Advances in Oncology Research and Treatments

Endostatin is a broad spectrum angiogenesis inhibitor and may interfere with the proangiogenic action of growth factors.

Keywords: Acute myeloid leukemia; Endostatin; Prognosis; Angiogenesis

Introduction

Acute Myeloid Leukemia (AML) is an aggressive hematologic malignancy characterized by accumulation of immature malignant myeloid cells in the bone marrow and blood due to their clonal proliferation without substantial maturation [1].

Angiogenesis is the formation of new vessels from an existing network of vasculature [2]. Irrespective of cellular origin, induction of angiogenesis requires a shi /switch towards activation/upregulation of inducers of angiogenesis over suppression of angiogenic inhibitors (herea er AI). Some key angiogenic activators include vascular endothelial growth factor A herea er VEGF (VEGF-A) [3], Matrix Metalloproteinases (MMPs), Placenta Growth Factor (PlGF), Fibroblast Growth Factor (FGF) and Hepatocyte Growth Factor (HGF) [4]. Endogenous inhibitors of angiogenesis include rombospondins (THSBs) endostatin, angiostatin and cytokines such as interleukin-12 [5].

e crucial role of angiogenesis in the growth, persistence, and metastases of solid tumors has been indicated in many studies [6,7]. ere is mounting evidence that angiogenesis is also signi cant in leukemia [8].

Endostatin, C-terminal fragment of collagen XVIII, is one of the most potent and speci c inhibitors of angiogenesis. Endostatin, originally isolated from medium of hemangioendothelioma, is generated from collagen XVIII through cleavage of an Ala-His linkage. On the cellular level, endostatin was shown to inhibit endothelial cell proliferation and migration and to induce apoptosis of endothelial cells [9,10].

Higher levels of serum endostatin have been associated with poor prognosis in patients with non-small cell lung carcinoma [11], and Non Hodgkin Lymphoma [12]. e results are not parallel to those in acute leukemia in which a limited number of the studies [13].

e aim of the study is to evaluate the prognostic role of endostatin in AML patients.

Patients and Methods

is study was conducted in Medical Oncology and Clinical Pathology Departments, Faculty of Medicine, Zagazig University during the period between January 2013 and February 2014.

It comprised 60 patients (28 women and 32 men); they were classi ed into 2 groups, Group I: Included 30 apparently healthy adult subjects (15 males, 15 females) with a mean age 35.8 ± 13.5 years. ey matched well with patients in terms of age and sex. Group II: Included 30 adult patients with newly diagnosed de novo AML (17 males, 13 females) with a mean age 38 ± 16.2 years. Patients and controls were subjected to the following:

(1) Complete history taking and thorough clinical examination particularly for pallor, petechiae, bruising, gum swelling, lymph node swelling and spleenomegaly.

(2) Routine laboratory Investigations.

- Complete blood count (CBC): by automated cell counter “Advia

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120", together with examination of Leishman stained peripheral blood smears for di erential leucocytic count.

- Liver, kidney functions tests and Lactate dehydrogenase using automated analyzer "Dimension RxL Max".

(3) Bone marrow Aspiration for Patients group only: Bone marrow smears were stained by Leishman and peroxidase stains and prepared for Immunophenotyping by ow cytometry: using Bectron Dickenson FacsCalibar device to detect the following markers (MPO, CD13,

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