

ASSOCIATION BETWEEN THE CLASSICAL CAROTIDAL ATHEROSCLEROTIC RISK FACTORS AND ISCHAEMIC HEART DISEASE

*Luiza Despina Demian*¹, *Mihaela Simona Popoviciu*¹, *A-R. Popa*¹,
*Diana Aron*¹, *Prună Camelia*¹, *Zourob Emadeldeen*²

¹ University of Oradea, Faculty of Medicine and Pharmacy, Emergency Clinical County Hospital, Department of Diabetes and Internal Diseases

² Neasher Hospital, Israel

Abstract

Coronarian risk factors (obesity, hypertension, diabetes, dyslipidaemia etc) can predict the existence and the likelihood of ischaemic heart disease development. We studied the vascular atherosclerotic changes that occurred in vascular system outside the coronary territory. The abdominal pelvic index was more relevant in detecting the influence of systolic arterial hypertension. Abdominal index was more useful for analyzing the influence of diastolic arterial hypertension, smoking, cholesterol, and disturbances in regulation of glycolysis. Because ischaemic heart disease is well correlated with abdominal index and showed no dependence on the abdominal pelvic index, we think that it is better to use only the abdominal pelvic index and body mass index. Multiple regression made for all variables including atherosclerotic plaques showed a better predictive value than excluding this last variable. These and other risk factors have predictive value for ischaemic heart disease.

keywords: *ischaemic heart diseases, atherosclerosis risk factors, carotidal atherosclerosis*

Materials and Methods

In this study we used the IMT (Intima Media Thickness) measurement index. It measures the thickness of intima caused by cholesterol deposition in intima and the proliferation of muscle cells in media.

Over the course of year 2005, 133 hospitalized patients that presented symptoms suggesting vertebral-basilar circulatory insufficiency, have been examined for extra cranial carotidal atherosclerotic changes. Carotidal Echo-Doppler (B module, pulse and color Doppler) was used to make the measurements. We took into consideration the

number of atherosclerotic plaques present in the studied carotidal segment as well as the presence of some of the classical coronary risk factors: hypertension, diabetes, hypercholesterolaemia, smoking and obesity (by measuring BMI (body mass index), AI (abdominal index) and AP (abdominal pelvic index). These factors were correlated with ischaemic ECG changes (primary changes of terminal phase and alteration in heart signal transduction) characterized as being either wide spread or localized.

Results and Discussions

In this group of patients, males were outweighed by females (57,4%) in number.

The age distribution for both the females and males showed a higher prevalence of those with ages between 51-60 years (39,84%) (fig. 1).

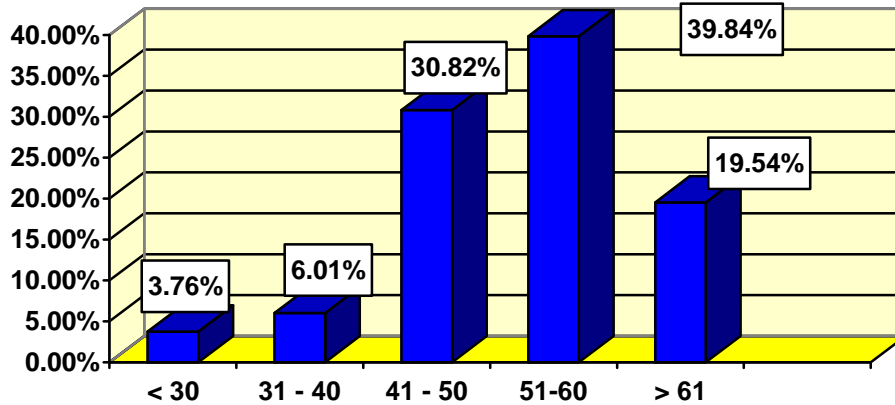


Figure 1. Age distribution of patients

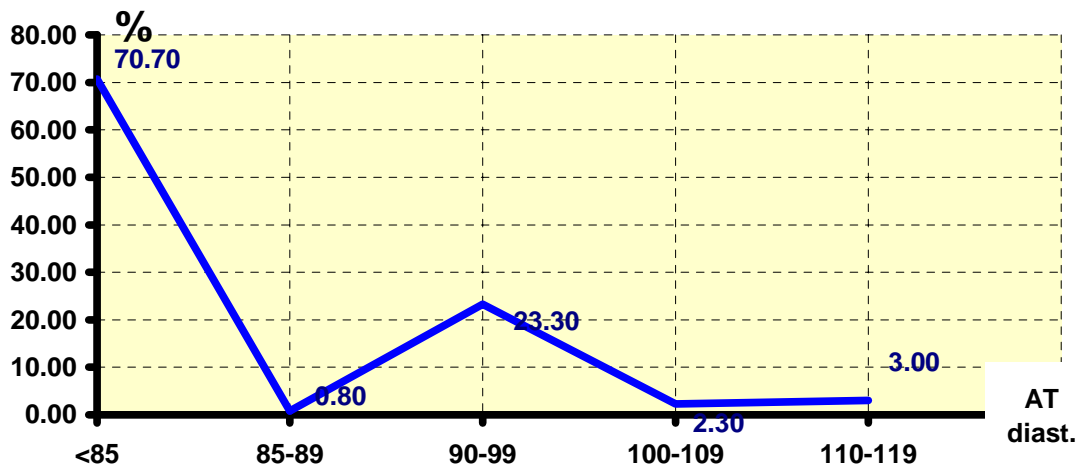


Figure 2. Prevalence of arterial hypertension as a function of diastolic arterial hypertension

Prevalence of arterial hypertension

Systolic arterial hypertension was present in 98 (73,7%) of the patients. Arterial hypertension distribution in patients was as follows, border form was found in 34 (25,6%), mild form in 53 (39,8%), medium level in 9 (68%), and sever form 2 (1,5%) patients.

Diastolic arterial hypertension was present in 39 (29,3%) of the patients. Arterial

hypertension distribution in patients was as follows, border form was found in 1 (0,8%), mild form in 31 (23,3%), medium level in 3 (2,3%) and severe form in 4 (3,0%) patients (fig. 2).

Obesity in studied patients

BMI was normal in 65 (48,9%) patients, 45 of patients (33,8%) were overweight, 21 (15,8%) had degree I obesity and 2 (1,5%) had degree II obesity (table 1, fig. 3).

Table 1. Body mass index

Batch	Nr.	Media	Variance	D.S.	E.S.	Min.	Me.	Max.	Modul	c.V.	e.M
Subjects	133	25,82	14,381	±3,79	±0,32	19,13	25,1	39,21	24,8	±14	±1,2

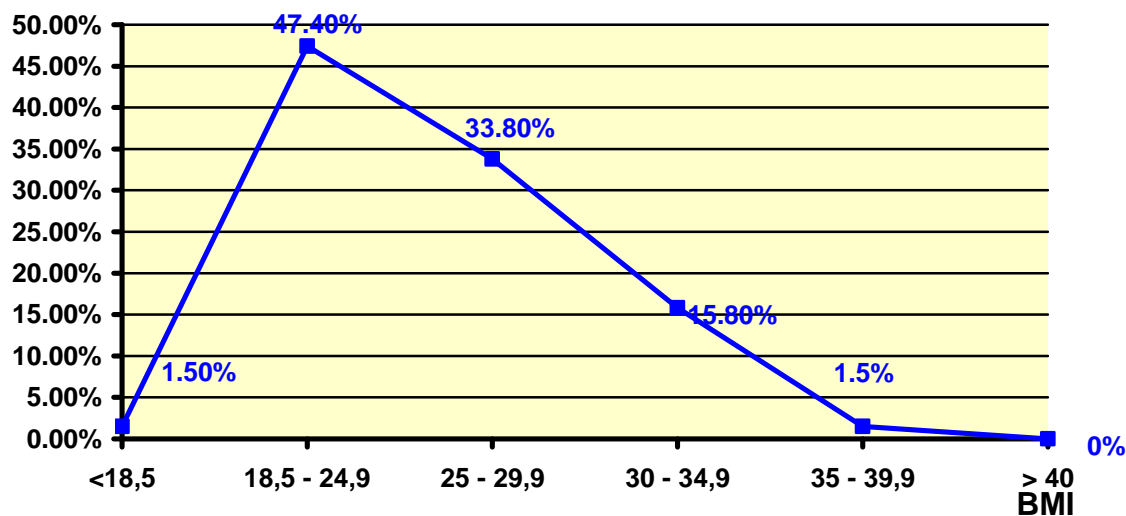


Figure 3. Distribution of obesity as a function of BMI

Abdominal obesity is an independent risk factor that increases cardiovascular risk of obesity. Distribution of abdominal fat in subjects with normal body weight is called abdominal distribution of adipose tissue and it also increases cardiovascular risk. Currently, abdominal obesity is quantified by using AI-

abdominal index or AP- abdominal pelvic index. We used both to discriminate between any possible differences (table 2,3).

Averages of AI and AP

Average AP- abdominal pelvic index was 0,919.

Table 2. Abdominal pelvic index

Batch	Nr.	Media	Variance	D.S.	E.S.	Min.	Me.	Max.	Modul	c.V.	e.M.
Patients	133	0,919	0.005	±0,07	±0,006	0,70	0,92	1,38	0,90	±8	±0,6

Table 3. Abdominal index

Bach	Nr.	Media	Variance	D.S.±	E.S.	Min.	Me.	Max.	Mo-dul	c.V.	e.M
Patients	133	0,54	0,011	0,10	0,009	0,40	0,53	1,53	0,48	±19	±1

Average abdominal index was over the normal limit value (0,543), having a normal average variance but lacking homogeneity. By comparing both indices, it was clear that abdominal index is more sensitive in detecting a higher number of subjects with abdominal obesity. This is an expected outcome because abdominal index is a ration between two

surface areas, whereas the abdominal pelvic index is done by measurements of different nature. Abdominal obesity, when using abdominal index, was more frequent in people of age under 45 (31 subjects - 73, 8%) than in those with age over 45 (11 subjects - 26, 2%) (p<0,009).

Prevalence of both abdominal obesity and distribution of abdominal tissue

The abdominal pelvic index was pathologic in 42 subjects (31,6%). The abdominal index was pathologic in 98 (73,7%) subjects (Figure 4). There are important differences in pathological values of indexes and so we can ask which one of the two can be given more recognition. Due to prevention, it is desirable to use an index that is more

sensitive in showing any pathological changes by indicating values that are over normal range. Therefore abdominal index is more advantageous. Due to the fact that recently it was proposed that it is enough to measure the abdominal circumference to appreciate the degree of obesity, without using a correction due to height, it is possible to use abdominal index alone.

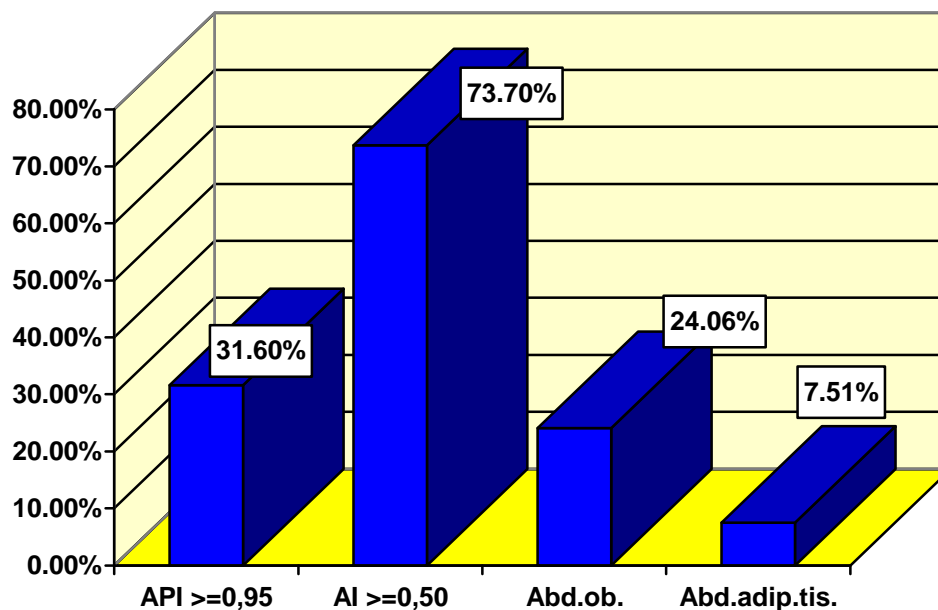


Figure 4. Abdominal pelvic index $\geq 0,95$, abdominal index $> 0,50$, and prevalence of abdominal obesity and abdominal adipose tissue distribution

Abdominal pelvic index was more relevant to show the dependence on the systolic arterial hypertension and abdominal index. It was more useful when we analyzed the dependence on diastolic arterial hypertension, smoking, blood cholesterol level, and disturbances in regulation of glycolysis.

To identify both an ongoing abdominal obesity and the distribution of abdominal adipose tissue, we need to correlate BMI with

abdominal pelvic index. The BMI with values over 25, associated with abdominal pelvic index $\geq 0,95$ for men, defines abdominal obesity. The same BMI but with normal abdominal pelvic index can be used to indicate the distribution of abdominal adipose tissue.

Ischaemic heart disease and obesity

Ischaemic heart disease in those under study had the following distribution: 6 (27,3%) for those with normal body weight,

11 (50%) for those overweight and 5 (22,3%) for those with obesity gr I (Kruskal - Wallis $p < 0,03$). We can conclude that ischaemic heart disease is dependent on body weight, overweight state being the most important one. The above data show that obesity is implicated in the development of ischaemic heart disease.

Ischaemic heart disease and abdominal obesity

In patients with ongoing ischemic heart disease 20 (90,9%) had abdominal obesity, quantified with abdominal index, and 2 (9,1%) had no abdominal obesity (fig.5). It is possible to calculate a relative risk of 3.57 before those with abdominal obesity could be considered as being able to develop ischemic heart disease. (Mantel - Haenszel $p < 0, 04$).

Patients with ongoing ischaemic heart disease had an abdominal pelvic index of 10 (45- 5%). This data shows that there is a correlation between ischaemic heart disease and the abdominal pelvic index. (Mantel - Haenszel $p > 0, 1$).

Because ischaemic heart disease is well correlated with abdominal index and show no dependence on the abdominal pelvic index, we can preferably use only these two: the abdominal pelvic index as well as BMI.

Prevalence of smoking

We found 35 (26,31%) were smokers. Most of them are in age group 45-49, the rest of them in 40-44. This is to their disadvantage because after 45 years of age, risk factors have a cumulative effect and the age itself becomes a risk factor too for ischaemic heart disease. Most of the smoker were male. ($p < 0,001$).

Average cholesterol blood level from a non-homogenous sample was 196,880mg/dl. The Oradea FRCI study showed that average cholesterol blood level in Bihor County was 207mg/dl. It was lower than what we determined in mechanical workers, but higher than what we found for mechanical assistants and those in control group (Table 4).

Table 4. Average cholesterol blood level of studied patients

Batch	Nr.	Media	Variance	D.S.	E.S.	Min	Me.	Max	Modul	c.V.	e.M.
Patients	133	196,88	1905,6	$\pm 43,6$	$\pm 3,7$	110	191	363	150	$\pm 22,1$	$\pm 1,9$

Prevalence of hypercholesterolaemia

57 (47,9%) of those under study had values of cholesterolemia higher than 200 mg/dl. This is a higher value than that found by Hâncu and all. in 1997 (41,2%) (Figure 5).

In Western Europe, the lowest levels of hypercholesterolaemia are found in Spain (36%); Italy having the highest levels (58%). Cholesterolaemia of 250mg/dl and higher

were found in 20 (15,03%) subjects. Evidently, cholesterolaemia shows important regional variations and is very much dependent on intake habits of people.

Ischaemic heart disease and cholesterolaemia

14 (63,6%) patients with ongoing ischemic heart disease had hypercholesterolaemia whereas 8 (36,4%) subjects had normal blood levels of cholesterol.

Hypercholesterolaemia increases the ischaemic heart disease risk with 2.33 times (Mantel - Haenszel $p < 0,04$). Higher blood

cholesterol levels in the group being studied, shows that they have an increased risk of developing ischemic heart disease.

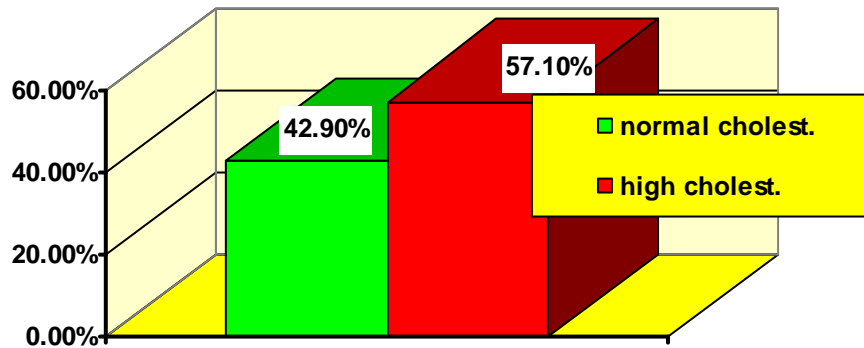


Figure 5. Prevalence of hypercholesterolaemia (>200 mg/dl)

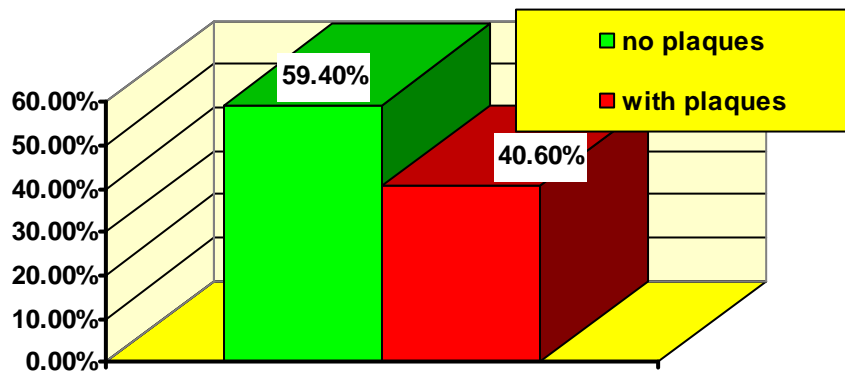


Figure 6. Prevalence of Atherosclerotic carotid plaques

Ischaemic heart disease and smoking

The results of Kruskal - Wallis ($p < 0,003$) test proves that development of ischaemic heart disease is also dependent on smoking.

Atherosclerotic carotid plaques and ischaemic heart disease.

54 (40,60%) subjects had atherosclerotic plaques. Out of them, 76,5% had atherosclerotic plaques in different locations of vessels. Non-specific changes related to atherosclerotic process were not taken into account (Figure 6).

These changes were clearly prevailing in subjects with ages ranging from 51 to 60 years. This group was followed by those with ages over 60 years.

ECG changes, related to ischaemia and its complications (including necrosis and BRS in 8 patients), were detected in 78 (58,64%) patients. 46,8 % of them had ECG changes that showed a wide spread ischaemia of either anterior wall or the other two walls. Any changes of ECG related to HVS were not taken into account. ECG changes were significant in patients in age group 51-60 years as well as in those over 61 years of age.

The presence of carotid atherosclerotic plaques was significantly correlated with ECG ischemia, age (51-60 years), hypercholesterolaemia, AI, BMI and diabetes. There was no correlation between of carotid atherosclerotic plaques and API, smoking and sex. A mild correlation was identified between of carotid atherosclerotic plaques and arterial hypertension. The number of plaques was also significantly correlated with the degree of ischaemic changes.

Conclusions

The abdominal pelvic index was more relevant in detecting the influence of systolic arterial hypertension. Abdominal index was

more useful for analyzing the influence of diastolic arterial hypertension, smoking, cholesterol, and disturbances in regulation of glycolysis.

Because ischaemic heart disease is well correlated with abdominal index and showed no dependence on the abdominal pelvic index, we think that it is better to use only the abdominal pelvic index and BMI.

Multiple regression made for all variables including atherosclerotic plaques showed a better predictive value than excluding this last variable.

These and other risk factors have predictive value for ischaemic heart disease.

REFERENCES

1. **Burke AP, Farb A, Malcom GT.** Coronary risk factors and plaque morphology in men with coronary disease who died suddenly. *N Engl J Med* 1997; 336(18): 1276-82.
2. **Alexander C M, Landsman PB, Grundy SM.** The influence of age and body mass index on the metabolic syndrome and its components (p 246-250) *Diabetes, Obesity and Metabolism*, Volume 10 Issue 2008; 3: 185-270.
3. **Davies MJ.** The pathophysiology of acute coronary syndromes. *Heart* 2000; 83(3): 361-6.
4. **Denke MA, Grundy SM.** Dyslipoproteinemias/ atherosclerosis: dietary therapy. In: Smith T, ed. *Cardiovascular Therapeutics*. Philadelphia: WB Saunders. 1996; 385-402.
5. **Diaz MN, Frei B, Vita JA.** Antioxidants and atherosclerotic heart disease. *N Engl J Med* 1997; 337(6): 408-16.
6. **Farmer JA, Gotto AM.** Dyslipidemia and other risk factors for coronary artery disease. In: Braunwald E, ed. *Heart Disease: A Textbook of Cardiovascular Medicine*. 5th ed. Philadelphia: WB Saunders. 1997; 1126-60.
7. **Gaziano JM.** Epidemiology of risk-factor reduction. In: Loscalzo J, Creager MA, Dzau VJ, eds. *Vascular Medicine*. Boston, Mass: Little, Brown 1996; 569-586.
8. **Kannel WB.** Contributions of the Framingham Study to the conquest of coronary artery disease. *Am J Cardiol* 1988; 62(16):1109-12.
9. **Labarthe DR.** Cardiovascular diseases: a global public health challenge. In: Labarthe DR, ed. *Epidemiology and Prevention of Cardiovascular Diseases*. Gaithersburg, Md: Aspen Publishers, Inc 1998; 3-16.
10. **Levine GN, Keaney JF Jr, Vita JA.** Cholesterol reduction in cardiovascular disease. Clinical benefits and possible mechanisms. *N Engl J Med* 1995; 332(8): 512-21.
11. **Libby P.** Atherosclerosis. In: Fauci A, et al, eds. *Harrison's Principles of Internal Medicine*. 14th ed. New York: McGraw-Hill Inc, 1998; 1345-52.
12. **Libby P.** Changing concepts of atherogenesis. *J Intern Med*. Mar 2000; 247(3): 349-58.
13. **Multiple Risk Factor Intervention Trial Research Group.** Relationship between baseline risk factors and coronary heart disease and total mortality in the Multiple Risk Factor Intervention Trial. Multiple

Risk Factor Intervention Trial Research Group. *Prev Med* 1986; 15(3): 254-73.

14. National Cholesterol Education Program. Summary of the second report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel II). *JAMA* 1993; 269(23): 3015-23.

15. Ridker P, Libby P. Nontraditional coronary risk factors and vascular biology: the frontiers of preventive cardiology. *J Investig Med* 1998; 46(8): 338-50.

16. Ridker PM. Evaluating novel cardiovascular risk factors: can we better predict heart attacks?. *Ann Intern Med* 1999;130(11): 933-7.

17. Ross R. The pathogenesis of atherosclerosis. In: Braunwald E, ed. *Heart Disease: A Textbook of Cardiovascular Medicine*. Philadelphia: WB Saunders 1997; 1105-25.

18. Ross R. The pathogenesis of atherosclerosis: a perspective for the 1990s. *Nature* 1993; 362(6423): 801-9.

19. Ross R. Atherosclerosis - an inflammatory disease. *N Engl J Med* 1999; 340(2):115-26.

20. Safar, Michel; Frohlich, Edward D. Atherosclerosis, large arteries, and cardiovascular risk/ volume editors, Michel E. Safar, Edward D. Frohlich: Basel; New York: Karger. Series: Advances in cardiology, v. 44, 2007.

21. Selwyn AP, Kinlay S, Libby P. Atherogenic lipids, vascular dysfunction, and clinical signs of ischemic heart disease. *Circulation* 1997; 95(1): 5-7.

22. Susan A Jebb, Jeremy Krebs. Lifestyle Determinants of Obesity, Obesity and diabetes, 2006; 33-47.

23. Weissberg PL. Atherogenesis: current understanding of the causes of atheroma. *Heart* 2000; 83(2): 247-52.

Correspondence Data:

LUIZA DEMIAN

e-mail: demianluiza@yahoo.com

telefon: 0723780612

fax: 0359463544