

COAGULATION DEFECTS IN ACUTE LEUKEMIA

Thesis By

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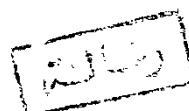
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Contents

* Introduction and Aim of The Work	1
* Review of Literature	
Mechanism of Hemostasis	4
A. Vascular Function	4
B. Platelets	7
C. Coagulation	11
+ The intrinsic coagulation cascade	14
+ The extrinsic coagulation cascade	25
+ The common pathway	27
+ The protein C-thrombomodulin mechanism	35
+ Formation of the fibrin clot	40
The Fibrinolytic Mechanism	46
Limiting Reaction	
+ Antithrombin III	58
+ Antiplasmin	63
+ α_2 -Macroglobulin	66
+ New Protease Inhibitors	68
Primary Fibrinolysis	69
Disseminated Intravascular Coagulation	
+ Pathophysiology	75
+ Laboratory abnormality	81
Acute Leukemia	
+ Etiology	92
+ Epidemiology	96
+ Classification	

-Morphologic classification	98
-Cytochemical classification	102
-Biochemical classification	104
-Immunological classification	107
+ Cytogenetics	111
+ Clinical Manifestation	114
+ Prognostic Factors	118
+ Bleeding Manifestation	120
* Material and Methods	127
* Results	155
* Discussion	215
* Summary	230
* References	232

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

List of Tables

Table 1: Components of The Hemostatic Mechanism	13
Table 2: Cytological features according to FAB classifica- tion of ALL	100
Table 3: Classification of ANLL	101
Table 4: Frequency of pretreatment hematological features in ALL patients	161
Table 5: Frequency of pretreatment hematological features in ANLL patients	162
Table 6: Epidemiological and laboratory findings in untreated ALL patients	163
Table 7: Epidemiological and laboratory findings in untreated ANLL patients	164
Table 8: Plasma factor II in acute leukemia patients expressed in percent of normal	165
Table 9: Plasma factor V in acute leukemia patients expressed in percent of normal	166
Table 10: Plasma factor VIII:C in acute leukemia patients expressed in percent of normal	167
Table 11: Plasma protein C in acute leukemia patients expressed in percent of normal	168
Table 12: Plasma AT III in acute leukemia patients expressed in percent of normal	169
Table 13: Serum FDP in acute leukemia patients expressed in ug/ml	170

Table 14: Plasma fibrinogen in acute leukemia patients	
expressed in mg/dl	171
Table 15: Serum albumin in acute leukemia patients	
expressed in g/dl	172
Table 16: SGPT in acute leukemia patients expressed in	
U/ml	173
Table 17: Direct serum bilirubin in acute leukemia patients	
expressed in mg/dl	174
Table 18: Indirect serum bilirubin in acute leukemia	
patients expressed in mg/dl	175
Table 19: Total serum bilirubin in acute leukemia patients	
expressed in mg/dl	176
Table 20: Coagulation factors and inhibitors in acute	
leukemia patients. Differences between groups . . .	177
Table 21: Liver parameters in acute leukemia patients.	
Differences between groups	178
Table 22: Coagulation factors in acute leukemia.	
Correlation study	179
Table 23: Liver parameters in acute leukemia patients.	
Correlation study	180

List of Figures

Figure 1: The consequence of Factor XII activation	15
Figure 2: The activation of Factor IX	20
Figure 3: Structural features of human factor VIII:C	22
Figure 4: γ -carboxyglutamic acid moities	28
Figure 5: The common pathway of thrombin generation	29
Figure 6: The activation of Factor V	30
Figure 7: Protein C-thrombomodulin mechanism	36
Figure 8: The structures of fibrinogen and the fibrin clot	42
Figure 9: The formation of the crosslinked fibrin clot	45
Figure 10: The structure of plasminogen and its mechanism of action	47
Figure 11: Degradation of fibrinogen	53
Figure 12: The mechanism of heparin action	62
Figure 13: The site of action of antithrombin and heparin within the coagulation cascade	64
Figure 14: Plasma factor II in acute leukemia patients	181
Figure 15: Plasma factor V in acute leukemia patients	182
Figure 16: Plasma factor VIII:C in acute leukemia patients	183
Figure 17: Plasma protein C in acute leukemia patients	184
Figure 18: Plasma antithrombin III in acute leukemia patients	185
Figure 19: Serum FDP in acute leukemia patients	186
Figure 20: Plasma fibrinogen in acute leukemia patients	187
Figure 21: serum albumin in acute leukemia patients	188
Figure 22: SGPT in acute leukemia patients	189
Figure 23: Direct serum bilirubin in acute leukemia patients	190

Figure 24: Indirect serum bilirubin in acute leukemia patients	191
Figure 25: Total serum bilirubin in acute leukemia patients	192
Figure 26: + Frequency distribution of F II in acute leukemia patients	
+ Frequency distribution of F V in acute leukemia patients	193
Figure 27: + Frequency distribution of F VIII:C in acute leukemia patients	
+ Frequency distribution of PC in acute leukemia patients	194
Figure 28: + Frequency distribution of AT III in acute leukemia patients	
+ Frequency distribution of FDP in acute leukemia patients	195
Figure 29: + Frequency distribution of fibr in acute leukemia patients	
+ Frequency distribution of alb in acute leukemia patients	196
Figure 30: + Frequency distribution of SGPT in acute leukemia patients	
+ Frequency distribution of DSB in acute leukemia patients	197
Figure 31: + Frequency distribution of ISB in acute leukemia patients	
+ Frequency distribution of TSB in acute leukemia patients	198

Figure 32: + Correlation between fibr and F II in ALL before treatment	
+ Correlation between F V and PC in ALL before treatment	199
Figure 33: + Correlation between F V and AT III in ALL (maintenece)	
+ Correlation between F V and fibr in ALL (maintenance)	200
Figure 34: + Correlation between PC and AT III in ALL (maintenance)	
+ Correlation between PC and fibr in ALL (maintenance)	201
Figure 35: + Correlation between AT III and fibr in ALL (maintenance)	
+ Correlation between PC and F II In ANLL before treatment	202
Figure 36: + Correlation between PC and F V in ANLL before treatment	
+ Correlation between PC and AT III in ANLL before treatment	203
Figure 37: + Correlation between fibr and FDP in ANLL before treatment	
+ correlation between FDP and bleeding in ANLL before treatment	204
Figure 38: + Correlation between F II and DSB in ALL before treatment	

+ Correlation between FDP and TSB in ALL before treatment	205
Figure 39: + Correlation between fibr and TSB in ALL before treatment	
+ Correlation between alb and SGPT in ALL before treatment	206
Figure 40: + Correlation between alb and SGPT in ALL before treatment	
+ Correlation between F II and alb in ALL (maintenance)	207
Figure 41: + Correlation between FDP and SGPT in ALL (maintenance)	
+ Correlation between AT III and alb in ANLL before treatment	208
Figure 42: + Platelet count versus bleeding tendency in ALL patients	
+ Platelet count versus bleeding tendency in ANLL patients	209
Figure 43: + Bleeding tendency versus cytological subtype in ALL patients	
+ Bleeding tendency versus cytological subtype in ANLL patients	210
Figure 44: + FDP versus bleeding tendency in ALL patients	
+ FDP versus bleeding tendency in ANLL patients .	211
Figure 45: + FDP versus cytological subtype in ALL patients	
+ FDP versus cytological subtype in ANLL patients	212

Figure 46: + Frequency distribution of platelets in bleeding and non-bleeding ALL patients	
+ Frequency distribution of PC in bleeding and non-bleeding ALL patients	213
Figure 47: + Frequency distribution of fibrinogen in bleeding and non-bleeding ALL patients	
+ Frequency distribution of FDP in bleeding and non-bleeding ALL patients	214

INTRODUCTION
AND
AIM OF THE WORK

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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