شبكة المعلومات الجامعية

# بسم الله الرحمن الرحيم


شبكة المعلومات الجامعية

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# التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

# 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


# بالرسالة صفحات لم ترد بالأصل

# بعض الوثائق الأصلية تالفة

Menoufiya University
Faculty of Medicine
Ophthalmic Department

# STUDY OF RETINAL VEIN OCCLUSION AND ITS DETERMINANT FACTORS

By

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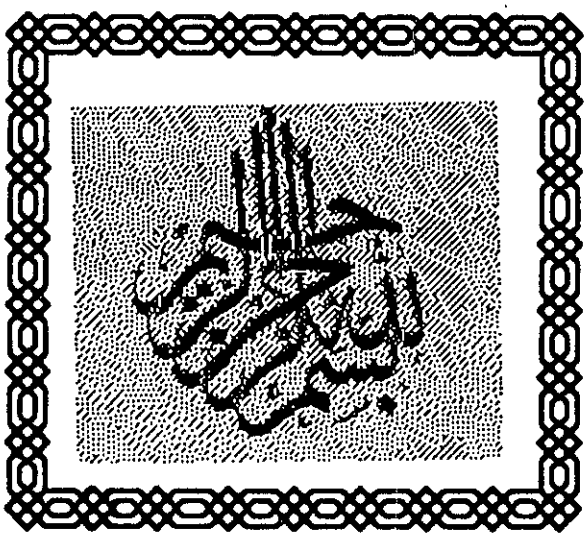

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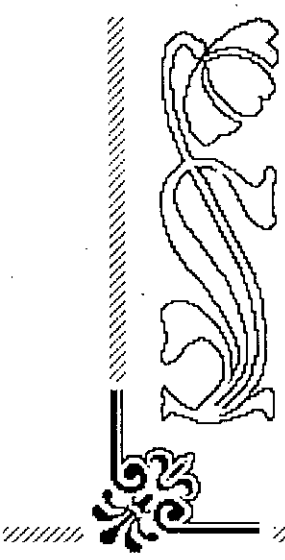

To

My father and my professor.

Prof. Dr.

Mohamed Kamel El-Sobky

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# لجنة المناقشة


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# CHAPTER 1

# INTRODUCTION

# INTRODUCTION

Retinal vein occlusion (RVO) is a common retinal vascular disorder, second in prevalence only to diabetic retinopathy (**Clarkson, 1989**).

Central retinal vein occlusion (CRVO) is a common cause of visual loss for which at the current time no effective and reliable form of treatment is available (**McAllister and Constable, 1995**).

Central retinal vein occlusion is usually seen as a disease of the elderly when it is frequently associated with arteriosclerotic or atherogenic disease elsewhere, glaucoma or factors which affect blood coagulability and viscosity. It is less common in younger people. (**Walters and Spalton, 1990**)

Conditions such as hyperlipidaemia, paraproteinaemias polycythaemia, increased platelet aggregation, and other hyperviscosity or hypercoagulability syndromes which result in changes in the composition of the blood (and its flow) have often been found in association with (CRVO). (**Dodson *et al.*, 1981**) and (**Dodson and Kritzinger, 1985**)

Hypertension and hyperlipidaemia are commonly found in patients with recurrent retinal vein occlusion and may therefore be important aetiological factors.

Treatment of hypertension and hyperlipidaemia might be of value to prevent recurrence of RVO (**Dodson *et al.*, 1985**).

Vasculitis of the venous wall (phlebitis)) has been frequently cited as a primary cause of CRVO in young adults (**Appen *et al.*, 1980**).

The frequent association of peripheral blood immunological abnormalities in patients with retinal vasculitis indicate the possible role of immunological mechanisms in its pathogenesis (**Wakefield *et al.*, 1986**).

The presence of a congenital anomaly in the central retinal vein at the lamina cribrosa causes turbulent flow and predisposes to thrombus formation.

Factors concerned in the causation of thrombosis are;

- Changes in the vessel wall.
- Changes in the blood flow and.
- Changes in the composition of the blood.

**(Walters and Spalton, 1990)**

Management of patients with retinal vein occlusion with or without glaucoma or ocular hypertension should include the identification and treatment of common conditions which appear to have an aetiological role in retinal vein occlusion (**Cole *et al.*, 1989**).

As with all ischaemic retinal disorders, neovascularisation (NV) is a serious complication of the vein occlusion. The identification of individual at risk for the development of neovascularisation of the iris (NVI) is particularly important in central retinal vein occlusion where ischaemic eyes may develop NVI whithin 100 days of the occlusion. About one third of eyes with NVI will progress to neovascular glaucoma if not treated by panretinal photocoagulation before angle closure (**Hayreh, *et al.*, 1983**).