



بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

قسم

نقسم بالله العظيم أن المادة التي تم توثيقها وتسجيلها
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بالرسالة صفحات
لم ترد بالأصل

بعض الوثائق
الأصلية تالفه

CD64 Expression on Blood Polymorphs in
Patients with sickle cell disease

Thesis

*Submitted for partial fulfillment of the degree of Master Degree
of Clinical and Chemical Pathology*

By

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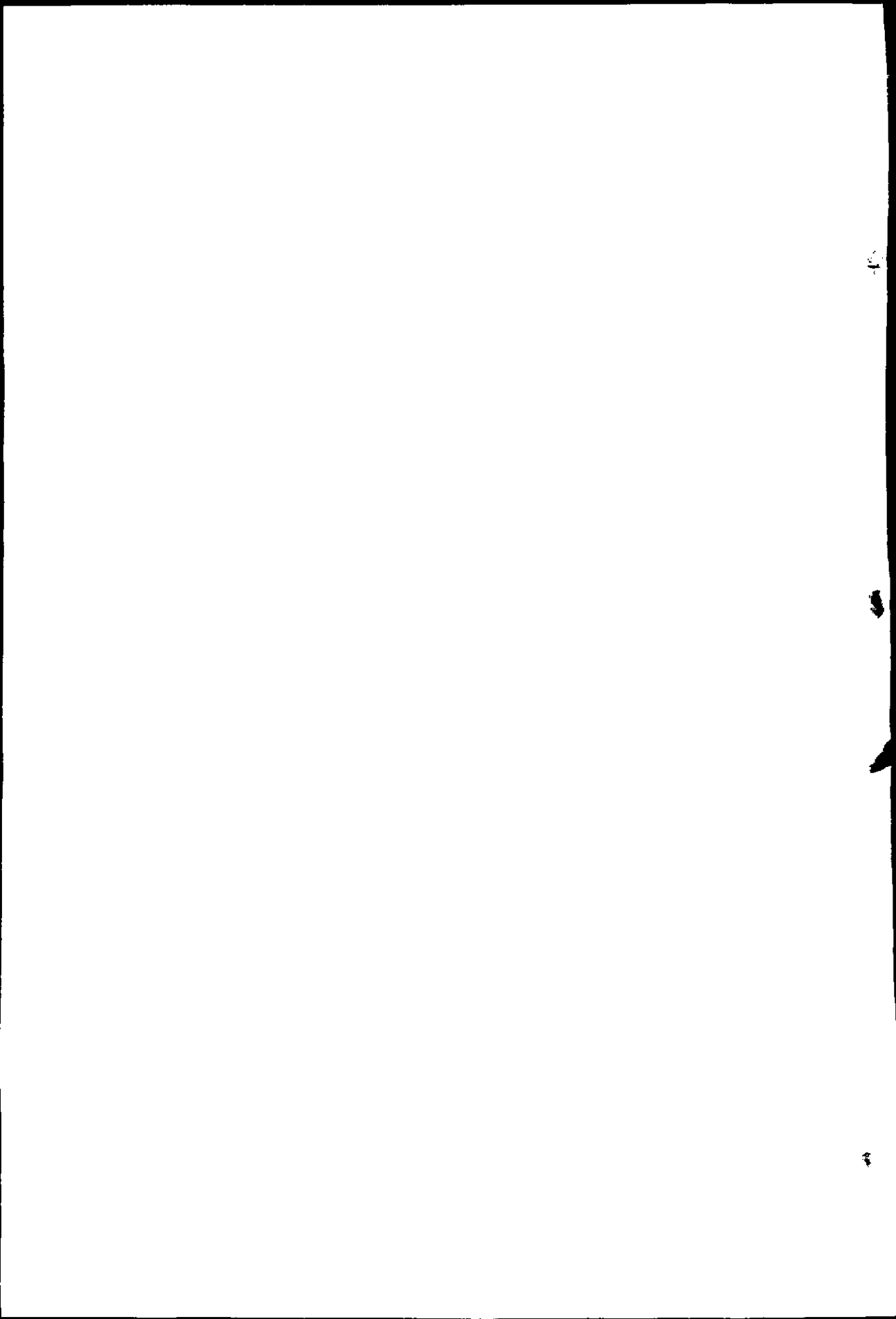
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2002*





To my parents

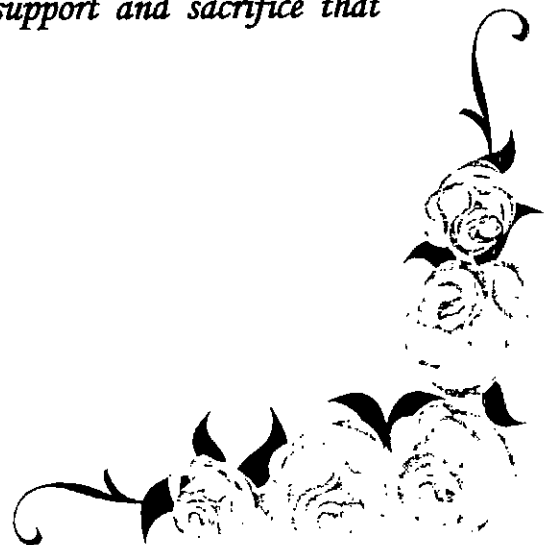
To my husband

To my little Salma

To all those who help me

Thank you

I would like to express my sincere thanks to my father, mother, and husband and to my lovely Salma for their patience, love, support and sacrifice that made this work possible.





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بعد فحص الرسالة بواسطة كل عضو منفردا وكتابة تقارير منفردة لكل منهم لامتدات اللجنة مجتمعة فسي

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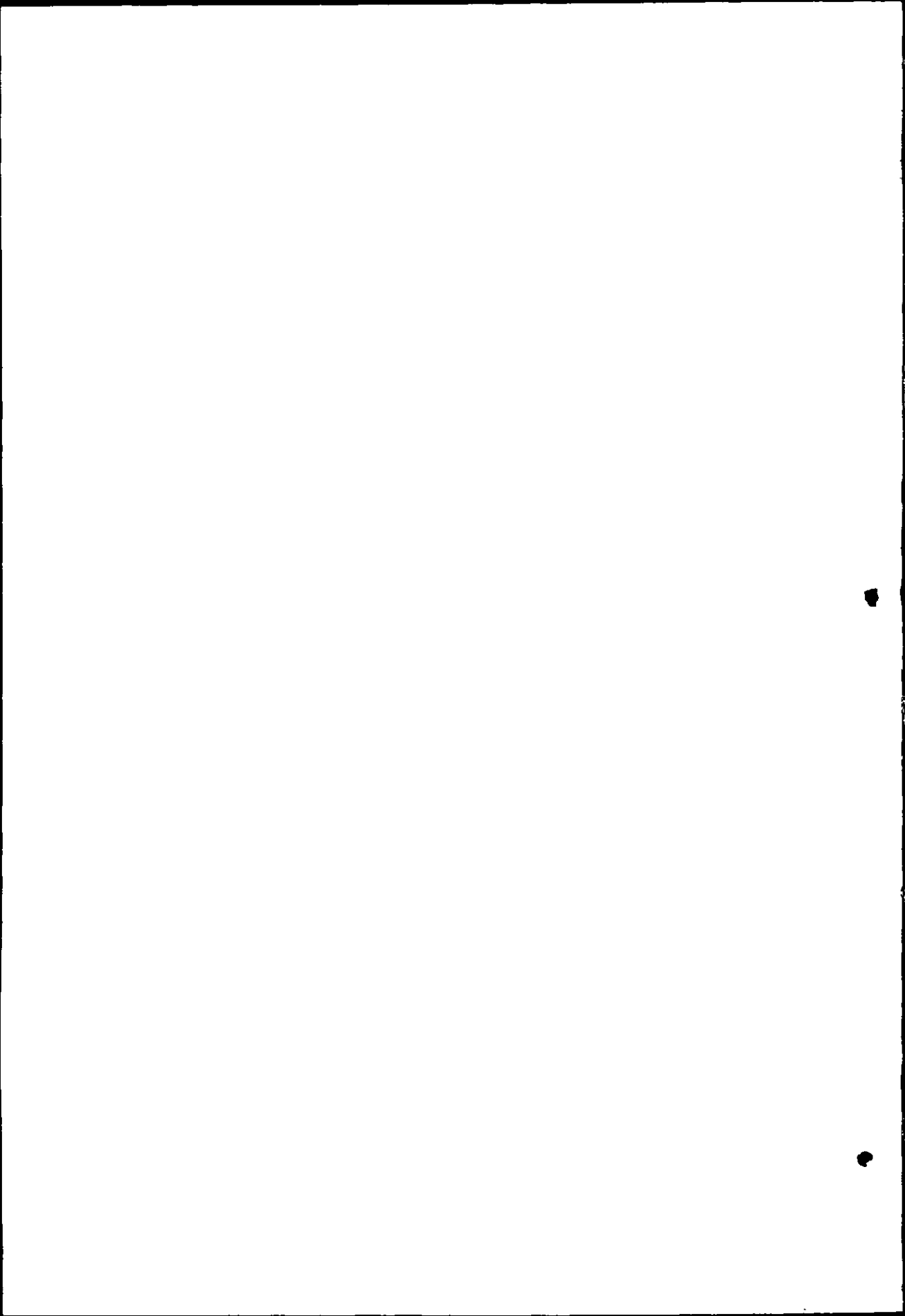
المصاحب للموضوع وعرضه وفيه النتائج واستخلاصها الشارح

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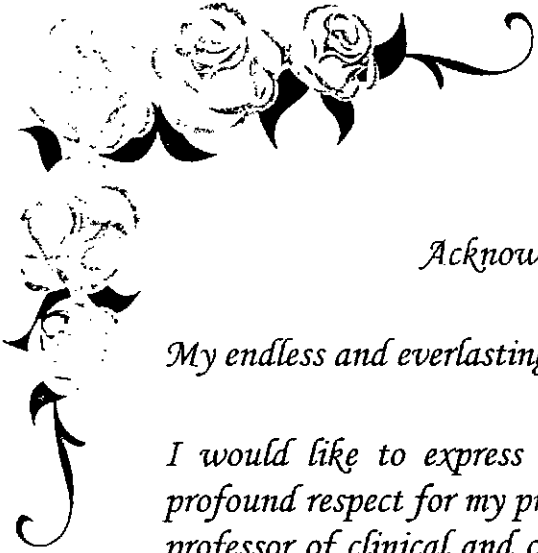
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Abstract

Vaso-occlusive crises of sickle cell disease are a major cause of morbidity and death. The pathogenetic mechanisms of vaso-occlusion remain controversial, although it is generally agreed that the increased binding of sickled erythrocytes to vascular endothelium is an important contributory factor. The suggestion that polymorphonuclear leukocytes (PMNs) may play a copathogenic role arises from the association of sickle cell crises with infection. The present work aimed to study the expression of CD64 and plasma level of sL-selectin as markers of polymorphonuclear leukocyte activation in sickle cell disease and to clarify the role of these markers in the pathophysiology of sickle cell disease. Forty patients with homozygous sickle cell disease were enrolled in this study, together with 15 healthy children, matched for age and sex, served as the control group. The studied patients were divided into 2 groups; Group I: those in a clinically steady state and Group II: those in acute painful crises. Diagnosis of sickle cell disease was based on full clinical assessment, complete blood picture, reticulocytic count, Hb electrophoresis. The present work showed a highly significant increase in percentage expression of CD64 on neutrophils in group II (patients with acute painful crisis) compared to healthy control and in group II compared to group I (steady state) ($p < 0.001$), while the increase in steady state compared to the control group was not significant ($p > 0.05$). Plasma sL-selectin level showed a highly significant increase in group I (sickle cell patients in steady state) and group II (sickle cell patients in crisis) compared to the healthy controls, also comparison of plasma sL-selectin level in both patient groups revealed a highly significant increase in group II compared to group I. There was a highly significant +ve correlation between percentage expression of CD64, and plasma level of sL-selectin as correlated to VOC/year, blood transfusion in sickle cell disease patients (group I and group II). On correlating the WBC and absolute neutrophil count to percentage expression of CD64 and plasma level of sL-selectin there was a highly significant +ve correlation in sickle cell patients group I and II. Regarding laboratory data, there was no significant correlation between percentage expression of CD64, and the erythrocyte related parameters (RBCs, HCT, MCH, MCV, total hemoglobin and reticulocytic count), platelets, and HbS% ($P > 0.05$) in sickle cell disease patients (group I and group II). A highly significant +ve correlation was found between sL-selectin and CD64 % in whole patient group [steady state + crisis]. Similarly a highly significant +ve correlation was found between sL-selectin and CD64 % in group I and in group II separately. Our results demonstrated that neutrophils are activated in sickle cell patients, especially during a vasoocclusive crisis by increased percentage expression of CD64 and increased plasma level of sL-selectin as neutrophil activation markers. We therefore postulate neutrophils to have an important role in the initiation and propagation of vasoocclusive crises in sickle cell disease.

Key words: Sickle cell disease – Vaso-occlusive crisis – CD64 – sL-selectin – Neutrophil.



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