Salwa Akl

بسم اللّه الرحمن الرحيم

مركز الشبكات وتكنولوجيا المعلومات

قسم التوثيق الإلكتروني

Salwa Akl

جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

نقسم باللّه العظيم أن المادة التي تم توثيقها وتسجيلها

علي هذه الأقراص المدمجة قد أعدت دون أية تغيرات

Salwa Akl

بعض الوثائق الأصلية تالفة
وبالرسالة صفحات لم ترد بالأصل

DYSAUTONOMIA IN PARKINSON'S DISEASE

THESIS
Submitted in partial fulfillment
For M.D Degree in
NEUROLOGY

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Zagazig University
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2000

Acknowledgement

I am truly indebted to Prof. Dr. Saher Hashim, Professor of Neurology, Faculty of Medicine, Cairo University, whose generous patience and professional guidance extends to all my work.

My most sincere gratitude and appreciation to Pro. Dr. Abdul Shafy Tabl, Professor of Internal medicine and Dean of Benha Faculty of Medicine, Zagazig University. for his valuable criticism, generous advice and support throughout the work.

I wish to express my sincere gratitude to Dr. Hussien Fathy. Assistant professor of Neurology Benha Faculty of Medicine, who taught me. the responsibility of the written word, for his professional guidance, he has generously given me much of his valuable time to complete this work..

My most since gratitude to Dr. Rizk Khodair, Assistant professor of Neurology, Benha Faculty of Medicine, for his constant effort, advice and support

I wish to express my appreciation to Dr. Mohamed Mansour, Assistant Professor of Pharmacology, Dr. Aly Alshazly. Lecturer o, urology and Dr. Roshdy Khalaf, lecturer of Internal medicine, Benha faculty of medicine, for their valuable help and cooperation

Special thanks to all my Professors and colleogues of Psychiatry and Neurology Dept., Benha Faculty of medicine, Zagazig University for their encouragement and support.

CONTENTS

LIST OF ABBREVATIONS

AD	:	Alzheimer's Disease.
AMP	:	Admenosine mono phosphate.
ANOVA	:	Analysis of variance.
BP	:	Blood pressure.
BPH	:	Benign prostatic Hypertrophy.
CBGD	:	Cortical –basal ganglionic degeneration.
CHAT	:	Choline acetyl transerase.
CNS	:	Central nervous system.
COMT	:	Catechol-O- methyltransferase.
CSF	:	Cerebrospinal fluid.
DLB	:	Dementia with Lewy bodies.
DLBD	:	Diffuse Lewy body disease.
DNA	:	Deoxyribonucleir acid.
DOPS	:	D,L threo 3,4 dihydroxyphenylserine.
DYN	:	Dynorphin.
ECG	:	Electrocardiogram.
EMG	:	Electromyography.
ENK	:	Enkephalin.
GABA	:	Gamma aminobutyric acid.
GAD	:	Glutamic acid decarboxylase.
GIT	:	Gastrointestinal.
GP	:	Globus-Pallidus.

Gpe	:	Globus pallidus external.
Gpi	:	Globus pallidus internal.
HRV	:	Heart rate variability.
HVA	:	Homovanillic acid.
HY	:	Hoehn and Yahr.
LAA	:	L-Aromatic amino acid decarboxylase.
MAO-B	:	Monoamine oxidase B.
MND	:	Motor neurone disease.
MPP^+	:	1-methy 1-4 pheny1, 1,2,3,6 tetrahydropyridine.
MPTP	:	1-methy 1-4 pheny 1, 1,2,3,6 tetrahydropyridine.
MRI	:	Magnetic resonance imaging.
MSA	:	Multiple system atrophy.
NADH	:	Reduced nicotinamide dinnucleotide.
NE	:	Norepinephrine.
OH	:	Hydroxyl radical.
OPCA	:	Olivopontocerebellar atrophy.
P	:	The probability.
PD	:	Parkinson disease.
PSP	:	Progressive supranuclearspalsy.
SD	:	Standard deviation.

SDS : Shy- drager syndrome.

SN : Substantia nigra.

SNC : Substantia nigra compacta.

SND : Striatonigral degeneration.

SNK : Student newman kelus.

SNr : Substantia nigra reticulate.

SoD : Superoxide dismutase.

SP : Substanse P.

STN : Subthalamic Necleus.

TOH : Tyrosine hydroxylase.

UPDRS : Unified Parkinson's disease rating scale.

WEM : The standard error of the mean.

Introduction And Aim of The Work

Introduction and Aim of The Work

Long time before James Parkinson's classic "An Essay on The Shaking plasy" (1817), ancient Egyptian and Indian sources reported many types of paralytic disorders and tremors. Non fully described the distinctive features of the syndrome which so justly perpetuates Parkinson's name **(Stern,1989 and Pearce,1989).**

The last 5 years have been marked by rapid developments in understanding the pathophysiology of Parkinson disease (PD) as well as by the introduction of a number of new drugs for symptomatic treatment of the disease. On the other hand the diagnosis of PD is still made on purely clinical grounds. Due to Continuing advances in therapy, it is increasingly important to recog-nize PD in its earliest stages and to distinguish it from other causes of Parkinsonism, for which prognosis and response to treatment differ **(Simuni and Stern,1998).**

Autonomic dysfunction in PD was first reported by James Parkinson himself. Abnormalities of salivation, sweating, bladder and bowel function are common features of the disease he described, and orthostatic hypotension, thought less common, is perhaps the most disturbing form of dysautono-mia **(Rajput and Rozdilsky,1976).**

The failure to increase heart rate with falling blood pressure and the lack of blood pressure overshoot with Valsalva in patients of PD may be due to inadequate incr-ease in serum norepinephrine on standing**(tanner et al., 1992)**.

Aim of the work

This work aimed at evaluation of the magnitude of problem of dysautonomia in Egyptian parkinson patients and its incedence and its impact on the activity of the patients. Moreover to forecast the prognosis of such patients

Review Of literature

Epidemiology and Etiology

Parkinson's disease is the commonest neurodegenerative disease after Alzheimer's disease, with an estimated incidence of 20/100 000 and a prevalence of 150/100 000 **(Schapira, 1999).**

A number of epidemiologic studies have been carried out to identify possible risk factors for the development of PD. Increasing age is the only unequivocal risk factor independent of disease prevalance in different population groups **(Mayeux et al.,1992).** It is a disease of the middle-to-late years with a mean age of onset of 60. In about 5% of patients, symptom onset occurs before age 40; in such cases, the disease is classified as young-onset PD **(Quinn et al.,1987).**

PD prevalence is highest in Europe and North America (between100-300/100,000) with lower rates in Asia and Africa (approximately 50/100,000) **(Schoenberg, et al.,1988).** PD is slightly more common in men than in women. In most studies, the incidence is lower among blacks than whites, although this observation might be biased by a number of socioeconomic factors such as access to health care and perception of the disease **(Bharucha, et al.,1988).** One epidemiological survey, conducted in Copiah County, Mississippi, used a door-to-door design to overcome these limitations. There was no difference

in age-adjusted PD prevalence between blacks and whites **(schoenberg, et al., 1985)**.

Abdul Baki et al., (1999) conducted an epidemolo-gical study using a door-to-door- design in a rural Egyptian community(Monofia), PD prevalence was 152/100.000, and the disease was common in women than in men.

The occurrence of parkinsonism as a late sequela of encephalitis lethargica in the 1920_s and 1930_s suggests infection as a possible etiologic factor. However, a viral etiology has never been shown, and both the clinical picture and pathology of postencephalitic parkinsonism and PD are distinctly different **(Poskanzer, et al., 1969).** A number of environmental factors have been implicated as possible risk factors for PD. The search for an environmental trigger has been fueled by a cluster of cases of parkinsonism that are clinically and pathologically indistinguishable from idiopathic PD, caused by exposure to 1-methyl, 4-phenyl,1,2,3,6-terahydropyridine (MPTP), a compound used by narcotic abusers in the 1970_s)**(Ballard, et al.,1985)**. It is unlikely that MPTP is present in the environment in sufficient quantities to be an important environmental risk factor for PD. However, the MPTP story has resulted in several pathogenetic theories that generally include oxidative stress as a possible common final pathway leading to nigral cell death and PD**(Tanner, et al.,1987)**. Rural living farming, well-water