

Miyokardiyal Köprüleşmenin Neden Olduğu Miyokard İnfarktüsü

ACUTE MYOCARDIAL INFARCTION RESULTING FROM MYOCARDIAL BRIDGING

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Özet

Koroner arterlerin miyokardiyal köprüleşmesi tesadüfen saptanan anjiyografik bir bulgu olarak düşünülmektedir, fakat birkaç rapor angina pectoris, miyokard iskemisi hatta infarktüsü ile ilişkisini göstermiştir. Burada biz, akut inferior miyokard infarktüsü ile başvuran ve anjiyografisinde sirkumfleks arterin orta kısmına yerleşmiş izole miyokardiyal tünelleşmenin olduğu genç bir hastayı sunmaktayız.

Anahtar kelimeler: İzole miyokardiyal köprüleşme, miyokard infarktüsü ve koroner anjiyografi

Summary

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Myocardial bridging of coronary arteries has been considered as an incidental angiographic finding; however, several reports suggest its association with angina pectoris, myocardial ischemia and even infarction. Herein we described a young patient presented with acute inferior myocardial infarction and whose coronary angiography showed isolated myocardial bridging located to middle portion of circumflex artery.

Keywords: Isolated myocardial bridging, myocardial infarction and coronary angiography

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Introduction

The muscle overlying the intramyocardial segment of epicardial coronary artery is termed as myocardial bridging (MB). It usually has a benign course, as coronary narrowing occurs during systole and coronary flow mostly occurs during diastole, but some reports showed that it might be associated with myocardial ischemia, infarction, atrio-ventricular block and sudden death [1-4]. In this article, we described a case presented with acute inferior myocardial infarction (MI) and whose coronary angiography revealed isolated MB located to middle portion of circumflex artery (Cx).

Case

M.S. a 47-year-old man presented to emergency department with 8 hours of crushing chest pain. Systematic questioning elicited no further significant symptom. Blood pressure was 150/90 mmHg, heart rate was 76/minute. Examination of the other systems was normal. Risk factors for coronary artery disease included history of smoking (one pack/day, for 20

years), hypertension (for five years) and alcohol consumption (one glass/day, for 10 years). Electrocardiography showed sinus rhythm and was consistent with an acute inferior MI. (Nearly 2 mm ST segment elevation in D2, D3, aVF leads, Figure 1). The patient was taken to coronary care unit (CCU). Aspirin (300mg/day), standard heparin (10000 U bolus and 1000 U/hour infusion), nitroglycerin (10µg/minute) was given. ST segment elevation gradually resolved and Q waves settled in leads DII, DIII, aVL 6 hours after the patient had been admitted to coronary care unit. Cardiac enzymes were slightly elevated at beginning, but elevated to a level more than four times the normal 6 hours later. On the sixth day of admission, the patient was taken to cardiac catheterization laboratory. The left coronary system was imaged at left and right oblique, right cranial and caudal, and antero-posterior cranial positions. Significant systolic luminal narrowing was observed in the mid segment of circumflex artery at right anterior oblique cranial positions. (Figure 2a-b). The right coronary system was normal. Thus, aspirin and beta-blocker were prescribed and the patient was discharged from the hospital as asymptomatic.

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Discussion

MB is often considered to be an incidental angiographic finding because coronary blood flow is a diastolic event, while compression from MB appears to be mostly systolic. However, it has been shown that diastolic flow can be disturbed, particularly at faster heart rates [2]. Moreover, previous reports have showed that MB may be associated with ischemia, myocardial infarction, atrioventricular block and sudden death [1-4]. Myocardial bridges have also been linked to myocardial stunning [5]. Iverson et al. [6] showed the relief of myocardial ischemia, confirmed by angiography and scintigraphic perfusion studies, in 9 patients with symptomatic MB. Teragawa et al. [7] using acetylcholine infusion into the LAD artery performed a spasm-provocation test in patients with chest pain. The authors observed that patients with MB experienced coronary spasm more frequently than patients without MB and concluded that MB increased the risk of coronary spasm [7].

Its usual localization is the middle portion of left anterior descending coronary artery. However, in our case MB had an unusual localization and furthermore it had led to inferior MI.

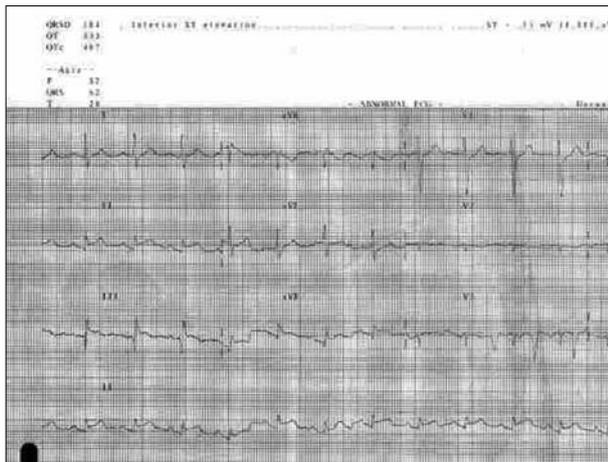


Figure 1. ECG shows acute inferior myocardial infarction.

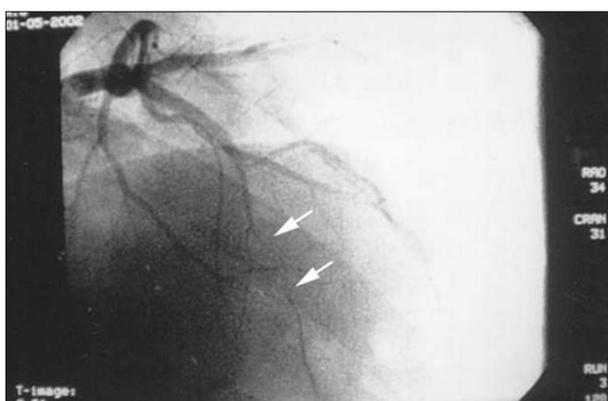


Figure 2a. Systolic compression at the mid-portion of Cx at the right anterior oblique view.

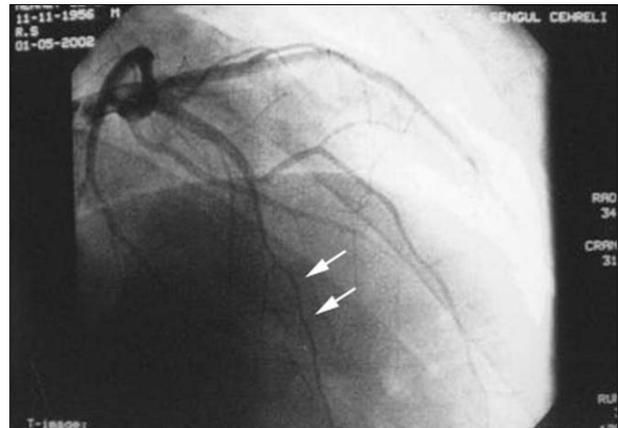


Figure 2b. Normalization of systolic compression at the mid-portion of Cx.

It is well known that main pathogenesis of acute coronary syndromes consists of atherosclerotic plaque disruption and thrombus formation [8]. In MB, although there is no atherosclerotic luminal narrowing, however, endothelial damage and vasospasm and superimposed thrombus at the bridged segment might have triggered the MI. Ciampricotti et al [3] have claimed that endothelial damage might occur because of the continuous mechanical stress, which predisposes the vessel segment to vasospasm. This hypothesis has been supported by intravascular ultrasound study revealing atherosclerotic involvement in MB as well [9]. Intracoronary Doppler measurements have also shown delayed luminal gain and reduced coronary flow reserve, phasic systolic vessel compression with a localized peak pressure, persistent diastolic diameter reduction, increased blood flow velocities and retrograde flow [10]. In addition, history of smoking and hypertension could have contributed to endothelial damage.

The treatment of MB is restricted to symptomatic patients and is based primarily on a pharmacologic approach. Beta-blockers are in general the first-line therapy [11]. The other therapeutic approaches consist of transluminal coronary intervention and surgery. Klues et al. [10] showed that intracoronary stent implantation, prevents external compression and increases in luminal diameter, thus, abolish all of the hemodynamic abnormalities induced by the bridges and improve clinical symptoms and objective signs of myocardial ischemia. Surgery has been the most frequently used method in symptomatic MB. Various techniques have been successfully employed, including coronary artery bypass surgery, either alone or combined with muscle resection, and supra-arterial muscle resection alone [6,12]. However, surgical treatment is worth considering in symptomatic patients when the area of ischemia supplied by the affected vessel is detected and previous medical treatment has been ineffective. In our case, because atherosclerotic luminal narrowing was not detected, percutaneous intervention was not performed. Therefore, we decided to follow the patient medically. In conclusion, MB is frequently thought as incidental angiographic finding but our case, together with those previously published, showed that it is not entirely an incidental finding, on the contrary, it may cause severe cardiac events.

References

1. Agirbasli M, Martin GS, Stout JB, Jennings HS 3rd, Lea JM 4th, Dixon JH Jr. Myocardial bridge as a cause of thrombus formation and myocardial infarction in a young athlete. *Clin Cardiol* 1997;20:581-83.
2. Schwarz ER, Klues HG, Vom DJ, Klein I, Krebs W, Hanrath P. Functional characteristics of myocardial bridging. A combined angiographic and intracoronary Doppler flow study. *Eur Heart J* 1997;18: 434-42.
3. Ciampricotti R, el Gamal M. Vasospastic coronary occlusion associated with a myocardial bridge. *Cathet Cardiovasc Diagn* 1988;14:118-20.
4. den Dulk K, Brugada P, Braat S, Heddle B, Wellens HJ. Myocardial bridging as a cause of paroxysmal atrioventricular block. *J Am Coll Cardiol* 1983;1: 965-9.
5. Chambers JD Jr Johns JP, Berndt TB, Davee TS. Myocardial stunning resulting from systolic coronary artery compression by myocardial bridging. *Am Heart J* 1994;128:1036-8.
6. Iversen S, Hake U, Mayer E, Erbel R, Diefenbach C, Oelert H. Surgical treatment of myocardial bridging causing coronary artery obstruction. *Scand J Thorac Cardiovasc Surg* 1992;26:107-11.
7. Teragawa H, Fukuda Y, Matsuda K, et al : Myocardial bridging increases the risk of coronary spasm. *Clin Cardiol* 2003;26:377-83.
8. Ridolfi RL, Hutchins GM. The relationship between the coronary lesions and myocardial infarct, ulceration of atherosclerotic plaques precipitating coronary thrombosis. *Am Heart J* 1977;93:468-86.
9. De Winter RJ, Kok WE, Piek JJ. Coronary atherosclerosis within a myocardial bridge, not a benign condition. *Heart* 1998;80:91-3.
10. Klues HG, Schwarz ER, vom Dahl J, et al. Disturbed intracoronary hemodynamics in myocardial bridging: early normalization by intracoronary stent placement. *Circulation* 1997;96:2905-13.
11. Schwartz ER, Klues HG, von Dahl J, Klein I, Krebs W, Hanrath P. Functional angiographic and intracoronary Doppler flow characteristics in symptomatic patients with myocardial bridging: effect of short-term intravenous beta-blocker medication. *J Am Coll Cardiol* 1999;27:1637-45.
12. Katznelson Y, Petchenko P, Knobel B, Cohen AJ, Kishon Y, Schacner A. Myocardial bridging: surgical technique and operative results. *Mil Med* 1996;161:248-50.