شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

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بالرسالة صفحات لم ترد بالاصل

SERUM SOLUBLE INTERLEUKIN - 2 RECEPTOR (IL-2R) IN PATIENTS WITH LYMPHOMAS CORRELATION WITH CLINICAL DATA, HISTOLOGICAL SUBTYPES AND STAGE OF THE DISEASE

MD THESIS

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BY

SAMIA ABD EL-WAHAB MOHAMMED
MBBCH, MS (ALEX. UNIVERSITY)

MEDICAL RESEARCH INSTITUTE
UNIVERSITY OF ALEXANDRIA
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SUPERVISORS

Prof. Dr. Mohammed Ragab Hussein
Professor of Haematology
Medical Research Institute
Alexandria University.

Prof. Dr. Hanaa M. Koheil
Professor of Oncology
Medical Research Institute
Alexandria University.

Dr. Samia Farag El-Belbessy
Assistant Professor of Haematology
Medical Research Institute
Alexandria University.

Dr. Magda M. Sultan
Lecturer of Haematology
Medical Research Institute
Alexandria University.

Dr. Raifa Mohammed Eid
Lecturer of Haematology
Medical Research Institute
Alexandria University.

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LIST OF ABBREVIATIONS

APC	Antigen - Presenting cell
ATLL	Adult T-cell leukaemia lymphoma
B	Basophil
BL	Blast
BCDF	B - cell differentiation factor
BCGF	B - cell growth factor
BM	Bone marrow
CD	Cluster of differentiation
CLL	Chronic Lymphocytic Leukaemia
CS	Clinical staging
E	Eosinophil
EBV	Epstein - Barr virus
F	Female
GM-CSF	Granulocyte-monocyte colony stimulating factor
Hb	Haemoglobin
HD	Hodgkin's disease
HTLV	Human T-cell leukaemia virus
IFN	Interferon
IgD	Immunoglobulin - D
IgM	Immunoglobulin - M
IL-2	Interleukin - 2
IL-3	Interleukin - 3
IL-5	Interleukin - 5
IL-2R	Interleukin - 2 receptor
LD	Lymphocyte depletion
LP	Lymphocyte predominance

LIST OF ABBREVIATIONS (Continued)

Ly	Lymphocyte
M	Male
MHC	Major Histocompatibility complex
MC	Mixed cellularity
Meta	Metamyelocyte
Meg	Megakaryocyte
Mon	Monocyte
Myel	Myelocyte
NHL	Non-Hodgkin's lymphoma
NRB	Normoblast
NS	Nodular sclerosis
PC	Plasma cell
PLL	Prolymphocytic leukaemia
Pr	Promyelocyte
PP	Present and productive
RS	Reed Sternberg cell
Seg	Segmented
St	Staff
sIL-2R	Soluble Interleukin - 2 receptor
SLVL	Splenic lymphoma (villous lymphocytes)
Tdt	Terminal deoxynucleotidyl transferase
WBC	White blood cell count

CONTENTS

INTRODUCTION

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Lymphoma

Lymphomas are heterogeneous group of neoplastic disorders originating from the immune system. The cells constituting this system are widely distributed and possess broad functional heterogeneity, so, lymphomas may originate in virtually any organ and show disparate histologic features, clinical behaviours and prognoses.[1]

The major determinants of clinical patterns and prognosis are the cell type of origin and the pattern of growth within involved lymph nodes.[2]

There are two major subgroups of lymphomas:

Hodgkin's disease (HD) and,

Non-Hodgkin's lymphomas (NHD).[3]

Hodgkin's disease (HD)

HD is a neoplastic disorder originating in lymphoid tissue and defined by the presence of Reed-Sternberg cells.[4] This lymphoma can occur at any age, however the age distribution is bimodal for both sexes with a peak in the late twenties followed by a decline to age 40 or 45 and a subsequent gradual increase with advancing age.[5] As regards the sex, the disease shows higher incidence in men versus women.[4]

Etiology and Pathogenesis

The etiology of HD remains unknown. That, it is true malignancy now seems certain, although, controversy remains concerning the origin and behaviour of the malignant cell in the histopathologic lesion.[4]

The bimodal age distribution of the disease has postulated two etiologic processes. The first hypothesis suggests an infectious cause of the disease in young adults and other environmental causes for lymphomas in older age groups.[6]

There have been several reports of HD developing in association with serologically documented primary Epstein-Barr Virus (EBV) infection. Molecular genetic techniques have detected the viral DNA in up to 20% of cases, they have also demonstrated that this DNA is clonal, thus, confirming the association of the virus with some Hodgkin's cases.[7] Moreover, it has been found that these EBV genomes are present in Hodgkin's cells regardless of the subtype. Furthermore, increased risk of the disease has been demonstrated among woodworkers and after tonsillectomy. There is also a familial association of HD with certain HLA antigens.[8]

Cytogenetic analysis of tissues involved with HD usually revealed a mixture of normal and abnormal Karyotypes. No consistent Karyotypic abnormality has been found. A number of cases have shown hypotetraploid, additional chromatin added to long arm of chromosome 14 (14 q+) and chromosomal breaking points at 11q23, 14q32 and 8q22 in up to 30% of the patients.[9]