سامية محمد مصطفى

شبكة المعلومات الجامعية

بسم الله الرحمن الرحيم

سامية محمد مصطفى

شبكة المعلومات الجامعية

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التوثيق الالكتروني والميكروفيلم

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سامية محمد مصطفى

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

يجب أن

تحفظ هذه الأقراص المدمجة بعيدا عن الغبار

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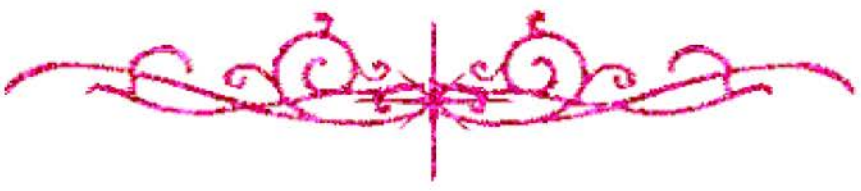
بعض الوثائق الأصلية تالفة



سامية محمد مصطفى

شبكة المعلومات الجامعية

بالرسالة صفحات لم ترد بالأصل

HEPATIC ENCEPHALOPATHY NEUROPSYCHIATRIC MANIFESTATIONS AND PATHOGENESIS

Thesis

Submitted for Partial Fulfillment of Master Degree

In "Neuro – Psychiatry"

By

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2001

بسم الله الرحمن الرحيم

﴿إِنَّمَآ أَمْرُهُۥٓ إِذَآ أَرَادَ شَيْـًٔا أَن
يَقُولَ لَهُۥ كُن فَيَكُونُ * فَسُبْحَٰنَ
ٱلَّذِى بِيَدِهِۦ مَلَكُوتُ كُلِّ شَىْءٍ
وَإِلَيْهِ تُرْجَعُونَ﴾

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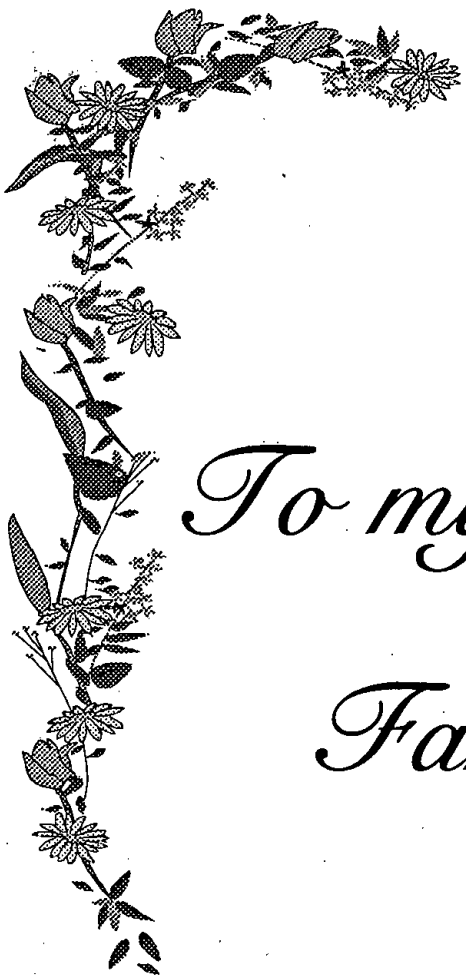
صدق الله العظيم



To my

Family

ACKNOWLEDGEMENTS

Thanks to God whose magnificent help is the first factor in every thing we can do in our life.

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ABBREVIATIONS

HE	: Hepatic Encephalopathy.
PSE	: Portal – Systemic Encephalopothy.
CNS	: Central Nervous System.
GABA	: Gamma – amine butyric acid.
C.S.F	: Cerebrospinal fluid.
EEG	: Electroencephalogram.
VEPS	: Visually evoked potentials.
CT	: Computed tomography.
MRI	: Magnitic resonance imaging.
Zn	: Zinc.
Cu	: Copper.
Fe	: Iron
Mn	: Manganese.
Mg	: Magnesium.
MDT	: Manual Dexterity test.
NCT	: Number Connection test.
LT	: Line tracing test.

CONTENTS

INTRODUCTION

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In schistosomal he... ation 70% of cases ... periportal fibrosis and ...0% develop genuine hepatic cirrhosis ... the whole of these cases, neuropsychiatric ... re found to be present in as many as 70% of ... high incidence (E... urg. ...96).

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... liver detoxic... makes the exoge... substances harmless for the body. With s... importa... the liver protects all the organs ... functioning, toxic metabolites injurious to the brain may be produced like ammonia and ... that are normally removed by the hepatobiliary route ...

INTRODUCTION

Egypt is considered a region with high rate of morbidity of chronic liver diseases in which hepatic encephalopathy is considered one of it's significant complications ***(El-Zayadi et al., 1992)***.

In schistosomal hepatic infestation 70% of cases develop periportal fibrosis and 30% develope genuine hepatic cirrhosis ***(Kock 1990)***. From the whole of these cases, neuropsychiatric manifestations were found to be present in as many as 70% of them, which is a very high incidence ***(Eisenburg, 1996)***.

A relationship between hepatic disorder and neuropsychiatric symptoms has long been speculated, in which a reduction in functioning liver cell mass and/or diversion of portal blood passing the liver via portal-systemic collaterals, are the two important ingredients which predispose to the disturbance in cerebral function, but the precise mechanisms involved are not known ***(Eddlston 1994)***.

Liver detoxication makes the exogenous substances harmless for the body. With such an important role, the liver protects all the organs of the body. When liver is malfunctioning, toxic metabolites injurious to the brain may be produced like ammonia and manganese, two substances that are normally removed by the hepatobiliary route and that in liver failure accumulate in the brain producing symptoms ***(Ananth et al., 1994 and Hazell 1999)***.

Hepatic encephalopathy represents a group of neuro-psychological signs and symptoms accompanying advanced, decompensated liver disease ***(Scharschmidt.1996)***.

Disorders of cerebral function, such as lack of concentration, impaired ability to think logically, loss of comprehension, impairment of space perception and recognition of numbers and letters can be very reliably detected by simple psychometric tests ***(Eisenburg 1996)***. Other psychiatric symptoms such as irritability and low threshold to anger, depression, deteriorating academic and work performance, cognitive impairment, psychosis and/or anxiety were encountered in clinical hepatic encephalopathy (HE) ***(R.Al-Jamal et al., 1998)***.

Trace elements such as zinc, copper, iron, manganese and magnesium can have profound biological effects in liver diseases, there functions are catalytic in nature i.e. that they act as direct activators of certain enzymes, or indirectly as essential components of vitamins or hormones. ***(Loomba et al 1995)***

Neuropsychiatric signs of zinc deficiency including irritability, emotional disorder, tremors and occasional cerebellar ataxia occur in patients with (HE) can be improved with the disease by correcting the zinc deficiency that compromises conversion of ammonia to urea ***(Elewa, 1990)***.

Patients with Wilson's disease have an impaired ability of the liver to excrete copper into the bile and this condition is characterized clinically by progressive cerebral damage involving particularly basal ganglia ***(Loomba. et. at 1995)***.

The severity of multi-organ failure in fulminant hepatic failure involves free radical damage caused by the presence of circulating iron, catalytic for free radical reactions ***(Evans et al 1994)***.

There are many symptoms that are common with manganese deficiency e.g. skeletal abnormalities and postural defects (ataxic), others are present due to its toxicity like chronic manganese poisoning characterized by severe psychiatric and neurological disorders clinically similar to Parkinson's disease ***(Barron. 1994)***.

Low values for serum magnesium have been reported in portal cirrhosis and epilepsy ***(Flink 1980)***.