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EFFECT OF CHEMOTHERAPY ON SERUM AND URINARY SODIUM AND POTASSIUM IN ACUTE CHILDHOOD LEUKEMIA

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INTRODUCTION AND AIM OF THE WORK

AIM OF THE WORK

The aim of this study is to estimate sodium and potassium levels , both in serum and urine, in acute childhood leukemia during the course of chemotherapy, to detect and try to prevent inappropriate changes in their levels.

REVIEW OF LITERATURE

ACUTE LEUKEMIA

Definition :

Acute leukemia is a primary malignant disease of blood forming organs. In its typical evolution, there is a progressive infiltration and replacement of normal bone marrow and lymphatic tissues by immature pregenitors of lymphoid and myeloid leukocytes (Edward, 1980) .

As the normal cellular elements accumulate in the bone marrow, they produce qualitative and quantitative changes in virtually all haemopoietic cells.

Regardless of its cellular origin or histopathologic appearance, each form of acute leukemia is associated with some degree of aberrant and usually diminished production of normal erythrocytes, granulocytes, and platelets. These deficiencies rather than the proliferation of leukemic cells per se, leads to the most important complications of this disease, e.g. anaemia , infection and hemorrhage (Edward, 1980).

Historical Notes :

Acute leukemia was defined as a separate clinical entity in 1889 by Epstein. In later years, further characterization were made when polychromatic stains became widely used for the study of blood cells. During the last 75 years, many morphologic subcategories of acute leukemia have been described, and much study and debate have been expended in evaluating the importance of these distinctions.

Histochemical, electron microscopic, and immunologic technique have been used to study different varieties of acute leukemia, looking for guides to therapy(Edward, 1980).

Classification :

A current classification for childhood leukemia is shown in table I . Subtypes of acute lymphocytic leukemia vary considerably with respect to clinical features and response to therapy, their classification in table I is based on the characteristics of surface

Incidence :

The overall incidence per year in the United States and in most Western European countries averages 3.5 per 100,000 inhabitants . It is the most common malignant disease in childhood, except for ages 2 through 9, during which time for unexplained reasons a relatively high frequency of acute lymphocytic leukemia appears, the incidence of acute leukemia like most cancers , increases with age . The overall incidence in males as compared with that in females is 1.3 : 1, the discrepancy being chiefly due to a greater incidence of acute leukemia in young boys and in older men (Gulter , 1975).

Miller (1968) , stated that the incidence of acute leukemia increases in individuals exposed to excessive irradiation and certain chemicals. The incidence is high in children born with a variety of congenital anomalies, and in patients with aplastic anaemia paroxysmal nocturnal hemoglobinuria and myeloproliferative disorders.

Etiology :

Although the cause of human acute leukemia remains obscure, it is increasingly evident that no single factor is responsible. Many influences must operate to produce the disease. High dose irradiation, chronic benzene exposure, or Down's syndrome alone do not produce leukemia, although they increase its overall incidence substantially.

In most instances, leukemia appears to result from the concentration of host susceptibility factors, chemical or physical injury to chromosomes, and clearly in animals and presumably in humans as well, the incorporation of genetic information of viral origin into susceptible stem cells (Edward, 1980).

Hereditary factors :- An inherited susceptibility to leukemia is suggested by the increased incidence of the disease observed in certain high risk families and in association with specific hereditary syndromes, and the high frequency of concordant leukemia in monozygous twins. Approximately 20% of such individuals will manifest the

disease within one year of their twin's diagnosis (Miller, 1968). Hereditary or congenital disorders such as Down's syndrome, Bloom's syndrome, Fanconi's anaemia, ataxia telangiectasia, and congenital agammaglobulinemia are also prone to terminate in acute leukemia.

Aquired Hemotologic diseases :-

Some hematologic diseases aquired during life have a remarkable tendency to terminate as acute leukemia. This development sometimes appears to be a coincidence, or it may relate to treatment given for the underlying disease, or reflect an intrinsic predetermination for the disease. There is a strikingly high frequency of acute leukemia in the myeloproliferative diseases, chronic granulocytic leukemia (CGL), polycythemia vera, and myelofibrosis, and in sideroblastic anaemia. Acute leukemia has been less commonly noted in patients with chronic lymphocytic leukemia, multiple myeloma, Hodgkin's disease, and paroxysmal nocturnal hemoglobinuria (PNH). (Edward, 1980).

Irradiation :

The leukemogenic potential of ionizing radiation has been amply demonstrated in animals and in humans.

Exposure to Chemicals :

The occurrence of acute leukemia in workers exposed to benzene was reported in 1908 by (Le Naire and Glaude, 1966). Similar, but less convincing, reports, have implicated chloramphenicol and phenyl butazone as leukemogenic (Daugan et al., 1965 and Gohen et al., 1967).

Acute leukemia was found to occur in patients with carcinoma of the ovary (Greenspan et al., 1974), also lung, and breast after 16 to 91 months of alkylating agent therapy. (Garfield, 1970). About one-half of these patient's were not given radiotherapy.

Viruses :-

The viral cause of a number of leukemias in mammals has been clearly documented.

Edward (1980) stated that there is increasingly convincing evidence to show that some acute leukemias are

associated with transmissible (infectious) RNA virus ,
closely related to the leukemogenic viruses of lower
mammals .

Clinical features :-

Edward 1980 , stated that the multiplicity of signs
and symptoms which may be produced by acute leukemia are
out lined in the following table :

TABLE 2 : Clinical Features of acute leukemia related to pathophysiology

Clinical symptoms	Signs	Cause
Fatigue weakness	Pallor, lethargy, weakness	Marrow failure, release of cellular ions and metabolites.
Weight loss	Weight loss	Reduced food intake, anaemia, hepatosplenomegaly, increased metabolism.
Bleeding in skin, mucous membranes, gums, gastrointestinal, genitourinary tracts.	Purpura, gum oozing or hypertrophy, hematuria, melena.	Marrow failure, DIC ^x
Infection of skin, throat, gums, respiratory or urinary tracts.	Fever, chills, tissue infiltrates, pyoderma gangrenosum.	Marrow failure, granulocytopenia, immunodeficiency
Headache, nausea, vomiting blurred vision, cranial nerve dysfunction.	Papilloedema, cranial nerve palsy, meningeal irritation.	Meningeal, CNS, or nerve infiltration and/or compression.
Bone pain and tenderness	Increased bone tenderness	Local leukemic infiltration.
Abdominal fullness anorexia	Hepatosplenomegaly, abdominal tenderness.	Infiltration of abdominal viscera.
Enlarged lymph nodes or tumor masses.	Enlarged lymph nodes masses in node areas, skin breast, testes.	Local tumor growth or infiltration.
Oliguria	Oliguria	Dehydration uric acid nephropathy, DIC.
Obstipation	Abdominal fullness tenderness.	Local infiltration, obstruction calcium magnesium imbalance.

Disseminated intravascular coagulation.