

Dedication

To My Mother (Nadia).

**To My sister and brother
(Kawther, Mohamed).**

**To everyone supported me until I
finished this work**

**Let me express deep thanks to all of
you.**

Acknowledgment

Primary my praise and thanks should be to Allah, the almighty most gracious and most merciful, who granted me the serenity, means of strength and practice to accomplish this work.

I am deeply indebted to my supervisor Dr: **Mansoor Mohammed Mansoor** for his valuable help and guidance during this study I am also great to his patience assistance and invaluable devices.

My appreciation is extend to all academic staff, technologist and other members of the department of haematology Sudan University of science and technology, and deep thanks to the staff of Khartoum teaching hospital.

Abstract

This study was hospital analytical case control, and it was conducted to evaluation of haemostatic mechanism among the Sudanese patient with renal diseases attending Khartoum teaching hospital throughout the period Between November 2009–march 2010.

60 patients were informed about the study, expected out come and agreement of participation was obtained, then the questionnaire was used to collect the information about patient's age, sex, blood groups as well as other diseases.

As well 5 ml of blood were taken from the patients 2.5 ml of which were taken in tri sodium citrate anticoagulant, and the remaining 2.5 were taken in EDTA anticoagulant and were used to evaluate the haemostatic mechanism (PT, PTT, Platelets counts).

As well computerized statistical package for social sciences version 11.5 was used to determine the samples size, and in subsequent data processing.

The result show mean of PT, APTT, Platelets count, and Bleeding time is 34.8, 39.2, 197, 6.1 Respectively.

There was significant for PT, APTT, Platelets count 0.000, 0.043, 0.004 Respectively

There was higher level significant variation in compared with control for PT, Bleeding time 0.006, 0.000 Respectively

Conclusion: it found that hemostatic mechanism was affected due to renal failure disease.

مستخلص الأطروحة

هذه دراسة وصفية تم إجراؤها لتحديد زمن البروثرومبين والثرومبوبلاستين و الصفائح الدموية و عامل التجلط فى المرضى المصابين بالفشل الكلوى الحاد و المزمّن الزائرون مستشفى الخرطوم . التعليمي فى الفترة ما بين نوفمبر 2009م الى مارس 2010م .

ستون مريض أعلموا حول الدراسة و تم الحصول على موافقة المشاركين . و تم إجراء الأستفتاء لهذه الدراسة .

تم أخذ خمسة مليلتر من المرضي من الدم 2.5 مليلتر أخذ فى مضاد و الباقي 2.5 مليلتر تم أخذه فى (Trisodium Citrate) التجلط التخثر وزمن PT وأستعمل لقياس. زمن البروثرومبين (EDTA) مضاد التجلط .(و تعداد الصفائح الدموية APTT الثرومبوبلاستين النشاط

تم استخدام برنامج الحزم الاحصائية للعلوم الاجتماعيه نسخه رقم 11.5 لتحديد حجم العينات و فى معالجة البيانات اللاحقة .

وزمن PT وجدت النتيجة بأنه كان هناك أطالة فى زمن البروثرومبين وزمن التجلط ونقصان فى تعداد APTT الثرومبوبلاستين النشاط الصفائح الدموية فى الفشل الكلوى الحاد فى مقارنة بالفشل الكلوى المزمن و كان هناك اختلاف هام مستوى أعلى مقارنة بالكنترول .لبروثرومبين و الثرومبوبلاستين و عامل التجلط و تعداد الصفائح الدموية

. وجد ان النتائج قد تأثرت نتيجة الفشل الكلوى الحاد والمزمن

LIST OF TABLES

Page No.	Subject	Table No
30	Showed Hemostatic parameters of patient compared with that of control.	Table 3-1
31	PT,APTT,Plateletes count,Bleeding time of patient with acute and chronic renal failure.	Table 3-2
32	Coagulation profile according to gender(Male and Female).	Table 3-3

List of Content

Page No.	Subject
I	Dedication
II	Acknowledgement
III	Abstract English:
IV	Abstract Arabic
V	List of tables
VI	List of content
Chapter one-Introduction and literature review	
1	1.1: Kidneys function in Hemostasis
1	1.1.1:excretion of metabolic waste products, foreign chemicals, drugs.
2	1.1.2:Regulation of water and electrolyte balance
3	1.1.3;Regulation of arterial pressure
3	1.1.4:Regulation of acid –base balance
3	1.1.5:Regulation of erythrocyte production
3	1.1.6:Regulation of 1,25Dihydroxy vitamin D3production
3	1.1.7:Glucose synthesis
4	1.2:Renal failure
4	1.2.1:classification
5	1.2.2:Acute renal failure
5	1.2.3:Chronic renal failure
5	1.2.4:Acute on chronic renal failure
5	1.2.5:Symptoms
6	1.2.6:Symptoms of kidney failure
7	1.2.7:Causes of Acute renal failure
8	1.2.8:Causes of chronic kidney disease
8	1.3Stages of kidney failure
8	1.3.1Glomerular filtration rate
9	1.4Use of term uremia
9	1.5Normal hemostasis
9	1.5.1Definition
9	1.5.2Components of normal hemostatic
10	1.5.3:Blood vessels
10	1.5.4:Endothelial cell function
12	1.5.5:Platelets
13	1.5.6:Platelets function in hemostatic process
14	1.6:Primay hemostatic
14	1.6.1:Platelets aggregation
16	1.6.2:Blood coagulation
16	1.6.3:Secondery hemostatic

17	1.6.4:Classification of coagulation factor
17	1.6.5:Extrinsic pathway
18	1.6.6:intrinsic pathway
18	1.7:Prothrombin time
19	1.8:Activate partial thromboplastin time
19	1.9:Bleeding time
20	1.10;Plateltes dysfunction in renal failure
20	1.11:Human kidney anatomy
21	1.12:Kidney anatomy
21	1.13:Kidney anatomy and excretion
23	1.14.1:Objectives
23	1.14.2:specific objectives
23	1.15:Rationale
Chapter Two- MATERIAL AND METHODS	
25	2.1: Study design
25	2.2: Study area
25	2.3: Study population
25	2.4: Sampling
25	2.5: Inclusion criteria
25	2.6. Exclusion criteria
25	2.7: Sample size
25	2.8: Tool of data collection
26	2.9: Data analysis
26	2.10: Approval consent
26	2.11: Safety assurance
26	2.13: Methodology
26	2.14: Requirement
26	2.14.1: Procedure
26	2.14.2: Platelets count
27	2.14.3.:Prothrombin time
27	2.14.4:Activated Partial thromoboplastin time
Chapter Three	
29	3. RESULT
Chapter Four	
33	4. Discussion, Conclusion & Recommendations
33	4.1 Discussion
35	4.2Conclusion
36	4.3Recommendations
References	
Appendixes	
39	Appendix: 1 Questionnaire

