Does Collateral Circulation Protect Against Acute Myocardial Infarction? Two Hemodynamically Important Collateral Circulation Examples: Case Report

Kollateral Dolaşım Akut Miyokard İnfarktüsüne Karşı Korur mu? Hemodinamik Olarak Anlamlı İki Kollateral Dolaşım Örneği

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ABSTRACT Coronary collateral vessels interconnect the major coronary arteries. They are small in (<200 μm) caliber. Collateral channels can not be seen in patients with normal or mildly diseased coronary arteries. The coronary collateral circulation is a complementary source of blood supply to myocardium jeopardized by stenosis of a coronary vessel. If they are adequate size, collaterals may protect against myocardial infarction in cases of total occlusion. The myocardial infarct size is smaller in patients with abundant collateral vessels than in patients without collaterals. Therefore total occlusion of a major epicardial artery may not lead to left ventricule dysfunction. Presented cases are important examples that the myocardium was protected against left ventricule dysfunction owing to total occlusion of one or more major coronary arteries.

Key Words: Collateral circulation; coronary angiography; acute coronary syndrome

ÖZET Koroner kollateral damarlar major koroner arterleri birbirine bağlar. Kalibrasyonları <200 μm daha küçüktür. Kollateral kanallar normal veya hafif daralmış koroner arterlerde görülemeyebilirler. Kollateral damarlar, tıkalı koroner arterin tehdit ettiği miyokarda kan sağlanmasında tamamlayıcı kaynaktırlar. Kollateraller eğer yeterli büyüklükte ise, tam tıkanma durumunda akut miyokard infarktüsüne karşı koruyabilirler. Gelişmiş kollateral damarları olan hastaların, kollaterali olmayanlara göre infarkt alanı daha küçüktür. Dolayısıyla bir major koroner arterin tam tıkanması sol ventrikül disfonksiyonuna neden olmayabilir. Sunulan olgular, bir veya daha fazla major koroner arterin tam tıkanmasının neden olduğu sol ventrikül disfonksiyonuna karşı miyokardı korunmuş önemli örneklerdir.

Anahtar Kelimeler: Kollateral dolaşım; koroner anjiyografi; akut koroner sendrom

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The coronary collateral vessels that interconnect major coronary arteries maintain myocardial perfusion despite the development of severe proximal atherosclerotic narrowing.[1] These channels may not be seen in normal or mildly diseased patients, but as coronary artery disease progresses and becomes more severe (>90 percent stenosis), a pressure gradient is generated between the anastomotic channels.[2] This gradient facilitates blood flow through the channels, which progressively dilate and finally become visible as collateral vessels. There is a great individual variability in the function of coronary collaterals among patients with chronic

stenosis. A hemodynamically important coronary collateral supply has a beneficial impact on the occurence of major ischaemic events.[3]

CASE REPORTS

CASE 1

A 42-year-old male patient, suffering from stable angina pectoris for 15 days was referred for coronary angiography. His coronary risk factors were diabetes mellitus lasting for two years, three packages/day cigarette smoking, high low density cholesterol (LDL-c: 189 mg/dl) and fibrinogen (500 mg/dl) levels. Electrocardiography revealed sinus rhythm and inferolateral 1 mm ST segment depression. Echocardiography was normal.

Coronary angiography revealed a totally occluded left anterior descending coronary artery (LAD), and critical stenosis of proximal portion of left circumflex (LCx) and right coronary artery (RCA). Additionally a great anastomosis between a proximal portion of the RCA and the distal site of the LAD was detected. In Figure 1, on right cranial projection, a great and hemodynamically important collateral vessels, like the main vessels between proximal RCA-distal LAD were detected. It was an important connection in terms of hemodynamics, because the echocardiography did not show any abnormal left ventricular contraction, despite total occlusion of the proximal LAD. Furthermore, Thrombolysis In Myocardial Infarction 2 retrograde flow was detected in the LAD. Patient was normal in resting position but symptomatic during exercise or daily ordinary activities. Total revascularization coronary bypass surgery was planned.

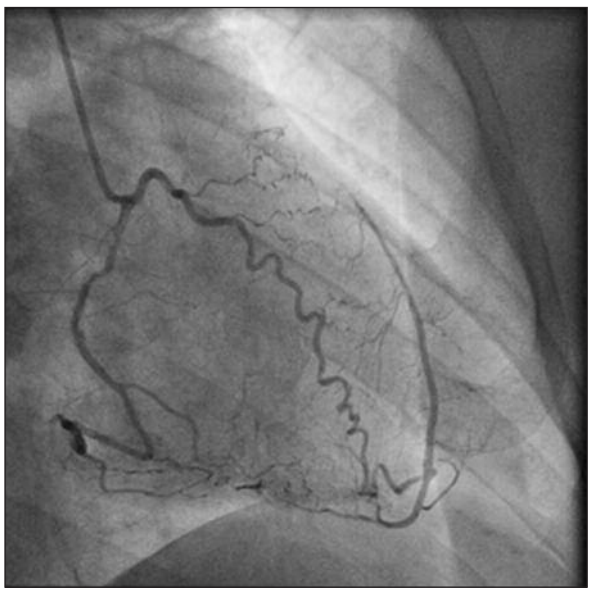

FIGURE 1: A great and hemodynamically important collateral vessel, like the main vessels, between proximal RCA-distal LAD on right cranial projection.

CASE 2

A 70-year-old female patient suffering from cholelithiasis and concomitant typical class 2-3 stable angina pectoris, according to Canadian classification, was referred for coronary angiography to determine the risk of the non-cardiac operation. She had no risk factors or familial genetic predisposition. Levels of LDL-c, fibrinogen, and hs-CRP were 112 mg/dl, 412 mg/dl, and 3.22 mg/lt respectively. Troponin T level was normal. Electrocardiography revealed atrial fibrillation and non-spesific ST/T segment abnormalities. Blood pressure and heart rate were normal. Although 2° mitral and aortic insufficiency and left atrial dilatation were detected on echocardiography, no significant ventricular contraction abnormality was revealed.

Coronary angiography revealed total occlusion of the LAD and 95% occlusion of the proximal portion of the diagonal bundle, along with a normal non-dominant LCx. The RCA was totally occluded at the proximal portion. A developed proximal side branch of the RCA, Kugel artery was interconnected with the posterolateral branch. Coronary angiography also revealed bridge collaterals between the proximal and distal portions of the RCA. A well developed ipsilateral collateral supply between proximal RCA-posterolateral branch of the RCA, Kugel artery was detected in Figure 2. This anastomosis was suggested to be an important interconnection in terms of hemodynamics. Although there was total occlusion of two major coronary arteries, the patient was asymptomatic in resting but became symptomatic during effort. Any contraction abnormality

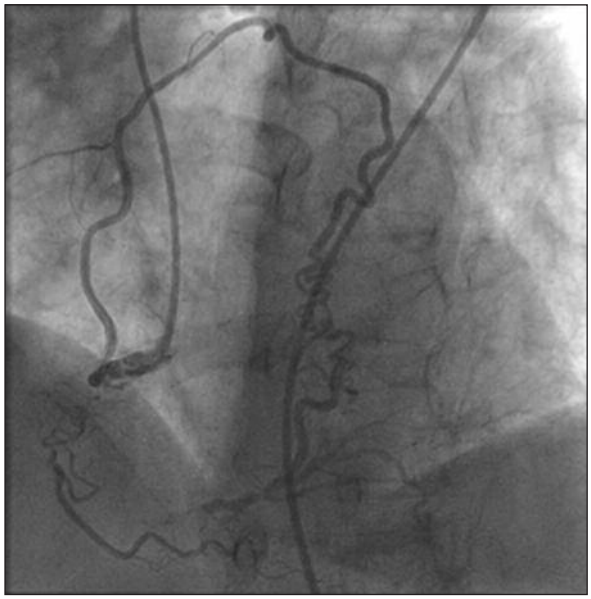

FIGURE 2: A well developed ipsilateral collateral supply between proximal RCA-posterolateral branch of the RCA, Kugel artery.

was not detected during echocardiographic examination. Therefore, total revascularization coronary bypass surgery was scheduled before non-cardiac surgery.

DISCUSSION

Coronary collateral vessels are potential channels which protect the effected myocardium against acute coronary ischaemia (ACS), if they are adequate in size. Flameng et al. reported that, a well developed collateral circulation supplies an equal perfusion with a coronary artery that has 90% stenosis.[4] This can protect the patient during ST elevation acute myocardial infarction (STEMI) or ACS.

In patients with chronic total occlusion of a major coronary artery without infarction, collateral-dependent myocardial segments show nearly normal baseline blood flow and oxygen consumption, but severely limited flow reserve. This finding helps explain the ability of collaterals to protect against resting ischemia but not against exercise-induced angina. Also, the size of the myocardial infarction is typically smaller than in patients without collaterals.[5]

A lot of factors that effect the development of coronary collateral circulation were determined. While diabetes mellitus and hypercholesterolemia have a negative effect on development of the coronary collateral circulation, preinfarction angina and severity of coronary stenosis affect it positively.[6-8] In our first case, although the patient has diabetes mellitus lasting for two years, he had a good proximal RCA-distal LAD anastomosis that had protected him against ACS.

The relationship between the collateral development and hypertension, which is an important risk factor for coronary artery disease, is not clear. Kyriakides et al, suggested that, hypertension has a positive effect on development of the collateral circulation, especially if left ventricular hypertrophy was present.[9]

Also, collateral circulation may be changed due to the infarct related coronary artery. It has been shown that, collateral development is better in acute RCA stenosis than other coronary arteries in early phases of acute myocardial infarction.[10]

Some biochemical factors like asymmetric dimethylarginine (ADMA) which is a competitive inhibitor of nitric oxide synthetase and arises from the methylation and hydrolysis of arginine residues during protein degradation effects the collateral development. It was showed that the relationship between poor coronary collateral growth and ADMA which inhibits angiogenesis on experimental studies on coronary collateral growth.[11]

In present two cases, the collateral vessels are well developed, almost as native coronary arteries. Therefore patients have been protected against ACS or STEMI.

In conclusion, coronary collateral circulation is an important entity during ACS or STEMI to protect myocardium against infarction, arrhythmia, angina, left ventricular dysfunction and sudden cardiac death.

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