीणा ग्रुप ऑफ हॉस्पिटल'ला ५ वर्षे पूर्ण होत आहेत. कित्येक ऋग्णांना त्याचा फायदा झालेला आहे. त्या एम.बी.बी.एस. पासून इंजिनिअर पर्यंत सगळ्या प्रकारचे ऋग्ण सामील आहेत. कित्येक ऋग्णांची लग्न सुध्दा झाले आहेत आणि त्यांना मुले सुध्दा झाली आहेत. आणि ते स्वस्थ जीवन जगत आहेत. या चिकित्सेचे वैशिष्ट्य म्हणजे ऋग्णांची डॉ. नंदेश्वर यांच्या मार्गदर्शनाखालीच पुढचे आयुष्य जगावे लागते. यात मुख्यतः होणारा त्रास इन्फेक्शनच्या अवस्थेमध्ये सेल्स परत अॅबनॉर्मल (विकृत) होतात आणि त्यामुळे पेशंटला त्रास होतो. इन्फेक्शनची (जीवाणू संक्रमण) चिकित्सा अॅन्टी बायोटिक्स दिल्यावर होते. अॅन्टी बायोटीक्स दिल्यावर परत पेशंट बरा होतो व नॉर्मल आयुष्य जगायला लागतो. संक्रमण किंवा इन्फेक्शन हे बॅक्टेरीया किंवा व्हायरस किंवा अॅनयेरोबीक जीवाणूमुळे होतो. इन्फेक्शनचा संबंध हा सिकल सेलशी नाही आहे. परंतु संक्रमित अवस्थेमध्ये शरीराचा प्राणवायू हा कमी होतो व आर. बी. सी. (लाल रक्तकण) हे पर अॅबनॉर्मल होतात. इन्फेक्शनच्या काळापुरते जर त्याची चिकित्सा योग्यवेळी नाही केली गेली तर पेशंटचा हिमोग्लोबीन, कमी होतो व पेशंटला क्रायसिस येतात.

वारंवार रुग्णांना ही कॅन्सेप्ट समजावून सुध्दा इन्फेक्शनच्या अवस्थेत झालेल्या त्रासाबद्दल त्यांची तक्रार असते. इन्फेक्शन हे वातावरणातून, जेवण, खाण्यापिण्याच्या वस्तूतून, किंवा एका संक्रमित व्यक्तीपासून दुसऱ्या संक्रमित व्यक्तीला होतो.

इन्फेक्शनपासून वाचण्यासाठी माणसाने स्वतःची काळजी स्वतः घ्यायला पाहिजे. जेवण हे झाकलेलं, गरम करूनच खाल्लं पाहिजे, उघड्यावरील खाण्यापिण्याच्या वस्तू खाणे हे टाळले पाहिजे, जेवणापूर्वी हात स्वच्छ धुतले पाहिजे, पाणी गरम करून प्यायला पाहिजे, एखाद्या व्यक्तीस सर्दी, पडसे झाले असल्यास त्याच्याजवळ जाऊ नये, इन्फेक्शन झालेल्या अवस्थेमध्ये सी.बी.सी., ब्लड टेस्ट, करावे व डब्ल्यू.बी. सी. काऊंट टी.एल.सी. काऊंट १०,०००-११,०००/- पेक्षा जास्त जर वाढलेलं असेल तर ते रुग्ण संक्रमित आहेत असे गृहित धरावे व त्याची पूर्ण अँटीबायोटीक्सचा कॉर्स करावा.

वीणा ग्रुप ऑफ हॉस्पिटलमध्ये आम्ही फक्त रुग्णांचे अँबनॉर्मल झालेले सेल नॉर्मल करून देतो व नॉर्मल झालेले सेल्स प्राणवायू वाहनाचे त्यांचे कार्य ते स्वतः करतात व रुग्ण आपोआप बरा होतो. इन्फेक्शन टाळणं हे रुग्णांच्या हातात आहे.त्यांनी जर योग्य दक्षता घेतली तर त्यांना इन्फेक्शन होणार नाही व ते स्वस्थ आयुष्य जगू शकतील.

विषाणू किंवा व्हायरसला योग्य चिकित्सा नसते. त्यामुळे व्हायरसने संक्रमित पेशंट १-१५ दिवस आजारी राहू शकतो. व्हायरस अँटीव्हायरस ट्रिटमेंट नसल्यामुळे त्याला तो त्रास सोसावा लागतो. तरी योग्यवेळी योग्य ती दक्षता घेतल्यामुळे आपण रुग्णांचे प्राण वाचवू शकतो. स्वस्थ जीवन जगू शकतो.

- डॉ. आशय नंदेश्वर

- डॉ. लिना नंदेश्वर

www.sicklecellanaemiatreatment.com

Inhibiting Function of Gene *Treatment of sickle cell anaemia*

Aim: To inhibit the function of 11th gene in patient suffering from sickle cell disease.

Object: To reduce the crisis of patient suffering from sickle cell by using Ayurvedic medicines.

Summary: Very less literature is available about sickle cell anaemia, so far as anecdotal part of it is concerned.

What is sickle cell disease (S.C.D)?

Sickle cell disease may be a Heterozygous or a Homozygous condition.

But more often it is Homozygous condition. According to my studies S.C.D is Homozygous: Heterozygous condition.

A patient of S.C.D. shows SS type of Haemoglobin.

But the condition of Morphology of R.B.C varies with the ratio of SS: A2: F:A

So if the Haemoglobin is totally of SS type then the R.B.C shows sickle -shaped pattern but if the percentage of A2 i.e. Adult Haemoglobin and F i.e. Faetal Haemoglobin is more than the R.B.C shows Poikilocytosis and Anisocytosis conditions. R.B.C shows different shapes like hexagonal, polygonal, teardrop cells, pencil cells like structures. We already know that valline is replaced by Glutamic Acid in S.C.D.

In SS: A2 type the friability of R.B.C outer layer is more so patients of these type require more blood transfusion.

But in SS: F type of Haemoglobin, the irregular shaped R.B.C agglutinate and forms clamp which causes ischemia and leads to crisis.

Signs & Symptoms of S.C.D patients :

- 1) In childhood they get rheumatoid arthritis like symptoms.
- 2) Sudden fall in Haemoglobin level.
- 3) Unbearable pain.
- 4) Involvement of multiple organ.
 - a) Hepatic failure,
 - b) Splenic failure.
 - c) Renal failure.
- 5) Due to frequent haemolysis is high bilirubin level because of which icterus is common in S.C.D patients
- 6) Bone crush is the chronic condition of S.C.D
- 7) Necrosis of the joints specially hip joint and shoulder joints.

What is Sickle cell trait?

Sickle cell trait is a condition where the patient is Phenotypical normal and Genotypically abnormal.

The allele shows S: A type of Haemoglobin.

So though S.C.D is autosomal recessive disorder but I have seen in few cases the parents were normal but the children were showing S type of Haemoglobin.

Treatment :

In Allopathy there is no specific treatment, but when I started treatment with Avurvedie medicines, we found the Morphological and Phenotypical changes in R.B.C and patient. The blood transfusion and the crisis of the patients stopped.

- Dr. Ashay. H. Nandeshwar

M.D. (Ayu)

Lecturer, Vasantdada Patil
Ayurvedic Medical Collage, Sangli.

Chapter - 1

Introduction

1.1 Research Question:

Can Ayurvedic medicines cure Sickle-Cell Disease? If it does, then to what extent and at what rate they cure the disease?

1.2 Intoduction :

When in my Biology class I was taught about Sickle-Cell Disease, as a junior high school student, I was much struck with the fact that Sickle-Cell Disease and most of the genetic diseases are incurable. The knowledge I gained about base substitution mutation in DNA and the Human Genome Project, showed me light to the possibility of a cure to the disease. Stem Cell therapy was the first thing that came into my mind, while thinking of a cure to SCD. Eventually I found that researches institutions across the globe. As a result, bone marrow transplant became a ray of hope for the sufferers of the disorder, but still, the scientists and doctors would assert the non-existence of a definite cure to the disease.

1.3 What is Sickle Cell Disease?

Sickle-Cell Disease (SCD), or sickle-cell anaemia (SCA) or drepanocytosis is one of the most life-threatening diseases ever discovered. The disease not only disturbs the life of the patients but also his family. As there has been no definite treatment or cure for the disease till date, the patients are very frequently admitted in hospitals and have to undergo unbearable pain. Some forms of Sickle-Cell Disease tell us that it is even more fatal than Cancer and AIDS. In spite of being a very deadly and widespread disorder in many parts of the country, Government of India has not taken any serious steps regarding eliminating or researching on the treatment and cure of SCD. This collection of clinical findings was unknown until the explanation of sickle cells was given by Chicago cardiologist and professor of medicine James B. Herrick. The disease was named “sickle-cell anaemia” by Vernon Mason in 1922.

Sickle cell disease has struck millions of people across the globe and is predominantly widespread among those whose ancestors are from sub-Saharan Africa, Spanish-speaking regions in the Western Hemisphere (South America, the Caribbean, and Central America), Saudi Arabia, India, and Mediterranean countries such as Turkey, Greece, and Italy. It affects an estimated 70,000 to 100,000 Americans. The disease occurs in about 1 out of every 500 African Americans births, and about 1 out of every 36,000 Hispanic Americans births. The trait of SCD occurs in about 1 in 12 African Americans. Sickle cell disease is a major public health concern. From 1989 through 1993, there was an average of 75,000 hospitalizations due to sickle cell disease in the United States, costing approximately \$475 million. During 2005, medical expenditures, in the US, for children with sickle cell disease averaged \$11,702 for children with Medicaid coverage and \$14,772 for children with employer-sponsored insurance. Sickle cell-related death among African-American children less than 4 years of age fell by 42% during 1999 to 2002. This coincides with the introduction of a vaccine that protected against invasive pneumococcal disease in 2000. The disease can be found in people living in parts of the

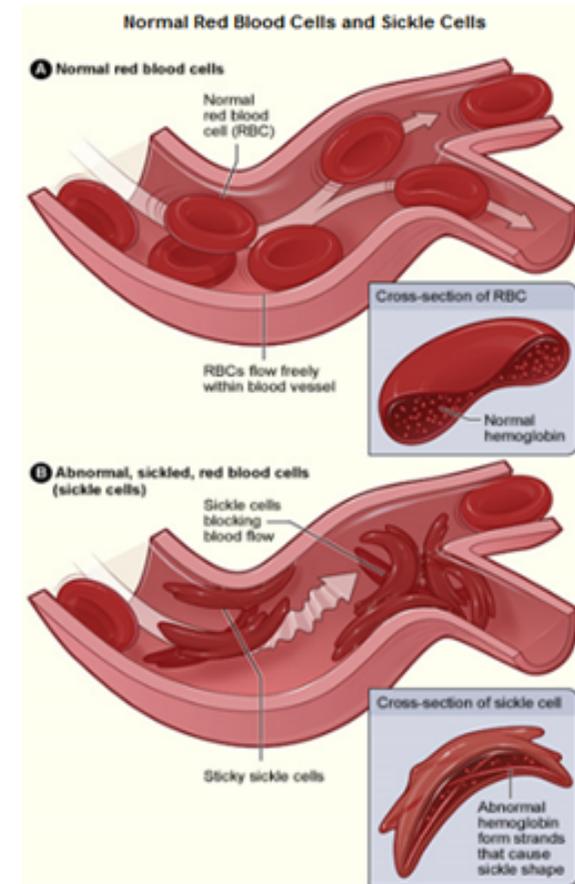
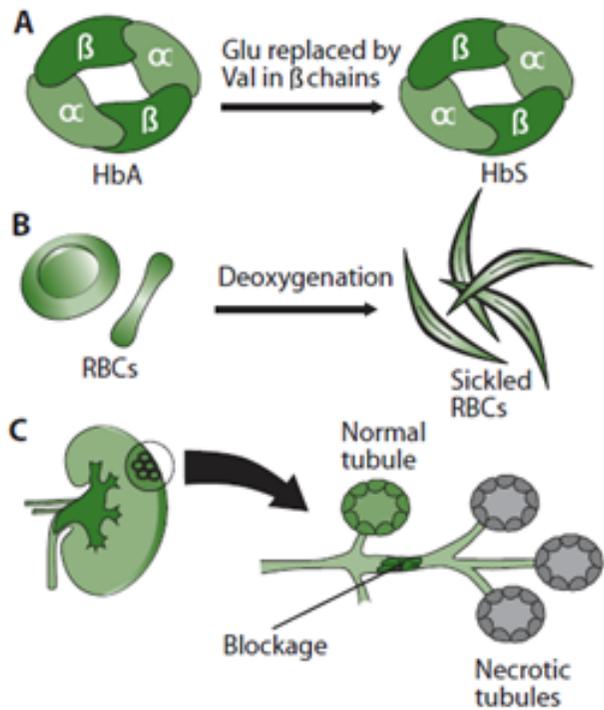
world where malaria is or was common. It is believed that people who carry the sickle cell trait are less likely to catch malaria.

Sickle cell anaemia is an inherited form of anaemia — a condition in which there aren't enough fit red blood cells to carry sufficient oxygen throughout the body. In normal condition, the red blood cells have biconcave shape and are flexible. They can move easily through the blood vessels. Whereas, in sickle cell anaemia and other forms of SCD, the red blood cells become rigid, sticky and are shaped like sickles or crescent moons. These irregularly shaped cells can get stuck in small blood vessels, which can slow or block blood flow and oxygen to parts of the body. There's no cure for most people with sickle cell anaemia. However, treatments can relieve pain and help prevent further problems associated with sickle cell anaemia.

Theoretical Basis:

Sickle cell anaemia is a genetic disorder which is inherited from parents to the offspring. It has been known to be caused by mutations in the DNA of the RBCs. Mutation is any event that changes genetic structure; any alteration in the inherited nucleic acid sequence of the genotype of an organism.

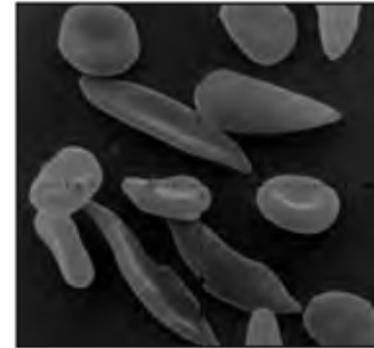
A mutation can just involve one nucleotide or it can even have an effect on a huge segment of the gene. It is often thought that a mutation which involves a smaller number of nitrogenous bases in the DNA will be less significant than one where a larger number of bases are changed. In the gene that controls sickle-cell anaemia a alteration of just one base lead to a protein with one amino acid distorted and the resulting disease. Haemoglobin is made of four polypeptide chains: two alpha chains and two beta chains. When an A to T base substitution occurs in the region of the gene coding for the 6th amino acid in the beta chain, the codon GAG (glutamic acid) becomes GTG (valine).



The resulting polypeptide is different and the haemoglobin formed is commonly known as HbS; the normal haemoglobin is known as HbA. The result of this is a slightly different structure, by one amino acid of the haemoglobin molecule which makes it crystallise at low oxygen levels (e.g. in the capillaries). The erythrocyte in which the haemoglobin can be found will then change from a biconcave shape into a sickle-cell shape (see Figure 404) and can block the small capillaries, and is less efficient at transporting oxygen. Even when the oxygen concentration increases again, the cells keep their sickle shape.

The symptoms of sickle-cell anaemia are acute anaemia, which causes physical weakness. The lack of oxygen may be severe enough to cause damage to the heart and kidneys or even death (in homozygous individuals). The gene for sickle-cell anaemia is co-dominant with the “normal” allele although the latter is expressed more strongly in the heterozygous individual. Heterozygous individuals (carriers) have some HbS but more normal haemoglobin. They may suffer from mild anaemia.

The selective advantage of being a carrier is found in malaria infested areas. Plasmodium (the protist causing malaria) cannot reproduce in erythrocytes with HbS. This means that individuals heterozygous for the sickle-cell trait have a reduced chance of contracting malaria. Natural selection has ensured that the sickle-cell trait is more common among people living in malaria-infested areas such as West Africa. As the African-American population largely originates from this area, the trait is found in frequencies higher than usual in this group. Carriers may not be aware of the fact that they possess the sickle-cell allele and are capable of passing it on to their children. If two carriers have a child, there is a 1 in 4 (25%) chance of the child having the disease.



Therefore, it is important that people who may be carriers, are tested to confirm the presence or absence of the sickle cell allele. If a female carrier is pregnant and the father is also a carrier, it is possible to test using amniocentesis or chorionic villi sampling (refer to Topic 4.2.6) to see if the child will have sickle-cell anemia. Should this be the case, then the parents may decide to discontinue the pregnancy. There are no easy answers to this problem. A great deal of research is being done about treating sickle-cell anemia including bone marrow transplant and/or blood stem cell treatment. Gene therapy is also a possibility but research is in an early stage.

Symptoms:

Signs and symptoms of sickle cell anaemia usually show up after an infant is 4 months old and may include:

1. Anaemia :

Sickle cells are fragile. They break apart easily and die, leaving you chronically short on red blood cells. Red blood cells usually live for about 120 days before they die and need to be replaced. However, sickle cells die after only 10 to 20 days. The result is a chronic shortage of red blood cells, known as anaemia. Without enough red blood cells in circulation, your body can't get the oxygen it needs to feel energized. That's why anaemia causes fatigue.

2. Episodes of pain :

Periodic episodes of pain, called crises, are a major symptom of sickle cell anaemia. Pain develops when sickle-shaped red blood cells block blood flow through tiny blood vessels to your chest, abdomen and joints. Pain can also occur in your bones. The pain may vary in intensity and can last for a few hours to a few weeks. Some people experience only a few episodes of pain. Others experience a dozen or more crises a year. If a crisis is severe enough, you may need hospitalization so that pain medication can be injected into your veins (intravenously).

3. Hand-foot syndrome:

Swollen hands and feet may be the first signs of sickle cell anaemia in babies. The swelling is caused by sickle-shaped red blood cells blocking blood flow out of their hands and feet.

4. Jaundice :

Jaundice is a yellowing of the skin and eyes that occurs because of liver damage or dysfunction. Occasionally, people who have sickle cell anaemia have some degree of jaundice because the liver, which filters harmful substances from the blood, is overwhelmed by the rapid breakdown of red blood cells. In people with dark skin, jaundice is visible mostly as yellowing of the whites of the eyes.

5. Frequent infections:

Sickle cells can damage your spleen, an organ that fights infection. This may make you more vulnerable to infections. Doctors commonly give infants and children with sickle cell anaemia antibiotics to prevent potentially life-threatening infections, such as pneumonia.

6. Delayed growth:

Red blood cells provide your body with the oxygen and nutrients you need for growth. A shortage of healthy red blood cells can slow growth in infants and children and delay puberty in teenagers.

7. Vision problems:

Some people with sickle cell anaemia experience vision problems. Tiny blood vessels that supply your eyes may become plugged with sickle cells. This can damage the retina — the portion of the eye that processes visual images.

Ayurveda :

Ayurveda is the oldest surviving complete medical system in the world. Derived from its ancient Sanskrit roots - 'ayus' (life) and 'ved' (knowledge) – and offering a rich, comprehensive outlook to a healthy life, its origins go back nearly 5000 years. It was expounded and practiced by the same spiritual rishis, who laid the foundations of the Vedic civilisation in India, by organising the fundamentals of life into proper systems. Ayurveda offers a unique blend of science and philosophy that balances the physical, mental, emotional and spiritual components necessary for holistic health.

General Observations about the treatment:

It was observed that after about 1 year of this treatment the patients stopped having blood transfusion as their haemoglobin level was rising. The crisis of the patients has also reduced during the first year of the treatment drastically. The patients reported to be fully cured from the basic problems caused by SCD such as joint pain, fever and weakness.

Methodology

2.1 Objectives of study:

1. To verify if the Ayurvedic medicines composed of plant extracts cure Sickle-Cell Disease.
2. To investigate the extent to which the Ayurvedic medicines cure SCD.
3. To find out the rate at which they cure SCD.

2.2 HYPOTHESIS

HYPOTHESIS 1 : The Ayurvedic medicines to a large extent help to cure SCD.

HYPOTHESIS 2 : All the symptoms which are directly related to abnormality in shape of RBCs will be cured.

HYPOTHESIS 3 : The medicines will show evident effects on the shape of RBCs in a year.

Survey:

20 patients who have been undergoing treatment since 1 year at the clinic were surveyed about the prognosis of their health problems. The following were the results:

Symptoms	Cured or Not
Chronic shortage of RBCs (Anaemia)	
Episodes of pain/crisis	
Hand-foot syndrome	
Jaundice	

Complication	Cured or Not
Strokes	
Acute chest syndrome	
Pulmonary hypertension	
Organ damage	
Blindness	
Skin ulcers	
Gallstones	

Questionnaire:

- Q. 1 How long have you been undergoing ayurvedic treatment of Dr. Nandeshwar?
- a. Less than a year
 - b. 1 year
 - c. More than a year

Q.2 Which of the following symptoms did you have before undergoing the treatment?

- a. Anaemia
- b. Pain/crisis
- c. Hand-foot syndrome
- d. Jaundice

Q3. How effectively have your symptoms (specified) subsided?

- a. Completely
- b. Somewhat
- c. Almost completely
- d. Not at all

Final Experiment:

To verify why the symptoms subsided in SCD cases, an experiment was conducted. In the experiment I planned to use the blood samples of 10 different patients, at a regular interval of 2 months.

2.3 Variables :

Dependent Variables (DV)		Total number of blood cells in the sample. Number of defected cells in the sample. Number of normal cells in the sample.
Controlled Variables	Independent Variables (IV)	Amount of blood used to prepare each slide.
	Fixed Variables (Constants)	Number of blood cells in the sample over a period of time
Uncontrolled Variables		Room temperature Humidity Slight Impurities Drops of solutions clinging on to the walls of test tubes, syringe, beaker, and thermometer.

Materials:

- Slides
- Droppers
- Leishman stain
- Distilled water
- Syringe with needles
- Cotton
- Spirit

Procedure 1:

Preparing the blood smears

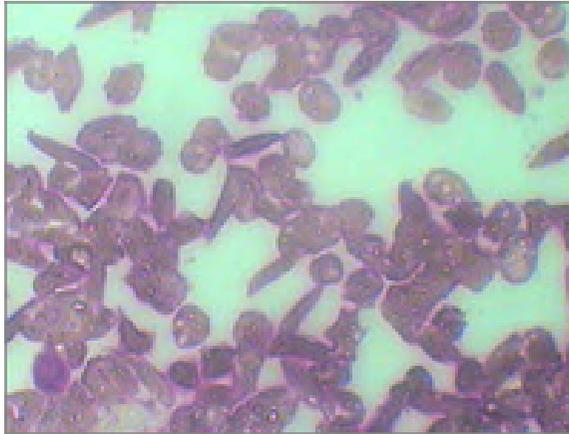
1. Distilled water was used to clean all the slides. The slides were then kept open to dry
2. 5 ml syringes were used to take out blood from the patients. In adult patients, the blood was taken out from a vein in the arm; and in young children the blood was taken from finger tips.
3. About 0.5 ml of blood was taken out from each patient.
4. Each blood sample was then added into a test-tube (cleaned and dried).
5. Separate droppers were used to transfer 1 drop of blood from each sample from the test-tubes to the slides.
6. The slides were then covered with Leishman stain, and were then allowed to dry for about 30 minutes each.

Procedure 2:

Viewing the smears under a microscope and counting the cells

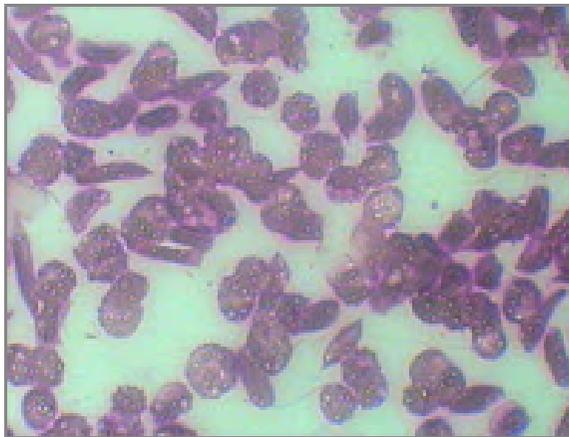
1. The prepared slides were then viewed under an optical microscope.
2. A digital camera was used to capture the images from the eye-piece.
3. The digital images were then zoomed on a computer and then counted.

At the Start of Treatment :



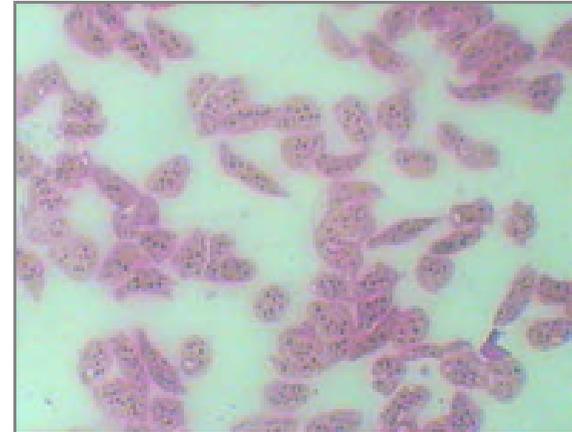
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
129	74	55	57.36434168527132%

2 Months of Treatment :



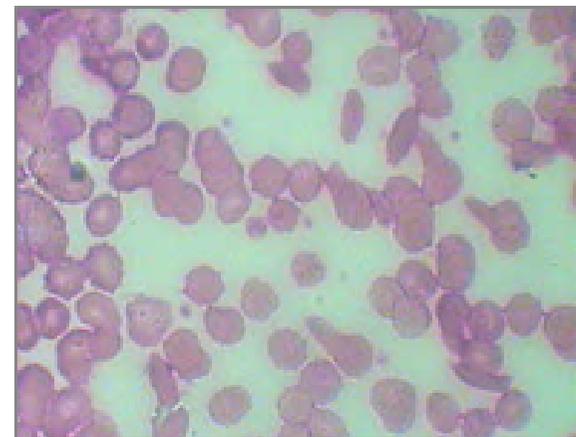
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
131	61	70	46.5648855%

4 Months of Treatment :



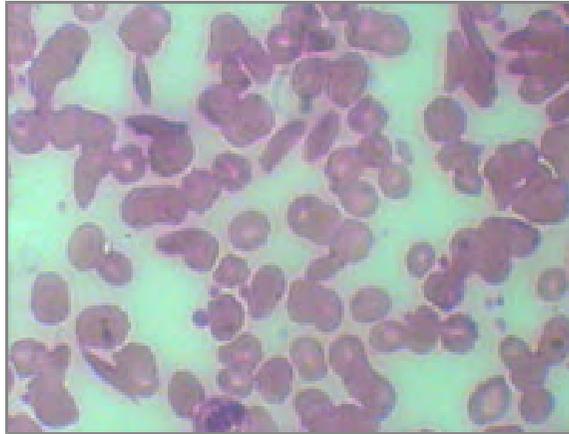
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
93	43	50	46.23655914%

6 Months of Treatment :



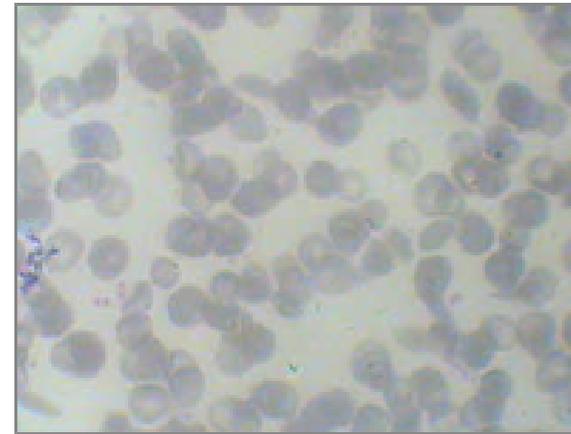
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
109	24	85	22.01834862385321%

8 Months of Treatment :



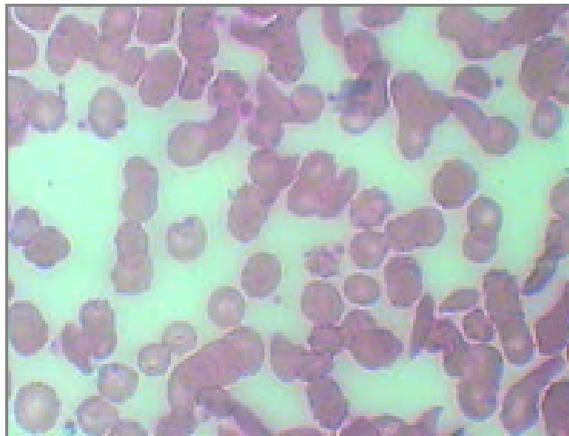
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
108	21	87	19.44444444%

12 Months of Treatment :



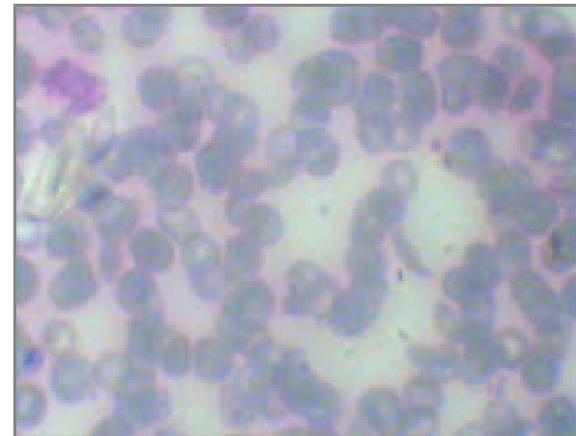
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
110	11	99	10%

10 Months of Treatment :



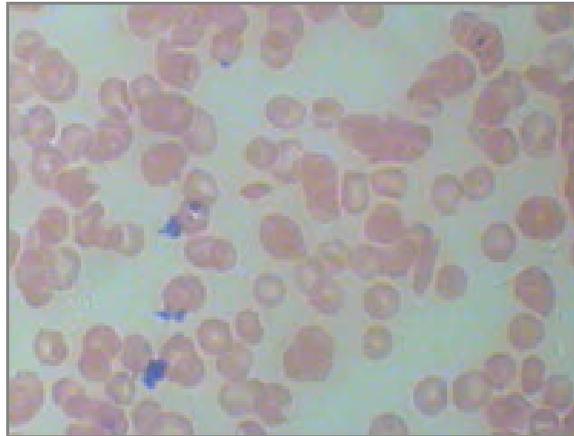
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
128	15	113	11.71875%

14 Months of Treatment :



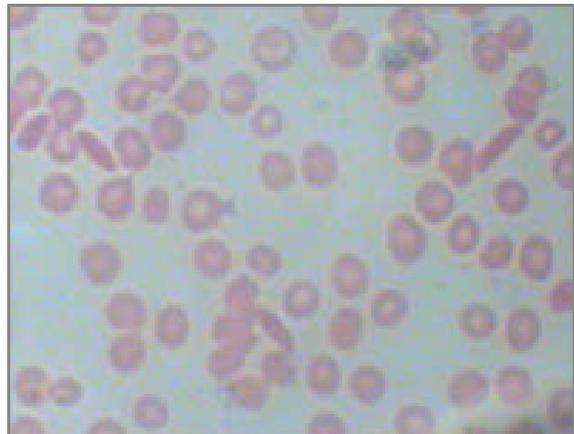
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
105	6	99	5.714285714%

16 Months of Treatment :



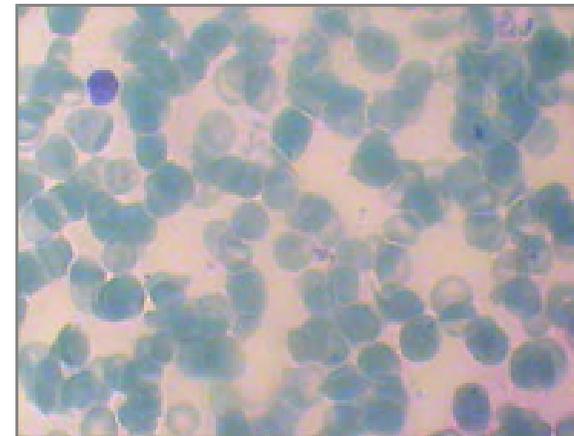
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
101	5	96	4.95049505%

18 Months of Treatment :



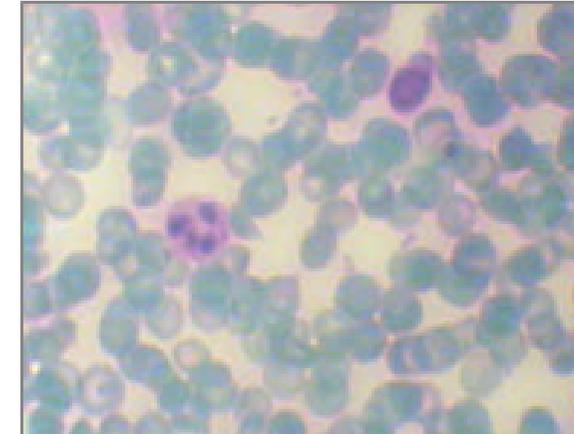
Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
87	4	83	4.597701149%

20 Months of Treatment :



Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
133	2	131	1.503759398%

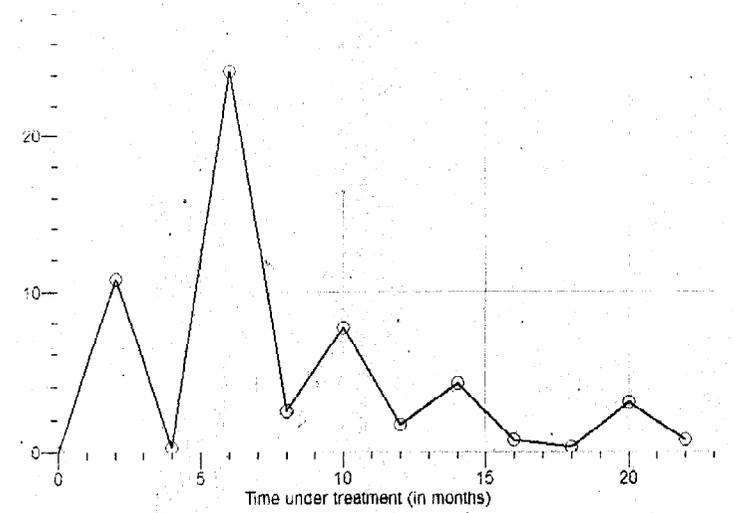
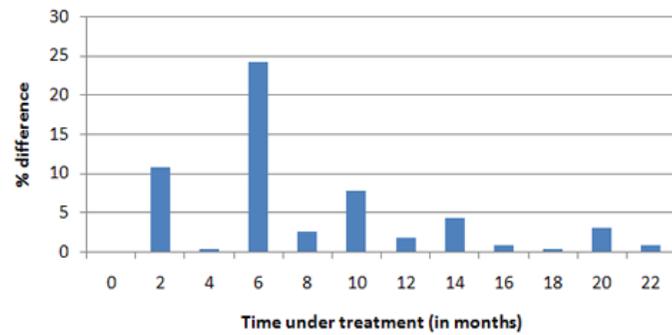
22 Months of Treatment :



Total number of RBCs observed	Number of defected cells observed	Number of normal Cells	% of defected cells
134	1	133	0.7462686567%

Statistical Analysis :

Measuring the effect of the medicine



Limitations and Scope for Improvement:

Improvements in the procedure:

1. Before using them the slides should be washed with an acid, then rinsed in distilled water and then air-dried. This is done to make the slides free of alkaline substances if the cells are going to adhere.
2. The slide should be made as soon as possible after the cells have been taken from the patient and collected in ethylenediaminetetraacetic acid (EDTA). If you wait too long, the cells will lose their shape and the true shape is extremely important for diagnosing. This was a very big limitation in the experiment.
3. The blood cells were not properly spread out in the above slides. This was a very significant limitation of the experiment. Due to the over-lapping of the blood cells definitely there must have been an error in counting. To overcome this limitation a thin capillary tube should have been used to take out the blood sample on to the slide.
4. The Science Advisory Board has laid down specific protocols for preparing blood smears. According to the protocol the procedure should include the use of Smear Fix© on the wet preparation of the slide gently into a coplin jar of acetic alcohol (3% acetic acid in 95% methanol). These two solutions are fixative which helps to keep the blood cells stable.

Complications

Sickle cell anaemia can lead to a host of complications, including:

- * Stroke. A stroke can occur if sickle cells block blood flow to an area of your brain. Stroke is one of the most serious complications of sickle cell anemia. Signs of stroke include seizures, weakness or numbness of your arms and legs, sudden speech difficulties, and loss of consciousness. If your baby or child has any of these signs and symptoms, seek medical treatment immediately. A stroke can be fatal.
- * Acute chest syndrome. This life-threatening complication of sickle cell anaemia causes chest pain, fever and difficulty breathing. Acute chest syndrome can be caused by a lung infection or by sickle cells blocking blood vessels in your lungs. It requires emergency medical treatment with antibiotics, blood transfusions and drugs that open up airways in your lungs. Recurrent attacks can damage your lungs.
- * Pulmonary hypertension. About one-third of people with sickle cell anemia will eventually develop high blood pressure in their lungs (pulmonary hypertension). Shortness of breath and difficulty breathing are common symptoms of this condition, which can ultimately lead to heart failure.
- * Organ damage. Sickle cells can block blood flow through blood vessels, immediately depriving an organ of blood and oxygen. In sickle cell anemia, blood is also chronically low on oxygen. Chronic deprivation of oxygen-rich blood can damage nerves and organs in your body, including your kidneys, liver and spleen. Organ damage can be fatal.
- * Blindness. Tiny blood vessels that supply your eyes can get blocked by sickle cells. Over time, this can damage the retina — the portion of the eye that processes visual images — and lead to blindness.
- * Skin ulcers. Sickle cell anaemia can cause open sores, called ulcers, on your legs.

- * Gallstones. The breakdown of red blood cells produces a substance called bilirubin. Bilirubin is responsible for yellowing of the skin and eyes (jaundice) in people with sickle cell anemia. A high level of bilirubin in your body can also lead to gallstones.
- * Priapism. Men with sickle cell anaemia may experience painful erections, a condition called priapism. As occurs in other parts of the body, sickle cells can block the blood vessels in the penis. This can damage the penis and eventually lead to impotence.

Tests and diagnosis

A blood test can be done for hemoglobin S — the defective form of hemoglobin that underlies sickle cell anemia. In the United States, this blood test is part of routine newborn screening done at the hospital. But older children and adults can be tested too.

In adults, a blood sample is drawn from a vein in the arm. In young children and babies, the blood sample is usually collected from a finger or heel. The sample is then sent to a laboratory, where it's screened for hemoglobin S.

If the screening test is negative, there is no sickle cell gene present. If the screening test is positive, further tests will be done to determine whether one or two sickle cell genes are present. People who have one gene — sickle cell trait — have a fairly small percentage of hemoglobin S. People with two genes — sickle cell disease — have a much larger percentage of the defective hemoglobin.

Treatments and drugs

Bone marrow transplant offers the only potential cure for sickle cell anemia. But, finding a donor is difficult and the procedure has serious risks associated with it, including death.

As a result, treatment for sickle cell anemia is usually aimed at avoiding crises, relieving symptoms and preventing complications. If you have sickle cell anemia, you'll need to make regular visits to

your doctor to check your red blood cell count and monitor your health. Treatments may include medications to reduce pain and prevent complications, blood transfusions and supplemental oxygen, as well as bone marrow transplant.

Medications

Medications used to treat sickle cell anemia include:

- * Antibiotics. Children with sickle cell anemia usually begin taking the antibiotic penicillin when they're about 2 months of age and continue taking it until they're 5 years old. Doing so helps prevent infections, such as pneumonia, which can be life-threatening to an infant or child with sickle cell anemia. Antibiotics may also help adults with sickle cell anemia fight certain infections.
- * Pain-relieving medications. To relieve pain during a sickle crisis, your doctor may advise over-the-counter pain relievers and application of heat to the affected area. You may also need stronger prescription pain medication.
- * Hydroxyurea (Droxia, Hydrea). This prescription drug, normally used to treat cancer, may be helpful for adults with severe disease. When taken daily, it reduces the frequency of painful crises and may reduce the need for blood transfusions. It seems to work by stimulating production of fetal hemoglobin — a type of hemoglobin found in newborns that helps prevent the formation of sickle cells. There is some concern about the possibility that long-term use of this drug may cause tumors or leukemia in certain people. Your doctor can help you determine if this drug may be beneficial for you.

Assessing Stroke Risk

Using a special ultrasound machine (transcranial), doctors can learn which children have a higher risk of stroke. This test can be used on children as young as 2, and those who are found to have a high risk of stroke are then treated with regular blood transfusions.

Blood Transfusions

In a red blood cell transfusion, red blood cells are removed from a supply of donated blood. These donated cells are then given intravenously to a person with sickle cell anemia.

Blood transfusions increase the number of normal red blood cells in circulation, helping to relieve anemia. In children with sickle cell anemia at high risk of stroke, regular blood transfusions can decrease their risk of stroke.

Blood transfusions carry some risk. Blood contains iron. Regular blood transfusions cause an excess amount of iron to build up in your body. Because excess iron can damage your heart, liver and other organs, people who undergo regular transfusions must often receive treatment to reduce iron levels. Deferasirox (Exjade) is an oral medication that can reduce excess iron levels. It can be used in people older than 2.

Supplemental oxygen

Breathing supplemental oxygen through a breathing mask adds oxygen to your blood and helps you breathe easier. It may be helpful if you have acute chest syndrome or a sickle cell crisis.

Bone marrow transplant

This procedure replaces bone marrow affected by sickle cell anemia with healthy bone marrow from a donor who doesn't have the disease. It can be a cure, but the procedure is risky, and it's difficult to find suitable donors. Researchers are still studying bone marrow transplants for people with sickle cell anemia. Currently, the procedure is recommended only for people who have significant symptoms and problems from sickle cell anemia.

Bone marrow transplant requires a lengthy hospital stay. After the transplant, you'll need drugs to help prevent rejection of the donated marrow.

Treating complications

Doctors treat most complications of sickle cell anemia as they occur. Treatment may include antibiotics, vitamins, blood transfusions, pain-relieving medicines, other medications and possibly surgery, such as to correct vision problems or to remove a damaged spleen.

Experimental treatments

Scientists continue to gain new insights into the symptoms and causes of sickle cell anemia. Some possible new treatments being studied include:

- * Gene therapy. Because sickle cell anemia is caused by a defective gene, researchers are exploring whether inserting a normal gene into the bone marrow of people with sickle cell anemia will result in the production of normal hemoglobin. Scientists are also exploring the possibility of turning off the defective gene while reactivating another gene responsible for the production of fetal hemoglobin — a type of hemoglobin found in newborns that prevents sickle cells from forming.
- * Butyric acid. Normally used as a food additive, butyric acid may increase the amount of fetal hemoglobin in the blood.
- * Clotrimazole. This over-the-counter antifungal medication helps to prevent a loss of water from red blood cells, which may reduce the number of sickle cells that form.
- * Nitric oxide. Sickle cell anemia causes low levels of nitric oxide, a gas that helps to keep blood vessels open and reduces the stickiness of red blood cells. Treatment with nitric oxide may prevent sickle cells from clumping together.
- * Nicosan. This is an herbal treatment in early trials in the U.S. Nicosan has been used to prevent sickle crises in Nigeria.

Immunity & Sickle Cell Anaemia

Definition :-

Resistance of the body against the pathogenic agents is known as 'Immunity'.

It is the ability of the body to resist different types of foreign bodies like bacteria, virus, and toxic substances etc. which enter the body.

Types :- 2 types - a) innate immunity

b) acquired immunity

a) Innate Immunity :-

It is also called a 'Natural immunity.' It is present from birth and it is the inborn capacity of the body to resist the entry of microorganisms into the body.

It includes -

- i) Phagocytosis of bacteria and other invaders by WBCs.
- ii) Cells of tissue macrophages system,
- iii) Resistance of skin

- iv) Presence of certain chemical compounds that lysozyme, basic polypeptide, natural killer lymphocyte.

b) Acquired Immunity :-

It is called as Adoptive Immunity also. Acquired Immunity is the resistance developed in the body against any specific foreign body like bacteria, viruses, toxins, vaccines or transplanted tissues. So it is also called as 'Specific immunity'.

Types of Acquired Immunity-

a) Active immunity &

b) Passive immunity

Acquired immunity developed by the entrance of any foreign, foreign body or a vaccine is called 'Active immunity'.

Acquired immunity developed by transferring antibodies, Serum or lymphocyte from an immune individual is called 'Passive Immunity'.

Types of Acquired Immunity -

a) Cellular immunity &

b) Humoral immunity

a) Cellular immunity -

Cellular immunity is by activation of T-lymphocytes which destroy their organisms, entering the body. This is also called as 'Cell Mediated immunity or T-cell immunity'.

It is a major defense mechanism against infections.

b) Humoral immunity -

Humoral immunity is by the activation of B-lymphocytes. This is also called 'B-cell immunity'.

Lymphocytes Are Responsible for Acquired Immunity -

A) Development of lymphocytes - In fetus, lymphocytes develop from bone marrow.

The pluripotent hemopoietic stem cells give rise to colony forming units (CFU) and lymphoid stem cells (LSC).

LSC give rise to

Lymphoblasts

lymphocytes

B) Location of lymphocytes - most extensively located in lymph nodes, but they are also found in special lymphoid tissues such as spleen, submucosal areas of gastrointestinal tract, thymus and bone marrow.

Humoral immunity

T lymphocyte - thymic & B-lymphocyte - Bursal.

C) Formation of antibodies and sensitized lymphocytes by a lymph nodes in response to antigens.

D) Thymus gland Pre processes the T-lymphocytes -

- 1) After origination of T-lymphocytes in the bone marrow 1st migrate to the thymus gland.
- 2) They divide rapidly and at the same time develop extreme diversity for reacting against different specific antigens.
- 3) One T-lymphocyte develops specific reactivity against one antigen then the next T-lymphocyte develops specificity against another antigen.
- 4) This continues until there are thousands of different types of T-lymphocytes with specific reactivities against many thousands of different antigens.
- 5) Different types of T-lymphocytes now leave the thymus and spread by blood throughout the body to lodge in lymphoid tissue everywhere.

E) Liver and Bone Marrow Preprocess the B-lymphocyte -

- 1) In human being, B-lymphocytes are known to be preprocessed in the liver during midfetal life and in the bone marrow during late fetal life and after birth.
- 2) B-lymphocytes are diff from T-lymphocytes in 2 ways - one is -instead of the whole cell developing reactivity against the

antigen, as occurs for the T-lymphocytes, the B-lymphocytes actively secrete antibodies that are reactive agents. Second is - B-lymphocytes have been greater diversity than the T-lymphocytes, thus forming many millions of types of B-lymphocytes antibodies with different specific reactivities.

Spleen -

- lymphoid organ
- function of spleen

a) Role in defense of body-

The macrophages in splenic pulp phagocytose the microorganisms and other foreign bodies. Spleen contains about 25% of T-lymphocyte and 15% of B-lymphocytes and forms the site of antibody production.

Function of Lymph -

It plays an important role in immunity by transporting lymphocytes.

Liver Functions -

- a) The reticuloendothelial cells (kuffer's cells) of liver play an important role in the defense of body.
- b) Liver is also involved in the detoxification of the foreign bodies. (Detoxification means Reduction of toxic properties of a poisonous substance).
- c) Foreign bodies like bacteria, antigens are swallowed and digested by reticuloendothelial cells of liver by means of phagocytosis.
- d) The reticuloendothelial cells of liver are also involved in production of some substances like interleukine and tumour necrosis factors which activate the immune system of the body.
- e) Detoxification - Removal of toxic property of the harmful agent occur's in 2 ways -
 - i) Destruction by means of metabolic degradation
 - ii) by converting toxic substances into non-toxic materials by means of conjugation with glucuronic acid or sulfates.

Effect of Sickle Cell Anaemia on Immunity :

So now it is very much clear that near about all the organs from skin to Bonemarrow are involved in immunity.

So in sickle cell anaemia due to cellular hypoxia the skin is dry and scaly with rashes. Spleen and liver are also damaged due to hypoxic changes and shows necrotic changes, due to necrosis liver and spleen also show dysfunction and do not take active part in immunization. Bone on later on stages goes in bone crush condition, equally the bone marrow and thymus and other glands also suffer from hypoxic shocks so their function in immunity also hamper in sickle cell anaemia.

So it is clearly seen that sickle cell patients are less immune and are more susceptible to infections.

Chapter - 4

Effect of Blood transfusion on sickle cell anaemia.

In my practice I have seen doctor's loading S.C.D. patients with Blood transfusion. Just like I.V. fluids. They are not bothered about Iron overload; I have not seen a single patient on Iron chelating drugs though patients shows hepatomegaly and spleenomegaly.

So according to me such type of blood transfusion ultimately becomes the cause to damage liver spleen and S.C.D. patient.

So there should be a change in attitude while giving blood transfusion to patients.

Then transmission of disease from contaminated blood, the chances are fewer that the patient gets infected due to presence of unidentical pathogen or if the blood is donated in window period.

So more blood transfusion more chances are there that patient will get infected with blood bound disorders.

Sickle Cell Anaemia & Multiple Drug Resistance i.e. M.D.R.

M.D.R. is a term with which every doctor is familiar and every doctor is encountering challenges against M.D.R. What is M.D.R.?

M.D.R. is a state where a patient does not respond to classical group of medicine or regularly used Antibiotics of lower generation of Antibiotics.

What causes M.D.R.?

- 1) All the Antibiotics should be prescribed by doctors.
- 2) Antibiotics should be in proper per kilogram body weight proportion and it should be taken for the proper course of time period, but many times some doctors prescribe Antibiotics in lower doses or in improper dose.
- 3) Some of the people who consider themselves as doctors and directly, bring medicines from the Pharmacists.
- 4) Some people directly purchase medicines from Pharmacists on pharmacist's advice as counter sale.

- 5) People who don't take the medicines as per doses or prescription are responsible to cause M.D.R.

Because of such reasons the pathogen or Bacteria becomes resistant to the Antibiotics and they are not completely eradicated i.e. it does not respond to the standard medication.

How MD.R. is related with Sickle Cell Anaemia patient ?

Sickle Cell Anaemic patients suffering increases in infection so if a patient is having M.D.R. then they will not respond to their regular Antibiotics and he will have to get hospitalized and will have to put on higher generation of Antibiotics.

So if Sickle Cell anaemic patient wants to lead a healthy life then they will have to take proper medication as advised by the proper Registered Doctor and should follow the proper course of treatment.

They should not take self medication because it can cost their life.

Case : One of my patient name unexposed due to medical ethics, his father used to tamper with the doses of Antibiotics, few months back he started getting frequent attack of fever, his father used to rush at me and used to shout at me.

Then one day I scold him and convince him that his son is having M.D.R. and he is not responding to cefexime he will have to start higher generation of Antibiotics. Then I took appointment of Dr. Sakhare from Wokhart hospital and started higher generation of the medicine and today the patient is completely well.

How to prevent M.D.R.?

1. Follow the instruction of your doctor.
2. Don't take Antibiotics on your own.
3. Don't stop Antibiotics yourself.
4. Don't purchase Antibiotics directly from pharmacists.
5. After getting relief from the symptoms of the illness don't stop the medicines finish the complete course.

Anaemia पाण्डु An Ayurvedic Concept

रक्तं जीवं इति स्थिति ।

Anaemia is defined as the decreased oxygen carrying capacity of the blood.

Pandu :- In this disease the complexion of the patient turns whitish yellow i.e. pallor. Hence it is termed as Pandu Roga.

Pathology of Anaemia :

व्यायाममम्लं लवणानिमद्यं मृदं दिवास्वप्नमतीव तीक्ष्णम् ।

निषेवमाणस्य प्रदूष्य रक्तं दोषस्त्वचं पाण्डुरतां नयन्ति ॥ सु.उ.त.अ.४३

- From this shloka it is clear that the above factors aggravates the Tridosha which in turn are responsible for vitiation of blood, resulting into whitish-yellow colour of the skin (pale colour).
- Here there is (impurity) of blood but not deficiency of rakta dhatu.
- Harit Samhita also mentioned about the vitiation of blood.
- According to charak Chkitsa 16, the signs & symptoms of disease are as follows. (सोऽल्प रक्तोऽल्पमेदस्को निःसारः। शिथिलेन्द्रियः । वैवर्ण्यं भजते)

- If we happen to see the sequence of the dhatu, Rasa dhatu comes first followed by Rakta, Maunsa, Meda etc. we realize that our physicians might have confused whether the pale colour of the skin is responsible for deficiency of blood or vice versa. But according to aetiology, without the cause / factors there is no disease; and hence it is clear that deficiency of blood results into pale colour and this is mainly due to impurity of pitta dosha. Earlier there were no parameters for measurement of Hb or else the Pandu disease would have been named as “Rakta-alpata.”

Modern :

In skin, there is network of blood capillaries. Epidermal layer is very fine & here the “Seeras” are absent. To some extent the Epidermal layer is transparent and due to this, redness of R.B.C’s is reflected on this layer. And so when there is deficiency of blood, the redness of R.B.C’s decreases and the skin colour appears pale - yellow.

After research of about 1 - 1 year on Anaemia, the following facts came forward, like :-

- 1) Modern medicines, inspite of having solutions, like Blood transfusion, could treat only general / common Anaemia and the other types of Anaemic patients were not treated as successfully,
- 2) If, only decreased oxygen carrying capacity is called Anaemia or deficiency of R.B.C’s is termed as Anaemia or Pandu, then it would have been much easier to treat this by simple means of Blood transfusion.
- 3) Practically it is difficult to co-relate the causes like iron deficiency or Vit B deficiency. These are directly contents and would be discussed in detail later on.
- 4) Our physicians have shown relation between discoloration of skin & Pitta, which is not true in reality. I have studied Anaemia and its complications in detail. To make it easier for the common man to understand & treat it, I have divided it in 2 parts.

i) Simple Anaemia (Sukh sadhya)

- a) Aahit aahar vihar.
- b) Abhighataj (Traumatic)
- c) Krumi-janya, Mrittika Janya.
- d) Agni Mandya Janya
- e) Due to Langhan
- f) Dehydration / Atisar janit.

ii) Complex Anaemia (Kashta Saadhya)

- a) Aplastic anaemia
- b) Sickle cell anaemia
- c) Haemophilia
- d) Haemolytic anaemia - i) thalasaemia

1) Simple Anaemia (Sukh Saadhya Raktalpata)

i) Agni maandya janya :-

Kaphaj Pandu :-

- 1) Having food / meal in spite of Agnimandya.
- 2) Having Heavy (Guru), Junk (Abhishandya), Oily / Snigdha, Pichhil food stuff high
- 3) Sleeping after having meal.
- 4) Consumption of Banana fruit, fish, Black gram.

2) Raktalpata Blood deficiency due to worms.

- The worms secrete an enzyme which increases the hunger, but most of the nutritious part of the consumed food is taken up by the worms themselves.
- During to this, there is no nourishment of the Rasa and Rakta dhatu which ultimately results into deficiency of blood.

3) Mrittika Bhakshan janya :-

- The consumed Mrittika (soil) is not digested.
- This is turn gets deposited in the strotasa.
- During to its Ruksha Character / nature it causes absorption of Rasa (dhatu) & liquid.
- This results in blood deficiency.

Treatment :-

- Krumijanya Krumi nirharan / worms expulsion.
- Mrittika janya The strotorodh (obstruction) caused due to Mrittika is removed by means of 'Lekhan Karma', followed by consumption of nutritious food.

3) Aahitkar aahar vihar -

- 1) The aggravation of Ruksha Guna is due to ingestion of Ruksha food or vaatal food leading to vaat Vridhi.
- 2) Owing to this, there is absorption of Drava-ansha (body fluid) and also constriction of strotasa.
Due to this (1) there is loss of body fluid.
(2) 'Constriction' of strotasa leading to malnourishment of Dhatu's. further leading to Rakta-alpata.

Treatment:- Snehan".

- 2) Consumption of Vidhahi food (irritants) & pitta aggravating foodstuffs, causes increase in the Drava Guna of Pitta which ultimately results into Agnimandhya causing production of Aam. OR if (here is increase in Aagni it further causes elevation of ushma Tikshna Guna. But here, rather than Rakta alpata there is possibilities of Kaamla (jaundice). Therefore Pitta mostly leads to kaamla & Kaamla is considered as subtype of Paandu.

Treatment :- of Kaamla

- 3) Rakta-alpata caused due to consumption of Abhishandya aahar or Rakta alpata due to Kupha is similar to that mentioned under the topic of Aagni mandhya.
- 4) **Langhan :-**
Excessive of Langhan leads to Vaat Prakop; further causing malnourishment which results in to Rakta alpata.
- 5) **Atisar Janya (Dehydration) :-**
 - Atisaar leads to excessive loss of body fluids.
 - This hampers the body fluid transportation (Vahan Karya) finally causing Anoxia.
 - Due to Anoxia, the Rakta & Medha Dhatus are unable to produce new R.B.C's resulting into Raktalpata.

Treatment :-

Since here Raktalpata alpala is due to loss of body fluids, treatment is done by providing the medium i.e. fluids, rather than giving iron supplements.

6) Abhigataja (Traumatic hemorrhage).

- Here Raktalpata is due to excessive blood loss which might be caused due to.
- Trauma or cuts / Laceration.
- M.C. or Miscarriage.

Treatment :- Blood transfusion.

Ayurveda :- This is treated by giving iron supplementation or substances having properties similar to iron.

Here ends the topic of simple Anaemia. After considering all the above mentioned points we conclude that if, any anaemic patient comes to us then,

- 1) Complete case history must be taken,
- 2) Keeping in mind, about all the causative factors, if we realize the main cause of the disease it becomes much easier to treat the disease.

Chapter - 7

Sickle Cell Anaemia (सिकल सेल अॅनिमियाची दहशत)

सिकल सेल अॅनिमिया हा सर्वात त्रासदायक व जीव घेणारा आजार आहे. या आजारामध्ये फक्त रुग्णच नव्हे तर त्याचा पूर्ण परिवार ग्रासला जातो. सिकल सेल अॅनिमियावर आजपर्यंत कोणत्याही प्रकारची खात्रीदायक चिकित्सा उपलब्ध नसल्यामुळे रुग्ण वारंवार रुग्णालयात भरती होत असतात आणि असहनीय वेदनांमधून त्याची सुटका जीव गेल्याशिवाय होत नाही. सिकल सेल अॅनिमिया हा कॅन्सर वा एचआयव्ही यांच्यापेक्षाही गंभीर स्वरूपाचा व्याधी आहे. इतका गंभीर असतानाही तो अनुवंशिक असल्याने सरकारकडून त्याचा गांभीर्याने विचार केला जात नाही.

सिकल सेल अॅनिमिया हा सर्वात आधी सर जेम्स हॅरिक यांना इरुला नामक आदिवासींमध्ये आढळला. १९१० मध्ये याचा प्रथम शोध लागला.

सिकल सेल अॅनिमियामध्ये मुख्यतः लाल रक्त पेशी या बाधित झालेल्या असतात. सामान्यतः लाल रक्त पेशी आकाराने गोल असतात. या व्याधीमध्ये गुणसुत्रांचा अकरावा जीन कारणीभूत आहे, असे आजपर्यंतच्या वैज्ञानिक संशोधनाने माहित (सिध्द) झालेले आहे. हा अकरावा जीन गोल रक्त पेशींना अर्धचंद्राकृति व अनेक वेगवेगळ्या प्रकारांमध्ये रूपांतरित करतो. प्राकृत लाल रक्त कणांचा (पेशींचा) जीवनकाल १२० दिवस असतो. पण सिकल सेल अॅनिमिक रुग्णामध्ये त्यांचा जीवनकाल २५-३० दिवस एवढा होतो. ज्यामुळे सिकल सेल रुग्णाला वारंवार रक्त दिले जाते. या रूग्णांमध्ये

लाल पेशी अर्धचंद्राकार व वेगवेगळ्या आकाराच्या असल्यामुळे एकत्रित जमा होण्याची वृत्ती असते. त्यामुळे या पेशी वाहून न जाता रक्तांच्या केशवाहीन्यांमध्ये असण्याची शक्यता जास्त असते. ज्या ठिकाणी शरीरात दबावाची क्षेत्रे जास्त असतात तेथे ही प्रक्रिया होण्याची शक्यता जास्त असते. यामुळे संबंधीत अवयवांना पोहचवला जाणारा रक्त पुरवठा कमी होतो किंवा बंद होतो. कमी होणाऱ्या स्थितीला इश्चेमिया असे म्हणतात. यामध्ये रूणाला असह्य तीव्र वेदना होतात. या वेदना मुतखड्याच्या वेदना किंवा प्रसवपीडा इतक्या तीव्र असतात.

रक्त पेशींच्या आकारांमुळे ऑक्सिजन वाहून नेण्याची क्षमता कमी असते. यामुळे आजारात शरीरातला एकूण एक अवयव ग्रासला जातो. यामुळे यकृत, प्लीहा (spleen), संधी, किडनी, अस्थि अशा प्रकारे सर्व अवयव हळूहळू कार्यहीन (Failure) होतात. याच स्थितीला Multiple organ involment म्हणतात. हे सर्व होत असताना यकृत (Liver) आणि मृत झालेले (necrosis) अवयव जीवाणू संक्रमणाचे स्थान बनतात ज्यामुळे हे रूण वारंवार जीवाणू संक्रमणाने ग्रसित होत असतात.

प्राकृत हिमोग्लोबिन हे 'AA' प्रकारचे असते आणि रूणांमध्ये 'S' प्रकारचा वेगळा हिमोग्लोबीन आढळतो. या 'S' प्रकारच्या हिमोग्लोबीनचे प्रमाण जितके जास्त तितका रूणाला त्रास जास्त असतो. याशिवाय या रूणांमध्ये 'F' प्रकारचे हिमोग्लोबीन व 'A₂' प्रकारचे हिमोग्लोबीनचे संघटित प्रकार सुध्दा आढळतात. या व्याधीचा विचार करता याचे पुढील प्रमाणे प्रकार पडतात.

- १) "SS" या प्रकारात हिमोग्लोबीनचा "SS" प्रकार आढळतो याचे प्रमाण ५०% पेक्षा जास्त असते.
- २) "SF" या प्रकारात हिमोग्लोबीनचे "S" आणि "F" प्रकार आढळतात.
- ३) "SFA₂" या प्रकारात हिमोग्लोबीनचे "S, F, A₂" आणि "F" प्रकार आढळतात.
- ४) "SA₂" या प्रकारात हिमोग्लोबीनचे S व A₂ प्रकार आढळतात.
- ५) "SA" या प्रकारात सिकल सेल ट्रेट असे म्हटले जाते. सिकल सेल ट्रेट चे रूण हे genetically abnormal असतात. पण शारिरिकदृष्ट्या ते प्राकृत असतात. त्यांना कोणत्याही प्रकारचा त्रास सिकल सेल मुळे होत नाही. रूण तपासत असताना सिकल सेल ट्रेट चे रूण सुध्दा येतात पण त्यामध्ये असे आढळून आले की, आलेल्या रूणांपैकी बऱ्याच रूणांचे निदान चुकीचे झालेले होते. त्यांना "SA" प्रकारचा नव्हे तर "SS" प्रकारचा व्याधी झालेला होता.

सिकल सेल अॅनिमियाचे निदान इलेक्ट्रोफोरेसीस व सॉल्युबिलिटी टेस्ट या पद्धतीने केले जाते. गर्भाला हा व्याधी आहे की नाही हे शोधण्यासाठी अॅन्जियोसिटोसिस या पद्धतीने केली जाते. परंतु असे कित्येक रूण आढळले आहेत ज्यांनी अॅन्जियोसिटोसिस केलेले होते व ते निगेटीव्ह होते. पण जन्मानंतर पॉझिटीव्ह आढळले. या आजारामध्ये रूणाला होणारा त्रास खालील प्रमाणे होतो.

- १) असहनिय जीव घेणाऱ्या वेदना ज्या कोणत्याही प्रकारच्या पेनकिलरनेही थांबत नाहीत. असेही रूण पाहिले गेले आहेत की जे एका दिवसात १० ते १४ फोर्टविन चे इंजेक्शन घेतात.
- २) Multiple organ Failure या आजारात रूणाची यकृत, प्लीहा, किडनी हे अवयव क्षत होतात, त्यांची कार्यक्षमता नाहीशी होते व रूण दगावतो.
- ३) Necrosis of joints (पेशींचा मृत्यू) – वक्षणसंधी किंवा हिप जॉईन्ट या आजारामुळे नेक्रोसिस होण्याचे सर्वसाधारण स्थान आहे.
- ४) जीवाणू संक्रमण – यकृत आणि मृत झालेली स्थाने ही जीवाणू संक्रमणास योग्य स्थाने असतात.
- ५) वरील सर्व कारणामुळे सिकल सेल ग्रसित रूणांची वयोमर्यादा साधारणतः ४० ते ४५ वर्षे असते.
- ६) SF, SFA₂, SA₂ हे रूण शारिरिक दृष्ट्या चांगले दिसतात परंतु यांच्यामध्ये अवयवांची क्षती (Multiple organ Failure) व Necrosis जास्त प्रमाणात आढळते.
- ७) व्याधी प्रतिकार क्षमता – सर्व पेशींवर परिणाम झाल्यामुळे व बोनमॅरोमधील बदलामुळे त्यांची व्याधी प्रतिकार क्षमता कमी असते व त्यामुळे ते वारंवार जीवाणू संक्रमणाने ग्रसित होत राहतात.
- ८) लाल रक्तकणांचा लवकर नाश झाल्यामुळे वारंवार त्यांना काविळीसारखे लक्षण आढळतात.
- ९) लहान मुलांमध्ये हात-पाय सुजून न्युमॅटिझम सारखी लक्षणे आढळतात. हा आजार समाजातील जवळ जवळ सर्व जातींमध्ये आढळून येतो.

यांच्यानुसार यांची जातीय टक्केवारी निम्न प्रकारे आढळते.

- | | |
|--------------------|--------|
| १) परधान आदिवासी – | २०.३१% |
| २) मराडमाली – | १६.१०% |
| ३) महार – | ११.८४% |

४) गोंड -	११.७४%
५) कुणभी -	९.३५%
६) वाणी -	९.०९%
७) ब्राह्मण -	४.१७%
८) कलार -	५.४१%
९) मुस्लीम -	३.७३%
१०) तेली -	४.८६%
११) राजपूत -	२.०४% इत्यादि.

चिकित्सेनंतर रूग्णामध्ये आढळून आलेले बदल.

- १) निलेश टेम्भूर्णीकर ६ वर्षांपासून ब्लड ट्रान्स्प्युजन थांबले आहे. व या वर्षी क्रायसेस सुद्धा आले नाहीत. हिमोग्लोबीन 11gm% पेक्षा जास्त असते. लाल रक्त पेशी संपूर्ण गोल झाल्या आहेत.
- २) प्रथम नावाच्या अडीच वर्षांच्या मुलात नखे बदलली गेली त्याचे कारण माहिती नाही.
- ३) चिकित्सेनंतर डब्ल्यूबीसी प्रमाण (श्वेत रक्तपेशी) अचानक वाढतात. ताप नसतो. तरीही वाढ होते. कारण माहिती नाही.
- ४) नेक्रोसिस झालेल्या स्थानी आणि खराब यकृत (Liver) जेव्हां दुरुस्त होत असतात. (ट्रीटमेंटच्या काळात) तेव्हां नेक्रोसिस झालेल्या रूग्णांत जीवाणू संक्रमण होत असते असे आढळते.
- ५) विविध आकार असणाऱ्या लाल रक्तपेशी गोलाकार होताना आढळतात. तरी दोन ते अडीच वर्षांनंतर रूग्ण स्थिर जीवन जगू लागतो.
- ६) तिसऱ्या वर्षी Infection पासून जास्त काळजी घ्यावं लागतं.



सिकल सेल बिमारी की उपचार पद्धती

सिकल सेल अॅनिमिया यह अनुवांशिक बिमारी है। गर्भ के गुणसुत्रो में हुए बदलाव से यह बिमारी होती है।

इसके बारे में सविस्तर जानकारी अंग्रेजी भाषा में दी जा चुकी है। इस बिमारी के S.S., S.F., S.F.A.₂, S.A. और S.A.₂ प्रकार होते हैं। S.A. यह ट्रेट होता है।

यह इस बिमारी की गुणात्मक अप्राकृत अवस्था है। लेकिन शारीरिक तथा प्राकृत अवस्था है।

अतः S.A. पेशन्ट को चिकित्सा की आवश्यकता नहीं है।

सिकल सेल अॅनिमिया के लक्षण :

- १) Hb या रक्त का अचानक कम हो जाना.
- २) पैरो मे या हाथों में सुजन आना.
- ३) इस बिमारी से किडनी, लिव्हर आदी अव्यय खराब हो जाते।
इस उपचार पद्धती से अप्राकृत लाल रक्त पेशीयों को प्राकृत किया व रखा जाता है। जिससे पेशन्ट सामान्य जिवन जी सकता है।



खायचे नाही

- ◆ दही
- ◆ जेवल्यावर लगेच पाणी प्यायचे नाही.
- ◆ केळी खायची नाही
- ◆ मिल्क शेक्स प्यायचे नाही
- ◆ शिळे अन्न खायचे नाही.
- ◆ ब्रेड, टोस्ट, मैद्याचे पदार्थ खायचे नाही
- ◆ दिवसा जेवल्यावर झोपायचे नाही.
- ◆ आंबवलेले पदार्थ इडली डोसा खायचे नाही.
- ◆ म्हशीचे दुध प्यायचे नाही.
- ◆ तुरीची दाळ खायची नाही.
- ◆ तंबाखु, सिगारेट, दारू यांचे सेवन करायचे नाही.
- ◆ समुद्री मासे, सुकवलेले मासे खायचे नाही.
- ◆ नुडल्स, चायनीज, अजीनोमोटो खायचे नाही.

खायचे पदार्थ

- ◆ गायीच्या दुधात एक चमचा गायीचे तूप टाकून प्यावे.
- ◆ सुका मेवा (ड्रायफ्रुट्स) फळ भाज्या भरपूर खाव्यात.
- ◆ कोमट पाणी, लिंबू पाणी प्यावे.
- ◆ जेवणात लिंबू, काळ्या गुळाचा खडा असावे.
- ◆ जेवल्यानंतर ताक प्यावे.
- ◆ नदीतील मासे, मटन, चिकन खाऊ खाऊ शकता.
- ◆ दररोज २ किलोमीटर फिरायला जावे.
- ◆ पोहणे, चालणे, खेळ नियमित पणे हवे.
- ◆ मुग डाळ, मसुर डाळ खावी.
- ◆ शक्यतो भातासाठी तांदुळ जुना वापरावा, ज्वारी, मका, आवळा, मुरब्बा लिंबाचे जुने लोणचे खावे.
- ◆ महिन्यातून एकदा चांगल्या क्वॉलिटीचा आईस्क्रीम खावा.

सिकल सेल ॲनिमिया आणि मर्फिज लॉ!

चिमुरड्यांना होणाऱ्या त्रासामुळे खचलो! रुग्णांना चिकित्सा देण्याच्या आधी, पूर्वकल्पना दिल्या तरीही, चिकित्सेच्या मध्ये मुलांना काही त्रास झाल्यास त्यांच्या पालकांकडून वाईट प्रतिक्रिया लगेचच यायची! तेव्हा असे वाटायचे की आपण हे का करतोय? सगळं सोडून द्यावस वाटायचं! पण तेव्हा **Murphy's Law** वं ५ वे सूत्र आठवले....

Left to themselves, things tend to go from bad to worse.

की.... जर मी थांबलो असतो तर जे रुग्ण आज या चिकित्सेने बरे झाले आहेत व जे काही बरे होत आहेत त्यांची अवस्था काय झाली असती?

– रेशन चिंचखेंड.....**spleen** (प्लीहा) काढावी लागली असती आणि एकट्या आईचा आधार कधी काळाच्या विळख्यात गेला असता कळतही नसतं....!

– प्रशांत ढगेदर ४ दिवसांनी **Fortwin** ची इंजेक्शन्स घेत असता....!

– निकिता सुसटे तिच्या दोन्ही किडनी कधीच खराब झाल्या असत्या!

– वेदान्त नरुले रामटेकेला दर महिन्याला **Blood Transfusion** ची गरज पडली असती. असे किती तरी रुग्ण **Sickle Cell anaemia** मुळे संपले असते पण आज ते बरे आहेत. आणि सर्वसामान्यासारखे त्यांचे आयुष्य व्यवस्थित जगत आहेत.

अशा रुग्णांना पाहिल्यानंतर वाटतं की, आपण जे पाऊल उचललेले आहे ते खरंच योग्य आहे. त्रास हा होणारच आहे.

Infection मध्ये मी हतबल होतो. रुग्णांना त्रास होतो. पण तो त्रास त्यांना सोसावाच लागणार आहे. जोपर्यंत ह्यावर अजून भर दिला जात नाही तोपर्यंत.

ही वाट मला एकट्यानेच पार पाडायची आहे... कोणतीही यंत्रणा सोबत नसताना. लोक निधी बद्दल बोलतात पण पैशांच्या निधीसाठी कुणी ही पुढे येत नाही. एकट्याने हा सर्व व्याप घेऊन चालायला जरा अवघड होते. पण रुग्णांकडे बघुन मी सर्व गोष्टींसाठी तयार होतो.

मला दुखत आहे

हा प्रश्न पुन्हा पुन्हा विचारला जात आहे. त्याचे पाय का दुखत आहेत? कमरेत का दुखत आहे?

- १) वेदना कधी होतात? विकृत रक्तपेशी जेव्हा रक्तवाहिन्यांमध्ये अडथळा निर्माण करतात तेव्हा पुढच्या अवयवांना रक्त मिळू शकत नाही. व रुग्णास दुखावयास लागते.
- २) दुखणे सुरु झाल्यावर तेथे मालीश करावी व शेख द्यावा.
- ३) दुखणे कमी करण्याच्या औषधीमुळे किडनी खराब होण्याची भिती असते म्हणून डॉक्टर आपणांस सांगतील तेवढ्याच पेन क्लिअर औषधांचा उपयोग करावा.
- ४) ट्रीटमेंट चालू असताना देखील हे पाहिले गेले आहे की इन्फेक्शन किंवा संक्रमणाच्या अवस्थेत सेल मध्ये ऑक्सीजन कमतरतेने सेल विकृत झाल्याने वेदना होतात.
- ५) इन्फेक्शन किंवा संक्रमण हे बॅक्टेरिया किंवा व्हायरस मुळे होते. सिकल सेल मुळे नाही.
- ६) इन्फेक्शन किंवा संक्रमणाची चिकित्सा केल्यावर दुखणे जाते.
- ७) अशा वेळी संयम बाळगावा.
- ८) सतत डॉक्टरांच्या संपर्कात रहावे.
- ९) ४ वर्षांच्या ट्रीटमेंट नंतर दुखणे फार कमी वेळा येते.
- १०) आम्ही इन्फेक्शन टाळू शकत नाही.

जर आपण इन्फेक्शन टाळू इच्छिता तर खालील गोष्टी लक्षात ठेवाव्यात.

- १) काहीही खाण्याच्या आधी हात स्वच्छ धुऊन घ्यावे.
- २) स्वच्छ भाड्याचाच वापर करावा.
- ३) उघड्यावरील खाद्यपदार्थ, दुकानातील तयार खाद्यपदार्थ खाऊ नये.
- ४) ज्यांना सर्दी खोकला आहे त्यांच्याजवळ जाऊ नये. शिंकल्यावर सर्दी पसरते.
- ५) डॉक्टर आपणास तापासाठी जी औषधे देतात ती सेवास्वरूप आहेत. ती पॅकेजमध्ये येत नाहीत. म्हणून त्या सेवेस सन्मानपूर्वक घ्यावे. तो आपला हक्क नाही.
- ६) ताप हा रुग्णाच्या स्वतःच्या वर्तणुकीमुळे किंवा आजुबाजुच्या वातावरणामुळे होतो तुम्ही डॉक्टरला माझी तबबेत खराब का झाली असे विचारू शकत नाही. कारण त्याचे जबाबदार तुम्ही स्वतः व तुमच्या आजूबाजूचे वातावरण आहे. त्रास झाल्यावर केवळ तो दुर होण्यासाठी औषधे विचारू शकता.

* Murphy's 10th Law - Mother Nature is a Bitch.

आई प्रकृती ही एक- कुत्री आहे. कारण ही कधी धोका देईल सांगता येत नाही. आज दि. २/१२/२०१० ला नागपूर किंवा विदर्भात थंडी कुठे जाणवत ही नाही. त्या ऐवजी आता अवकाळी पाऊस आल्याने रुग्ण **Viral Infection** नी त्रस्त झाले. त्या रुग्णांची सांत्वना करता करता मी थकलो. त्यामुळे माझी लढाई ही आई प्रकृतीशी! "**Mother Nature**" शी आहे.

त्यामुळे हे सगळं असताना, माझ्या दोन स्त्री रुग्णांना कोणताही त्रास न होता त्या गोवा आणि इतर ठिकाणी फिरून आल्यावर **Thank you!** बोलल्या आणि माझा कामाचा जोर वाढला. आता मी **Mother Nature** शी दोन हात करायला नव्याने तयार झालोय.

लोकांना वाटत आपण पैसे दिले की आपली जबाबदारी संपली आता आपला मुलगा किंवा मुलगी बरी झालीच पाहिजे...! आणि त्यांनी मात्र कर्तव्य विमुक्त होऊन रहायचं. वारंवार **Infections** बदल सांगून ही बाहेरचे ब्रेडचे पदार्थ, बटाटे वडे, तेलकट पदार्थ खालणे हा काही समजुतदारपणा नव्हे!

पण काही लोक मला असे ही भेटले ज्यांनी मी सांगितलेल्या प्रत्येक गोष्टीला प्रामाणिकपणे अंगीकृत केलं. माझ्या एका रुग्णाच्या आईने प्रत्यक्ष शाळेत जाऊन स्वतःच्या मुलाला वेगळं बसवण्याची तरतूद केली. त्यामुळे सर्दी-खोकल्यासारख्या त्रासातून त्याचे रक्षण केले. अशाचप्रकारे सर्व पालकांनी सुध्दा जे सांगितले जाते त्या निदर्शनांचे काटेकोरपणाने पालन करणे आवश्यक आहे. त्यामुळे **Infection** मुळे होणारा त्रास कमी होईल.

Preface

I am very happy to write preface for my first edition of sickle cell Anaemia.

I hereby pay my homage to Dr. Laxmidutt Shukla Sir, Er Professor in Samhita Department at K.G.M.P. Ayurvedic Collage without whose support and teaching, I would not be able to practice Ayurveda so keenly.

Dr. K. R. Kohli Director of Ayurveda whose personality and ability to conduct was always encouraging to me

Dr. Tayade sir, Dr. Makwana Sir, Dr. Ramesh wawre Sir, taught me basic of Ayurveda Master Kaustubh Chahande a student of I.B. is the Architect of this Research Presentation.

I pray Lord Buddha for the peace and harmony of this country.

- Dr. A. H. Nandeshwar

www.sicklecellanaemiatreatment.com

विशेष सूचना

- * सभी पेशन्ट से निवेदन है की बरसात शुरू हो गई है तो बाहर का कुछ भी खाद्य पदार्थ न खाये ।
- * बाहर का खाद्य पदार्थ खाने से या बाहर का जुस, शरबत पिने से बिमारी हो सकती है तो उसका सेवन ना करे।
- * पेशन्ट हो या साधारण व्यक्ती खाना खाते समय अपने हात अच्छी तरह धो ले।
- * फल फ्रुट अच्छी क्वालीटी के लें और अच्छी तरह धोके खाये ।
- * जब भी पानी पिये उबालकर पिये ।
- * खाद्य पदार्थ हमेशा ढक कर रखे ।
- * खाना हमेशा गरम करके खाये ।
- * बासी खाना ना खाये ।
- * तेल में तले हुए पदार्थ पेशन्ट को नहीं खिलाये ।
- * घर अच्छी तरह साफ रखें और मच्छर मक्खी घर में आने न पाये इसका भी ध्यान रखे।
- * मिल्क शेक नहीं पिना है।
- * दुध और फल एक साथ ना लें।
- * पेशन्ट बरसात में भिगने न पाये इसका ध्यान रखें ।

