ORIGINAL ARTICLE

Relationship between body fat distribution and blood disorders in patients with Visceral Obesity with and without Diabetes Mellitus

Gino Avellone, Vincenzo Di Garbo, Valentina Guarnotta, Elvira Prestipino, Domenico Campisi, Giovanni Abruzzese, Rosa De Simone, Gilia Raneli and Giuseppe Licata

Department of Clinical Medicine, Lipid and Thrombosis Research Center, University of Palermo, Italy

ABSTRACT

Background

Visceral obesity can be considered the "primum movens" for cardiovascular complications, especially when the visceral obesity is associated to diabetes mellitus.

Aim of the study

The aim of our study has been to demonstrate in patients with visceral obesity metabolic, hemorheologic, hemostatic and inflammatory abnormalities already identified by researchers as predisposing factors for cardiovascular events.

Methods

Our study included 261 patients. We divided these patients in four groups: a control group of 30 subjects; a group of 28 patients with peripheral obesity; a group of 30 patients with visceral obesity; a group of 38 patients with visceral obesity and type 2 diabetes mellitus. We measured metabolic parameters like serum triglycerides, total cholesterol and HDL-Cholesterol, apolipoproteins A-1 and B, blood glucose, plasmatic insulin and HOMA/IR levels, hemorheologic parameters like blood viscosity, plasma viscosity and haematocrit, hemostatic, fibrinolytic and inflammatory parameters like fibrinogen, hs-CRP, factor VII, baseline PAI-1 and t-PA (Ag), post venous occlusion PAI-1 and t-PA levels.

Results

In the groups of visceral obesity with and without diabetes mellitus, we observed higher metabolic parameters except of Apolipoprotein A-1 and HDL-Cholesterol, which were lower than the control group and the peripheral obesity group. Patients with visceral obesity with and without diabetes mellitus had hemorheologic disorders and showed higher hemostatic, fibrinolytic and inflammatory parameters levels than control group and peripheral obesity group. In contrast mean levels of t-PA (Ag) post VO were significantly lower in visceral obesity groups than control subjects and peripheral obesity group.

Conclusions

Our findings suggest that metabolic, hemorheologic, inflammatory, hemostatic and fibrinolytic disorders predispose to cardiovascular and cerebral events, and provide a brief assessment model of the cardiovascular risk.

Key words: *Abdominal Visceral Fat, Hemorheology, Blood Coagulation, Inflammation, C-reactive protein, Lipids*

Corresponding Author: Prof. G. Avellone, Via De Gasperi, 203, 90146 Palermo
E-mail: ginoavellone@libero.it

INTRODUCTION

Cardiovascular events are the first death cause in patients with visceral obesity, a key component of metabolic syndrome.[1,2] This syndrome has a dramatic social impact in industrialized countries both for high economic costs and for disabling complications.[3]

The metabolic syndrome phenotype off springs from different environmental factors, such as an excessive caloric intake and a lack of physical activity, supported by a genetic predisposition. It is characterized from a cluster of components:[4] visceral adiposity, insulin-resistance and a slight glycaemic disorder, hypertension, atherogenic dyslipidemia (hypertriglyceridemia, small dense low-density lipoprotein (LDL), low high density lipoproteins (HDL), thrombotic, inflammatory and fibrinolytic disorders.[5,6,7]

Patients with metabolic syndrome have a strong and increased risk of mortality from coronary heart disease, such as congestive heart failure, unstable angina, and strokes.

The Botnia Study reports a 12% mortality due to cardiovascular events in patients with metabolic syndrome and a 2,2% mortality in subjects without metabolic syndrome.[8] The ARIC study (Atherosclerosis Risk in Communities) shows a two-fold cardiovascular risk in patients with metabolic syndrome.[9]

Patients with visceral obesity are exposed to cardiovascular risk factors including hyperinsulinemia, pro-inflammatory disorders, dyslipidemic disorders and hypercoagulative features, consisting of increased levels of clotting factors (tissue factor, factor VII and fibrinogen) as well as of inhibition of the fibrinolytic pathway (increased plasminogen activator inhibitor-1 and decreased tissue plasminogen activator activity).[10] At the same time, visceral obesity increases the risk for venous and arterial thromboembolism caused by the adipose tissue activation and