*Available online at* www.ijpab.com

ISSN: 2320 – 7051
*Int. J. Pure App. Biosci.* **3 (1):** 167-170 (2015)

*Review Article*

INTERNATIONAL JOURNAL OF PURE & APPLIED BIOSCIENCE

# A Comprehensive Review on Some Medically Important Infections of Human

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## ABSTRACT

*Sickle cell anemia is an inherited form of anemia-a condition in which there aren't enough healthy red blood cells to carry adequate oxygen throughout our body. Normally, the red blood cells are flexible and round, moving easily through the blood vessels. In sickle cell anemia, 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. Thalassemia is a form of inherited autosomal recessive blood disorders characterized by abnormal formation of hemoglobin. Thalassemia is caused by variant or missing genes that affect how the body makes hemoglobin, the protein in red blood cells that carries oxygen. There has been a rise in concern for this genetic disorder of blood especially in children and many awareness campaigns and initiatives in greater interest of public health are undertaken in India in association with various hospitals and NGOs.*

*Malaria is an infectious vector-borne disease spread by mosquito bite to humans and other animals caused by Plasmodium group of protozoan parasites. The disease is widespread in tropical and subtropical regions including Sub-Saharan Africa, Asia, and Latin America. Malaria causes symptoms that typically include fever, headaches, fatigue and vomiting. Dengue fever, also called breakbone fever (associated muscle and joint pains), is caused by the dengue virus and is a mosquito-borne tropical disease. Symptoms in infected humans are similar to measles which include fever, headache, muscle and joint pains, and a characteristic skin rash. Sometimes, it develops into grievous dengue hemorrhagic fever which is a life-threatening disorder. Diabetes mellitus is caused in pet animals due to inadequate production of insulin by the islet beta cells in the pancreas. Glucose in the urine causes the diabetic animal to excrete large volumes of urine. In turn, this creates dehydration and the urge to drink large amounts of water.*

***Keywords:*** *Dengue fever, Diabetes, Malaria, Sickle cell anemia, Thalassemia.*

## INTRODUCTION

Sickle cell anemia is caused by a mutation in the gene that tells the body to make hemoglobin — the red, iron-rich compound that gives blood its red color. Hemoglobin allows red blood cells to carry oxygen from the lungs to all parts of our body. In sickle cell anemia, the abnormal hemoglobin causes red blood cells to become rigid, sticky and misshapen.

The sickle cell gene is passed from generation to generation in a pattern of inheritance called autosomal recessive inheritance. If only one parent passes the sickle cell gene to the child, that child will have the sickle cell trait. With one normal hemoglobin gene and one defective form of the gene, people with the sickle cell trait make both normal hemoglobin and sickle cell hemoglobin[1].

Dengue can be acquired through a single mosquito bite both in human and in non-human primates. Several species of mosquito within the genus *Aedes*, principally *A. aegypti* are responsible for transmission of Dengue. They typically bite during the day, particularly in the early morning and in the evening and can transmit the infection throughout the year. Other *Aedes* species that transmit the disease include *A. albopictus*, *A. polynesiensis* and *A. scutellaris*[2].

Diabetes mellitus is caused due to inadequate production of insulin by the islet beta cells in the pancreas. Glucose in the urine causes the diabetic animal to excrete large volumes of urine. In turn, this creates dehydration and the urge to drink large amounts of water[3].

People with thalassemia make less hemoglobin and have fewer circulating red blood cells than normal, which results in mild or severe microcytic anemia. Thalassemia can cause significant complications, including iron overload, splenomagaly, bone deformities, and cardiovascular illness. Thalassemia may confer a degree of protection against malaria (specifically, malaria caused by the protozoan parasite *Plasmodium falciparum*. Various thalassemias resemble another genetic disorder affecting hemoglobin, sickle-cell disease[4].

In severe cases malaria can cause yellow skin, seizures, coma or death. These symptoms usually begin 10-15 days after being bitten. In those who have not been appropriately treated disease may recur months later. Most deaths are caused by *Plasmodium falciparum*. The disease is transmitted by the bite of an infected female *Anopheles* mosquito. This bite introduces the parasites from the mosquito's saliva into a person's blood. The parasites then travel to the liver where they mature and reproduce[5].

**Prevalence and epidemiology of Sickle cell anemia in India**

α-thalassemia is seen practically in every community of the country with varying frequency while sickle gene is confined mainly to dravidians and predravidians tribes inhabiting malaria endemic regions. It is also seen among certain caste groups from coastal areas of Odisha and Andhra Pradesh. Sickle hemoglobin was first detected by Lehman and Cutbush in 1952 among the tribals from Nilgiris[1].

**Implications of the genetic basis of infection**

Infection is a significant contributor to morbidity and mortality in sickle cell disease (SCD). The sickle gene confers an increased susceptibility to infection, especially to certain bacterial pathogens, and at the same time infection provokes a cascade of SCD-specific pathophysiological changes. Historically, infection is a major cause of mortality in SCD, particularly in children. Hemoglobinopathies including thalassemia with an estimated 10,000 live births each year and Sickle cell disease (SCD) with an estimated 5,200 live births each year are a major public health problem in India[1].

**Clinical diagnosis of dengue**

Virus isolation in cell cultures, nucleic acid detection by PCR, viral antigen detection (such as for NS1) or specific antibodies (serology) are the tests employed for diagnosis of dengue fever. Virus isolation and nucleic acid detection are more accurate than antigen detection[2].

**Metabolic complications associated with diabetes fever**

Initially, diabetic patients have incompetency to metabolize enough sugar and have an increase in appetite and a desire to consume more food. It is equally important to maintain a strict schedule for insulin injections[3].

**Cause of thalassemia**

Both α- and β-thalassemias are often inherited in an autosomal recessive manner. Cases of dominantly inherited α- and β-thalassemias have been reported. For the autosomal recessive forms of the disease, both parents must be carriers for a child to be affected. The risk is 25% for each pregnancy for an affected child when both parents carry a hemoglobinopathy trait[4].

**Incidence of the trait**

An estimated 60-80 million people in the world carry the β-thalassemia trait. Genetic counseling and genetic testing are recommended for families who carry a thalassemia trait. Countries such as Bangladesh, Nepal and Pakistan are seeing a large increase of thalassemia patients due to lack of genetic counseling and screening.

An estimated 1,000 people live with thalassemia major in the United States, and an unknown number of carriers. Because of the prevalence of the disease in countries with little knowledge of thalassemia, access to proper treatment and diagnosis can be difficult[4].

**Therapeutic correction**

Multiple blood transfusions can result in iron overload. Medications with deferoxamine, deferiprone, or deferasirox for iron overload related to thalassemia may be treated by chelation therapy. These treatments have resulted in improved life expectancy in those with thalassemia major. Bone marrow transplantation may offer the possibility of a cure in young people who have an HLA-matched donor. Success rates have been in the 80–90% range. Common side effects of medications include: nausea, vomiting and diarrhea. It however is not effective in everyone and is probably not suitable in those with significant cardiac issues related to iron overload[4].

**Pathophysiology of malaria**

Within the red blood cells, the asexual reproduction of the parasites occurs, periodically breaking out of their host cells to invade fresh red blood cells. The classical descriptions of waves of fever arise from simultaneous waves of merozoites escaping and infecting red blood cells. Some *P. vivax* sporozoites produce hypnozoites that remain dormant for periods ranging from several months (7–10 months is typical) to several years and do not immediately develop into exoerythrocytic-phase merozoites. After a period of dormancy, they reactivate and produce merozoites. Hypnozoites are responsible for long incubation and late relapses in *P. vivax* infections, although their existence in *P. ovale* is uncertain[5].

**Detection of the parasites**

PCR based detection of the parasite's DNA have been developed. Laboratory diagnosis of the infection includes microscopic examination of blood using blood films, or with antigen-based rapid diagnostic tests[5].

**Treatment recommended**

The recommended treatment for malaria is a combination of antimalarial medications that includes an artemisinin. Quinine along with doxycycline may be used. The risk of disease can be reduced by preventing mosquito bites by using mosquito nets and insect repellents, or with mosquito-control measures such as spraying insecticides and draining standing water[5].

The course of infection in dengue is divided into three phases: febrile, critical, and recovery. The characteristic symptoms of dengue are sudden-onset fever, headache (typically located behind the eyes), muscle and joint pains, and a rash. The febrile phase involves high fever, potentially over 40 °C (104 °F), and is associated with generalized pain and a headache; this usually lasts two to seven days. Nausea and vomiting may also occur. A rash and petichiae like red erythematous spots occurs in 50–80% of those with symptoms. The fever itself is classically biphasic or recurrent in nature[2].

## CONCLUSION

There is no cure for most people with sickle cell anemia. However, treatments can relieve pain and help prevent further problems associated with sickle cell anemia. The present review was constructed to portray the genetic alteration and involvement in the infection along with the incidence and prevalence of the disease among Indian population. There is no evidence from randomised controlled trial to support zinc supplementation in thalassemia. Mortality from the procedure is about 3%. There are no randomized controlled trials which have tested the safety and efficacy of non-identical donor bone marrow transplantation in persons with β- thalassemia who are dependent on blood transfusion. For most of its human life cycle the malarial parasites reside within the liver and blood cells and are protected from host immune defense. However, some of the parasites in the circulation are destroyed in the spleen. Cerebral malaria is defined as a severe *P. falciparum*-malaria presenting with neurological symptoms, including coma, or with a coma that lasts longer than 30 minutes after a seizure. For controlling the incidences of dengue fever the primary method of controlling *A. aegypti* is by eliminating its habitats by spraying organophosphate or pyrethroid insecticides or biological control agents in open stagnant water.

Dietary control and daily injections of insulin can regulate most diabetic dogs, allowing them to lead active, healthy lives. Oral hypoglycemic agents used for treating diabetes in people have not been effective in dogs, but research is continuing in this area.

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