بسم الله الرحمن الرحيم

Sudan University of Sciences and Technology

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Evaluation of Haemostatic Mechanisms in Sudanese Patients With Gastrointestinal Bleeding

تقويم آلية التخثر لدى المرضى المصابين بأمراض النزف المعوي بمركز محمد صالح إدريس للنزف المعوي

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بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قال الله تعالى :

{يَرْفَعِ اللَّهُ الَّذِينَ آمَنُوا مِنكُمْ وَالَّذِينَ أُوتُوا الْعِلْمَ دَرَجَاتٍ}

صدق الله العظيم

سورة المجادلة الآية 11

Dedication

To source of my life

My father and My mother

To source of my interesting in life

My husband

To source of my happiness

My sisters and my brothers

To all patients suffering from gastrointestinal bleeding with my hopeness to get cure.

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First of all the thanks is for Allah

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lastly the great thanks is for my family to assistance, supports and thank care for this research.

Abstract

This study was carried out at Mohammed Salih Idres center for gastrointestinal bleeding during the period from January to feb 2010,

prothrombin time (PT), activated partial thromboplastin time (APTT) and platelet count were measured to 50 gastrointestinal bleeding patients, 43 were males (86%) and 7 females (14%) with mean age 47 years, compared with 30 healthy individual as control group.

All sample were analyzed to determine the effect of GI bleeding on coagulation and correlate coagulation parameters investigated to age, gender and causes of GIT bleeding.

The results showed that PT was significantly prolonged in patients than controls (P=0.00), APTT was slightly prolonged in patients (P=0.05) and platelet count was significantly decreased in patients than control (P=0.00).

Patients with oesphagus variances were having a normal intrinsic and extrinsic pathway and platelet count; Patients with portal hypertension were having a decrease platelet count only, and Patients with other causes of GIT bleeding were having a defect in the intrinsic and extrinsic pathway and decrease in platelet count.

No statistically significant correlation was found between coagulation parameters investigated and gender and age in those patients.

ملخص الأطروحة

أجريت هذه الدراسة بمركز محمد صالح إدريس للنـزف المعـوي فـي الفـترة مـن يناير الى فبراير 2010م تم فيها قياس زمن البروثرومبين وزمن الثرومبوبلاسـتين المنشط جزئيا كما تم قيـاس عـدد الصـفائح الدمويـة وذلـك لمعرفـة تـأثير النـزف المعوي على هذه القياسات بالاضافه الى معرفة العلاقة بين هذه القياسات وعمر المريض وجنسه ومسبب النزف المعوي.

شملت هذه الدراسة 50 مريضا 43 ذكور (86%) و 7 من الاناث (14%).

كان متوسط العمر لديهم 47 سنه و 30 شخصا طبيعيا كان متوسط العمـر لـديهم 21 سنه.

أظهـرت النتـائج معـدلات أكـثر مـن الطبيعيـة لكـل مـن زمـن البروثرومـبين والثرومبوبلاستين المنشط جزئيا ونقصان في عدد الصفائح الدمويه فـي المرضـى مقارنه بالمجموعة الضابطة.

المرضي الذين يعانون من دوالي المرئ لم تكـن لـديهم أي مشـاكل فـي المسـار الداخلي او الخارجي لعمليه التجلط ومعدل طبيعي في عدد الصـفائح الدمويـة امـا المرضي الذين يعانون من فرط التوتر البابي كان لديهم نقصان في عدد الصـفائح الدموية فقط, والمرضي الذين يعانون من أسباب مختلفة أخرى كان لديهم اعتلال في المسار الداخلي والخارجي لعملية التجلط و كذلك نقصـان فـي عـدد الصـفائح الدموية .

لم يتضح وجود علاقة احصائية بين زمن البروثرومـبين والثرومبوبلاسـتين المنشـط جزئيا وعدد الصفائح الدموية وبين جنس وعمر المريض.

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Abbreviations

a2-PI	a2-plasmin inhibitor
ADP	Adenosin diphosphate
APTT	Activated partial thrombopastin
AT	Anti thrombin
ATP	Adenosine tri phosphate
CHO	charbohydrate
D.W	Distilled water
ECS	Endothelial cells
EHPVO	Extra hepatic partal verous obstructive
FDP	Fibrin degradation product
FFP	Fresh frozen plasma
FSP	Fibrin split product
GDP	Guanosine di phosphate
GIT	Gastrointestinal tract ATP
GP	Glycoprotein
GRP	Gastrin releasing peptide

GTP	Guanosine tri phosphate
HMWK	high molecular weigh kininogen
HPS	Hermansky pudlak syndrome
IBD	inflammatory bowel disease
LACT	Lipoproten-associated coagulation in hibitor
NSAIDs	non-steroidal anti-inflammatory drugs
O.V	Oesophegus varaces
PA	tissae plasma activator
PAE-1	plasminogen Activeator in hibfor
PAI-1	plasmin activator inhibitor-1
PC	Protein C
PDGF	platelet drived growth factor
PF4	platelet factor
PGE2	ProstaglandinsE2
PHT	Portal hypertension
PIVKAs	pratein induced by vitamin K antagonists
PPF	Periportal fibrosis
PPP	Platelet poor plasma
PT	Prothombin time
VWF	Von willebrand factor

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