

REPUBLIQUE DU CAMEROUN

PAIX-TRAVAIL-PATTIE

MINISTERE DE L'ENSEIGNEMENT SUPERIEUR

DIRECTION DE L'ENSEIGNEMENT SUPERIEUR  
PRIVE

REPUBLIC OF CAMEROON

PEACE-WORK-FATHERLAND

MINISTRY OF HIGHER EDUCATION

DEPARTMENT OF PRIVATE EDUCATION

**CAPITOL HIGHER INSTITUTE OF HEALTH**  
  
**SCIENCES AND BEAUTY THERAPIES**

P.O BOX: 875, BAMENDA.  
MOTTO: HOPE IS THE KEY

**A CASE STUDY REPORT ON SICKLE CELL  
ANAEMIA IN A CHILD CARRIED OUT AT THE  
REGIONAL HOSPITAL BAMENDA  
FROM 27 SEPTEMBER TO 29 OCTOBER 2016**

**SUBMITTED IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR  
THE AWARD OF HIGHER NATIONAL DIPLOMA (HND) IN NURSING**

**PRESENTED BY:  
NCHULA VICTORIA SHIZA**

**SUPERVISED BY:  
Dr MFONFU DANIEL**

**April 2017**

## **CERTIFICATION**

This is to certify that the case study on sickle cell anaemia was carried out at the Regional Hospital Bamenda, North West Region from 27 September to 29 October 2016. This is original by NCHULA VICTORIA SHIZA.

Student **Nchula Victoria Shiza** Signature\_\_\_\_\_ date: 25/04/2017

Supervisor: **Dr Mfonfu Daniel** Signature\_\_\_\_\_ date: 25/04/2017

Dean of studies **Dr Mfonfu Daniel** Signature\_\_\_\_\_ date: 25/04/2017

President of Jury **Dr Mfonfu Daniel** Signature\_\_\_\_\_ date: 25/04/2017

## **DEDICATION**

This piece of work is dedicated to my parents Mr. and Mrs. Boma Martin Nchula, who because of their moral and financial support made it possible for me to achieve this piece of work..

## **ACKNOWLEDGEMENTS**

It will be unwise to say that this work has been done without the academic, moral and material assistance of some people.

Special thanks goes to the manger of CAPITOL, higher institute Mr. Ngalah Edward for creating an institution and for choosing the Regional Hospital Bamenda Northwest Region for me to carry out my case study.

Thanks go to the entire staff of CAPITOL for their relentless efforts just to make sure that the students are equipped with the best knowledge.

Great thanks goes to the entire administration and hospital staff for their assistance given to us to realize our objectives.

Thanks to the supervisor who despite all the commitments took time to guide the researcher towards the success of this of this project.

Special thanks goes to my parents whom God has given grace to sponsor this work.

Thanks so much and God almighty will surely prolong their lives so that they can reap the fruits of their labour.

And lastly gratitude goes to classmates' friends and neighbours for love encouragement and support they gave to realize this work and their tolerance made the researcher to understand that winners do not quit and quitters don't win.

## LIST OF ABBREVIATION

ABBREVIATION	FULL MEANING
VOC	Vaso Occlusive Crises.
SCD	Sickle Cell Disease
DOA	Date Of Admission
DOD	Date Of Discharge
COD	Condition Of Discharge
Hb	Hemoglobin
BRH	Bamenda Regional Hospital
SCA	Sickle Cell Anaemia

## LIST OF FIGURES

Figure one (organigramme).....

## LIST OF TABLES

<b>Table 1: LAB RESULTS FOR BLOOD</b> -----	<b>26</b>
Table 2: LAB RESULTS FOR STOOL-----	27
<b>Table 3: DAILY DRUG CHART</b> -----	<b>28</b>
<b>Table No 4: Nursing care plan 1</b> -----	<b>30</b>
<b>Table No 5: Nursing care plan 2</b> -----	<b>31</b>
<b>Table 6: Nursing care plan 3</b> -----	<b>31</b>
<b>Table No 7: Nursing care plan 4</b> -----	<b>32</b>
<b>Table No 8: Nursing care plan 5:</b> -----	<b>32</b>
<b>Table 9: DAILY EVALUATION OF THE PATIENT</b> -----	<b>33</b>
<b>Table 10: VITAL SIGN CHART</b> -----	<b>33</b>

## TABLE OF CONTENTS

<b>CERTIFICATION</b> -----	2
<b>DEDICATION</b> -----	3
<b>ACKNOWLEDGEMENTs</b> -----	4
<b>LIST OF ABBREVIATION</b> -----	5
<b>LIST OF FIGUREs</b> -----	6
<b>LIST OF TABLEs</b> -----	7
Table of content -----	8
<b>CHAPTER ONE – INTRODUCTION</b> -----	<b>9</b>
<b>1.1 INTRODUCTION</b> -----	<b>9</b>
<b>1.2 MOTIVATION</b> -----	<b>9</b>
<b>1.3 GENERAL OBJECTIVES</b> -----	<b>9</b>
<b>1.4 SPECIFIC OBJECTIVES</b> -----	<b>9</b>
<b>1.5 BRIEF DESCRIPTION OF PLACE OF STUDY</b> -----	<b>10</b>
<b>CHAPTER TWO - LITERATURE REVIEW</b> -----	<b>12</b>
<b>2.1 Definition of Sickle Cell anaemia</b> -----	<b>12</b>
<b>2.2 Distribution of Sickle Cell Anaemia in the World</b> -----	<b>13</b>
<b>2.3 INHERITANCE</b> -----	<b>13</b>
<b>2.4 CAUSES OF SICKLE CELL ANAEMIA</b> -----	<b>14</b>
<b>2.5 PATHOPHYSIOLOGY</b> -----	<b>16</b>
<b>2.6 Signs and symptoms</b> -----	<b>17</b>
<b>2.7 TREATMENT OF SICKLE CELL ANAEMIA</b> -----	<b>20</b>
<b>2.8 Preventing of Complications</b> -----	<b>21</b>
<b>2.9 Management of sickle cell anaemia</b> -----	<b>22</b>
<b>2.10 PROGNOSIS OF SICKLE CELL ANAEMIA</b> -----	<b>24</b>
<b>2.11 PREVENTION OF SICKLE CELL ANAEMIA</b> -----	<b>24</b>
<b>2.12 DEFINITION OF NURSING CARE PLAN</b> -----	<b>24</b>
<b>2.13 Objectives of nursing care plan</b> -----	<b>24</b>
<b>2.14 The 14 basic need proposed by virginal Henderson</b> -----	<b>24</b>
<b>2.15 Nurses responsibility in drug administration</b> -----	<b>24</b>
<b>CHAPTER THREE - PRESENTATION OF CASE</b> -----	<b>25</b>
<b>3.1 THE DEMOGRAPHICAL IDENTIFICATION OF THE CASE</b> ----	<b>25</b>
<b>3.2 Condition on admission</b> -----	<b>25</b>
<b>3.3 PROVISIONAL DIAGNOSIS BY MD</b> -----	<b>25</b>
<b>3.4 CLERKING / ASSESSMENT BY NURSES</b> -----	<b>25</b>
<b>3.5 Table 1: LAB RESULTS</b> -----	<b>26</b>
<b>3.6 Doctor’s diagnosis after results</b> -----	<b>27</b>
<b>3.7 Medical prescription and treatment by dr. after results</b> -----	<b>27</b>
<b>3.8 Table 2: DAILY DRUG CHART</b> -----	<b>28</b>
<b>Table No 3: Nursing care plan: Date -28/09/2016</b> -----	<b>30</b>
<b>3.14 DAILY EVALUATION OF THE PATIENT</b> -----	<b>33</b>
<b>3.15 VITAL SIGN CHART</b> -----	<b>33</b>
<b>3.16 EVOLUTION OF THE STATUS OF PATIENT</b> -----	<b>33</b>
<b>CHAPTER FOUR - REVIEW MEDICATION</b> -----	<b>34</b>
<b>CHAPTER FIVE – DISCHARGE SUMMARY</b> -----	<b>42</b>
<b>CHAPTER SIX - CONCLUSION</b> -----	<b>44</b>



## **CHAPTER ONE - INTRODUCTION**

### **1.1 INTRODUCTION**

A case study is a description of a real life problem or a situation which requires the analysis of the main issues. These issues needs to be discussed and related to be discussed related to academic literature and conclusion drawn about on why the conditions occur and how best to respond to it. In Cameroon it serves as the academic requirement for the acquisition of HND

Consequently, the researcher decided to choose a case study on sickle cell anaemia because statistics have shown that, there is a high incidence rate of sickle cell anaemia now as compared to other times (source why?).

### **1.2 MOTIVATION**

The researcher was motivated to carry out this study on sickle anaemia in order to know how it can be prevented and the risk factors to avoid producing sicklers. This is so due to increased rate at which husbands abandon their wives and children because they cannot cope with the child's medical requirement thus allowing the wife to bear the load and suffering alone. This reason regarded my interest in this disease so as to adverse the community on its hazards so it can be prevented'

### **1.3 GENERAL OBJECTIVES**

Successfully manage the case of sickle cell anaemia as a member of the medical and nursing team and to submit the report of this case study in partial fulfilment to obtain the HND in nursing.

### **1.4 SPECIFIC OBJECTIVES**

- ❖ Description of place of study (diagram )
- ❖ Give a brief description of place of study
- ❖ Fig organigram of place of internship and source
- ❖ Identify the patient
- ❖ Describe the circumstances of arrival of the patient.
- ❖ Admit the patient.
- ❖ State the provisional diagnosis on admission state source.
- ❖ Administer any emergency medications
- ❖ Clerk/assess the patient.
- ❖ Administer the medications prescribed by the medical officer. Monitor and record side effects on the patient.
- ❖ Describe nurses responsibility in the administration of drugs to patients.
- ❖ Establish daily drug chart
- ❖ State results of confirmatory diagnostic test

- ❖ Develop and implement nursing care plans
- ❖ Describe the evaluation of the patient and vital signs
- ❖ Review the medications administered
- ❖ Write the discharge summary
- ❖ Identify positive findings weaknesses, make recommendations make conclusions.

## **1.5 BRIEF DESCRIPTION OF PLACE OF STUDY**

According to the presidential decree were changing all provinces to regions in Cameroon the formal Bamenda provincial hospital now call Bamenda hospital formally located at up station was created in 1940

On the 5<sup>th</sup> of April 1965, the present Bamenda regional hospital was transferred to its present site located at the central town of Bamenda 1km away from food market and along the road to Ntarikon. It is a research centre a dialysis centre, a teaching hospital and also a referral hospital for district hospital in the region.

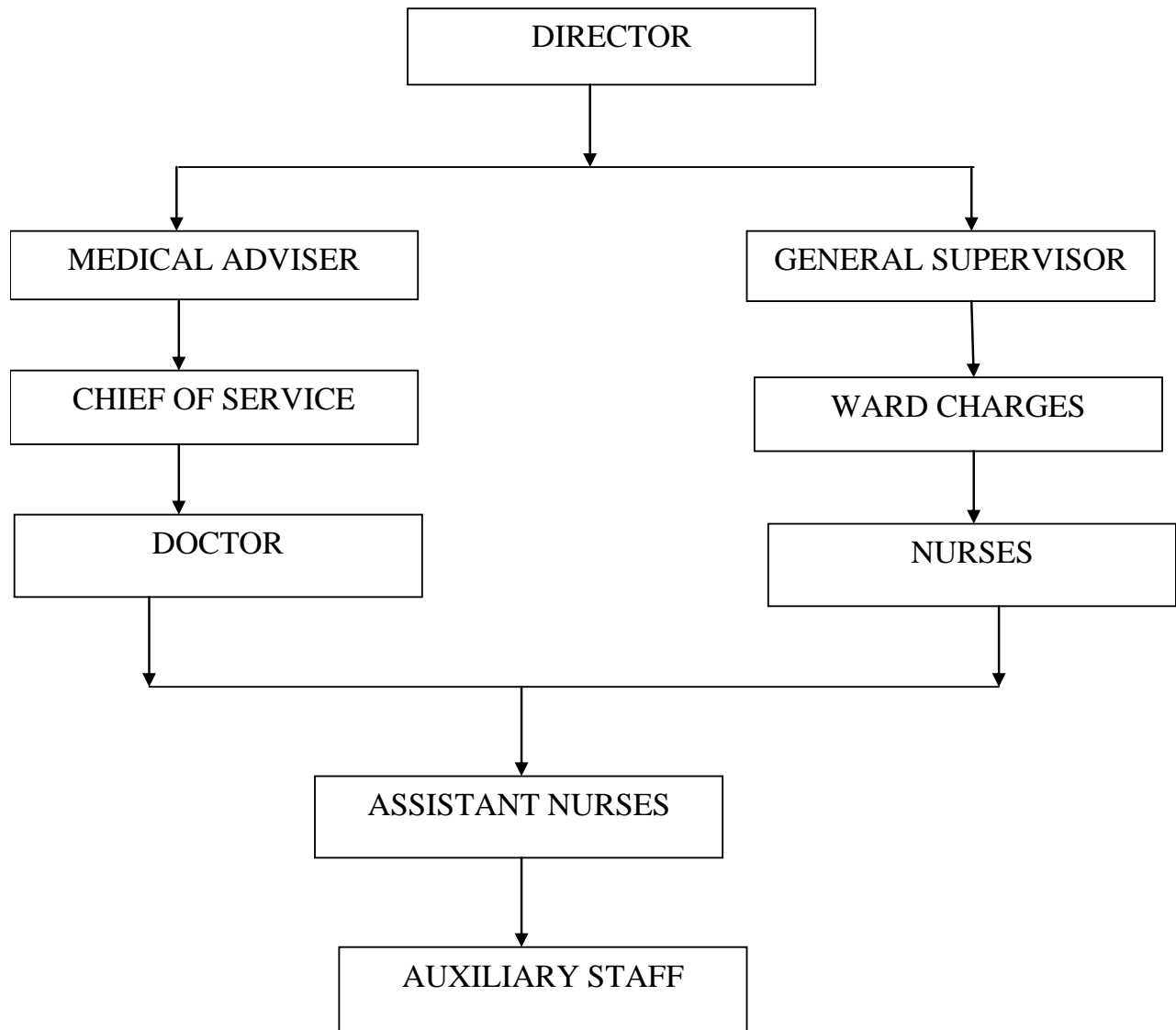
The hospital has many unites where different activities are carried out. Some of these units are as follows.

- i. Male medical ward
- ii. Female medical ward
- iii. Male surgical word
- iv. Female surgical ward
- v. Paediatric ward
- vi. Gynaecological ward
- vii. Ophthalmology department
- viii. Physiotherapy department
- ix. The maternity section
- x. The pharmacy
- xi. The medical laboratory
- xii. The operating theatre
- xiii. Reanimation unit
- xiv. Dialysis department
- xv. EPI section
- xvi. The dental department
- xvii. The TB unit

The above departments are headed by ward charges, SRN are also present on the departments and run their activities in two shifts. The general supervisor, is in charge of all the Nurses in BRH

The hospital has a bed capacity of 400beds with more than 200 personnel including 28doctors amongst which are specialists.

ORGANIGRAM



Source: General Supervisor

## CHAPTER TWO - LITERATURE REVIEW

### 2.1 Definition of Sickle Cell anaemia

Sickle-cell anaemia is a form of sickle-cell disease in which mutant haemoglobin genes are inherited from both parents. (Report by the Secretariat; World Health Organization).

Sickle cell anaemia is inherited form of anaemia — a condition in which there aren't enough healthy red blood cells to carry adequate oxygen throughout the body. Normally, the red blood cells are flexible and round, moving easily through the blood vessels. In sickle cell anaemia, the red blood cells become rigid and 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 (Mayo Clinic Staff).

Sickle-cell anaemia (SCA) also known as drepanocytosis, is a hereditary blood disorder, characterized by an abnormality in the oxygen-carrying haemoglobin molecule in red blood cells. This leads to a propensity for the cells to assume an abnormal, rigid, sickle-like shape under certain circumstances. Sickle-cell anaemia is associated with a number of acute and chronic health problems, such as severe infections, attacks of severe pain ("sickle-cell crisis"), and stroke, and there is an increased risk of death.

Sickle-cell anaemia occurs when a person inherits two abnormal copies of the haemoglobin gene, one from each parent. A person with a single abnormal copy does not experience symptoms and is said to have sickle-cell trait.

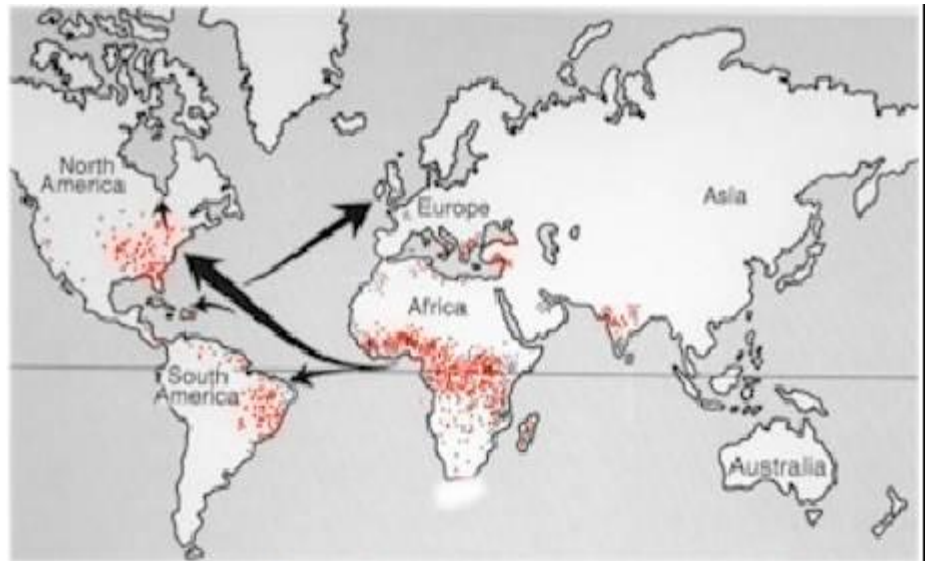
([http://en.wikipedia.org/wiki/Sickle-cell\\_disease](http://en.wikipedia.org/wiki/Sickle-cell_disease))

However, the disease may also present in the mandible. The oral manifestation and radiographic findings of a sickle cell patient with a left mandibular neuropathy along with dental management guidelines are presented in the context of interdisciplinary care (Davis V 1992)

Haemoglobin is the protein in red blood cell that carries oxygen. It is made up of two alpha chains and two beta chains. They are made up by the alpha and beta genes. The four main types of sickle cell anemia are caused by different mutation in the genes (Rebecca A. Morrison et al: genetic sick booklet)

### 2.3 Distribution of Sickle Cell Anaemia in the World

Sickle-cell anaemia is particularly common among people whose ancestors come from Sub-Saharan Africa, India, Saudi Arabia and Mediterranean countries. Migration raised the frequency of the gene in the American continent. In broad terms, the prevalence of the sickle-cell trait (healthy carriers who have inherited the mutant gene from only one parent) ranges between 10% and 40% across equatorial Africa and decreases to between 1% and 2% on the north African coast and <1% in South Africa. The sickle-cell gene has become common in Africa because the sickle-cell trait confers some resistance to falciparum malaria during a critical period of early childhood, favouring



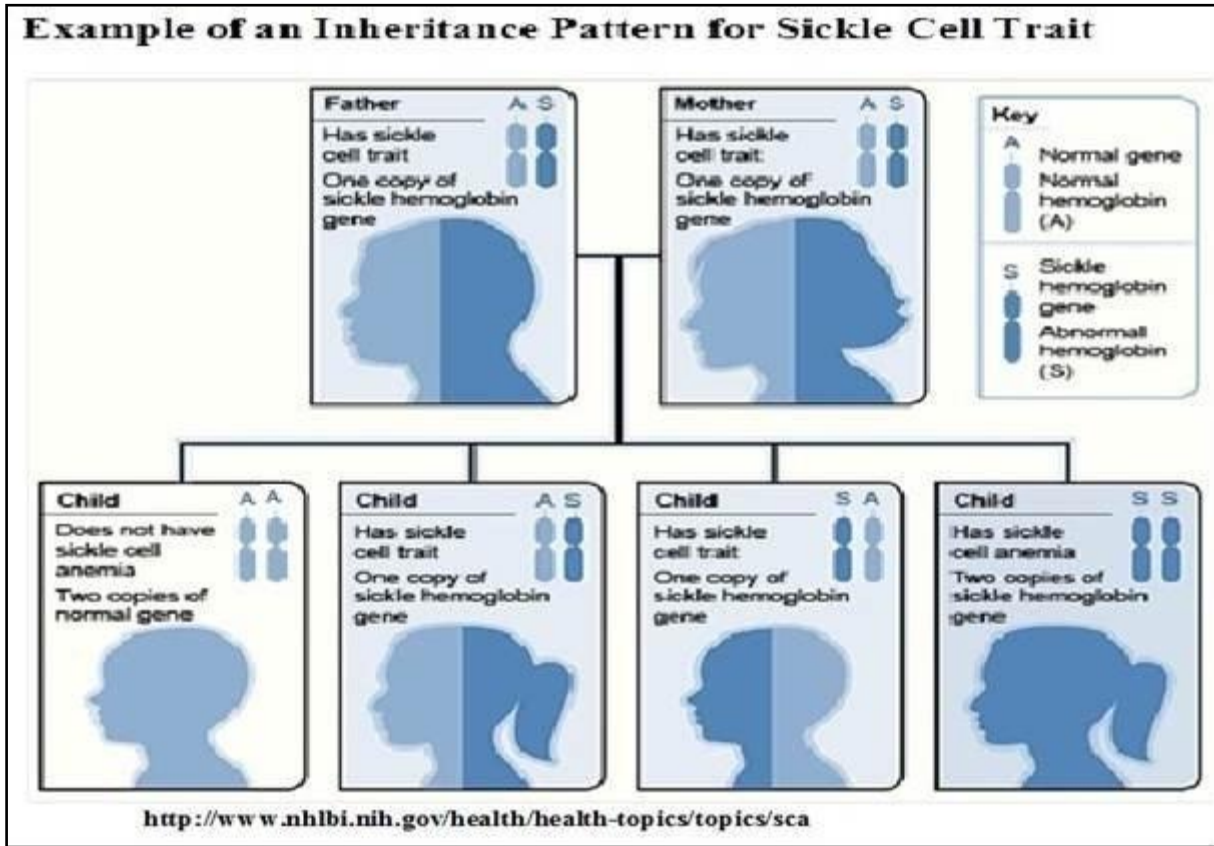
Debra A. Vedro, and Rebecca A. Morrison, Children's Medical Center of Dallas, Dallas, Texas

**Fig I – Distribution of SCA in the world**

survival of the host and subsequent transmission of the abnormal haemoglobin gene. Although a single abnormal gene may protect against malaria, inheritance of two abnormal genes leads to sickle-cell anaemia and confers no such protection, and malaria is a major cause of ill-health and death in children with sickle-cell anaemia. There is increasing evidence that malaria not only influences outcome but also changes the manifestations of sickle-cell anaemia in Africa. (Report by the Secretariat; World Health Organization)

### 2.3 INHERITANCE

Sickle-cell anaemia has an autosomal recessive pattern of inheritance from both parents. The types of haemoglobin a person makes in the red blood cells depend on what haemoglobin genes are inherited from her or his parents. When both parents have sickle-cell trait, a child has a 25% chance of sick anaemia, 25% will not carry any sickle-cell alleles, and 50% will have the heterozygous condition.



**Fig ii – Inheritance of SCA**

## 2.4 CAUSES OF SICKLE CELL ANAEMIA

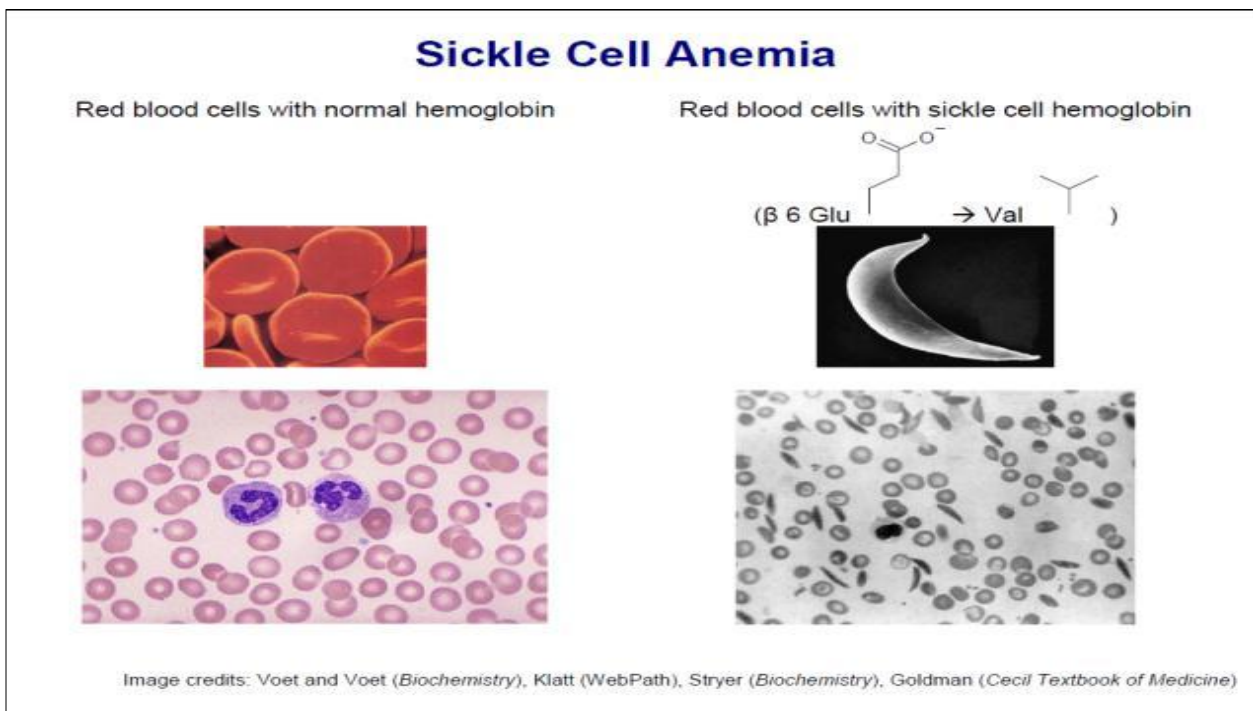
Sickle cell anaemia is caused by a mutation in gene that tells the body to make haemoglobin (a protein in red blood cells that carry oxygen). Haemoglobin is the red iron compound that gives blood its red colour. Haemoglobin enables red blood cells to carry oxygen from the lungs to all parts of the body.

In sickle cell anaemia, the abnormal haemoglobin causes RBC to become rigid and sticky. The sickle cell gene is passed from generation to generation in pattern of inheritance called autosomal recessive inheritance. This means that the mother and the father must pass across the defective gene for a child to be affected. If only one parent passes on the sickle cell gene to the child, then the child is said to have the sickle cell trait with one normal haemoglobin gene and the defective form of the gene.

People with the sickle cell trait synthesize the normal haemoglobin and the sickle cell haemoglobin. This blood may contain some sickle cell but generally they don't experience the symptoms. However, they are carriers of the disease which means that they can pass on this defective gene to their offspring with each pregnancy.

## 2.5 PATHOPHYSIOLOGY

Sickle-cell anaemia covers a wide spectrum of illness. Most affected people have chronic anaemia with a haemoglobin concentration of around 8 g/dl. The main problems arise from the tendency of the red blood cells to become sickle-shaped and block capillaries at low oxygen tension. In children, sickle-shaped red blood cells often become trapped in the spleen, leading to a serious risk of death before the age of seven years from a sudden profound anaemia associated with rapid splenic enlargement or because lack of splenic function permits an overwhelming infection. Between 6 and 18 months of age affected children most often present with painful swelling of the hands and/or feet (hand-foot syndrome). Survivors may also suffer recurrent and unpredictable severe painful crises, as well as “acute chest syndrome” (pneumonia or pulmonary infarction), bone or joint necrosis, priapism or renal failure. (Report by the Secretariat; World Health Organization)



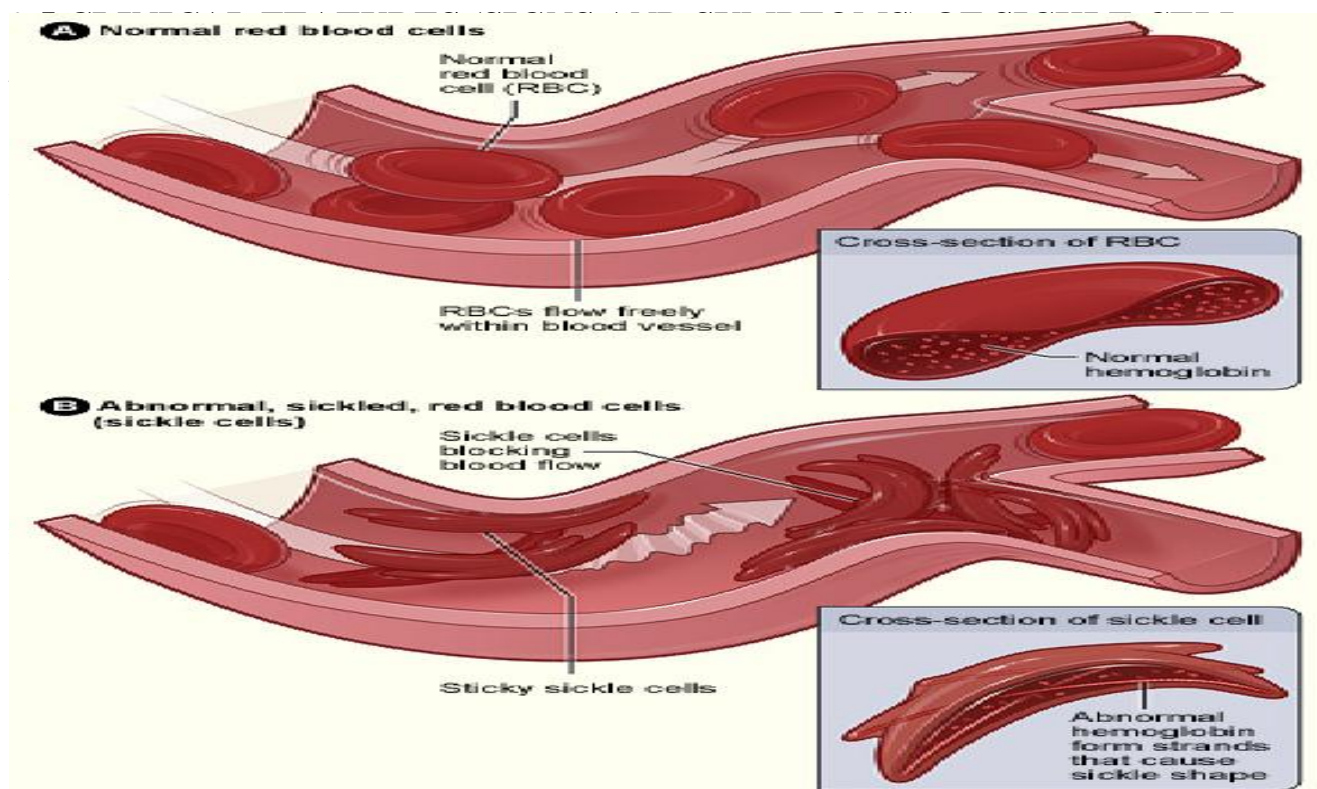
**Fig iii- blood film in SCA**

There are two essential pathological processes.

- ❖ Haemolysis
- ❖ Vaso-occlusion.

Haemolysis results in anaemia and a functional deficiency of nitric oxide which results in vascular endothelial damage and may be responsible for complications such as pulmonary-hypertension, priapism and stroke.

Vaso-occlusion cause acute and chronic ischaemia and is responsible for acute pain and organ damage (1332-SC-Clinica-standard-WEB.).



*Figure A shows normal red blood cells flowing freely in a blood vessel. The inset image shows a cross-section of a normal red blood cell with normal hemoglobin. Figure B shows abnormal, sickled red blood cells blocking blood flow in a blood vessel. The inset image shows a cross-section of a sickle cell with abnormal (sickle) hemoglobin forming abnormal strands.*

**Fig iv – sickling of RBC and vaso occlusion**

**2.6 Signs and symptoms** This pain is called a sickle cell crisis. Sickle cell crises often affect the bones, lung, abdomen and joints. The pain usually last from hours to as long as a week or more.

Many people who have sickle cell anaemia also have chronic pain, especially in their bones. Chronic pain often lasts for weeks or months and can be hard to bear and mentally draining chronic pain may limit your daily activities.

Almost all people who have sickle cell anaemia have painful crises at some point in their lives. Some have this crisis less than a year. Others may have crises once a



month or more. Repeated crises can damage the bones, kidneys, lungs, eyes, heart, and liver. This type of damage happens more often in adults than in children.

### Signs and symptoms of infection

Fever – Cough - Vomiting and / or diarrhoea – Crankiness - Rapid breathing  
Pale colour - Unusual sleepiness – dyspnoea

### .Vaso-occlusive crisis

The vaso-occlusive crisis is caused by sickle-shaped red blood cells that obstruct capillaries and restrict blood flow to an organ resulting in ischaemia, pain, necrosis, and often organ damage.

### Splenic sequestration crisis

Because of its narrow vessels and function in clearing defective red blood cells, the spleen is frequently affected. It is usually infarcted before the end of childhood in individuals suffering from sickle-cell anaemia. This autosplenectomy increases the risk of infection from encapsulated organisms; preventive antibiotics and vaccinations are recommended for those with such asplenia.

Splenic sequestration crises are acute, painful enlargements of the spleen, caused by intra-splenic trapping of red cells and resulting in a precipitous fall in haemoglobin levels with the potential for hypovolemic shock. Sequestration crises are considered an emergency.

### Acute chest syndrome

Acute chest syndrome (ACS) is defined by at least two of the following signs or symptoms: chest pain, fever, pulmonary infiltrate or focal abnormality on a chest X-ray, respiratory symptoms, or hypoxemia. It is the second-most common complication and it accounts for about 25% of deaths in patients with SCA, majority

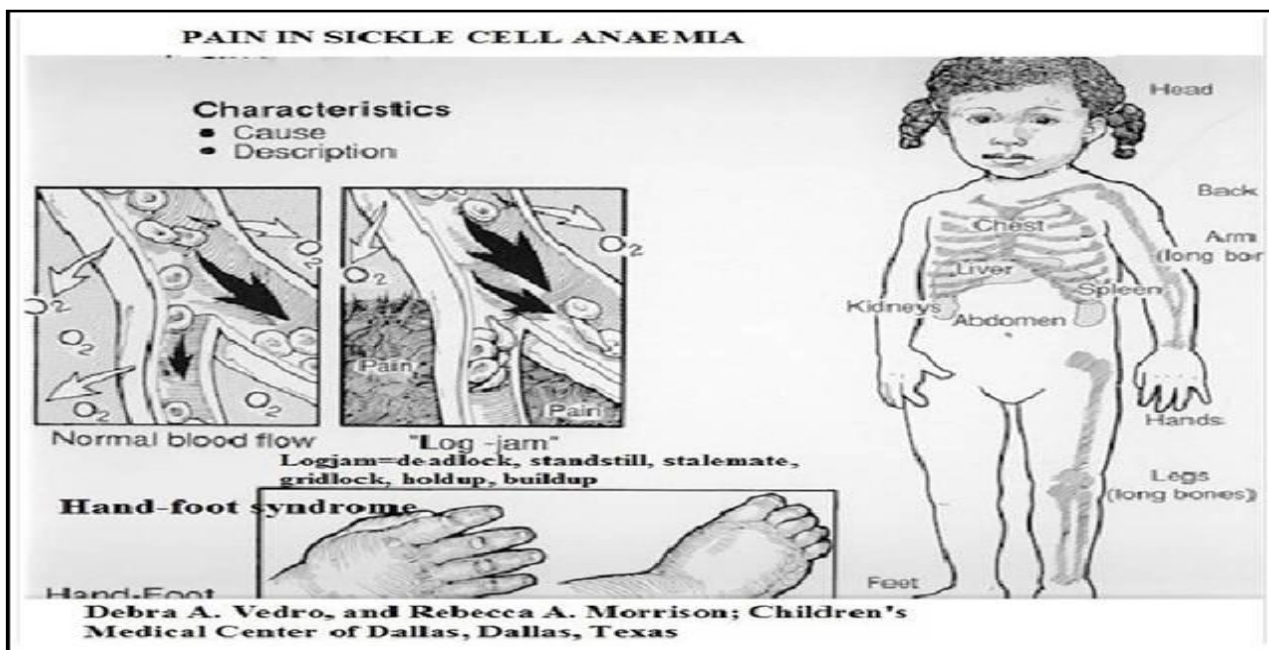


Fig v – pain in SCA

of cases present with vaso-occlusive crises then they develop ACS. Nevertheless, about 80% of patients have vaso-occlusive crises during ACS.

### **Aplastic crisis**

Aplastic crises are acute worsening of the patient's baseline anaemia, producing pallor, tachycardia, and fatigue. This crisis is normally triggered by parvovirus, which directly affects production of red blood cells by invading the red cell precursors and multiplying in and destroying them. Parvovirus infection nearly completely prevents red blood cell production for two to three days. In normal individuals, this is of little consequence, but the shortened red cell life of SCA patients results in an abrupt, life-threatening situation. Reticulocyte counts drop dramatically during the disease (causing reticulocytopenia), and the rapid turnover of red cells leads to the drop in haemoglobin. This crisis takes 4 days to one week to disappear. Most patients can be managed supportively; some need blood transfusion.

### **Haemolytic crisis**

Haemolytic crises are acute accelerated drops in haemoglobin level. The red blood cells break down at a faster rate. This is particularly common in patients with coexistent G6PD deficiency.

### **Dactylitis**

One of the earliest clinical manifestations is dactylitis, presenting as early as six months of age, and may occur in children with sickle-cell trait. .

### **DIAGNOSIS** ([http://en.wikipedia.org/wiki/Sickle-cell\\_disease](http://en.wikipedia.org/wiki/Sickle-cell_disease))

In HbSS, the complete blood count reveals haemoglobin levels in the range of 6–8 g/dl with a high reticulocyte count (as the bone marrow compensates for the destruction of sickled cells by producing more red blood cells).

Abnormal haemoglobin forms can be detected on **haemoglobin electrophoresis**

An acute sickle-cell crisis is often precipitated by infection. Therefore, a urinalysis to detect an occult urinary tract infection, and chest X-ray to look for occult pneumonia, should be routinely performed.

A test to see if an unborn child has the disease takes either a blood sample from the foetus or a sample of amniotic fluid. Since taking a blood sample from a foetus has greater risks, the latter test is usually used.

Neonatal screening provides not only a method of early detection for individuals with sickle-cell disease, but also allows for identification of the groups of people that carry the sickle cell trait.

## **2.7 TREATMENT OF SICKLE CELL ANAEMIA**

(<http://www.nhlbi.nih.gov/health/health-topics/topics/sca>)

Sickle cell anaemia has no widely available cure. However, treatments can help relieve symptoms and treat complications.

The goals of treating sickle cell anemia are to relieve pain; prevent infections, organ damage, and strokes; and control complications (if they occur).

Blood and marrow stem cell transplants may offer a cure for a small number of people who have sickle cell anaemia.

### **Treating Pain**

Mild pain often is treated at home with over-the-counter pain medicines, heating pads, rest, and plenty of fluids. More severe pain may need to be treated in a day clinic, emergency room, or hospital.

The usual treatments for acute (rapid-onset) pain are fluids, medicines, and oxygen therapy (if the oxygen level is low). Fluids help prevent dehydration, a condition in which the body doesn't have enough fluids. Fluids are given either by mouth or through a vein. The doctor may prescribe antibiotics if the patient has an infection.

Treatment for mild-to-moderate pain usually begins with acetaminophen (Tylenol<sup>®</sup>) or non-steroidal anti-inflammatory drugs (NSAIDs), such as ibuprofen.

### **Hydroxyurea**

Severe sickle cell anaemia can be treated with a medicine called hydroxyurea. This medicine prompts the body to make fetal hemoglobin. Fetal hemoglobin, or hemoglobin F, is the type of hemoglobin that newborns have.

In people who have sickle cell anemia, fetal hemoglobin helps prevent red blood cells from sickling and improves anemia.

Taken daily by mouth, hydroxyurea reduces how often painful sickle cell crises and acute chest syndrome occur. Many people taking hydroxyurea also need fewer blood transfusions and have fewer hospital visits.

Hydroxyurea can reduce the number of white blood cells in your blood, which can raise your risk for infections.

## **2.8 Preventing of Complications**

Blood transfusions are commonly used to treat worsening anaemia and sickle cell complications. A sudden worsening of anaemia due to an infection or enlarged spleen is a common reason for a blood transfusion.

Some, but not all, people who have sickle cell anaemia need regular blood transfusions to prevent life-threatening problems, such as stroke, spleen problems, or acute chest syndrome.

### **Infections**

Infections can be a major complication of sickle cell anaemia throughout life, but especially during childhood. Often, infections can be prevented or treated.

To prevent infections in babies and young children, treatments include:

- Daily doses of antibiotics. Treatment may begin as early as 2 months of age and continue until the child is at least 5 years old.
- All routine vaccinations (including a yearly flu shot), plus the pneumococcal vaccine.

### **Eye Damage**

Sickle cell anaemia can damage the blood vessels in the eyes and the retinas. The retinas are the thin layers of tissue at the back of the eyes. Regular check-ups with an eye doctor who specializes in diseases of the retina can help detect eye damage.

### **Strokes**

Stroke prevention and treatment are now possible for children who have sickle cell anaemia. Starting at age 2, children who have sickle cell anemia should have routine ultrasound scans of the head. This is called trans cranial Doppler (TCD) ultrasound. These scans are used to check the speed of blood flow to the brain.

TCD scans allow doctors to find out which children are at high risk of stroke. Doctors can treat these children with routine blood transfusions to reduce the risk of stroke.

## **Treating Other Complications**

Acute chest syndrome is a severe and life-threatening complication of sickle cell anemia. If acute (sudden) failure of the liver and kidneys also occurs, it's called acute multiple organ failure.

Treatment for these complications usually occurs in a hospital and may include oxygen therapy, blood transfusions, antibiotics, pain medicine, and balancing body fluids.

Leg ulcers (sores) due to sickle cell anaemia can be very painful. Ulcers can be treated with cleansing solutions and medicated creams or ointments.

Skin grafts might be needed if the leg ulcers are on-going. Bed rest and keeping the legs raised to reduce swelling are helpful. If you have a lot of pain from leg ulcers, the doctor may recommend a strong pain medicine.

The doctor might recommend gallbladder surgery if the presence of gallstones leads to gallbladder disease.

Priapism (a painful erection in males) can be treated with fluids, medicines, or surgery.

## **2.9 Management of sickle cell anaemia**

### **Folic acid and penicillin**

Children born with sickle-cell disease will undergo close observation by the paediatrician and will require management by a haematologist to assure they remain healthy.

These patients will take a 1 mg dose of folic acid daily for life.

From birth to five years of age, they will also have to take penicillin daily due to the immature immune system that makes them more prone to early childhood illnesses.

### **Malaria chemoprophylaxis**

The protective effect of sickle-cell trait does not apply to people with sickle cell disease; in fact, they are more vulnerable to malaria, since the most common cause of painful crises in malarial countries is infection with malaria. It has therefore been

recommended that people with sickle-cell disease living in malarial countries should receive anti-malarial chemoprophylaxis for life.

### **Vaso-occlusive crisis**

Most people with sickle-cell disease have intensely painful episodes called vaso-occlusive crises. The frequency, severity, and duration of these crises, however, vary tremendously. Painful crises are treated symptomatically with analgesics; pain management requires opioid administration at regular intervals until the crisis has settled. For milder crises, a subgroup of patients manage on NSAIDs (such as diclofenac or naproxen). For more severe crises, most patients require inpatient management for intravenous opioids; patient-controlled analgesia (PCA) devices are commonly used in this setting. Diphenhydramine is also an effective agent that is frequently prescribed by doctors in order to help control any itching associated with the use of opioids.

### **Acute chest crisis**

Management is similar to vaso-occlusive crisis, with the addition of antibiotics (usually a quinolone or macrolide, since cell wall-deficient bacteria are thought to contribute to the syndrome), oxygen supplementation for hypoxia, and close observation. Should the pulmonary infiltrate worsen or the oxygen requirements increase, simple blood transfusion or exchange transfusion is indicated. The latter involves the exchange of a significant portion of the patient's red cell mass for normal red cells, which decreases the percent of haemoglobin S in the patient's blood. The patient with suspected acute chest syndrome should be admitted to the hospital with worsening A-a gradient an indication for ICU admission

### **Hydroxyurea**

The first approved drug for the causative treatment of sickle-cell anaemia, hydroxyurea, was shown to decrease the number and severity of attacks and shown to possibly increase survival time. This is achieved, in part, by reactivating fetal haemoglobin production in place of the haemoglobin S that causes sickle-cell anaemia.

### **Transfusion therapy**

Blood transfusions are often used in the management of sickle-cell disease in acute cases and to prevent complications by decreasing the number of red blood cells (RBC) that can sickle by adding normal red blood cells.

## **Bone marrow transplants**

Bone marrow transplants have proven to be effective in children. Bone marrow transplants are the only known cure for SCA. However, bone marrow transplants are difficult to obtain because of the specific HLA typing necessary. Ideally, a twin family member (syngeneic) or close relative (allogeneic) would donate the bone marrow necessary for transplantation

## **2.10 PROGNOSIS OF SICKLE CELL ANAEMIA**

About 90% of patients survive to age of 20 and close to 50% survive beyond the fifth decade.

## **2.11 PREVENTION OF SICKLE CELL ANAEMIA**

People who are at high risk of having a child with sickle cell anaemia and are planning to have children may want to consider genetic counselling. A counsellor can explain the risk (likelihood) of having a child who has the disease. He or she can help explain the choices that are available.

The best choice for couples with each having HbAS is to avoid marriage.

## **2.12 DEFINITION OF NURSING CARE PLAN**

This is a deliberate and systematic phase of the nursing process involving decision making and problem solving.

Nurses make use of collected data and diagnostic statement to formulate goals and design interventions and actions to manage, prevent, eliminate or reduce the patient's problems.

## **NURSING CARE PLAN**

Nursing process is an organized systematic method of giving individual care to patient which focuses on human responses to potential alteration of health

## **2.13 Objectives of nursing care plan**

To be able to carry out proper nursing supervision and monitoring of vital signs

To plan and combat unsanitary conditions

To identify and propose solutions that will be of help to the patient as concern his/her health

To monitor care and progress in order to plan future care

To provide nursing care

To advise patient on nutrition and fluid which is part of the therapy

## **2.14 The 14 basic need proposed by virginal Henderson**

1) The need to breathe normally

- 2) The need to eat and drink adequately
- 3) The need to eliminate body waste
- 4) The need to move and maintain desirable posture
- 5) The need to sleep and rest
- 6) The need to clothing, dress and undress
- 7) The need to maintain temperature and other vital signs within normal range
- 8) The need to keep body clean, well robed and protect skin
- 9) Avoid danger in their environment and avoid injury other
- 10) Communicate with others in expressing emotions
- 11) Worship according to one's faith
- 12) Play or participate in various form of recreation
- 13) Work in the sense that there is sense of accomplishment
- 14) Learn, discover or satisfy the anxiety that lead to normal development and health and use the available health facilities

### **2.15 Nurses responsibility in drug administration**

Here check for the 7 Rights

- 1) Right patient
- 2) Right drug
- 3) Right route
- 4) Right dose
- 5) Right documentation
- 6) Right administration
- 7) Right time

Discuss with the patient before administering g the drug, the route of administration and if the patient will feel any pain or not.



## CHAPTER THREE - PRESENTATION OF CASE

### 3.1 THE DEMOGRAPHICAL IDENTIFICATION OF THE CASE

Name – MN  
Age – 4 Years  
Sex – Female  
Blood group – O  
Nationality – Cameroonian  
Occupation – Child (pupil)  
Religious – Presbyterian  
Contact person – Mother  
Address – Mile 6 Mankon  
Bed Number – Bed 10(a)  
Ward – B – Ward (children ward)  
Date of admission – 28 / 09 / 16

**3.2 Condition on admission:** The patient came into B-ward at 9.00am on 29-09-2016 after having been consulted by the Doctor in the paediatric general consultation. The patient was accompanied by the mother. Although very weak she managed to walk in to the ward. Her complaints were taken and vital signs monitored. **The patient was a known sicklier.**

The patient came into the ward presenting with Fever, Running stomach, General body weakness and pain, Anorexia, Pallor and No vomiting. The vital signs were:

Temperature – 38°C, Body weight – 15kg, Pulse – 77b/m,  
Respiration – 18 cycles/minute

### 3.3 PROVISIONAL DIAGNOSIS BY MD

Known Sickle Cell Anaemia (SCA) / scabies

### MEDICAL DOCTORS PRESCRIPTION BEFORE LAB RESULTS

ORS 1 sachet in 1Litre of water  
1litre daily x 3 days

Zinc tabs 20mg daily x 10 days  
Ibuprofen syrup  
10mls bid x 5 days  
Folic Acid tabs 50mg  
1 daily x 30 days

### 3.4 CLERKING / ASSESSMENT BY NURSES (HISTORY TAKING AND EXAMINATION.

On arrival into the ward the patient looked weak physical examination the patient hand scabies, dry lips pale skin and sunken eyes. The patient has been sick for over one week with the following complains fever, running stomach, general body

#### PAST SURGICAL HISTORY

The patient has never been operated upon .

#### PAST MEDICAL HISTORY

The patient has been admitted several times before because of malaria and sickle cell crises

#### FAMILY HISTORY

In the patient's family there is no other case of SCA.

#### SOCIAL HISTORY

The patient is the second child to the parents, a class 2 pupil and she sleeps under a mosquito net.

#### PHYSICAL ASSESSMENT (VITAL SIGNS)

Temperature – 38°C  
Body weight – 15kg  
Pulse – 77b/m  
Respiration – 10cm

### 3.5 Table 1: LAB RESULTS FOR BLOOD

Parameters		Result	Ref. Range
WBC	H	32.3 x 10 <sup>3</sup> /w	
Lymph #		6.5 x 10 <sup>3</sup> /w	
Mid #	H	3.1 x 10 <sup>3</sup> /w	
Gran #	H	22.7 x 10 <sup>3</sup> /w	
Lymph %		20.1%	
Mid %		9.5%	
Gran %	H	70.4%	
HGB	L	8.0g/dl	
RBC	L	3.39 x 10 <sup>3</sup> /w	
MCV	L	25.1%	
MCH	L	74.1fL	
MCHC	L	23.5pg	
RDW-CV	H	25.1%	
RDW-SD	H	59.0fL	
PCT		2.85 x 10 <sup>3</sup> /w	
MPV		8.3fL	

Parameters		Result	Ref. Range
PDW		14.0	
PCT		0.236%	

Table 2: LAB RESULTS FOR STOOL

Type	Test	Result	Range	Remark
Whole blood	MP (microscopy)	Positive	-	-
Stool	Stool examination	- Macroscopy: Colour: Brownish - Macroscopy: consistency: mucoid, watery - Macroscopy: Protozoans seen: No Protozoan see - Macroscopy: cells and crystals, yeast cell +, red blood cell ++ leucocytes +, charcot leyden crystals + (CLC)	- - - -	-
		Microscopy: Helminths: NO Helminth seen		
		M.P – positive Hb 8.g/dc		
	Stool exam	Yeast cell + , Red blood cell ++ CLC +, Mucoi'd		

### 3.6 Doctor's diagnosis after results

Malaria / enteritis (yeast cells, amoebiasis) / SCA

### 3.7 MEDICAL PRESCRIPTION AND TREATMENT BY DR. AFTER RESULTS

Start malaria protocol with

1m Arthemeter (paluter) 30MG 12hourly x 3days

iv ceftriaxone 1.5mg daily 10 days

Drink ORS freely

1 ibumol syrup 10mls bd x 6 days 30days

After rounds the medications where modified

Continue malaria protocol with Arthemeter.

Ceftriaxone inj 700mg / 24hrl

Gentamycin inj 50mg / 24hrl

Folic acid 1 tab / daily

Drink much water

After the 6 doses of Arthemeter taken in 5 days the child was relayed on orals.  
 Combiat tab 20 / 120mg  
 2 – 0 – 2 / day for 3 days

**3.8 Table 3: DAILY DRUG CHART**

Date	Time	Drug	Dose	Route	Frequency	Remarks
28-09-16	Morning	ORS	1.5ml	PO	Daily	Served
		Zinc	20mg	PO	Daily	Served
		Ibumol syrup	10mg	PO	bd	Served
		Folic acid	50mg	PO	Daily	Served
		Ibumol syrup	10mg	PO	bd	Served
		Zinc	20mg	PO	Daily	Served
	Evening	Ibumol syrup	10mg	PO	bd	Served
29-09-16	Morning 6am	Arthemeter	0.3cc	1m	bd	Served
		ORS	1.5ml	PO	Freely	Served
		Ibumol syrup	10mg	PO	bd	Served
	Afternoon 2pm	Ceftriazone	1.5mg	Lv	Daily	Served
	Evening 6pm	Arthemeter	0.3cc	1m	bd	Served
		Ibumol syrup	10mg	PO	bd	Served
30-09-16	Morning 6am	Arthemeter	0.3cc	1m	bd	Served
		Zinc	20mg	PO	Daily	Served
		Ibumol syrup	10mg	PO	bd	Served
		Folic acid	50mg	PO	Daily	Served
		ORS	1.5ml	PO	Freely	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served
		Paramol	300mg	Iv	If fever	Served
	Evening	Arthemeter	0.3cc	1m	bd	Served
		Ibumol syrup	10mg	PO	bd	Served
01-10-16	Morning	Arthemeter	0.3cc	1m	bd	Served
		Zinc	20mg	PO	Daily	Served
		Ibumol syrup	10mg	PO	bd	Served
		Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served
	Evening	Arthemeter	0.3cc	1m	bd	Served
		Ibumol syrup	10mg	PO	bd	Served

Date	Time	Drug	Dose	Route	Frequency	Remarks
	Off	Arthemeter	0.3cc	1m	bd	Served
Date	Time	Drug	Dose	Route	Frequency	Served
02-10-16	Morning	Combiat tab	20/120mg (2tabs)	PO	bd	Served
		Zinc	20mg	PO	Daily	Served
		Ibumol syrup	10mg	PO	bd	Served
		Folic acid	50mg	PO	Daily	Served
		Ibumol syrup	10mg	PO	bd	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served
	Evening	Combiat	20/120mg (2tabs)	PO	bd	Served
Ibumol syrup		10mg	PO	bd	Served	
03-10-16	Morning	Combiat	20/120mg (2tabs)	PO	bd	Served
		Zinc	20mg	PO	Daily	Served
		Ibumol syrup	10mg	PO	bd	Served
		Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Lv	Daily	Served
	Evening	Combiat	20/120mg (2tabs)	PO	bd	Served
		Ibumol syrup	10mg	PO	bd	Served
04-10-16	Morning	Combiat	20/120mg (2tabs)	PO	bd	Served
		Zinc	20mg	PO	Daily	Served
		Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served
		Blood transfusion	300cc	Iv	-	Served
	Evening	Combiat	20/120mg (2tabs)	PO	bd	Served
05-10-16	Morning	Zinc	20mg	PO	Daily	Served
		Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served

Date	Time	Drug	Dose	Route	Frequency	Remarks
06-10-16	Morning	Zinc	20mg	PO	Daily	Served
		Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served
07-10-16	Morning	Zinc	20mg	PO	Daily	Served
		Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served
07-10-16	Morning	Zinc	20mg	PO	Daily	Served
		Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served
08-10-16	Morning	Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served
09-10-16	Morning	Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served
10-10-16	Morning	Folic acid	50mg	PO	Daily	Served
	Afternoon	Ceftriazone	1.5mg	Iv	Daily	Served
		Gentamycine	50mg	Iv	Daily	Served

**Table No 4: Nursing care plan 1: Date -28/09/2016** - Need: to eat and drink adequately

Nursing diagnosis: Risk for fluid volume deficit related to inadequate fluid intake

Objectives	Nursing intervention.	Rationale	Evaluation.
To rehydrate adequately so as to maintain fluid volume	Estimate the child's daily fluid requirements. Monitor the child's usual fluid consumption and make necessary adjustments. Encourage the child to take fluids. Observe for signs of dehydration	Optimizing fluid intake ensures that the child gets needed fluid Dehydration exacerbates crises	The child shows signs of adequate hydration within the first 24 hours
	Record intake and output	Recording enables you to monitor daily	

Objectives	Nursing intervention.	Rationale	Evaluation.
		fluid intake and spacing through out the day	

**Table No 5: Nursing care plan 2:** Date – 29 09 – 2016. Need: To move and maintain desirable posture

Nursing diagnosis: Discomfort related to pain due to inflamed limbs

Objectives	Nursing intervention.	Rationale	Evaluation.
Reduce pain in 24 hours	Distract patient with films songs	This will cause the pain receptors to be less stimulated	The patient stopped crying and was moving about within 24 hours
	Do massages	This will reduce cramps and the heat from the massage will cause the blood vessels to dilate	
	Administer analgesics, such ibumol	There will be a mark reduction of pains	

**Table 6: Nursing care plan 3** Date –30/09/2016 - Need: to stop peripheral tissue damage due anaemia

Nursing diagnosis: **risks of peripheral tissue ischaemia related to anaemia evidenced as** pallor of mucous membranes and swollen limbs

objective	Intervention	Rationale	Expected out come
To promote skin integrity and proper tissue perfusion to prevent hypoxia	* Instruct child to avoid physical exertion, emotional stress, low oxygen environments such overcrowding,	Decreased activity and exposure reduce body's need for oxygen	The child has no shortness of breath and show no sign of hypoxia
	Administer blood transfusions as ordered	Packed cells increase number of red blood cells available to carry oxygen to tissue cells. Transfusions prompt circulation	

**Table No 7: Nursing care plan 4:** Date: 01/10/2016; Need: to prevent infection  
 Nursing diagnosis: : Risk for infection related to chronic disease and splenic malfunction

Objective	Intervention	Rationale	Expected out come
To Prevent The child from developing infections	Ensure adequate nutrition by providing high-calories high-protein diet. Ensure that the child's immunizations are up to date. Report any signs of infection to physician immediately	This will reduce the rate or risk infection if any	The child is free of infection
	Isolate the child from possible sources of infection. Instruct parents about signs of infection and encourage them to seek prompt health care	Restriction of persons with infections agents prompt care for infection reduces the change of sickle-cell crisis	

**Table No 8: Nursing care plan 5:** date 02/10/2016; Need: to reduce anxiety  
 Nursing diagnosis: : anxiety related to knowledge deficit of the illness

Objective	Intervention	Rationale	Expected out come
Alleviate fear	Review basics of sickle-cell anaemia Teach the the family about signs and symptoms of crisis	Knowledge of disease helps ensure compliance with treatment regimen and adherence to preventive measures	Fear is reduced to a minimum level



**3.14 Table 9: DAILY EVALUATION OF THE PATIENT**

Date	Time	Observation	Signature
28-09-16	Morning	Ever, diarrhoea, general body weakness, joint pains anoixia	Served
29-09-16	Morning	Fever, loss of appetite	Served
30-09-16	Morning	Fever, body weakness	Served
01-10-16	Evening	Fever	Served
02-10-16	Evening	Pains	Served
03-10-16	Morning	Body weakness	Served
04-10-16	Morning	Fever, body weakness	Served
05-10-16	Morning	Cough	Served
06-10-16	Morning	Cough	Served
07-10-16	Morning	Cough	Served
08-10-16	Evening	No complain	Served
09-10-16	Evening	No complain	Served
10-10-16	Morning	No complain	Served

**3.15 Table 10: VITAL SIGN CHART**

Date	T°C	Body weigh	Pulse	Respiration	Bowel	Urine	Vomiting
28-09-16	38°C	15kg	77b/m	18c/m	4	3	
29-09-16	39.4°C	15kg	77	18c/m	4	4	
30-09-16	36.7°C	15kg	77	18c/m	3	5	
01-10-16	37.4°C	15kg	77	18c/m	-	2	
02-10-16	39°C	15kg	77	18c/m	2	3	
03-10-16	37.2°C	15kg	77	18c/m	1	4	
04-10-16	36.7°C	15kg	77	18c/m	1	6	
05-10-16	38.9°C	15kg	77	18c/m	3	2	
06-10-16	36°C	15kg	77	18c/m	2	3	
07-10-16	35.9°C	15kg	77	18c/m	2	3	
08-10-16	36.8°C	15kg	77	18c/m	1	4	
09-10-16	36.5°C	15kg	77	18c/m	2	5	
10-10-16	37°C	15kg	77	18c/m	-	2	

**3.16 EVOLUTION OF THE STATUS OF PATIENT**

**The patient got well and was discharged.**

## CHAPTER FOUR - REVIEW MEDICATION

The medications received by the patient were

- i. ORS
- ii. Zinc tabs
- iii. Ibumol syrup
- iv. Folic acid tabl
- v. Artemeter
- vi. Ceftriaxon
- vii. Gentamycin
- viii. Paramol
- ix. Combiat tab 20/120mg

### 4.1) ZINC

\* Group metal: It is called an essential trace element, because very small amounts are necessary for human health.

\* Mode of action: Zinc acid in the growth of sickle cell children. Zinc also inhibits the activities of plasma ribonuclease an enzyme present in SCA. Zinc supplimentents in patients with SCA results in a significant improvement in secondary sexual characteristics. The normal zatron of plasma ammonia concentrations and the reversal of abnor malities in dark adaptation.

Zinc is needed for the proper maintenance of the human body. It is found in several systems and biological reactions and it is needed for immune function, wound healing, blood clothing hydroid function.

\* Dosage 20mg daily

\* Route by mouth P.O

\* Side effect

Nausea, vomiting, diarrhea, metallic taste, kidney and stomach damage, it causes burns when use on broken skin, stinging, itches, tingling.

\* Caution

Zinc should not be inhale through the nose, as it may cause permanent lose of smell.

Diabetes: Large dose of zinc can lower blood sugar in people with diabetes.

Zinc is risky when consumed in high doses by pregnant and lactating women.

It should be use cautiously with those having allergy.

\* Drug reaction

- Antibiotics (Quinolone antibiotics thetiacyclines) react with zinc. It decrease how much is absorbed.

- Cisplatin (platinol AQ) interacts with zinc. This drug is used to treat cancer when combined with zinc it increases its effect.

- Contraindication

Allergies

#### 4.2) IBUMOL SYRUP

\* Group (NSAID) (Analgesic)

\* Mode of action

Ibuprofen and paracetamol are the active ingredients of ibumol used to provide pain relief in various conditions, inflammation and fever.

The combination of ibuprofen and paracetamol blocks the production of chemicals in the body that are responsible for pain, fever, swelling and inflammations.

\* Indication

It is indicated for the relief of headache, from musculo-skeletal origin, feverishness, muscular menstrual and dental pain.

\* Forms of the drug

This combination is available as tablets, capsules and as syrup

\* Caution

The combination of ibuprofen and paracetamol should be taken with a meal to minimize gastro-intestinal irritation.

- Prolonged use can cause liver and kidney failure, gastro-intestinal damage should be checked.

\* Contraindication

Asthma, stomach ulcer, bleeding disorders, if you are taking blood thinning medication allergic to aspirin or other medication, you are taking other medication.

Route by mouth

Side effects

G.I.T disorders, headache, dizziness, drowsiness, depression, swelling (feet / ankles) rash / itch, difficulty breathing, blood in vomit, dark tarry stool, yellow-tinged skin and eyes.

Drug reaction

Asthma medication, reduces the effect of asthma medication, risk of asthma attack.

Cardia glycosides digoxin risk that heart failure may be more severe.  
Cholestyramine. Reduce effect of paracetamol symptoms  
Ciclosporin. Potential ibuprofen toxicity.  
Methotrexate. Potential methohexate toxicity.  
Diuretics. Potential risk of kidney damage

Dosage 10ml  
(Ibuprofen + paracetamol Health 24)

#### 4.3. FOLIC ACID

\* Group vitamin ( )

\* M.O.A Mechanism of action

The average volume of folic acid levels in the serum are 7-36nmol/L, in the erythrocyte 320-1300nmol/L. Folic acid is needed for the proper development of the human body it is involved in producing the genetic material called D.N.A and in numerous other bodily functions.

\* Contraindication

Diverticular disease, ulcer from stomach acid, ulcerated colon, several blood transfusion problem with food passing via the esophagus, high amounts of ascorbic acid in urine.

\* Form of Drug

It can be seen as injections or tablets

\* Indications

Use to prevent and treat low blood levels of folate, as well as its complications including tired blood, anaemia, inability of the bowels to absorb nutrients properly ulcerative colitis liver disease, alcoholism and kidney dialysis cancer, heart disease, weak bones, liver disease pregnant women.

\* Side effects

Long term high doses of folic acid might cause abdominal cramps, diarrhoea, rash, sleep disturbance, irritability, confusion, nausea, stomach upset, behaviour changes, skin reactions, seizures, gas, excitability.

\* Caution

Using folic acid, vit b6, and B12 intravenously (by iv) or by mouth might worsen narrowed arteries. Folic acid should not be used by people recovering from this procedure (to widen narrowed arteries angioplasty)

\* Drug interaction

Fosphenytoin (Cenobion) interacts with folic acid methotrexate (MTX)  
Rheumatex) interacts with folic acid.

Phenobarbital (Luminal) interacts with folic acid

Primidone (Mysoline) interacts with folic acid

Pyrimethamine (Daraprim) interacts with folic acid

Dosing

By mouth 25-100mcg (50) per day.

To prevent neural tube defect at least 400mg (micrograms) per day taken by women.

Route IV, P.O

[www.eebn.com/vit-amin-supplements/...](http://www.eebn.com/vit-amin-supplements/...)

#### 4.4 ARTEMETER

Group antimalarial

MOA

Pharmacokinetic data in humans are sparse, with no data demonstrating the rate or extent of absorption or the systemic distribution of artemether. After parenteral administration, artemether is rapidly hydrolyzed to the active metabolite dihydroartemisinin.

\* Indication

Antimalarial drug: It is indicated for the treatment of all kinds of malaria, including the chloroquine resistant malaria and the first aid of fatal malaria.

\* Contraindication

Artemether is contraindicated in patients with hypersensitivity to artemether or other artemisinin compounds.

Side effects

Bradycardia, electrocardiogram abnormalities, gastrointestinal disturbances (nausea, abdominal pain) diarrhea, dizziness injection site pain skin reaction, fever.

\* Drug interaction

Studies and review in the literature demonstrated that the active substance of Artemether has no interaction with other drugs on decreasing therapeutic effect and increasing toxicity and side effect in human bodies.

Dose = 0.3cc

Route = Im

Forms of the drug = injections only

\* Caution

Allergy, history of heart disease or recent heart attack, heart rhythm disorder, liver or kidney disease low levels of potassium or magnesium.

[www.drugs.com/cdi/artemether](http://www.drugs.com/cdi/artemether)

#### 4.5. PARAMOL INJECTION: GENERIC NAME (ACETAMINAPHEN)

\* Group Analgesic

\* MOA

The pain killers in paramol work in two different ways. Paracetamol is thought to work by reducing the production of prostaglandins in the brain and spinal cord. The body produces prostaglandins in response to injury and certain diseases. One of their effects is to sensitize nerve endings causing pains (presumably to prevent us from causing further harm to the area). As paracetamol reduces the production of these nerve sensitizing prostaglandins it is thought it may increase our pain remains, we don't feel it as much.

Dihydrocodeine is a slightly stronger pain killer known as an opioid. Opioid pain killers work by mimicking the action of natural pain-reducing chemicals called endorphins that are produced in the brain and spinal cord. Codeine acts on the same opioid receptors as natural endorphins and this blocks the transmission of pain signals sent by the nerves to the brain. This means that even though the cause of the pain may remain, less pain is actually felt.

Paramol injection 1 tablet contains two active ingredients, paracetamol and dihydrocodeine.

\* Indication

Short-term up to three days treatment of acute moderate pain such as; headache, migraine, period pain, toothache, back pain, muscular and joint pains and nerve pain (neuralgia).

\* Side effects

Rash, itching, swelling especially of the face, tongue, throat, severe dizziness, trouble breathing.

\* Caution

Allergies, diabetes, phenylketonuria, pregnancy lactating. Do not use with any other drug containing acetaminophen.

\* Contraindication

Daily alcohol use, when combine with acetaminophen may be danger you liver.

\* Dose = 0.3cc

\* Route = IM and PO

\* Forms of the drug = Injectables and tablets, ephedrine

\* This drug reacts negatives with other drugs containing acetaminophen.

#### 4.6. COMBIAT TABS

Group antimalarials

#### MOA

This drug interferes with the genus plasmodium and destroys its.

\* Contraindication

History of arrhythmia, bradycardia and congestive heart failure accompanied by reduced left ventricular action.

#### Side effect

Headache, dizziness, loss of appetite, weakness, fever chills, hredness, muscle, joint pain, nausea, vomiting abdominal pain, cough, trouble sleeping, chest pain.

\* Caution

Allergy, electrolyte disturbance, hepatic impairment.

\* Dosage 20/120mg (2 table bid)

\* Route P.O

\* Indication severe malaria

Forms of the drug = Tablet

#### 4.7. CEFTRIATONE

Group cephalosporins

M.O.A

It inhibits the action of bacterial cell wall synthesis and growth of bacteria

Indication

Septicemia, pneumonia, meningitis

Dose

1g daily 2-4 daily in severe infection or 50mg/kg twice daily

Route im, iv

Side effect

Diarrhea, abdominal pains, mouth soreness, body rashes, hypersensitivity reaction.

Contra indication

History of hypersensitivity

#### 4.8. GENTAMYCINE

Group aminoglycoside

MOA

They are bacteriocidal active against some gram positive and gram negative organisms

Indication

Septicaemia, neonatal infection, meningitis central nervous system infection, pyelonephritis, prostatitis

Dose 2mg/kg daily given every 8 hours and should not exceed 7 days.

Side effect

Vestibular and auditory damage if given in prolonged or overdose.

Caution = Allergy

Route = IV

Forms of the drug = injectables



The patient did not manifest any of the above side effects.

#### 4.9. ORS (ORAL REHYDRATION SOLUTION)

##### MOA

It enhances the reabsorption of water and electrolyte

Contains an alkalinizing agent to counter acidosis

It also replaces electrolytes in case of deficit

It should be slightly hyposmolar

##### Indication

Given in fluid and electrolyte loss

Dosage should be given according to fluid loss usually 200-400mls of solution after every loss motion.

**Observation:** the patient did not react to any of these medications

## CHAPTER FIVE – DISCHARGE SUMMARY

Date of admission: 28 / 09 / 2016

Date of discharge: 10-10- 16

Diagnosis on admission (DOA): Sickle cell crises / scabies

Diagnosis on discharge (DOD): Malaria / sickle cell anaemia, and Enteritis due to yeast cells and amoebiasis

### TREATMENT RECEIVED IN THE WARD

ORS 1sacket in 1L water

1l daily x 3 days

Zinc tab 29mg

20mg daily x 10days

Ibumol syrup

10mls bd x 5days

Folic acid 50mg

1 daily x 30 days

Ceftriaxone 700m

Daily x 10 days

Centamycine 50mg

Daily x 10 days

RESPONDS TO TREATMENT – Good  
CONDITION ON DISCHARGE- Better

### 5.6 HOME TREATMENT

Fixim syrup 100mg / 5m

5ml 2times / day for 1 week

Folic acid 1 tab / daily

Drink much water

Ascaribial solution

Apply in the evening for 3 days

Cyteal (atron)

Use to wash the body

#### 5.7 ADVICE ON DISCHARGE

The child should continue sleeping under the mosquito net, eat balance diet, much fruits and vegetable, make sure the child's medication are given and the environment should be kept clean.

5.8 APPOINTMENT DATE = 27 – 10 – 2016

5.9 FOLLOW UP (APPOINTMENT OR BY TELEPHONE) IF APPOINTMENT IS NOT RESPECTED.

The appointment was respected and the doctor confirm the child was okay

## CHAPTER SIX - CONCLUSION

### 6.1 POSITIVE FINDINGS

The child always took her medications

### 6.2 DIFFICULTIES ENCOUNTERED

Removal of cannula of infusion because of playing

### 6.3 PROPOSED SOLUTION

Parents were advised to control the children

### 6.4 RECOMMENDATION

I will like to suggest that youths and the entire community should be educated on the outcome and suffering involve in having a sickle cell child. They should be counselled do sickle cell test and those with AS should be advised no to get married.

### 6.5 CONCLUSION

**We recommend premarital counselling for the screening of sickle cell traits – AS. Couples who both have sickle cell traits AS should be advised not to get married**

## REFERENCE

[http://www.healthsystem.virginia.edu/uvahealth/peds\\_hrpregnant/sickcell.cfm](http://www.healthsystem.virginia.edu/uvahealth/peds_hrpregnant/sickcell.cfm)

Hockenberry, M. and Wilson, D. (2007). *Wong's Nursing Care of Infants and Children (8th ed.)*. St Louis: Mosby.

Jakubik, L. D., Cockerham, J., Altmann, A., & Grossman, M. B. (2003). The ABCs of

pediatric laboratory interpretation: Understanding the CBC with differential and LFTs. *Pediatric Nursing*, 29(2), 97-103.

Jakubik, L. D. & Thompson, M. (2000). Care of the child with sickle cell disease: Acute complications. *Pediatric Nursing*, 26(4), 373-379.

Rausch, M. & Pollard, D. (1998). Management of the patient with sickle cell disease.

*Journal of Intravenous Nursing*, 21(1), 27-39.

Freie HMP. Sickle cell disease and hormonal contraception. *Acta Obstet Gynecol Scand* 1983;62:211-7.

3. Diav-Citrin O, Hunnisett L, Sher GD, et al. Hydroxyurea use during pregnancy: a case report in sickle cell disease and review of the literature.

*Am J Hematol* 1999;60:148-50.

<http://www.mayoclinic.org/diseases-conditions/sickle-cell-anemia/basics/definition/con-20019348>

National Heart, Lung, and Blood Institute. The management of sickle cell disease. Fourth edition; 2002.

Ware RE, Helms RW. Stroke with transfusions changing to hydroxyurea (SWiTCH). In:

Clinicaltrials.gov: St. Jude Children's Research Hospital; 2010.

**<http://www.nhlbi.nih.gov/health/health-topics/topics/sca>[http://en.wikipedia.org/wiki/Sickle-cell\\_disease](http://en.wikipedia.org/wiki/Sickle-cell_disease)**

World Health Organization, Sickle-cell anaemia, Report by the Secretariat, Fifty-Ninth World Health Assembly A59/9; A59\_9-en

M. A. Bender, Sickle Cell Disease, Critical Elements of Care, The Center for Children with Special Needs, Seattle Children's Hospital, Seattle, WA

Louise D. Jakubik, Nursing Care of the Child with Sickle Cell Disease: Acute Complications, Nursing of Children Network Conference 2010

©2008-2010 Nurse Builders. Concurrent\_Session\_IIIB