

بسم الله الرحمن الرحيم





شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم



جامعة عين شمس

التوثيق الإلكتروني والميكروفيلم

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Plasma level of D-dimer in patients with Chronic Idiopathic Urticaria

Thesis

*Submitted for partial fulfillment of a Master Degree
in Dermatology, Venereology and Andrology*

By :

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*Faculty of Medicine
Ain Shams University
2008*

بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

وَاقِلْ رَأْيِي زَادَ نِيَّ حَلِيمًا

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سُورَةُ النَّازِعَاتِ آيَةُ 114

Acknowledgements

*I would like to express my deepest thanks and respect to **Dr. Nader Fouad M. Ragab** Professor of Dermatology, Venereology and Andrology, Ain Shams University, for his valuable supervision, guidance and kind advice throughout this work.*

*Special thanks and deepest gratitude to **Dr. Samar Abdallah M. Salem**, assistant professor of Dermatology, Venereology and Andrology, Ain Shams University, for her good support, continuous supervision and unlimited help during this work.*

I would like to thank my family for their kindness, support, and much needed encouragement.

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Protocol for thesis

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INTRODUCTION

Chronic urticaria (CU) is a common skin disorder characterized by the recurrent eruption of short-lived wheals accompanied by redness and itching for at least six weeks (**Greaves, 2000**). However, it is not a single disease but a cutaneous reaction pattern for which there are multiple potential causes. The clinical expression of the disease varies from one patient to another in duration of activity, morphological features of the lesion and its histopathological basis (**Tharp et al., 1990**). Chronic idiopathic urticaria (CIU) is a common form of chronic urticaria in which no precise causal factors can be identified (**Champion, 1990**).

Results of a number of in vitro studies performed suggest an auto-immune pathogenesis for chronic idiopathic urticaria, but several aspects remain ill-defined or even contradictory. It was generally accepted that auto-antibodies to IgE or to high affinity IgE receptors which were commonly regarded as the most relevant pathogenic factor in the disease, can be detected in sera of a proportion ranging from 25% to 50% of patients with chronic

idiopathic urticaria (**Hide et al., 1993, Niimi et al., 1996, Tong et al., 1997**).

Recently, an activation of coagulation cascade via the tissue factor pathway has been observed in most of patients with chronic idiopathic urticaria. Also, indirect evidence of the possible involvement of coagulation cascade came from the observation that the proportion of skin test-positive patients rises substantially if autologous plasma is injected instead of autologous serum (about 80% Vs 50%) suggesting that clotting destroys a plasma factor that is some how involved in the skin reaction (**Asero et al., 2006**).

The recent information indicates that chronic urticaria is characterized by an activation of the extrinsic pathway of coagulation leading to the formation of thrombin which might be responsible for generating C5a and increasing vascular permeability. On the other hand, the excess of fibrin formation is counter-balanced by enhanced fibrinolysis as demonstrated by the increase in D-dimer levels (**Asero et al., 2007**).

D-dimer is a breakdown product by the degradation of cross linked fibrin by the plasmin due to any etiology **(Freyburger et al., 1998)**. Also, it is produced in all situations in which coagulation and fibrinolysis are activated **(Goldstien et al., 2001)**.

Thrombus formation is normally followed by an immediate fibrinolytic response by plasmin causing the release of fibrin degradation products -predominately containing D-dimer- into the circulation. So, the absence of D-dimer implies that thrombosis is not occurring and negative D-dimer assays have an important role in excluding any diagnosis of coagulation **(Kelling et al., 2004)**.

In recent studies D-dimer levels were found to be higher in patients with slight to moderate chronic idiopathic urticaria than in a group of age and sex matched normal controls and were significantly elevated in a selected group of patients with severe exacerbation of chronic idiopathic urticaria proving that the coagulation cascade is activated up to the production of fibrin as a result of the generation of

thrombin. That was further supported by the dramatic drop of D-dimer levels which were extremely high during an acute phase of severe chronic urticaria and perfectly normal after remission (**Asero et al., 2007, Asero et al., 2008**).

New therapies targeting anticoagulants are currently considered in treatment of chronic urticaria according to clinical studies which were performed to investigate the effect of warfarin in treating patients with chronic idiopathic urticaria unresponsive to anti-histamines, these patients responded to warfarin showing complete resolution of symptoms and some of them had a dramatic response (**Berth et al., 1988, Barlow and Greaves, 1992, Parslew et al., 2000**).

Also, a case report study showed a marked clinical improvement in a patient with chronic urticaria after been treated with subcutaneous heparin and remained completely recovered after fifteen months of treatment. If heparin was discontinued or the dose was reduced deterioration occurred. This supports the involvement of coagulation cascade in the pathogenesis of chronic urticaria. (**Chua and Gibbs, 2005**).

Aim of the Work:

The aim of this work is to estimate the plasma levels of D-dimer in patients with chronic idiopathic urticaria, to assess its possible role in CIU and to correlate such levels with the disease severity.

Patients And Methods:

This study will include:

- Twenty patients with CIU after taking their consent.
- Ten patients with CU with a known cause are also selected after taking their consent.
- Ten healthy persons will be also included as a control group.
- All persons (patients & controls) will be subjected to:
 - 1) Full history taking.
 - 2) General examination.
 - 3) Dermatological examination.
 - 4) Other investigations as required to exclude possible causes of chronic urticaria.
 - 5) Blood samples will be obtained from patient and controls, and plasma D-dimer levels will be measured by the ELISA Method.

- Statistical analysis of the results will be performed.

The Thesis will include:

- 1 - Introduction and Aim of the Work.
- 2 - Review of Literature.
- 3 - Patients and Methods.
- 4 – Results.
- 5 – Discussion.
- 6 - Summary and Conclusion.
- 7 – References.
- 8 - Arabic Summary.

References:

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