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**STUDY OF PLASMA ANTITHROMBIN III AND
PROTEIN C IN EGYPTIAN CHILDREN WITH
CHRONIC RENAL FAILURE**

THESIS



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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

﴿ قَالُوا سُبْحَانَكَ لَا عِلْمَ لَنَا
إِلَّا مَا عَلَّمْتَنَا إِنَّكَ أَنْتَ
الْحَكِيمُ الْحَكِيمُ ﴾

صَدَقَ اللَّهُ الْعَظِيمَ

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To ...

My Family

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	Page
Mechanisms of Thrombosis in Hemodialysis Circuits	43
Subjects and Methods	49
Results	68
Discussion	100
Summary and Conclusion	112
Recommendations	114
References	115
Arabic Summary	

LIST OF ABBREVIATIONS

13-HODE	13-hydroxy octadecadienoic acid
α_2 AP	α_2 antiplasmin
Ab	Antibody
ACT	Activated clotting time
ADP	Adenine diphosphate
APS	Anti-phospholipid antibody syndrome
ARF	Acute renal failure
AT III	Anti-thrombin III
ATP	Adenosine triphosphate
AVF	Arteriovenous fistula
AVG	Arteriovenous graft
BTG	B-thromboglobulin
BUN	Blood urea nitrogen
Ca	Calcium
cAMP	Cyclic adenosine monophosphate
CGN	Chronic glomerulonephritis
CRF	Chronic renal failure
DIC	Disseminated intravascular coagulopathy
DVT	Deep venous thrombosis
EDRF	Endothelial-derived relaxing factor
ESRD	End-stage renal disease
FgDP	Fibrinogen degradation products
FnDP	Fibrin degradation products
FPA	Fibrinopeptide A
FPB	Fibrinopeptide B
HAT	Heparin associated thrombocytopenia
Hb	Hemoglobin
HD	Hemodialysis
HS	Heparan sulphate
IC Hge	Intracranial hemorrhage
IgG-ACA	Immunoglobulin anticardiolipin antibody

IX	Christmas factor
LMW	Low molecular weight
LPC	Lysophosphatidyl choline
NS	Nephrotic syndrome
PAF	Platelet-activating factor
PAI	Plasminogen activator inhibitor
PAP	Plasmin α_2 antiplasmin
PC	Protein C
PF1,2	Prothrombin fragments (1+2)
PGI ₂	Prostaglandin I ₂
PL	Phospholipid
PS	Protein S
PUV	Posterior urethral valve
r-HuEPO	Recombinant human erythropoietin
rt-PA	Recombinant tissue plasminogen activator
SLE	Systemic lupus erythematosus
TAT	Thrombin-antithrombin
TF	Tissue factor
TFPI	Tissue factor pathway inhibitor
t-PA	Tissue plasminogen activator
TXA ₂	Thromboxane A ₂
u-PA	Urokinase-like plasminogen activator
UTI	Urinary tract infection
V	Labile factor, proaccelerin, accelerator globulin
VII	Stable factor, proconvertin
VIII	Anti-hemophilic globulin (AHG), antihemophilic factor (AHF)
vWF	von Willebrand factor
WBACT	Whole blood activated clotting time
X	Stuart-Prower factor
XI	Plasma thromboplastin antecedent (PTA)
XII	Hageman factor

LIST OF TABLES

	Page
Table (1): Physiologic and pathologic platelet activators.	8
Table (2): Methods of assessing thrombogenicity in hemodialysis.	20
Table (3): Causes of hemodialysis access thrombosis.	38
Table (4): rt-PA in AVF thrombosis. Therapeutic protocol.	42
Table (5): The important clinical data of CRF patients under HD.	69
Table (6): Routine laboratory data of patients under HD.	71
Table (7): Results of AT III, PC, fibrinogen and D-dimer of HD patients pre-, post-dialysis and from a peripheral vein.	72
Table (8): Data of CRF patients on conservative treatment.	73
Table (9): Data of the control group.	74
Table (10): AT III, PC, fibrinogen and D-dimer in HD patients (predialysis) versus control group.	75
Table (11): AT III, PC, fibrinogen and D-dimer statistical values in HD group (post-dialysis) versus control group.	76
Table (12): AT III, PC, fibrinogen and D-dimer in peripheral venous samples of HD patients versus control group.	77
Table (13): AT III, PC, fibrinogen and D-dimer values of patients under conservative treatment versus those of controls.	78
Table (14): Comparison of the routine laboratory data of CRF patients under HD with those of patients under conservative treatment.	79

	Page
Table (15): Comparison of AT III, PC, fibrinogen and D-dimer in HD patients (pre-dialysis) versus those under conservative treatment.	80
Table (16): AT III, PC, fibrinogen and D-dimer in HD patients (post-dialysis) versus those under conservative treatment.	81
Table (17): AT III, PC, fibrinogen and D-dimer in peripheral venous samples of HD patients versus conservative group.	82
Table (18): Immediate effect of HD on AT III, PC, fibrinogen and D-dimer of HD patients (pre- versus post-dialysis).	84
Table (19): Comparison of the plasma levels of AT III, PC, fibrinogen and D-dimer of HD patients in pre-dialysis fistula samples with samples from peripheral veins.	85
Table (20): Effect of gender on some of the studied laboratory data of HD patients.	87
Table (21): Effect of the type of dialyzer filter on some laboratory parameters of HD patients.	88
Table (22): Effect of mode of heparinization of HD patients.	90
Table (23): Immediate effect of the mode of heparinization on the level of AT III and PC.	92
Table (24): Relation of thrombotic tendency to the laboratory data of patients under HD.	93
Table (25): Effect of thrombocytopenia on some laboratory data of patients under HD.	94
Table (26): Summary of the important correlations in the study.	96

LIST OF FIGURES

	Page
Fig. (1): Interplay of the coagulation, fibrinolysis and anticoagulation systems.	6
Fig. (2): Pathways to the formation of fibrin and major mechanisms of their localization.	10
Fig. (3): The initiation of coagulation by tissue factor.	11
Fig (4): Positive feed-backs of coagulation.	13
Fig. (5): Major inhibitors of coagulation.	14
Fig (6): Schematic diagram of the fibrinolytic system	18
Fig. (7): Factors influencing hemodialysis-related thrombogenicity and their interactions.	21
Fig. (8): Primary arteriovenous fistula.	37
Fig. (9): Cuffed double-lumen silastic catheter (Permcath).	37
Fig. (10): General scheme of the complex interaction between plasma proteins and blood cells after their exposure to dialysis membranes.	44
Fig. (11): Comparison of PC level between control group, HD group (pre-, post-dialysis and peripheral venous samples) and patients under conservative treatment.	83
Fig. (12): Results of AT III, PC, fibrinogen and D-dimer of HD patients pre-, post-dialysis and from peripheral veins.	86
Fig. (13): Effect of the type of dialyzer filter on laboratory parameters of HD patients.	89

	Page
Fig. (14): Effect of mode of heparinization of HD patients on AT III, PC, fibrinogen and D-dimer.	91
Fig. (15): Effect of thrombocytopenia on some laboratory data of patients under HD.	95
Fig. (16): Correlation of fibrinogen with hemoglobin in HD patients.	97
Fig. (17): Correlation of fibrinogen with AT III in HD patients.	97
Fig. (18): Correlation of fibrinogen with AT III in patients under conservative treatment.	98
Fig. (19): Correlation of PC and fibrinogen in patients under conservative treatment.	98
Fig. (20): Correlation between PC and D-dimer in patients under conservative treatment.	99

Introduction

INTRODUCTION

Patients undergoing hemodialysis (HD) are subjected to a special risk of thrombotic complications (*Lai et al.*, 1991).

There is a high incidence of cerebrovascular accidents leading to a 10 to 20% mortality rate in both adult and pediatric dialysis populations, together with recurrent thrombosis of the vascular accesses that carries the threat of pulmonary embolism (*Brenner and Rector*, 1991).

Latent activation of coagulation has been suggested as the cause of elevated thrombogenic risk among dialysis patients. Besides, certain types of filters are more thrombogenic than others (*Hakim and Schulman*, 1989 and *Schultze et al.*, 1992).

Antithrombin III (AT III) is a plasma glycoprotein synthesized by the liver and probably the vascular endothelial cells (*Bauer and Rosenberg*, 1995). It inhibits thrombin and factors IXa and Xa, its activity is promoted when it binds to heparan sulphate or to heparin (*Ward*, 1995a).

A decrease in plasma AT III during dialysis was reported by some investigators. However, other investigators failed to confirm this observation (*Muller*, 1992).

Protein C (PC) is an important naturally occurring anticoagulant and fibrinolytic agent, the deficiency of which is associated with a thrombotic diathesis (*Lai et al.*, 1991). Information regarding the effect of hemodialysis on AT III and PC is either controversial (as in AT III) or limited (as in PC).

The D-dimer is the terminal fragment in the degradation of fibrin (*Gaffney et al.*, 1980). The presence of D-dimer in plasma is an indirect marker of a coagulation activation followed by a reactive thrombolysis. In patients with chronic renal failure (CRF) without dialysis, fibrinogen and fibrin degradation products (FgDP, FnDP) were reported to be significantly increased, denoting an activation of fibrinolysis (*Opatrny*, 1997).