# COAGULATION DEFECTS IN ACUTE LEUKEMIA

Thesis By

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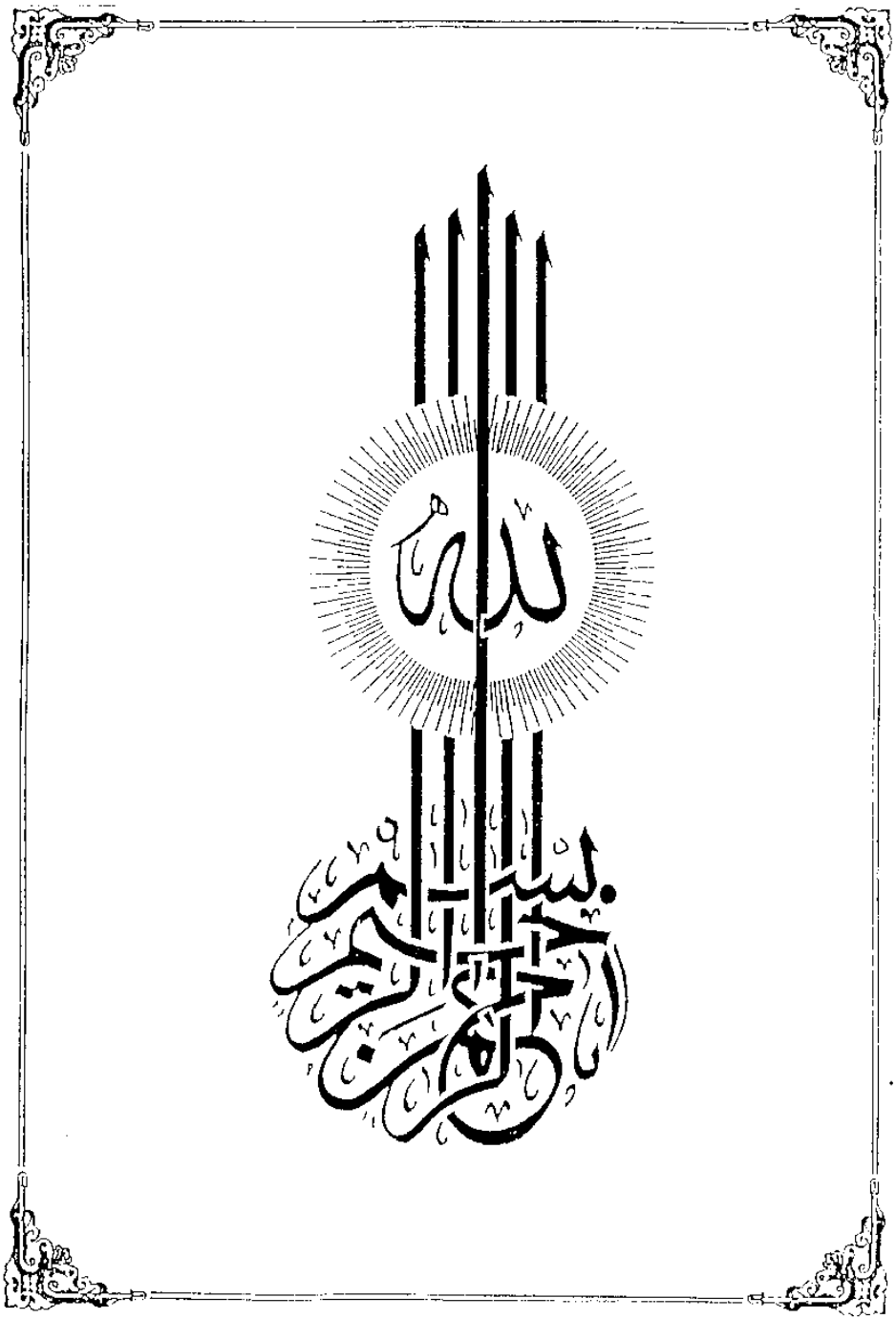

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## Abbreviations

**Diagnostic groups:**

C = Control

ALL = Acute lymphoblastic leukemia

ANLL = Acute nonlymphoblastic leukemia

APL = Acute promyelocytic leukemia

Suffixes:

B= before treatment

A= after treatment

M= in complete remission under maintenance therapy

R= in relapse

**Coagulation factors:**

FII = factor II

FV = factor V

FVIII:C= factor VIII:C

PC = protein C

ATIII = antithrombin III

FDP = fibrinogen degradation products

Fibr = fibrinogen

**Liver parameters:**

alb = albumin

GPT = glutamic pyruvate transaminase

BD = bilirubin direct

BI = bilirubin indirect

BT = bilirubin total

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# INTRODUCTION
# AND
# AIM OF THE WORK

## Introduction And Aim Of The Work

Haemorrhage is a common and often catastrophic complication of acute leukemia. In a great majority of patients, bleeding is due to thrombocytopenia resulting from bone marrow replacement by leukemic cells ( **Bick and Wilson, 1984** ). However, in some patients with acute leukemia severe coagulation defects may occur ( **Amer et al, 1988; Joseph and Hebert, 1969; Kreis et al, 1984** ). Khalifa and co-workers reported prolonged TT in ALL and prolonged PTT as well as TT in ANLL patients at diagnosis ( **Khalifa et al, 1988** ).

Defective or decreased synthesis of clotting factors in the prothrombin complex may be a common problem in acute leukemia related to leukemic cell infiltration in the liver. In addition, impaired synthesis of other, non-vitamin K dependant factors may occur ( **Lisiewicz, 1978; Bick, 1980** ).

The hepatocytes also synthesise proteins with anticoagulant action. The two most important are protein C and antithrombin III. The former is a vitamin K-dependant plasma glycoprotein, which is activated by thrombin and the endothelial cell cofactor thrombomodulin ( **Keisel, 1979; Esmon and Owen, 1981**). Its anticoagulant properties include a selective inactivation of factors $V_a$ and $VIII_a$ ( **Walker et al, 1979; Marler et al, 1981** ) as