EFFECTS OF HYPERTENSION ON CORONARY COLLATERAL CIRCULATION AND LEFT VENTRICULAR WALL MOTION IN PATIENTS WITH CORONARY ARTERY DISEASE

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It is generally accepted that coronary collateral developement is proportional to the severity of the arterial obstructive disease. In the present study the relation between systemic hypertension, left ventricular hypertrophy and coronary collateral circulation in patients with coronary artery disease has been investifated.

97 patients with coronary artery disease were enrolled to the study. 44 patients had systemic hypertension, the rest of the patients formed the control group. In all of the patients coronary angiography and contrast left ventriculography was performed, and wall motion index (WMI) was defined. Collateral filling of the obstructed vessels were classified on the presence and extent of distal epicardial coronary artery filling by angiography. Echocardiography was performed in every patient and interventricular wall thickness was measured, thickness more than 1.1 cm was accepted as hypertrophic interventricular septum. Coronary collateral index (CCI) was found to be 2.36±0.65 in the hypertensive group and 1.65±1.04 in the control group ($p<0.05$). In subgroup analysis the mean CCI was 2.18±0.62 in hypertensive subjects with an occluded LAD and 1.18±0.95 respectively ($p=0.001$, $p<0.005$).

The effect of the degree of coronary collateral circulation (CCC) was significant by different in the two groups ($p=0.008$, $p<0.05$). There was a significant difference in the relation between CCC and type of occluded artery ($p=0.008$, $p<0.05$). Mild correlation was found between CCC and thickness interventricular septum ($r=0.60$, $p=0.001$, $p<0.005$). The results indicate that patients with hypertension and CAD who have one occluded coronary artery, have an increase in CCC corresponding to the degree of left ventricular wall thickness and also supply protected left ventricular wall motion.

Key words: *Coronary collateral circulation, coronary artery disease, left ventricular wall motion.*

Coronary collateral vessels are the alternative coronary blood reserves to the myocardium in the region perfused by the occluded epicardial coronary artery. In the normal human heart, a network of tiny vasculary channels exists within the myocardium and these channels interconnect with one another as well as with the major coronary arteries. Imbalance of myocardial oxygen supply and demand over time may induce changes in some of these collateral channels, resulting dilatation and transformation into larger vessels with greater blood-carrying capacity[1-4].

It is generally accepted that coronary collateral development is proportional to the severity of the arterial obstructive disease. Many investigators have also reported that in the presence of complete arterial occlusion 80% of patients have collateral vessels supplying the distal segment of the occluded artery[5]. However, some patients with totally occluded coronary arteries do not have any evidence of angiographic visualisation of distal part of the occluded artery despite the favourable pressure gradient between the donating and receiving coronary artery. This can be depend on diffuse atherosclerotic disease in the distal part of the occluded artery, genetically differences and inadequate collateral development[5].

In clinical practice, although there is a total occlusion of major coronary artery or the history of myocardial infarction, we can face with normal left ventricular function or without Q vawe. This is the result of coronary collateral circulation[6-8].

In the present study, we have investigated the coronary circulation in relation to the presence of systemic hypertension and left ventricular hypertrophy in patinets with coronary artery disease.

METHODS

We enrolled 97 patients to the study who underwent diagnostic coronary angiography and had total luminal obstruction in one of three major coronary arteries. 44 patients had systemic hypertension which formed the study group and rest of them were the control group. Patients were classified in to two groups: those with and without hypertension and also subgroups were defined according to the occluded artery; LAD or RCA. The diagnosis of hypertension was made if the diastolic pressure was > 90 mmHg, and systolic > 150 mmHg.

Coronary Angiography

Coronary angiography and contrast left ventriculography was performed and wall motion index (WMI) was defined by giving scores to the degree of contractility and abnormal segment motion (Normal :0, each hypokinetic segment:1, each diskinetic or akinetic segment:2, aneurysmatic segment:3)

Collateral filling of the obstructed vessels was classified based on the presence and extent of distal epicardial coronary artery filling by coronary angiography, as follows; (0:no filling, Class1: filling of the side branches, Class 2: partial filling of the epicardiial segment, Class 3: complete filling of the epicardial segment)

Echocardiogaphy

Echocardiography was performed using Ving-Med CFM 800. M-mode and 2D echocardiography recordings analysed by a second investigator who was unaware of the clinical characteristics of the subjects. Interventricular wall thickness was measured by using American Society of Echocardiography (ASE) criteria and >1.1 cm thickness was accepted hypertrophic interventricular septum[9].

Statistical Analysis

All parametric data are expressed as mean ±SD. As a statistical method, ANOVA, linear regression analysis, 2-tailed t test and chi-square tests were used. $p<0.05$ was considered significant.

RESULTS

Patients characteristics were shown in Table 1. There was no statistically significant

differences between hypertansive and control groups with respect to following parameters; age, sex and atherosclerotic risk factors. Coronary collateral index (CCI) was found mean 2.36±0.65 in hypertensive group and 1.65±1.04 in control group. Significant differences was found between two groups ($p<0.05$). In subgroup analysis the mean CCI was found 2.18±0.62 in hypertensive patients with occluded LAD and 1.18± 0.95 in normotasive patients. Significant differences was also found between two mean values ($p=0.001$, $p<0.005$) (Figure 1-2).

The effect of the degree of coronary collateral circulation (CCC) was compared between hypertensive and control group (Table 2). Statistically significant differences were encountered ($p=0.008$, $p<0.05$)(Figure 3).

The relation between (CCC) and type of the occluded artery is shown in Table 3. There was statictically significant differences between the two groups ($p=0.008$, $p<0.05$) (Figure 4). Mild correlation was found between CCI and thickness of interventricular septum ($r=0.60$, $p=0.001$, $p<0.005$), and also negative mild correlation was found between CCI and WMI ($r=0.68$, $p=0.001$).

Important differences were found in mean CCI values in patients with totally occluded RCA subgroup ($p=0.52$, $p>0.05$). When the total occluded vessels were compared for determining CCI. A significance was found in right coronary artery group ($p=0.001$, $p<0.05$). Statistically significant differences were found

Table I. Patients Characteristics.

Patients (n= 96)	**Hypertensive Group (n= 44)**	**Non-Hypertension Group (n= 52)**
Age	53 ± 10	52 ± 8
Male/ Female	38: 6	43 : 9
RCA (% 100 Occ.)	17 (39%)	19 (36%)
LAD (% 100 Occ.)	27 (61%)	33 (64%)
Blood cholesterol > 250	11 (24%)	9 (18%)
Diabetes mellitus	5 (11%)	8 (15%)
Cigarettes smoking	35 (79%)	34 (65%)
Family history	12 (27)	17 (32%)
Systolic blood pressure (mmHg)	165 ± 27	112 ± 10
Diastolic blood pressure (mmHg)	99 ± 22	75 ± 7
Echocardiographic septal diameter (cm)	1.23 ± 0.09	1.01 ± 0.08
MI history	19 (43%)	35 (67%)
Q wave on ECG	18 (41%)	37 (71%)

Table 2: Comparement of the degree of CCI in patients with and without hypertension.

CCI	**HT** (44 patients)		**Normotansive** (52 patients)	
	n	**%**	**n**	**%**
0	0	(0)	8	(15)
I	4	(10)	8	(31)
II	20	(45)	14	(27)
III	20	(45)	14	(27)

Table 3: The degree of CCI in LAD and RCA groups.

CCI	**LAD** (60 patients)		**RCA** (36 patients)	
	n	**%**	**n**	**%**
0	8	(13)	0	(0)
I	18	(30)	2	(6)
II	22	(37)	12	(33)
III	12	(20)	22	(61)

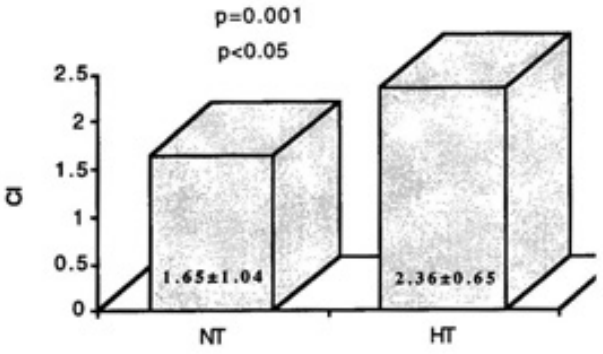


Figure 1. CI values in HT and NT group.

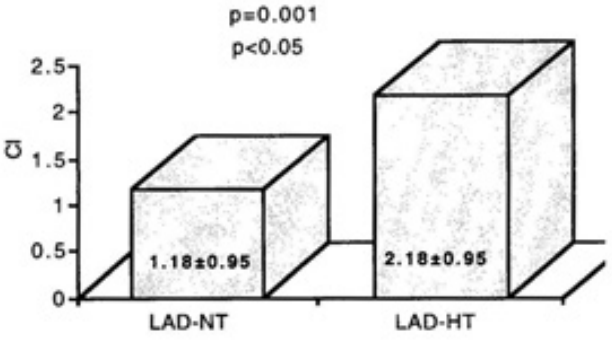


Figure 2. CI values in LAD-NT and RCA-HT subgroups.

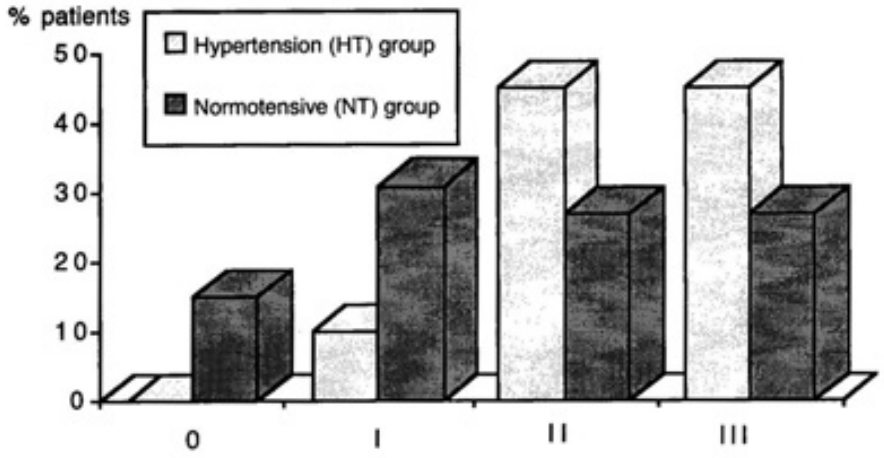
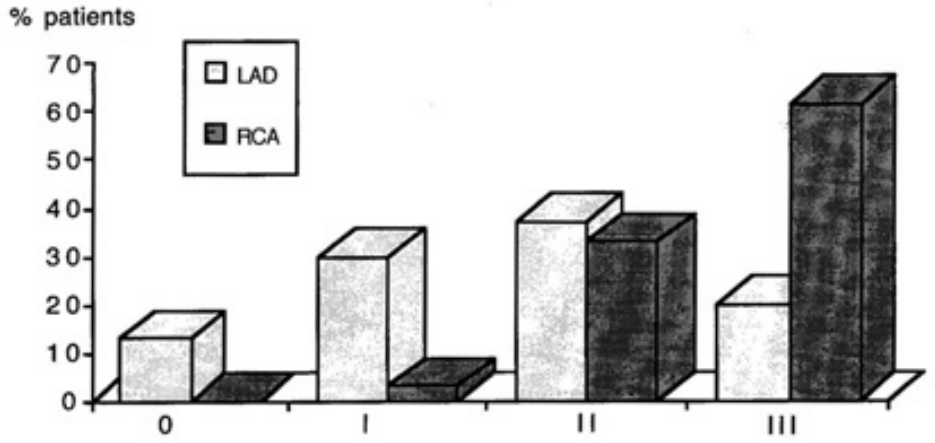


Figure 3:
The degree of coronary collateral development in HT and NT group.

Figure 4:
The comparement of coronary collateral development in LAD and RCA group.



between groups and subgroup analysis in the comparement of differences of WMI values. Also in the evaluation of subgroups which were separated by occluded vessels, the differences in mean WMI values was found important in LAD occluded subgroups ($p=0.001$, $p<0.05$).

DISCUSSION

In our study, CI values was significantly higher in coronary artery disease patients with hypertension (HT) than without HT. In the literature, there is only two studies that has searched the relation between HT and coronary collaterals[10-11]. Zenon studied on a group who had 95% or higher coronary artery stenosis in LAD or RCA. Besides this the other coronary arteries had no critical stenosis[10]. In Karpanaou study; patients who had the same perfusion scores were separated to 1, 2 and 3 vessels disease and groups were compared with each others[11]. In these two studies, although the patient selections were different from our study, our results was similar with them.

Coronary collaterals was obtained 70.6% in HT patients and 46.8% in non-HT group.

Patients who had one occluded vessel (LAD or RCA) was enrolled to the study because of determining the development of collateral vessels in the ischemic myocardial areas, the vessel that was taking the collaterals must be totally occluded.

In the comparement of otal occluded LAD and RCA, the development of coronary collateral (CC) in RCA was better than LAD in the Karpanou and Kyriakides studies[10,11]. In the source of this, metabolic, humoral, neurologic and right ventricular pressure difference effect the RCA resistance and CC development. In systole, the pressure of right ventricule is lower than left ventricle; so in systole right ventricular perfusion continue despite left ventricle. This is why RV have better CC than LV. In our study, CI values was found significantly higher in HT patients with totally occluded LAD group. However CI values of RCA occluded HT patients was different from control group. Results also showed that during HT period, coronary collateral development accompanying increasing in left ventricular muscle, but HT did not promote right ventricular hypertrophy. This might suggest RCA dependent collateral growth in HT. Some reports assert that CC has no positive affect on left ventricular wall motion, but they have many study limitations such as; groups are separated with the existence of collateral development rather than the degree of well developed CC[12-15]. Helfert, Cally and Robin took into account only the collateral vessels filling, but preservation of viable myocardium in the infarct related coronary artery region not only existened with collateral vessels but also filling of the distal segment of the occluded artery[12]. Also in these studies patient study group consisted of 75% or higher stenosed vessels were determined for the development of CC. Althogh 75-90% luminal narrowing in coronary artery could cause an ischemia or stimulate collateral growth, this was not severe enough to promote angiographically visualised collaterals and retrograde filling. We took into account the degree of the occluded artery filling in the determination of CC. We obtained normal left ventricular function in 59% of the patients with well developed CC and 89% of the patients with insufficent CC had ventricular wall motion abnormalities (diskinesia, akinesia and aneurysm). These results were similar with Levine[5] study. In our study, 10% of HT patients had no collateral vessels. This might depend on genetically differences, diffuse atherosclerotic changes in the distal portion of the occluded artery. All of those patients had ventricular aneurysm.

In acute phase of myocardial infarction, well developed CC is not a rule, but CC could be seen in 40% of patients during the infarction[16]. However fixed coronary stenosis will usually stimulate collateral growth with luminal narrowing of more than 80-90%. Total occlusion may also act as an initiating event. Of these events, gradual and intermittent occlusion appears to be most effective in generating collateral growth. The frequency and duration of ischemic stimuli are critical and must be sufficent to initiate cellular and biochemical changes that will ultimately act in collateral development. After acute phase of the myocardial infarction, CC incidence can be 75-100% in total occlusion, 17-42% in

subtotal occlusion of the infarct related artery[17].

In 6 patients with no -or poor- CC of infarct related artery had a normal left ventricular wall motion. It might depend on technical and machinary limitation of coronary angiography such as: film quality, visualisation of the distal coronary filling in late period and not to repeating the angiography after nitroglyserin injection.These results indicate that patients with hypertension and CAD who had occluded one coronary artery, have an increase in CC corresponding to the degree of left ventricular wall thickness, and also supply protected left ventricular wall motion.

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