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شبكة المعلومات الجامعية

بسم اللّه الرحمن الرحيم

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شبكة المعلومات الجامعية

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

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جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

قسم

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بالرسالة صفحات
لم ترد بالأصل

Study Of Toxicity Of Amiodarone-Anti-Arrhythmic Drug-In Therapeutic Doses On Thyroid And Lung Functions In A Period Of Six Months

Thesis

Submitted To The Faculty Of Medicine, Tanta University
For Partial Fulfillment Of The Requirements Of The Master Degree
In Clinical Toxicology

By

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بسم الله الرحمن الرحيم

قالوا سبحانك لا علم لنا إلا ما علمتنا إنك أنت العليم الحكيم.

صدق الله العظيم

سورة البقرة (٣٢)

Acknowledgement

I wish to express my deepest gratitude and thankfulness to Dr. Mona Heshmat, Professor and Head of Department of Forensic Medicine and Toxicology, Faculty of Medicine, Tanta University for devoting much of her valuable time in the supervision of this work with continuous guidance and encouragement. Moreover, Professor Mona has made it possible for me to benefit from her thorough knowledge, great experience and valuable advice.

My cordial gratitude goes to Dr. Mostafa El-Kholy, Assistant Professor of clinical toxicology, Faculty of Medicine, Zagazig University, for suggesting the subject of this thesis, his great help in supervision and sincere guidance throughout the work.

I am deeply indebted to Dr. Mona El-Gohary, Lecturer of forensic medicine and toxicology, Faculty of Medicine, Tanta University, for her infinite help and guidance in performing this study thesis.

I wish to express my profound gratitude to Dr. Mai Salama, Assistant Professor, Cardiology Department, Faculty of Medicine, Tanta University, for her help in all stages of performing of this thesis.

Last but not least, I am greatly thankful to all members of the Department of Forensic Medicine and Toxicology, Faculty of Medicine, Tanta University. All members of the Department have helped me throughout my study in such a way.

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AIM OF THE WORK

INTRODUCTION

Introduction

Amiodarone is a benzofuran derivative approved by the Food and Drug Administration (FDA) for the treatment of patients with life threatening ventricular tachyarrhythmias when other drugs are ineffective or not tolerated (Braunwald, 1996).

It is a class III antiarrhythmic agent discovered in 1961, and was originally marketed as an antianginal agent in 1967 (Lancellotti et.al., 1998).

Although amiodarone is considered one of the most effective and commonly used antidysrhythmic agent, its effects are still the subject of debate. Of the greatest concern is amiodarone-induced pulmonary toxicity. Amiodarone has recently been suggested as a possible cause of pulmonary fibrosis (Card et.al., 1998). Amiodarone-induced pulmonary toxicity is one of the most life-threatening complications of this therapy. It is easily missed by any physician who is suddenly confronted with nonspecific pulmonary complaints during amiodarone treatment therapy (Jessurun et.al., 1998).

In view of its high iodine content, the drug was studied for its possible influence on thyroid function. These studies demonstrated that amiodarone exerts important effects on peripheral metabolism of thyroid hormones and thyroid function as well. It inhibits conversion of T_4 to T_3 in vivo. (Figge et.al., 1990, Polikar et.al., 1993).

Hepatic dysfunction from amiodarone is also well recognised (Harris et.al., 1983). The liver enzymes rise in patients on large doses of amiodarone, but clinically evident hepatic dysfunction has not been detected. Cirrhosis occurs uncommonly and may be fatal (Braunwald E, 1996).

Other adverse effects of amiodarone include neurological dysfunction, photosensitivity, bluish skin discoloration, corneal microdeposits and gastrointestinal disturbances. (Davies et.al., 1992).

Aim of the work

The aim of this study is to evaluate the toxic effects of Amiodarone anti-arrhythmic drug given in therapeutic doses over a period of 3 to 6 months on thyroid and pulmonary functions and epidemiological study of the cases admitted to the Cardiology Department of Tanta University Hospital; Giving way to learning when is the best time to stop the drug or propose a safe dose regimen.

REVIEW OF LITERATURE