Sickle Cell Anemia

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Abstract **- This paper reviews Sickle cell anemia. Sickle cell anemia is a homozygous form of HbS (HbSS).from one place instead of glutamine with valine in place 6 of the ye- globin chain.the cells also lead to polymerization and vaso-occlussion in the vasculature. Type β - globin is found inshort arm of chromosome 11. The combination of two tununts ant-globin subunits form hemoglobin S (HbS). Less -oxygen conditions, the absence of polar amino acids in the sixth ye-globin chain promotes diversityhemoglobin polymerization, which reverses red blood cells and then cuts and reduces their stiffness.**

INTRODUCTION

Sickle cell disease (SCD) is one of the hospitals portem haemoglobinopathies Visible with haemolytic anemia, an additional tendency to infectionand vaso-occlusion that occurs in almost all vascular beds leading to ischemic tissue damage by physical inactivity and premature death. The outcome is difficult to predict, and few effective treatment options are available. The spread of SC a grid of people from Suriname, Netherlands Antilles and African countries.1 Recent survey (with a30% response) involving more than 400 Dutch hospitals departments where patients with hemoglobinopathies

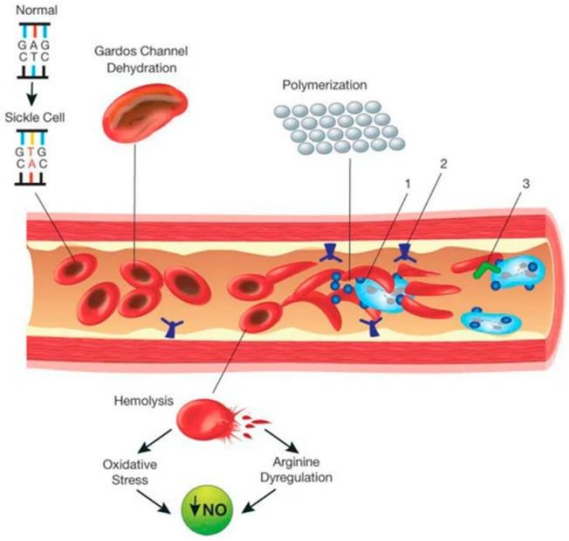
HISTORY SICKLE CELL

This collection of clinical findings was unknown until the scissors were described in 1904 by Chicago's cardiologist and medical professor James B. Herrick (1861 - 1954) named Ernest Edward Irons (1877 - 1959) who discovered a unique and long scissors - cells formed "in the blood of Walter Clement Noel, a 20-year-old Canadian dentist after Noel admitted the Chicago Presbyterian Hospital in December 1904 with anemia. graduated and returned to the capital of Grenada for dentistry, died of pneumonia in 1906 and was buried in Catholic cemeteries as Sauteurs in northern Grenada.

PATHOPHYSIOLOGY

Loss of red cell elasticity is central to the pathophysiology of sickle cell disease. Normal red blood cells stretch and form a biconcave disk, allowing cells to stumble over capillaries. [45] In sickle cell disease, low oxygen uptake promotes red cell disease and repeated episodes of sickling damage to the cell membrane and reduces cell density. These cells fail to return to normal when normal oxygen reuptake is restored. As a result, these strong blood cells are less likely to become paralyzed as they pass through smaller capillaries, leading to vessel blockage and ischemia. The real anemia--¬ of the disease is caused by haemolysis, the destruction of red cells, due to its condition. Although the bone marrow is trying to compensate for the formation of new red cells, it is not the same as the level of destruction Healthy red blood cells usually work for 90-120 days, but sic cells

DIAGRAM PATHOPHYSIOLOGY



TYPES OF SICKLE CELL DISEASE

There are several types of sickle cell disease:

Sickle Cell Anemia (SS): When a child inherits one beta globin (sickle cell gene) gene from each parent, the child has Sickle Cell Anemia (SS). People with high rates of sickle cell anemia are those from Africa and India.

Sickle Hemoglobin- C Disease (SC): People with Sickle Hemoglobin-C Disease (SC) have more and more different genes into their beta genes that produce both hemoglobin C and hemoglobin S. Sickle Hemoglobin-C disease can

Sickle Beta-Plus Thalassemia: People with Sickle Beta Thalassemia (SB) also contain genetically modified beta globin. The severity of the disease varies according to the amount of normal beta globin produced.

Sickle Hemoglobin-D Disease: By research, hemoglobin D, which replaces the gene beta globin, has been found to interact with the hemoglobin type of scissors.

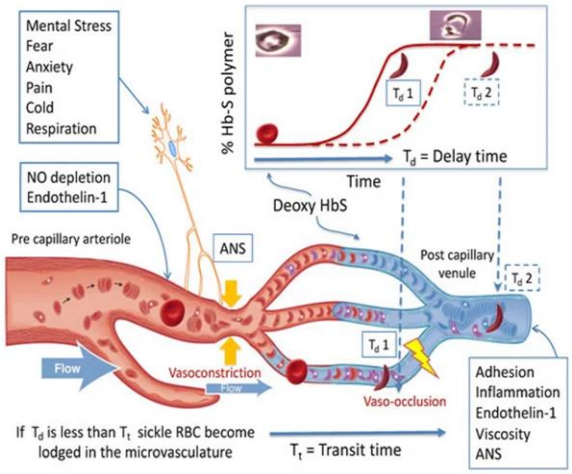
Sickle Hemoglobin-O Disease: Hemoglobin O, another substitute for beta globin, also interacts with hemoglobin. People with Sickle Hemoglobin- O disease (SO) may have symptoms of sickle cell anemia.

VASOOCCLUSION

Vasoocclusion (VOC) is thought to be the causative agent of painful complications, severe splenic shock, and shrinkage(painful construction and prolonged penile erection). Painful problems,disrupting the SCD symptom, is defined as severe painlasts 2 hours or more caused by SCD. The most affected sites include arms, legs, back,

stomach, chest and head. Pain problems do not include otherscauses / types of pain in SCD such as dactylitis, asthma syndrome, upper right quadrant syndrome, osteomyelitis,and appendicitis. It is a common cause of hospital-sation and generalized pain (defined as 2 or more episodes of pain three-year) is associated with a lower standard of living and an increased risk of death

DIAGRAM VASOOCCLUSION



MANAGEMENT OF SICKLE CELL DISEASE

As a chronic disease, the natural history of SCD is a disease replaced by expiration dates included by critical events, known as complications, which leads to patients seeking health care as well frequent hospitalizations. (ACS), acute splenic sequestration (ASS), based on indistinct symptoms including pain, fever, anemia, increased jaundice, and leg cramps ulcers. Other conditions include pregnancy, dehydration, and very cold weather. During the rising life span of people with SCD, there has been increased awareness on the importance of improvement and quality of life such as preventing damage to large organs. SCD is associated with increasing death. Causes of deaths in the USA, UK, and Jamaica include diseases, ACS, ASS and aplastic problems manage critical events, and reduce the last organ damage.

CONCLUSION

SCD, in its various forms, is a highly debilitating disease affecting more and more people around the world. Endless haemolytic anemia and vaso-occlusion in almost all organs lead significant illness and premature death Important issue on treating patients with stem cells is an early identification of high-risk studies of adverse outcomes, to begin with treatment before the development of a degenerative organ damage. This is very important given power adverse medical effects such as hydroxyurea, chronic red cell transplants and bone marrow transplants.

REFERENCES

[1] J. K. Onwubalili, "Sickle cell anaemia and reincarnation beliefs in Nigeria," The Lancet, vol. 2, no. 8364, p. 1423, 1983.
[2] E. Nzewi, "Malevolent ogbanje: recurrent reincarnation or sickle cell disease?" Social Science and Medicine, vol. 52, no. 9, pp. 1403–1416, 2001.
[3] Konothy Ahulu FD; Effect of Environment on Sickle Cell Disease in West Africa; Epidemiological and Clinical Considerations, 2004.C.V. Mosby Co, St. Louis.
[4] Desai DV, Hiren D; Sickle Cell Disease. History and Origin. The Internet Journal of Hematology, 2004; 1(2).
[5] Hoflman R, Benz JE, Shattil SJ, Furie B, Cohen HJ, Silberstein LE; Methaemoglobinemia in Haematology Basic Principle and Practice, 2nd Ed. U.S.A: Churchill Livingstone Inc, 1995.