It was discussed that in a meta-analysis of 17 studies consisting of 20839 patients treated with clopidogrel showed a 2.7-fold higher risk for stent thrombosis (ST) and a 1.5-fold higher risk for mortality following percutaneous coronary intervention (PCI) in HTPR patients (2). However, we found no statistically significant difference between the study and control groups in terms of ST (2.9% vs. 2.6%, p=0.82) and cardiovascular mortality (2.9% vs. 4%, p=0.34) in the first 6-month follow up (1). First of all, in the abovementioned meta-analysis, non-Western patients were excluded from the study because of different pharmacodynamic response to P2Y12-inhibitors across races. In addition, there is no long-term outcome follow-up (just the first month follow up data were available) in 6 of the 17 studies compromising 4694 of 20839 patients. Despite these methodological differences there may be some confounding variables altering our study results as previously mentioned in the limitations section:

One of the major reasons for ST and stent malapposition could not be evaluated in our study because there was no feasibility of IVUS or OCT when the stent deployed. Another issue about ST is that this entity could be affected by the type and size of stent. In our study, as we specified in limitation section, we do not have data enclosing stent size and type (BMS or DES). We accept that not covering stent type and size could have played a role in evaluation of results.

The prevalence of HTPR varies from study to study. There are many reasons for this disharmony: race, dietary habitudes, concomitant drug use, time from clopidogrel ingestion to study platelet functions, technique used, and cut-off levels for platelet reactivity. In our study, platelet functions were studied only once (24 hours after clopidogrel ingestion) and Multiplate analyzer was used. Platelet function assessment more than once, as performed in GRAVITAS (3) trial, could predict more accurate outcomes regarding mortality and ST. Another issue concerning platelet function is cut-off levels of assays. In the GRAVITAS (3) trial, when HTPR cut-off level is chosen as 230 PRU (Verify Now), <230 PRU was not associated with a lower risk of the primary end-point at 60 days [hazard ratio (HR), 0.62; 95% confidence interval (CI), 0.25–1.51; p=0.30] and at 6 months after PCI (HR, 0.71; 95% CI, 0.41–1.23; p=0.22). However, when the cut-off level is chosen as 208 PRU, <230 PRU showed a lower risk of the primary end-point at 60 days (HR, 0.18; 95% CI, 0.04–0.79; p=0.02) and at 6 months (HR, 0.43; 95% CI, 0.23–0.82; p=0.01). In our study, Multiplate analyzer was used and HTPR was defined with a cut-off level of 200 and the area under the aggregation curve as described by the manufacturer. According to a previously conducted study with Multiplate analyzer (4), an ADP test value >468 AU seems to be the optimal cut-off level to separate patients with high risk of stent thrombosis. Our study was conducted to evaluate not only ST but also find the prevalence of HTPR and associated risk factors, and a cut-off level of 200 was more reasonable than 468. However, there could be a more precise conclusion about ST and mortality if we have chosen 468 as the cut-off level.

Adnan Kaya
Department of Cardiology, Faculty of Medicine, Düzce University; Düzce-*Turkey*

References

1. Tekkeşin Aİ, Kaya A, Çakıllı Y, Türkkan C, Hayıroğlu Mİ, Borklu EB, et al. The first six-month clinical outcomes and risk factors associated with high on-treatment platelet reactivity of clopidogrel in patients undergoing coronary interventions. Anatol J Cardiol 2016; 16: 967-73. Crossref
2. Aradi D, Kirtane A, Bonello L, Gurbel PA, Tantry US, Huber K, et al. Bleeding and stent thrombosis on P2Y12-inhibitors: collaborative analysis on the role of platelet reactivity for risk stratification after percutaneous coronary intervention. Eur Heart J 2015; 36: 1762-71.
3. Price MJ, Angiolillo DJ, Teirstein PS, Lillie E, Manoukian SV, Berger PB, et al. Platelet reactivity and cardiovascular outcomes after percutaneous coronary intervention a time-dependent analysis of the Gauging Responsiveness with a VerifyNow P2Y12 assay: Impact on Thrombosis and Safety (GRAVITAS) Trial. Circulation 2011; 124: 1132-7. Crossref
4. Sibbing D, Braun S, Morath T, Mehilli J, Vogt W, Schomig A, et al. Platelet reactivity after clopidogrel treatment assessed with point-of-care analysis and early drug-eluting stent thrombosis. J Am Coll Cardiol 2009; 53: 849-56. Crossref

Address for Correspondence: Dr. Adnan Kaya
Düzce Üniversitesi Tıp Fakültesi, Kardiyoloji Anabilim Dalı
81100, Konuralp, Düzce-*Türkiye*
E-mail: adnankaya@ymail.com

Fractional flow reserve guided stenting of a myocardial bridge

To the Editor,

Myocardial bridging (MB) is a common congenital coronary anomaly. The treatment is debated in symptomatic forms. Percutaneous coronary intervention (PCI) could be a possible solution; however, in these cases the major adverse cardiac event rate is high (1).

A 52-year-old man presented with chest pain provoked by emotional stress. Laboratory tests and transthoracal echocardiography were normal. Treadmill test was indicated according to Bruce protocol that demonstrated silent ischemia at 125 Watts workload. Beta blocker was uptitrated (bisoprolol 2.5–10 mg daily).

Despite the oral medical therapy, the patient remained symptomatic. Coronary angiography showed MB in the mid left anterior descendent artery (LAD) with lumen compression (minimal lumen diameter: 0.26 mm, reference vessel diameter: 2.6 mm, and lesion length: 25.4 mm) but without any atherosclerotic lesions. A fractional flow reserve (FFR) measurement proved significant myocardial ischemia (Pd/Pa=0.69). After FFR measurement, the lesion was stented with a 3.0×38 mm paclitaxel eluting stent (Promus Premier, Boston Sci, US) at 14 atm. Control angiography

revealed good angiographic result, and final FFR (Pd/Pa=0.96) verified improved hemodynamics. After the procedure, the patient had no complaints and at the 18-months control multislice CT angiography excluded the restenosis.

Drug eluting stent implantation with a longer stent than the visible bridge was safe and effective in this patient during the follow-up period. PCI seems a reasonable treatment in symptomatic MBs; however, patient selection and procedural aspects remain unclear in the absence of comparative clinical trials.

Angina pectoris-like symptoms could be caused by several reasons beyond atherosclerotic coronary disease. To hold the MB responsible for the symptoms, its pathological role must be proved. In a recent publication by Hakkem (2), the FFR measurement was done with dobutamine provocation in the symptomatic bridge. The most severe hemodynamic alteration was found in diastolic FFR; therefore, the authors are suggested to use this value in the MB patients.

Dynamic compression caused by the MB is unique and this kind of coronary lesion differs from other atherosclerotic lesions. The high incidence of procedural failures like stent thrombosis (3), coronary perforation (4), and early restenosis (5) suggest that the stents' mechanical properties, diameter, and length are the determining factors for a successful intervention. High inflation pressures may be required for optimal stent implantation despite the higher risk of coronary perforation.

Basically the stent recoil means the percentage by which the diameter of a stent decreases from its expanded diameter (when the balloon is inflated at nominal pressure) to its relaxed diameter (when the balloon is retrieved from the stent). We have to calculate with a dynamic stress component as well, which is caused by the myocardium mass above the lesion. The given device's resistance to this permanent, cyclic force can make a difference between various stent types. On the contrary e.g., the pushability seems to be a less important feature when preparing for stenting a MB on the mid segment of the LAD.

Funding sources: This project was supported by the Semmelweis University, Budapest.

György Bárczi[1], Ibolya Csécs[1], Zoltán Ruzsa[1,2], Béla Merkely[1]
[1]Semmelweis University Heart and Vascular Center; Budapest-*Hungary*
[2]Department of Invasive Cardiology, Bács-Kiskun County Hospital, Teaching Hospital of the Szent-Györgyi Albert Medical University; Kecskemét-*Hungary*

References

1. Corban MT, Hung OY, Eshtehardi P, Rasoul-Arzrumly E, McDaniel M, Mekonnen G, et al. Myocardial bridging: contemporary understanding of pathophysiology with implications for diagnostic and therapeutic strategies. J Am Coll Cardiol 2014; 63: 2346-55. Crossref
2. Hakeem A, Çilingiroğlu M, Leesar MA. Hemodynamic and intravascular ultrasound assessment of myocardial bridging: fractional flow reserve paradox with dobutamine versus adenosine. Catheter Cardiovasc Interv 2010; 75: 229-36. Crossref
3. Derkacz A, Nowicki P, Protasiewicz M, Reczuch K, Szczepanik-Osadnik H, Witkowska M. Multiple percutaneous coronary stent implantation due to myocardial bridging--a case report. Kardiol Pol 2007; 65: 684-7.
4. Hering D, Horstkotte D, Schwimmbeck P, Piper C, Bilger J, Schultheiss HP. Acute myocardial infarct caused by a muscle bridge of the anterior interventricular ramus: complicated course with vascular perforation after stent implantation. Z Kardiol 1997; 86: 630-8.
5. Kurşaklıoğlu H, Barçın C, İyisoy A, Köse S, Amasyalı B, Işık E. Angiographic restenosis after myocardial bridge stenting. Jpn Heart J 2004; 45: 581-9. Crossref

Address for Correspondence: György Bárczi, MD
Semmelweis University Heart and Vascular Center, Budapest, Hungary
Városmajor u. 68. 1122 Budapest-*Hungary*
Phone.+36 - 1- 4586841 Fax.+36-1-45868428
E-mail: barczigyorgy@gmail.com

DOI:10.14744/AnatolJCardiol.2017.7406

Pulmonary valve and pulmonary artery myxomas

To the Editor,

Myxomas arising from the pulmonary valve and pulmonary artery are very rare. The mechanisms of these myxomas remain unknown; however, it is supposed that they arise in situ or from a dislodgement of myxomas from remote sites (1). Eck reported the first case of pulmonary valve myxoma in a premature neonate in 1935 (2). Later in 1955, Blodorn (2) reported an autopsied case of myxomas involving both the pulmonary valve and pulmonary artery (2). Until present, only two decades of such cases have been reported worldwide. The myxoma could be found at any age, from neonate to very aged patients, with a slight male predominance.

The myxomas located near the pulmonary valve may influence opening and closing of the valve leading to valvular stenosis and (or) insufficiency. As some patients were asymptomatic, the myxomas were discovered by incidental findings during routine examinations, whereas majority presented with circulatory or institutional symptoms. Physical examinations, electrocardiography, and chest X-ray films might not offer specific diagnostic evidences.

Myxomas may be misdiagnosed as pulmonary valve stenosis, pulmonary artery embolism, or pulmonary valve vegetation and lead to an inappropriate therapy, such as anticoagulation or thrombolysis (3). Transthoracic or transesophageal echocardiography and cardiac computed tomography are reliable diagnostic means. Computed tomography could clearly show the location, size, and mobility of the myxoma as well as the relation between myxoma and cardiac system. The feature of pulmonary artery myxoma in cardiac magnetic resonance imaging was reported to be a hypointense mass (4). A moving mass on echocardiography or a filling defect on computed tomography could be helpful in