INTRODUCTION

Leukemia is a malignant disease that originates in a cell in the marrow. It is characterized by the uncontrolled growth of developing marrow cells. There are two major classifications of leukemia: myelogenous or lymphocytic, which can each be acute or chronic ***(Cancer Treatment Centers of America, 2009).***

Acute lymphoblastic leukemia (ALL) is the most common malignancy diagnosed in children, representing nearly one third of all pediatric cancers. In acute lymphoblastic leukemia, a lymphoid progenitor cell becomes genetically altered and subsequently undergoes dysregulated proliferation, survival, and clonal expansion. Although a few cases are associated with inherited genetic syndromes, the cause remains largely unknown. ***(Ie viseur et al., 2008).***

Acute myeloid leukemia (AML) Consists of a group of malignant disorder, characterized by the replacement of normal bone marrow with abnormal primitive hematopoietic cells if untreated, the disorder uniformly results in death, usually from infection or bleeding ***(Bhatia and Neglia , 2006).***

Human hepcidin is a 25–amino acid peptide first identified in human urine and plasma. The hepatocytes are the main cellular source of hepcidin but other studies also detected hepcidin synthesis in bacteria-activated neutrophils and macrophages, at a lower level than in hepatocytes. ***(Park et al., 2001).***

Hepcidin inhibits the cellular efflux of iron by binding to, and inducing the degradation of, ferroportin, the sole iron exporter in iron-transporting cells. In turn, hepcidin synthesis is increased by iron loading and decreased by anemia and hypoxia. Additionally, hepcidin synthesis is greatly increased during inflammation, trapping iron in macrophages, decreasing plasma iron

concentrations and causing iron-restricted erythropoiesis characteristic of anemia of inflammation ***(Ganz, 2003).***

The central involvement of hepcidin in iron regulation and its pathologies should make the eventual hepcidin assay useful for the diagnosis of iron disorders and the monitoring of their treatments. The development of hepcidin agonists and antagonists may provide useful therapeutics for the treatment of iron disorders ***(Ganz et al., 2008).***

Hepcidin, the systemic regulator of iron homeostasis is activated by proteins responsible for hereditary hemochromatosis, bone morphogenetic proteins (BMPs), and inflammatory cytokines. Three recent publications now identify a novel hepcidin suppressor, the transmembrane serine protease TMPRSS6 (also known as matriptase-2), which is required to sense iron deficiency. ***(Muclcenthaler, 2008)***.

Anemia is a common complication in patients with hematologic malignancies, and is caused by a variety of mechanisms, including neoplastic cell infiltration into the bone marrow, hemolysis, nutritional deficiencies, and defects in erythropoiesis as a result of the disease itself or cytotoxic therapy ***(Littlewood and Mandelli, 2002)***.

Hepcidin concentrations are elevated in multiple cancer and leukemia patient populations raising the possibility that functional iron deficiency may play a role in the etiology of anemia in cancer patients ***(Arvedson et al., 2008)***.

The lack of references related to the clinical significance of hepcidin in hematological malignancy in children stimulates us to study its role in explanation of anemia in such cases in Egypt.